ACHIEVING EXCELLENCE IN THE OSCE

Part II

Neurology, Respirology and Rheumatology

This book complements

Achieving Excellence in the OSCE - Part I

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

Part II

Neurology to Rheumatology

A.B.R. Thomson

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“The Democratization of Knowledge”

2012
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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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➢ 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 Neurology Chapter

1. Perform a focused physical examination of the cranial nerves (CN).
2. Perform a focused physical examination for the causes of sudden blindness.
3. Perform a focused physical examination for the causes of retinal hemorrhage.
4. Perform a focused physical examination to distinguish between papilladema vs papillitis.
5. Perform a focused physical examination to determine the cause of unequal pupils (anisocoria).
6. Perform a focused physical examination for the causes of ptosis.
7. Perform a focused physical examination for Horner’s syndrome.
8. Perform a focused physical examination for the causes of the Argyll Robertson pupil.
9. Perform a focused physical examination of the patient with a large pupil which reacts slowly to light and accommodation (Holmes-Adie Syndrome).
10. Perform a focused physical examination for the causes of unilateral or bilateral ptosis.
11. Perform a focused physical examination for a CN III lesion (of the left side in this example).
12. Perform a focused physical examination for a CN IV lesion (of the right side in this example).
13. Perform a focused physical examination for a CN VI lesion (on the left side in this example).
14. Perform a focused physical examination to determine the cause of a person’s diplopia.
15. Perform a focused physical examination for the causes of facial weakness/paralysis (CN VII lesion).
16. Perform a focused physical examination for the causes of facial pain.
17. Perform a focused physical examination for a lesion in the cavernous sinus, cerebellopontine angle, jugular foramen, pseudobulbar and bulbar palsy (multiple cranial nerve palsies), and its causes.
18. Take a directed history and perform a focused physical examination for a lesion at the cerebellopontine angle.
19. Take a directed history and perform a focused physical examination to determine the causes of dysarthria (disorder of articulation).

20. Perform a focused physical examination for posterior inferior cerebellar artery thrombosis.

21. Take a directed history and perform a focused physical examination for the jugular foramen syndrome.

22. Perform a focused physical examination for the causes of nystagmus/vertigo.

23. Take a directed history to determine the causes of facial pain.

24. Perform a focused physical examination to distinguish between an intramedullary from an extramedullary cord lesion.

25. Take a directed history and perform a focused physical examination for lateral medullary syndrome.

26. Take a directed history and perform a focused physical examination to distinguish between pseudobulbar and bulbar palsy.

27. Perform a focused physical examination for disorders of the cerebellum.

28. Perform a focused physical examination to distinguish between sensory ataxia and cerebellar ataxia.

29. Perform a focused physical examination to localize a spinal cord lesion to a specific lumbar or sacral nerve root level.

30. Perform a focused physical examination to distinguish between total spinal cord transaction, or incomplete cord compression.

31. Perform a focused physical examination to detect the following sensory syndromes.

32. Perform a focused physical examination for the causes and site of spinal cord compression.

33. Perform a focused physical examination for causes of spastic paraesthesia.

34. Take a directed history and perform a focused physical examination for tabes dorsalis.

35. Perform a focused physical examination to determine the nerve roots involved in spinal cord disease (sensory dermatomes).

36. Perform a focused physical examination for closed spina bifida.

37. Perform a focused physical examination to determine if a person has a UMN or an LMN lesion.
38. Perform a focused physical examination for loss of corticospinal inhibition.

39. Perform a focused physical examination for hemisection of the spinal cord (Brown-Sequard syndrome).

40. Perform a focused physical examination for a lesion in the spinal canal at any level below T10 (cauda equina syndrome).

41. Perform a directed physical examination to establish the neurological cause of a brachial plexus lesion, and the cervical rib syndrome.

42. Perform a focused physical examination for syringomyelia.

43. Perform a focused physical examination for muscle or UMN nerve root disease in the muscle groups of the upper and lower body.

44. Perform a focused physical examination of the sensory dermatomes of the peripheral nervous system, and give their signature zones.

45. Perform a focused physical examination to establish the neurological cause of wasting of the small muscles of the hand.

46. Perform a focused physical examination of the cutaneous sensory innervation of the hand.

47. Perform a focused physical examination for carpal tunnel syndrome.

48. Perform a focused physical examination for ulnar nerve palsy (C8, T1).

49. Perform a focused physical examination for the cause of a carcinomatous neuropathy.

50. Perform a focused physical examination for the causes of benign intracranial hypertension (pseudotumour cerebri).

51. Perform a directed physical examination for the causes of peripheral neuropathy.

52. Perform a focused physical examination for Charcot-Marie-Tooth disease (features of hereditary motor and sensory neuropathy).

53. Perform a focused physical examination for Charcot-Marie-Tooth (CMT) disease (hereditary peroneal muscular neuropathy).

54. Perform a focused physical examination for inflammatory polyradiculoneuropathy.

55. Take a directed history and perform a focused physical examination for common peroneal nerve palsy (aka: lateral popliteal nerve palsy [L4,5].

56. Take a directed history of the causes of muscle weakness.

57. Perform a focused physical examination for (Becker) muscular dystrophy (MD).
58. Take a directed history and perform a focused physical examination for myotonia dystrophia.

59. Take a directed history and perform a focused physical examination for limb girdle dystrophy.

60. Take a directed history and perform a focused physical examination for myasthenia gravis (MG).

61. Take a directed history and perform a focused physical examination for motor neuron disease (MND).

62. Perform a directed physical examination for extrapyramidal disease.

63. Perform a focused physical examination of tremor.

64. Take a directed history and perform a focused physical examination for chorea.

65. Take a directed history and perform a focused physical examination for dementia.

66. Take a directed history to differentiate between delirium and dementia.

67. Take a directed history for seizures.

68. Take a directed history and perform a physical examination for a cerebral vascular accident (CVA).

69. Perform a focused physical examination for the causes of paraplegia.

70. Perform a focused neurological examination to determine the location of an arterial cerebral occlusion.

71. Perform a focused physical examination to determine if a lesion affects functions of the dominant cerebral hemisphere.

72. Perform a focused physical examination to determine the presence of parietal lobe dysfunction.

73. Take a directed history to differentiate between a carotid or vertebrobasilar transient ischemic attack (TIA).

74. Perform a focused physical examination to determine the location of lesions causing sensory loss.

75. Perform a focused physical examination for coma.

76. Take a directed history to detect disease of the frontal, parietal or temporal lobe, or the motor cortex.

77. Take a directed history for delirium.

78. Perform a focused physical examination to distinguish between meningeal irritation, versus lesion of the sciatic nerve or its spinal roots (Lasègue’s sign).
79. Perform a focused physical examination for meningitis (the numbers in brackets represent valves for sensitivity)
80. Take a directed history to determine the cause of a patient’s dizziness.

**OSCE Questions in Respirology Chapter**

1. Perform a directed physical examination for asymmetry in the expansion of the chest
2. Perform a directed physical examination of the pulmonary system for tracheal deviation
3. Take a directed history for cough.
4. Take a directed history of hemoptysis
5. Perform a directed physical examination of the pulmonary system for consolidation, collapse, effusion, or fibrosis.
6. Perform a focused physical examination to distinguish between the major causes of dullness at a lung base.
7. Perform a directed physical examination for clubbing.
8. Perform a directed physical examination for sarcoidosis.
9. Causes of slow resolution or recurrence of pneumonia
10. Take a directed history for asthma.
11. Perform a focused physical examination for asthma.
12. Take a focused history and perform a directed physical examination for chronic bronchitis.
13. Take a directed history to differentiate between bronchial asthma, chronic bronchitis, and emphysema.
14. Take a directed history for the harmful effects of cigarette smoking
15. Take a focused history and perform a directed physical examination for bronchiectasis.
16. Perform a directed physical examination of the pulmonary system in the patient with suspected mediastinal compression (e.g. carcinoma of the lung).
17. Perform a directed physical examination for Pancoast’s (superior pulmonary sulcus tumor) syndrome (often from cancer [often non-small cell] of the apex of the lung, infiltrating C8, T1, 2; may also occur with lymphoma, or by spread of lymph node metastases in breast or lung cancer).
18. Take a directed history and perform a focused physical examination for pulmonary hypertension.

19. Perform a focused physical examination for acute respiratory distress syndrome (ARDS).

20. Take a directed history and perform a focused physical examination to determine the possible presence of a deep vein thrombosis (DVT).

21. Take a focused history for the causes of pneumothorax.

22. Take a focused history for the causes of lung abscess.

23. Take a directed history and perform a focused physical examination for fibrosing alveolitis.

**OSCE Questions in Rheumatology Chapter**

1. Take a directed history for a musculoskeletal (MSK) disorder.

2. Perform a focused physical examination of the joints of the upper and lower body and from the abnormal articular findings, give the most likely diagnosis.

3. Take a directed history for the common side effects of nonsteroidal anti-inflammatory drugs.

4. Perform a focused physical examination of the elbow.

5. Take a directed history and a focused physical examination for features differentiating diseases affecting the elbow.

6. Perform a focused physical examination of the shoulder.

7. Perform a focused physical examination for causes of shoulder pain.

8. Take a directed history of back pain.

9. Perform a focused physical examination of back pain.

10. Perform a focused physical examination of the hip.

11. Perform a directed physical examination of the knee.

12. Perform a focused physical examination of the ankle.

13. Perform a focused physical examination of the feet.

14. Perform a focused physical examination for complications of rheumatoid arthritis (RA), and its complications.

15. Take a directed history and perform focused physical examination to distinguish rheumatoid arthritis from osteoarthritis.

16. Perform a focused physical examination for diseases that may have positive rheumatoid factor.
17. Perform a focused physical examination for the causes of spondyloarthritis.

18. Take a directed history for ankylosing spondylitis.

19. Perform a focused physical examination for ankylosing spondylitis.

20. Perform a focused physical examination for primary vs secondary osteoarthritis.

21. Perform a focused physical examination for psoriatic arthritis.

22. Perform a focused physical examination for ankylosing spondylitis.

23. Perform a focused physical examination for primary vs secondary osteoarthritis.

24. Perform a focused physical examination for psoriatic arthritis.

25. Perform a directed history and perform a focused physical examination for systemic lupus erythematosus (SLE) and its complications.

26. Perform a directed history and perform a focused physical examination for scleroderma and its complications.

27. Perform a directed history and perform a focused physical examination for Raynaud’s phenomenon (white->blue->red fingers/toes in response to cold temperature).

28. Perform a directed history and perform a focused physical examination for systemic lupus erythematosus (SLE) and its complications.

29. Take a directed history and perform a focused physical examination for vasculitis.

30. Take a directed history and perform a focused physical examination for the causes of vasculitis.

31. Take a directed history and perform a focused physical examination for causes of polymyalgia rheumatica-like syndromes.

32. Take a directed history of the cause of aseptic necrosis of the bone. (acronym: ASEPTIC)

33. Perform a directed physical examination for Charcot’s joint (neuroarthropathy).

34. Perform a focused physical examination for polymyositis/dermatomyositis.

35. Perform a focused physical examination for Marfan’s syndrome.
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3. Perform a focused physical examination for the causes of retinal hemorrhage.
4. Perform a focused physical examination to distinguish between papilladema vs papillitis.
5. Perform a focused physical examination to determine the cause of unequal pupils (anisocoria).
6. Perform a focused physical examination for the causes of ptosis.
7. Perform a focused physical examination for Horner’s syndrome.
8. Perform a focused physical examination for the causes of the Argyll Robertson pupil.
9. Perform a focused physical examination of the patient with a large pupil which reacts slowly to light and accommodation (Holmes-Adie Syndrome).
10. Perform a focused physical examination for the causes of unilateral or bilateral ptosis.
11. Perform a focused physical examination for a CN III lesion (of the left side in this example).
12. Perform a focused physical examination for a CN IV lesion (of the right side in this example).
13. Perform a focused physical examination for a CN VI lesion (on the left side in this example).
14. Perform a focused physical examination to determine the cause of a person’s diplopia.
15. Perform a focused physical examination for the causes of facial weakness/paralysis (CN VII lesion).
16. Perform a focused physical examination for the causes of facial pain.
17. Perform a focused physical examination for a lesion in the cavernous sinus, cerebellopontine angle, jugular foramen, pseudobulbar and bulbar palsy (multiple cranial nerve palsies), and its causes.
18. Take a directed history and perform a focused physical examination for a lesion at the cerebellopontine angle.
19. Take a directed history and perform a focused physical examination to determine the causes of dysarthria (disorder of articulation).

20. Perform a focused physical examination for posterior inferior cerebellar artery thrombosis.

21. Take a directed history and perform a focused physical examination for the jugular foramen syndrome.

22. Perform a focused physical examination for the causes of nystagmus/vertigo.

23. Take a directed history to determine the causes of facial pain.

24. Perform a focused physical examination to distinguish between an intramedullary from an extramedullary cord lesion.

25. Take a directed history and perform a focused physical examination for lateral medullary syndrome.

26. Take a directed history and perform a focused physical examination to distinguish between pseudobulbar and bulbar palsy.

27. Perform a focused physical examination for disorders of the cerebellum.

28. Perform a focused physical examination to distinguish between sensory ataxia and cerebellar ataxia.

29. Perform a focused physical examination to localize a spinal cord lesion to a specific lumbar or sacral nerve root level.

30. Perform a focused physical examination to distinguish between total spinal cord transaction, or incomplete cord compression.

31. Perform a focused physical examination to detect the following sensory syndromes.

32. Perform a focused physical examination for the causes and site of spinal cord compression.

33. Perform a focused physical examination for causes of spastic paraparesis.

34. Take a directed history and perform a focused physical examination for tabes dorsalis.

35. Perform a focused physical examination to determine the nerve roots involved in spinal cord disease (sensory dermatomes).

36. Perform a focused physical examination for closed spina bifida.

37. Perform a focused physical examination to determine if a person has a UMN or an LMN lesion.
38. Perform a focused physical examination for loss of corticospinal inhibition.
39. Perform a focused physical examination for hemisection of the spinal cord (Brown-Sequard syndrome).
40. Perform a focused physical examination for a lesion in the spinal canal at any level below T10 (cauda equina syndrome).
41. Perform a directed physical examination to establish the neurological cause of a brachial plexus lesion, and the cervical rib syndrome.
42. Perform a focused physical examination for syringomyelia.
43. Perform a focused physical examination for muscle or UMN nerve root disease in the muscle groups of the upper and lower body.
44. Perform a focused physical examination of the sensory dermatomes of the peripheral nervous system, and give their signature zones.
45. Perform a focused physical examination to establish the neurological cause of wasting of the small muscles of the hand.
46. Perform a focused physical examination of the cutaneous sensory innervation of the hand.
47. Perform a focused physical examination for carpal tunnel syndrome.
48. Perform a focused physical examination for ulnar nerve palsy (C8, T1)
49. Perform a focused physical examination for the cause of a carcinomatous neuropathy.
50. Perform a focused physical examination for the causes of benign intracranial hypertension (pseudotumour cerebri).
51. Perform a directed physical examination for the causes of peripheral neuropathy.
52. Perform a focused physical examination for Charcot-Marie-Tooth disease (features of hereditary motor and sensory neuropathy).
53. Perform a focused physical examination for Charcot-Marie-Tooth (CMT) disease (hereditary peroneal muscular neuropathy).
54. Perform a focused physical examination for inflammatory polyradiculoneuropathy.
55. Take a directed history and perform a focused physical examination for common peroneal nerve palsy (aka: lateral popliteal nerve palsy [L4,5]).
56. Take a directed history of the causes of muscle weakness.
57. Perform a focused physical examination for (Becker) muscular dystrophy (MD).
58. Perform a focused physical examination for myotonia dystrophia.
59. Perform a focused physical examination for limb girdle dystrophy.
60. Perform a focused physical examination for myasthenia gravis (MG).
61. Perform a focused physical examination for motor neuron disease (MND).
62. Perform a directed physical examination for extrapyramidal disease.
63. Perform a focused physical examination of tremor.
64. Take a directed history and perform a focused physical examination for chorea.
65. Take a directed history and perform a focused physical examination for dementia.
66. Take a directed history to differentiate between delirium and dementia.
67. Take a directed history for seizures.
68. Take a directed history and perform a physical examination for a cerebral vascular accident (CVA).
69. Perform a focused physical examination for the causes of paraplegia.
70. Perform a focused neurological examination to determine the location of an arterial cerebral occlusion.
71. Perform a focused physical examination to determine if a lesion affects functions of the dominant cerebral hemisphere.
72. Perform a focused physical examination to determine the presence of parietal lobe dysfunction.
73. Take a directed history to differentiate between a carotid or vertebrobasilar transient ischemic attack (TIA).
74. Perform a focused physical examination to determine the location of lesions causing sensory loss.
75. Perform a focused physical examination for coma.
76. Take a directed history to detect disease of the frontal, parietal or temporal lobe, or the motor cortex.
77. Take a directed history for delirium.
78. Perform a focused physical examination to distinguish between meningeal irritation, versus lesion of the sciatic nerve or its spinal roots (Lasègue’s sign).

79. Perform a focused physical examination for meningitis (the numbers in brackets represent values for sensitivity).

80. Take a directed history to determine the cause of a patient’s dizziness.
Introduction

- The language of neurology

- Agnosia
  - Failure to recognize, whether visual, auditory or tactile; Related to receptive dysphagia.

- Apraxia
  - Inability to carry out purposive movements in absence of motor paralysis, sensory loss or ataxia; Related to expressive dysplagia.

- Dysarthria
  - Difficulty with articulation
  - Causes
    - UMN lesion of the cranial nerves – pseudobulbar or bulbar
    - Extra pyramidal disease – monotonous speech
    - Cerebellar disease – altered rhythm of speech
    - Mouth ulcerations
    - Hearing
    - Alcohol intoxication

- Dysphasias
  - Disorder in use of symbols for communication, whether spoken, heard, written or read.
    - Expressive – Lesion of post. inf. 3rd frontal convolution
    - Receptive – Lesion of post. sup. temporal cortex, an angidengyrus of parietal lobe.
  - Dominant higher center disorder of the use of language (handedness: 94% of right-handed people and 50% of left-handed people have a dominant left hemisphere for language and math)
  - Screening flowing speech “Describe the room”
  - Comprehension “Touch your chin”, “Is this your right hand?”
    - Ask patient to name two objects you point to
    - Say “British constitution”
  - Writing
    - Conductive aphasic patients have impaired writing (dysgraphia) while receptive aphasic patients have abnormal content of writing.
    - Patients with dominant frontal lobe lesions may also have dysgraphia
  - Receptive
    - No understanding for spoken (auditory dysphasias) or written words in absence of deafness or blindness
    - Cannot follow verbal or written commands (“touch your nose, then your chin, then your ear”)
- Cannot repeat “No ifs, ands or buts”
- Lesion in posterior part of first temporal gyrus of the dominant hemisphere (Wernicke’s area)
  o Expressive dysphasia
    - Motor apraxia – the patient understands spoken or written words, but cannot answer correctly
    - Automatic (recite a list, such as days of the week); emotional speech, maybe preserved
    - Lesion in posterior part of the third frontal gyrus (Broca’s area) of the dominant hemisphere
  o Nominal dysphasia
    - Specific objects cannot be named, but person may give a long answer to try to explain what the object is (circumlocution)
    - Lesion of posterior temporoparietal area of dominant hemisphere; encephalopathy; increased intracranial pressure (poor localizing value, and may occur in the recovery phase of receptive, expressive or conductive dysphasia)
  o Conductive dysphasia
    - Poor naming, poor repetition of statements, good following of commands
    - Lesion of fibers joining Wernicke’s and Broca’s areas, or lesion in arcuate fasciculus

➢ Dysphonia
  o Decreased volume and altered tone of speech
    - Damage to larynx or recurrent laryngeal nerve palsy

➢ Echolalia
  o Parrot-like repetition by the subject of statements or acts made before them.

➢ Epilepsy
  o A paroxysmal transitory disturbance of brain function, ceasing spontaneously, with a tendency to recurrence.

➢ Myoclonus
  o A brief shock-like contraction of a number of muscle fibres, a whole muscle or several muscles, either simultaneously or successively.

➢ Perseveration
  o Meaningless repetition of an activity

➢ Verbigeration
  o Meaningless repetition of words or sentences
• Neuroanatomy Refresher

➢ CNS lesion localization


• Spinal cord section

Adapted from: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto, 2005, page 155.
General neurological history and physical examination

- Localization of Neurologic Disorders
  - Level
    - Supratentorial
    - Posterior fossa
    - Spinal cord
    - Peripheral
  - Extent
    - Focal
    - Diffuse
Useful background: Take a thorough neurological history (mnemonic “SHOVE”)

- Syncope, speech defect, swallowing difficulty
- Headache
- Ocular disturbances; diplopia, field defects
- Vertigo
- Epilepsy; seizures
- History pertaining to motor and sensory components of the cranial nerves and limbs, e.g. pain paraesthesia, weakness, incoordination


Useful background: Directed history for disorders of the central (CNS) and peripheral nervous system (PNS) (e.g. the Neurological system).

- Where is the lesion and what is the lesion? (example - loss of consciousness (LOC)/(syncope vs. seizure))
  - Complete vs. partial
  - Duration
  - Changes in body position (e.g. loss of balance, fetal position, prone)
  - Associated symptoms (eg tongue biting, body movements, incontinence)
  - Preceding symptoms (e.g. light-headedness)
  - Post attack symptoms (e.g. confusion, sleepiness)
  - Previous diagnosis of systemic disorders (e.g. cardiovascular problems)
  - Current medications
  - Collateral /corollary information (e.g. bystanders)

- Course
  - Onset (e.g. thunderclap)
  - Pattern (e.g. worse in the morning=increased ICP)
  - Differences from previous headaches (type/pattern)
  - Associated symptoms (e.g. nausea and/or vomiting, neck stiffness)
  - Preceding symptoms/aura
  - Systemic conditions (e.g. infections)
  - Current medications/addictions
Abbreviations: CNS, central nervous system; LOC, loss of consciousness; PNS, peripheral nervous system


Cranial nerves

- Remember: You need to establish where the lesion is, and what the likely lesion is.

Useful background: Cranial nerve supply

- Olfactory
  - Smell
- Optic
  - Vision
- Oculomotor
  - All ocular muscles, except superior oblique and lateral rectus
  - Ciliary muscle
  - Sphincter papillae
  - Levator palpebrae superioris
- Trochlear superior oblique muscle
  - Tested by asking patient to look down and inwards
- Trigeminal
  - Sensory for face, cornea, sinuses, nasal mucosal, teeth, tympanic membrane and anterior two thirds of tongue
  - Motor to muscles of mastication
- Abducens (External rectus muscle)
- Facial
  - Motor to scalp and facial muscles of expression
  - Taste in anterior two thirds of tongue (via chorda tympani)
  - Nerve to stapedius muscle
- Auditory and vestibular components
- Glossopharyngeal
  - Sensory for posterior one third of tongue, pharynx and middle ear
  - Taste fibres for posterior one third of tongue
  - Motor to middle constrictor of pharynx and stylopharyngeus
- Vagal
  - Motor to soft palate, larynx and pharynx (from nucleus ambiguus)
  - Sensory and motor for heart, respiratory passengers and abdominal viscera (from dorsal nucleus)

- Spinal accessory
  - Motor to sterno mastoid and trapezius
  - Accessory fibres to vagus

- Hypoglossal
  - Motor to tongue and hyoid bone depressors


1. Perform a focused physical examination of the cranial nerves (CN).

- I (Olfactory) smell (sensory) (detecting non irritating odours)
  - Coffee, mint, vinegar

- II (Optic)-vision (sensory)
  - Visual acuity (Snellen visual chart)
  - Visual fields by confrontation
  - Colour test
  - Reflex
    - Papillary light reflex (perform at time of fundoscopy: tests CN III)
  - Accommodation to light
  - Red reflex
  - Fundoscopy
    - Retinal vessels
    - Optic disc
    - Macula
    - Lesions

- III (Oculomotor), [IV (trochlear), VI (abducens)] (motor)
  - Eye alignment
  - Gaze (6 position movements)
  - Endpoint nystagmus
  - III only – lid elevation, pupillary constriction to light (direct and consensual) or on a near object (accommodation)
  - Papillary light reflex
  - Ciliary muscle
  - Sphincter papillae
  - Levator palpebrae superiors

- IV (Trochlear) – look in and down (superior oblique)
VV (Trigeminal)
- Sensory - Pain, temperature and light touch for same side of face, cornea, sinuses, nasal mucosa, teeth, tympanic membrane, anterior 2/3 of tongue? or VII
- Motor - Mouth, open symmetrically, open against resistance, move jaw against resistance, clench teeth, chewing (masseter and lateral pterygoid muscles)
- Reflex
  - Corneal reflex (afferent limb), jaw jerk (afferent and efferent limbs)
  - Glabeller reflex (limb)
- Asymmetry of face
  - V1 = ophthalmic
    - Forehead and tip of nose
    - Afferent limb of the corneal reflex
  - V2 = maxillary
    - Medial aspect of cheek
    - Afferent limb of jaw jerk reflex
    - Chin, except angle of the jaw(C2)
  - V3 = mandibular
    - Innervates jaw muscles

VII (Facial)
- Motor (scalp and fascial muscles of expression; all facial movements except eyelid elevation [CN III])
  - Raise eyebrows, wrinkle forehead (all facial movements except lid elevation [CN III] or mouth and jaw movement [CN V])
  - Open/close eyes
  - Smile, showing teeth
  - Puff out cheeks
  - Phonation
- Sensory
  - Speech, say ‘PAH’ (via the chorda tympani)
  - Taste, anterior 2/3 of tongue taste to (posterior 1/3 of tongue [CN IX])
VIII (Vestibulo-cochlear) – auditory and vestibular components (sensory)
- Hearing (whisper test)
- Local sound and vibration (S12 Hz tuning fork)

IX (Glosopharyngeal)
- Sensory: posterior third of tongue, pharynx, nasopharynx, middle ear; voice-hoarse, nasal, taste; phonation
  - Motor
    - Initiate swallow (middle constrictor of pharynx, and stylopharyngeus)
    - Gag reflex (afferent limb, IX; efferent limb, X)
    - Ipselateral palate elevation (with CN X)
    - Soft palate, larynx, pharynx (nucleus ambiguus)

- X (Vagus)
  - Motor
    - Gag reflex (afferent limb, IX; efferent limb, X)
    - Soft palate, larynx, pharynx (nucleus ambiguus)
    - Ipselateral palate elevation (with CN IX)
    - Swallowing, phonation
  - Reflex
    - Gag reflex (afferent limb, IX; efferent limb, X)
  - Secretory
    - Parotid gland
    - Afferent and efferent pathways to heart, lung and, GI tract (nucleus solitaries)

- XI (Spinal accessory) (motor)
  - Rotate head against resistance (sternocleidomastoid muscle)
  - Shrug shoulders (trapezius muscle)
  - Necessary fibers to vagus nerve

- XII (Hypoglossal)
  - Motor to tongue and hyoid bone depressors
  - Tongue movement (deviation, fasciculation, atrophy, pushing tongue against teeth)
  - Speech: say ‘AH’ (dysarthria)

Clinical gem: Uvula- deviates to strong side; jaw and tongue-deviate to weak side

Adapted from: McGee S. R. Saunders/Elsevier 2007, page 691; Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto 2005, pages 157-158.
Useful background: Multiple cranial nerves abnormalities

<table>
<thead>
<tr>
<th>CN Combination</th>
<th>Common cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unilateral III, IV, V1, VI</td>
<td>Cavernous sinus lesion</td>
</tr>
<tr>
<td>Unilateral V, VII, VIII</td>
<td>Cerebellopontine angle lesion</td>
</tr>
<tr>
<td>Unilateral IX, X, XI</td>
<td>Jugular foramen syndrome</td>
</tr>
<tr>
<td>Bilateral X, XI, XII</td>
<td>Bulbar palsy (LMN), pseudobulbar palsy (UMN)</td>
</tr>
</tbody>
</table>

Adapted from: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto* 2005, page 162.

**The Eye: CN II**

Useful background: Visual field defects

- Monocular defects are usually due to a problem in the affected eye, whereas binocular visual field defects are usually intracranial in origin
- III Nerve palsy: affected side dilated (mydriasis), ptosis, weak extraocular muscle except lateral rectus and superior oblique
- Causes of sudden blindness
  - Brain
    - Trauma- ocular or post head injury
    - CVA
    - Migraine
    - Hysteria Vitreous hemorrhage, especially in diabetics
  - CN II
    - Cranial arteritis
    - Toxins eg methanol
    - Retrobulbar neuritis
  - Retinal vessels
    - Embolism of retinal artery
    - Thrombosis of retinal vein
  - Retina
    - Retinal detachment
  - Intraocular
    - Acute glaucoma

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 82.
Useful background: Lesions of the visual fields

**ANTERIOR LESIONS**

1. Constricted visual field

2. Arcuate scotoma

3. Altitudinal defect

4. Central scotoma

**CHIASMAL LESIONS**

1. Bitemporal hemianopia

2. POSTCHIASMAL LESIONS

3. Left homonymous superior quadrantanopia

4. Left homonymous inferior quadrantanopia

5. Left homonymous hemianopia with macular sparing

1. Perform a focused physical examination for the causes of sudden blindness.

- Artery
  - Embolus
  - Arteritis

- Vein
  - Thrombus

- Ret. Neurotic, including toxins

- Retinal detachment
  - Trauma
  - Tumour
  - Toxemia
  - Myopia

- Retinal hemorrhage
  - Diabetes
  - Edwards

- Glaucoma

- CVA

- Migraine

- Hysteria

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

Q: In the context of an abnormal examination of the eyes, what is Eales’ disease.
A:  
  - Periodic vitreous hemorrhage and pre-retinal (subhyaloid) hemorrhages.

Useful background: Causes of uveitis

- (Uveal tract= iris, ciliary body and choroid)

- Miscellaneous, systemic disease
  - GI
    - Ulcerative colitis
    - Crohn’s disease
  - MSK
    - Ankylosing spondylitis
- Rheumatoid arthritis
- Reiters disease
- Behcets disease

- Lung
  - Sarcoidosis

➢ Infections
  - Bacterial: TB
  - Spirochaetal: Sy. Relapsing fever, Weil’s disease
  - Protozoal: Malaria, toxoplasmosis
  - Nematode larvae: Toxocara of dog or cat

➢ Secondary to ocular disease
  - Ophthalmitis
  - Trauma

➢ Idiopathic


➢ Visual field defects (CN II)

<table>
<thead>
<tr>
<th>Lesion Location</th>
<th>Anatomy</th>
<th>Signs and symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ One eye</td>
<td>Anterior to optic chiasm</td>
<td>- Glaucoma&lt;br&gt;- Retinal hemorrhage&lt;br&gt;- Optic neuropathy&lt;br&gt;- Central retinal artery occlusion (leads to potential monocular blindness [amaurosis fugax])</td>
</tr>
<tr>
<td>➢ Both eyes (bitemporal hemianopia)</td>
<td>At optic chiasm</td>
<td>- Upper &gt; lower – inferior chiasmal compression (pituitary adenoma)&lt;br&gt;- Lower &gt; upper – superior chiasmal compression</td>
</tr>
<tr>
<td>➢ Both eyes (homonymous hemianopia)</td>
<td>Behind optic chiasm</td>
<td>- Cerebral infarcts&lt;br&gt;- Hemorrhage&lt;br&gt;- Tumors</td>
</tr>
</tbody>
</table>

Source: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto*, 2005, page 158.
Useful background: Causes of gradual blindness

- CNS - Migraine
- CN III - Optic neuritis
  - Aatrophy
  - Papilloedema
- Retina - Retinal degeneration
- Intraocular - Glaucoma
- Lens - Cataracts

3. Perform a focused physical examination for the causes of retinal hemorrhage.

- Brain
  - ↑ ICP, including subarachnoid
- Eye
  - Eye – trauma
- Retinal vessels
  - Artery
    - HBP
    - Arteritis
  - Vein
    - Thrombosis
- Retina
  - Retinal detachment, including tumour
- Hematology
  - Anemia (severe)
  - Bleeding diathesis (severe)
- Diabetes

Abbreviations: CNS, central nervous system; ICP, intracranial pressure; HBP, hypertension

Useful background:

- **Causes of concentric diminution (tunnel vision)**
- **Brain**
  - Anterior calcarine cortex
  - Migraine
  - Hysteria
  - Occipital cortex
- **CN II**
  - Papilloedema
  - Retro-neuritis
- **Retinal vessels**
- **Retinal disease**
- **Glaucoma**
- **Causes of bitemporal hemianopia (Central chiasmal lesions)**
  - Pituitary or peri-sellar tumour
  - Inflammatory, vascular or traumatic lesions
- **Binasal hemianopia**
  - Bilateral lesions confined to the uncrossed optic fibres.
- **Causes of homonymous hemianopia (HN)**
  - Optic tract lesions – usually due to tumours, which produce a progressive hemianopia, which bisects the macula, commonly due to thrombosis of the posterior cerebral vessels.
- **Causes of homonymous quadrantanopia**
  - Anteriorly placed lesions of the optic radiation, especially temporal lobe tumours. More posterior lesions of the optic radiation become more hemianopic.

Abbreviations: HN, homonymous hemianopia

Useful background: Causes of central scotoma*

- **Brain**
  - Demyelinating disorders (multiple sclerosis)

- **CN II**
  - Optical nerve compression by tumor, aneurysm
  - Toxins – methanol, tobacco, lead, arsenical poisoning
  - Hereditary disorders – Friedreich’s ataxia, Leber’s optic atrophy

- **Retinal vessels**
  - Ischemia
    - central retinal artery occlusion (thromboembolism)
    - temporal arteritis
    - syphilis
    - idiopathic acute ischemic neuropathy

- **Retina**
  - Secondary to retinitis pigmentosa

- **Intraocular**
  - Glaucoma

- **Metabolic**
  - Vitamin B\textsubscript{12} deficiency

*Scotoma is a small patch of visual loss within the visual field.


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

**Q1:** What is the ‘reversed’ Argyll Robertson pupil?

**A1:**
- The pupil react to light but not to accommodation
- Seen in Parkinsonism caused by encephalitis lethargic

**Q2:** What causes miosis?

**A2:**
- Old age
- Pilocarpine (treatment for glaucoma)

**Q3:** What non-neurological conditions cause an eccentric pupil?

**A3:**
- Trauma
- Iritis
Useful background:

➢ Cause of eye pain
  o Cornea, conjunctiva (↑ by blinking)
    - Corneal abrasions
    - Foreign bodies
    - Keratitis)
  o Iris (photophobia)
    - Inflammation of the iris
    - Middle layer of the eye corneal irritation
  o CN II (↑ by moving eye)
    - Optic neuritis
  o Artery (forehead)
    - With brow or temporal pain (e.g. may indicate temporal arteritis)
  o Intraocular (headache, nausea)
    - Acute angle-closure glaucoma

Adapted from: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto 2005, page 201.

Useful background: Cause of dilated pupils and contracted pupils

➢ Dilated
  o CN III
    - Third nerve lesion
    - Holmes – Adie syndrome (degeneration of nerve to the ciliary ganglion)
  o Iris
    - Blunt trauma to the iris (pupil may be irregularly dilated and reacts sluggishly to light – post – traumatic iridoplegia)
  o Lens
    - Lens implant
    - Iridectomy
  o Drugs
    - Mydriatic eye drops
    - Drug overdose, eg. cocaine, amphetamine
    - Poisoning, eg. Belladonna
  o Coma, death
    - Deep coma
    - Death

• Contracted
  o Old age
  o CN III
  o Pons
- Argyll Robertson pupil (distinguish)
- Pontine lesion
- Narcotics
  - Sympathetic
    - Horner syndrome
  - Drugs
    - Pilocarpine eye drops


➢ Causes of papilledema
  - Increased intracranial pressure
    - Space occupying lesion (causing raised intracranial pressure), or a retroorbital mass
    - Benign intracranial hypertension (pseudotumour cerebri) (small or normal sized ventricles)
      - Idiopathic
      - Oral contraceptive pill
      - Addison’s disease
      - Drugs- e.g. nitrofurantoin, tetracycline, vitamin A, steroids
      - Head trauma
  - Increased formation of CSF- e.g. choroids plexus papilloma (rare)
  - Decreased absorption of CSF
    - Tumour causing venous compression
    - Subarachnoid space obstruction from meningitis
  - Decreased outflow
    - Hydrocephalus (large cerebral ventricles)
    - Obstruction (a block in the ventricle, aqueduct or outlet to the fourth ventricle) e.g. tumour
  - Communicating hydrocephalus
  - Systemic hypertension (grade 4)
  - Central retinal vein thrombosis


Useful background:

➢ Causes of optic neuritis
  - Infective
    - Local: retinitis, periostitis
    - Systemic: syphilis, toxoplasmosis, typhoid fever, mumps
  - Toxins
    - Methyl alcohol
    - Lead
- Benzene
- Tobacco

○ Metabolic
  - Diabetes mellitus
  - B12 deficiency
  - Intestinal or uterine haemorrhage

○ Demyelinating disease eg.
  - Multiple sclerosis (MS)
  - Devic’s disease
  - Schilder’s disease

○ Hereditary degenerations
  - Leber’s disease
  - Marie’s disease
  - Freidreich’s ataxia

○ Giant cell arteritis

○ Trauma


➢ Causes of Optic Atrophy
  ○ Glaucoma
  ○ Chronic papilledema
  ○ Retinal lesions
    - Chorio-retinitis, meningitis
    - Intra-ocular hemorrhage, etc.
  ○ Optic neuritis (retrobulbar neuritis)
  ○ Pressure on an optic nerve
    - Tumour
      ▪ Pituitary
      ▪ Optic Nerve
    - Aneurysm
      ▪ Intracavernous aneurysm of internal carotid artery
    - Paget’s disease
  ○ Division of optic nerve
    - Surgery
    - Trauma
  ○ Lesions of optic tract

2. Perform a focused physical examination to distinguish between papilladema vs papillitis.

<table>
<thead>
<tr>
<th>Papilladema</th>
<th>Papillitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Optic disc</td>
<td>➢ Optic disc swollen</td>
</tr>
<tr>
<td>o Swollen without venous pulsation</td>
<td>o Swollen</td>
</tr>
<tr>
<td>➢ Visual acuity</td>
<td>➢ Visual acuity</td>
</tr>
<tr>
<td>o Normal (early)</td>
<td>o Poor</td>
</tr>
<tr>
<td>➢ Blind spot</td>
<td>➢ Blind spot</td>
</tr>
<tr>
<td>o Large</td>
<td>o Large central scotoma</td>
</tr>
<tr>
<td>➢ Visual fields of peripheral constriction</td>
<td>➢ Visual fields of peripheral constriction</td>
</tr>
<tr>
<td>o Usually slow onset of bilateral</td>
<td>o Onset usually sudden and unilateral</td>
</tr>
<tr>
<td>o Colour vision normal</td>
<td></td>
</tr>
<tr>
<td>o Eye movement- no pain</td>
<td>o Painful</td>
</tr>
</tbody>
</table>


➢ Causes of retinitis pigmentosa
   o Congenital (associated with cataract and deaf mutism)
   o Laurence Moon Biedl syndrome
   o Hereditary ataxia
   o Familial neuropathy i.e. Refsum’s disease

Abbreviation: CSF, cerebrospinal fluid


Useful background: Cervical sympathetic pathway to the eye

➢ Mid brain (superior colliculus)
➢ Tectospinal tract
➢ C8, T1 and 2 ventral roots
➢ Cervical sympathetic trunk
➢ Internal carotid and cavernous nerve plexus
➢ Ophthalmic division of the trigeminal nerve

3. Perform a focused physical examination to determine the cause of unequal pupils (anisocoria).

- **Definition:**
  - Anisocoria is a difference ≥ 0.4 mm in diameter of the pupils
  - Represents either a problem with the papillary constrictor muscle (parasympathetic denervation, iris disorder, pharmacologic pupil) or the papillary dilator muscle (sympathetic denervation, simple anisocoria)

- Normal variant (20%)
- CN III Palsy
- Drug
- Unilateral blindness (affected eye is dilated)/ eye disease
  - Iritis
  - acute angle closure glaucoma
  - trauma
  - previous surgery

- Horner’s syndrome
- Pharmacological
- Blindness or amblyopia in one eye (pupil larger in the affected eye)
- Cerebrovascular accidents
- Severe head trauma
- Hemianopia due to optic tract involvement


“A lot of things in medicine that make sense, don’t work out”

Grandad
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 (NID – 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: Pupils

<table>
<thead>
<tr>
<th>Finding</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Detecting intracranial structural lesion in patients with coma</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anisocoria &gt; 1mm</td>
<td>39</td>
<td>96</td>
<td>9.0</td>
<td>0.6</td>
</tr>
<tr>
<td>Absent light reflex in at least one eye</td>
<td>83</td>
<td>77</td>
<td>3.6</td>
<td>0.2</td>
</tr>
</tbody>
</table>


4. Perform a focused physical examination for the causes of ptosis.

- Unilateral
  - Third nerve palsy
  - Horner’s syndrome
  - Myasthenia gravis
  - Congenital or idiopathic
Causes of Horner’s syndrome

- Neck
  - Malignancy - e.g. Thyroid
  - Trauma or surgery

- Lower trunk brachial plexus lesions
  - Trauma
  - Tumor

- Carotid arterial lesion
  - Carotid aneurysm or dissection
  - Pericarotid tumours (Raeder’s syndrome - sweating not affected since tumor involves internal carotid artery)
  - Cluster headache

- Brainstem lesions
  - Vascular disease (especially the lateral medullary syndrome)
  - Tumour
  - Syringobulbia

- Syringomyelia (rare)

- Lung
  - Carcinoma (usually squamous cell carcinoma) of the apex of the lung


5. Perform a focused physical examination for Horner’s syndrome.

- Miosis
  - No sympathetic activity to balance parasympathetic action: paralysis of the dilator of the pupil

- Ptosis
  - Damage to sympathetic nerves of eyelids, with paralysis of upper tarsal muscle.
  - Often, slight elevation of browlid, due to paralysis of lower tarsal muscle.
  - Rarely, enophthalmos due to paralysis of the muscle of muller.

- Anhydridosis (not always present)

- Associated neurological signs
  - Ipsilateral
  - Nystagmus
  - V (pain/ temperature)
o IX, X
o Lower cranial nerves, recurrent laryngeal nerve palsy (hoarseness)
o Loss of cerebellar function
o Contralateral – loss of pain/temperature over trunk and limbs

➢ Associated non-neurological signs (see causes below)
o Clubbing, weak finger abduction, abnormal respiratory examination of lung apices, lymphadenopathy, thyroid mass, (carcinoma), carotid aneurysm or bruit
o Test for syringomyelia, with central cord, lesions (look for disassociated sensory loss, and possible bilateral Horner’s syndrome)

Useful background: Causes of abnormal reaction to light or to accommodation

➢ Pupil fails to constrict to light, but does constrict with accommodation (Argyll Robertson pupils)
o Syphilis, tertiary
o Diabetes
o Alcohol (Wernicke encephalopathy)

➢ Pupil fails to constrict to light and to accommodation (Adie’s pupil)

➢ Pupil fails to constrict to light, and actually dilates (Marcus Gunn pupil, or afferent pupillary defect, seen in optic neuritis or severe retinal damage such as central retinal artery occlusion)


6. Perform a focused physical examination for the causes of the Argyll Robertson pupil.

➢ Infection o Neurosyphilis – tabes dorsalis
  o Brainstem encephalitis
  o Sarcoidosis

➢ Infiltration o Pinealoma
  o Tumors of the posterior portion of the third ventricle

➢ Metabolic o Diabetes mellitus and other conditions with autonomic neuropathy
o Lyme disease

➤ Degenerative
  o Multiple sclerosis
  o Syringobulbia


7. Perform a focused physical examination of the patient with a large pupil which reacts slowly to light and accommodation (Holmes-Adie syndrome).

  o Near vision
    - ↓ constriction in response to near vision.
    - ↓ re-dilation after near vision.
  o If a strong and persistent stimulus is used
    - The pupil contracts excessively to a very small size
    - When the stimulus is removed, the pupil slowly returns to its former size (known as the “myotonic” pupil).
  o Segmental palsy and segmental spontaneous movement of iris

➤ Ankle reflexes – absent


“Hard work and good intentions are admirable, but in the successes of life, you have to put the puck in the net.”

Grandad
Useful background: Tonic pupil (Adie’s Pupil)

**Tonic pupil (Adie’s pupil)**

- **Dilated right pupil**

- **Exposure to light**
  - No light reaction

- **Near response**
  - Pupil constricts

- **Pilocarpine eye drops**
  - Pupil constricts markedly

➢ The patient in this figure has a right tonic pupil. At baseline, there is anisocoria with the right pupil larger than the left (first row). The dilated pupil fails to react to light (second row) but constricts slowly (i.e. ‘tonic’ contraction) when the patient focuses on a near object (third row). After instillation of dilute pilocarpine eye drops (fourth row), the pupil constricts markedly.

Useful background: The relative afferent papillary defect (Marcus Gunn Pupil)

- This shows a patient with an abnormal right optic nerve.
- The pupil that dilates during the swinging flashlight test has the 'relative afferent papillary defect' and is labelled the 'Marcus Gunn pupil'.


Useful background: Causes of cataract

- Inherited
  - Rubella syndrome
  - Down's syndrome
  - Hepatolenticular degeneration
  - Galactosemia
  - Dystrophia myotonica

- Senility

- Heat, radiation
Secondary to ocular disease
- Glaucoma
- Ophthalmitis
- Trauma (contusion cataract)

Metabolic
- Diabetes mellitus
- Hypoparathyroidism (lamellar cataract)
- Corticosteroid therapy

Miscellaneous causes
- Atopic eczema

Heat and irradiation


Useful background: Causes of retinal hemorrhage

CNS
- Subarachnoid hemorrhage
- Raised IC pressure

Retinal vessels
- Arteritis (PN, cranial arteritis, etc)
- Retinal vein thrombosis

Retina
- Trauma and retinal detachment

Hypertension

Diabetes

Hematology
- Severe anaemia, especially PA
- Bleeding diathesis-defect in platelets, vessels or coagulation factors

Affected pupil size may be dilated, depending on the cause of the CN III palsy, (e.g. intracranial aneurysm).

Abbreviation: PA, pernicious anemia

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 82.
SO YOU WANT TO BE A NEUROLOGIST!

Q: What is the neurological changes associated with hyperparathyroidism
A:  
- Cataracts
- Papilloedema
- Basal ganglia defects
- Benign intracranial hypertension


---

**Eye movement: CN III, IV, VI**

8. Perform a focused physical examination for the causes of unilateral or bilateral ptosis.

- **Unilateral**
  - Congenital (usually bilateral) – always partial
  - Oculomotor nerve lesion – usually unilateral, complete, frontalis overaction
  - Cervical sympathetic lesion (Horner’s)
  - Myasthenia gravis
  - Myopathy (senile) – facioscapulohumeral, dystrophia, myotonica, trauma
  - Tabes dorsalis, syphilis
  - Hysterical – unilateral, no frontalis overactivity, complete
  - Ideopathic

(Exclude congenital microphthalmos, contralateral exophthalmos)

- **Bilateral**
  - Myasthenia gravis
  - Myotonica dystrophia
  - Ocular myopathy or oculopharyngeal dystrophy
  - Mitochondria dystrophy
  - Tabes dorsalis
  - Congenital
  - Bilateral Horner’s syndrome (e.g. syringomyelia)

Useful background: Cardinal positions of gaze

Adapted from: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto* 2005, page 159

9. Perform a focused physical examination for a CN III lesion (of the left side in this example).

Useful background: The eye muscles and nerve innervations

Adapted from: Talley N. J., et al *Maclennan & Petty Pty Limited* 2003, Figure 10.9, page 366.
Useful background: Gaze defects of CN III, IV, VI

<table>
<thead>
<tr>
<th>Gaze defect</th>
<th>Location of lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Looks down and out (including ptosis)</td>
<td>o CN III palsy (look for pupil involvement)</td>
</tr>
<tr>
<td>➢ Can’t look in and down (difficultly walking downstairs)</td>
<td>o CN IV palsy</td>
</tr>
<tr>
<td>➢ Can’t move affected eye laterally</td>
<td>o CN VI palsy</td>
</tr>
<tr>
<td>➢ Slow adduction of ipsilateral eye and nystagmus in abduction of contralateral eye</td>
<td>o Medial longitudinal fasciculus (MLF)</td>
</tr>
<tr>
<td></td>
<td>o Internuclear ophthalmoplegia (suggests Multiple Sclerosis)</td>
</tr>
</tbody>
</table>

Source: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto*, 2005, page 159.

“Is there an upper age limit as to who should be offered a liver transplantation? Remember that the patient’s physiology is more important than chronology when making the selection decision.”
Useful background: The eye

➢ Eye movements
  o With the eyes turned *laterally* - the *elevators* and depressors are the *superior* and inferior recti, respectively
  o With the eyes turned *medially* - the *elevators* and depressors are the *inferior* and superior obliques, respectively

➢ Diplopia is caused by CN III, IV, VI disease or disease of extraocular muscles, e.g. trauma, tumour, vascular disease, multiple sclerosis, syphilis.

➢ Exophthalmos
  o Inflammation (cellulitis)
  o Thrombus (cavernous sinus thrombosis)
  o Bleeding or tumour behind the eye.
  o Thyroid disease


Useful background: Common causes of third nerve palsy

➢ Infection
  o Encephalitis
  o Basal meningitis
  o Carcinoma at the base of the skull

➢ Infiltration
  o Parasellar neoplasms
  o Meningioma at the wing of sphenoid
  o Tumors, collagen, vascular disorder, syphilis.

➢ Vascular
  o Ophthalmoplegic migraine
  o Aneurysms of posterior communicating artery (painful ophthalmoplegia).

➢ Degenerative
  o Multiple sclerosis

➢ Metabolic
  o Hypertension
  o Diabetes (pupil-sparing CN III palsy)

➢ Trauma

SO YOU WANT TO BE A NEUROLOGIST!

Q: What are the eponymous syndrome in which the third cranial nerve (CN III) is involved?

A: ❧ Weber’s syndrome: ipsilateral third nerve palsy with contralateral hemiplegia. The lesion is in the midbrain.

❖ Benedikt’s syndrome: ipsilateral third nerve palsy with contralateral involuntary movement such as tremor, chorea and athetosis. It is due to a lesion of the red nucleus in the midbrain.

❖ Claude’s syndrome: ipsilateral oculomotor paresis with contralateral ataxia and tremor. It is due to a lesion of the third nerve and red nucleus.

❖ Nothnagel’s syndrome: unilateral oculomotor paralysis combined with ipsilateral cerebellar ataxia.


Useful background: Conditions causing isolated palsies of cranial nerves III, IV and VI

<table>
<thead>
<tr>
<th>Condition</th>
<th>Oculomotor Nerve (III)</th>
<th>Trochlear Nerve (IV)</th>
<th>Abducens Nerve (VI)</th>
<th>Mixed*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proportion (%)**</td>
<td>31</td>
<td>10</td>
<td>45</td>
<td>14</td>
</tr>
<tr>
<td>Etiology (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Head trauma</td>
<td>13</td>
<td>34</td>
<td>11</td>
<td>18</td>
</tr>
<tr>
<td>Neoplasm</td>
<td>11</td>
<td>5</td>
<td>19</td>
<td>29</td>
</tr>
<tr>
<td>Ischemic</td>
<td>25</td>
<td>22</td>
<td>17</td>
<td>7</td>
</tr>
<tr>
<td>Aneurysm</td>
<td>17</td>
<td>1</td>
<td>3</td>
<td>11</td>
</tr>
<tr>
<td>Other</td>
<td>13</td>
<td>9</td>
<td>22</td>
<td>19</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>21</td>
<td>29</td>
<td>28</td>
<td>16</td>
</tr>
</tbody>
</table>

* ‘Mixed’ refers to combinations of cranial nerves III, IV, or VI
** ‘Proportion’ is ratio of palsies affecting designated cranial nerve to total number of palsies affecting cranial nerve III, IV and VI.

Useful background: Causes of a red and painful eye

<table>
<thead>
<tr>
<th>Disease</th>
<th>Distribution of redness</th>
<th>Corneal surface</th>
<th>Pupil</th>
<th>Vision</th>
<th>Iris</th>
<th>Discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacterial conjunctivitis</td>
<td>o Peripheral conjunctiva o Bilateral (central sparing)</td>
<td></td>
<td>- Normal</td>
<td>o Normal</td>
<td>o Normal</td>
<td>o Normal</td>
</tr>
<tr>
<td>Acute iritis</td>
<td>o Around cornea o Unilateral</td>
<td>- Dull</td>
<td>o Irregular shape</td>
<td>o Miotic</td>
<td>o Slowly reactive</td>
<td>o ↓/ blurred</td>
</tr>
<tr>
<td>Acute closure glaucoma</td>
<td>o Around cornea o Unilateral</td>
<td>- Dull</td>
<td>o Oval partially dilated</td>
<td>o Non-reactive</td>
<td>o ↓/ blurred</td>
<td>o Corneal edema</td>
</tr>
<tr>
<td>Corneal ulcer/abrasion</td>
<td>o Around cornea o Unilateral</td>
<td>- Dull</td>
<td>o Normal</td>
<td>o Reactive</td>
<td>o Defect shadow</td>
<td>o Watery/ muco-purulent</td>
</tr>
<tr>
<td>Subconjunctival hemorrhage</td>
<td>o Localised hemorrhage o No posterior limit</td>
<td></td>
<td>- Normal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conjunctival hemorrhage</td>
<td>o Localised hemorrhage o Posterior limit present</td>
<td></td>
<td>- Normal</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

10. Perform a focused physical examination for a CN IV lesion (of the right side in this example).

Looking down and left

- Right eye is weak

Head tilted left

- Eyes aligned

Head tilted right

- Right hypertropia

- Simple inspection (first row) reveals that the right eye lags behind left eye, indicating that the weak muscle is indeed on the right side) e.g. right superior oblique.
- Tilting the head away from the affected side (e.g. to the left side, away from the weak right superior oblique, aligns the eyes normally.
- Tilting the head toward the affected side (e.g. to the right side, third row) brings out a prominent right hypertropia (e.g. the right eye is higher than the left eye).

Adapted from: McGee S. R. Evidence Based Physical Diagnosis. 2nd Edition. Saunders/Elsevier, St. Louis, Missouri, 2007, Figure 55-6, page 684.


"Life is a memory so our treasured thoughts never need to die"

Grandad
11. Perform a focused physical examination for a CN VI lesion (on the left side in this example).

Paralysis of the CN VI causes esotropia, and an inability to fully abduct the affected left eye.


12. Perform a focused physical examination to determine the cause of a person’s diplopia.

- Diplopia is worst when looking down and to the left, indicating that the weak muscle is either the left inferior rectus muscle or right superior oblique muscle.
- Side-by-side images
  - Only lateral (VI) or medial (III) cranial nerves
- One-above-the-other
  - Superior (IV) or inferior (III) oblique, inferior rectus (III)
- False image
  - Pale, peripheral, poorly seen
- Loss of lateral image with covering one eye at point of maximum separation indicates that the covered eye is causing the diplopia.
- Persistence of both images with covering one eye is due to dislocated lens, astigmatism, or false reporting

Q: What is ‘Fisher’s one and a half syndrome’?

Horizontal eye movement is absent, and the other eye is capable only of abduction (“one and a half movements are paralysed”). The vertical eye movements and the pupils are normal. The cause is a lesion in the pontine region involving the medial longitudinal fasciculus and the parapontine reticular formation on the same side. This results in failure of conjugate gaze to the same side, impairment of adduction of the eye, and nystagmus on abduction of the other eye.


Q: What are the structures in close proximity to the CN VI nucleus and fascicles?

A: 
- Facial and trigeminal nerves
- Corticospinal tract
- Median longitudinal fasciculus
- Parapontine reticular formation
- Temporal bone


Q: How would you distinguish congenital from non-congenital Horner’s syndrome?

A: In congenital Horner’s, there are all the usual features of miosis, ptosis, enophthalmos, and elevation of the lower lip, plus there would be heterochromia of the iris (i.e. the iris remains grey-blue).
SO YOU WANT TO BE AN OPHTHALMOLOGIST!

Q: What are the eponymous syndromes affecting CN VI?
A: ➢ Pons Infarction
  - Raymond’s syndrome: ipsilateral CN VI paralysis and contralateral paresis of extremities
  - Millard-Gubler syndrome, in which there is ipsilateral VI and VII palsy, with contralateral hemiplegia.
  - Foville’s syndrome has all the features of Millard-Gubler paralysis, plus lateral conjugate gaze palsy.

➢ Gradening’s syndrome
  - Inflammation of the tip of the temporal bone, involving V and VI, as well as the greater superficial petrosal nerve
  - This results in unilateral paralysis of the lateral rectus, nerve, pain in the distribution of V (particularly V₁), and excessive lacrimation

➢ Others
  - Duane’s syndrome: widening of the palpebral fissure on abduction, and narrowing on adduction
  - Gerhardt’s syndrome: bilateral abducens palsy
  - Mobius syndrome: paralysis of extraocular muscles, especially abducens, with paresis of facial muscles


Useful background: Causes VI nerve palsy

➢ Hypertension
➢ Diabetes
➢ Raised intracranial pressure (false localizing signs)
➢ Multiple sclerosis
➢ Basal meningitis
➢ Encephalitis
➢ Nasopharyngeal carcinoma
➢ Acoustic neuroma

Face

Useful background: The anatomy of the facial nerve (CN VII)

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 74.

Useful background: Ramsay-Hunt Syndrome (herpes zoster of geniculate ganglion)

- Pain in ear and mastoid region
- Facial paresis or spasm
- Deafness, dizziness or hyperacusis
- Vesicles on auricle or anterior fauces
- Ipsilateral taste loss in anterior two-thirds tongue


13. Perform a focused physical examination for the causes of facial weakness/paralysis (CN VII lesion).

- Supranuclear
  - Cortico-pontine tract lesions
  - Sub-thalamic and corpus striatum lesions
  - Temporal lobe lesions
- Nuclear and infra-nuclear
- Pontine
  - Polio
  - DS
  - Neoplasms
- Cerebello-pontine angle
  - Acoustic neuroma
  - Meningioma
  - Basilar artery aneurysm
  - Guillain-Barré syndrome
  - Chordoma
  - Chronic meningitis, including carcinomatous

- Internal auditory canal
  - Acoustic neuroma
  - Geniculate herpes

- Facial canal
  - Bell’s palsy
  - Chronic otitis, cholesteatoma or mastoidectomy
  - Head injury
  - Hypertension in children
  - Sarcoidosis
  - Leukemic infiltrate

- Face
  - Forceps delivery
  - Stab wounds
  - Parotid tumours
  - Leprosy

- Unilateral
  - UMN (sparing of forehead)
    - Melkersson-Rosenthal syndrome (facial palsy, recurrent facial edema, and plication of the tongue)
    - Myasthenia (may mimic bilateral facial nerve palsy)
  - LMN lesion
    - Idiopathic (Bell’s palsy)
    - Herpes zoster
    - Cerebellopontine angle tumors
    - Parotid tumors
    - Old polio
    - Otitis media
    - Stroke (hemiplegia)
    - Skull fracture

- Bilateral
  - Facial nerve (VII) damage
    - Guillain-Barré syndrome
Sarcoidosis
- Bilateral parotid disease
- Lyme disease
- Mononeuritis multiplex
  - Muscle disease
  - Myopathy, myasthenia gravis

Abbreviations: MS, multiple sclerosis; LMN, lower motor neurons; UMN, upper motor neurons


14. Perform a focused physical examination for the causes of facial pain.

- **CNS**
  - Migrainous neuralgia (‘cluster’ headache)

- **Skull / spine**
  - Cervical spondylosis, Paget’s of skull

- **Disease of teeth, sinuses, ear, nose or throat**

- **CN VII**
  - Post-herpetic neuralgia
  - Trigeminal neuralgia

- **TMJ**
  - Temporo-mandibular arthritis (Costen’s syndrome)

- **Ear**
  - Acoustic neuroma

- **Heart**
  - Myocardial ischemia

- **Blood vessels**
  - Cranial arteritis
  - Aneurysm of posterior communicating artery- posterior inferior cerebellar artery

- **Miscellaneous**
  - Atypical facial pain – Constant, nagging deep pain not corresponding to any anatomical sensory distribution
  - MS - facial

Adapted from: Burton J.L. *Churchill Livingstone* 197, page 74.
SO YOU WANT TO BE A NEUROLOGIST!

Q: In the context of CN VII, what are the Raeder’s paratrigeminal syndrome, and the superior orbital fissure syndrome?

A: Raeder’s paratrigeminal syndrome - Severe retro-orbital pain succeeded by ipsilateral miosis and ptosis
   Superior orbital fissure syndrome – Boring retro-orbital pain and paresis or cranial nerves III, IV, V and VI

SO YOU WANT TO BE A NEUROLOGIST!

Q1: What is the component of the facial nerve (CN VII). Which is sensory and what does it supply?

A1: o The nervus intermedius of Wrisberg
    o Taste sensation from the anterio two thirds of the tongue
    o Probably, cutaneous impulse from the anterio wall of the external auditory canal.


Q2: How do you localize the site of the facial nerve palsy?

A2: o Involvement of the nuclei in pons – associated ipsilateral sixth nerve palsy.
    o Cerebellopontine angle lesion – associated fifth and eight nerve involvement.
    o Lesion in the bony canal – loss of taste (carried by the lingual nerve) and hyperacusis (due to involvement of the nerve to stapedius.

Q3: What reflexes involve the facial nerve?

A3: o Corneal reflex
    o Palomomental reflex
    o Suck reflex
15. Perform a focused physical examination for a lesion in the cavernous sinus, cerebellopontine angle, jugular foramen, pseudobulbar and bulbar palsy (multiple cranial nerve palsies), and its causes.

<table>
<thead>
<tr>
<th>Site</th>
<th>Affected cranial nerve</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cavernous sinus</td>
<td>o Unilateral III, IV, V and VI</td>
</tr>
<tr>
<td>Cerebellopontine angle lesion (usually a tumor)</td>
<td>o Unilateral V, VII and VIII</td>
</tr>
<tr>
<td>Jugular foramen lesion</td>
<td>o Unilateral IX, X and XI</td>
</tr>
<tr>
<td>Bulbar (LMN) and pseudobulbar (UMN) palsy</td>
<td>o Combined bilateral X, XI and XII</td>
</tr>
</tbody>
</table>

SO YOU WANT TO BE A NEUROLOGIST!

Q: In the patient with Bell’s palsy, what is Bell’s phenomenon?
A: Bell’s palsy is a peripheral mononeuropathy of CN VII which affects the peripheral nerve and results in both UMN and LMN lesions. In a person with lower (peripheral) CN VII damage, the eyelid of the affected side cannot be closed, so the eyeball on that side moves (upwards when the person closes the eyelid of the unaffected side (synkinesis), using the intact orbicularis muscle contraction on that side.

Useful background: Causes of multiple cranial nerve palsies

- Inherited
  - Arnold Chiari malformation

- Infection
  - Guillain Barre syndrome (spares sensory nerves)
  - Tuberculosis
  - Sarcoidosis

- Infiltration
  - Nasopharyngeal carcinoma
  - Hematological malignancy,
  - Brainstem tumor (e.g. in the cerebellopontine angle) have similar signs

- Vascular
  - Brainstem vascular disease causing crossed sensory or motor paralysis (i.e. cranial nerve signs on one side and contralateral long tract signs).

- Trauma

- Metabolic
  - Paget’s disease
  - Mononeuritis multiplex (rarely, e.g. diabetes mellitus)

Abbreviations: LMN, lower motor neurons; MS, Multiple sclerosis; UMN, upper motor neurons


16. Take a directed history and perform a focused physical examination for a lesion at the cerebellopontine angle.

- Definition
  - Symptoms and signs related to cranial nerves VIII and IX.
  - The cerebellopontine angle is the shallow triangular fossa lying between the cerebellum, lateral pons and the inner third of the petrous temporal bone.
  - This angle extends from the trigeminal nerve (above) to the glossopharyngeal nerve (below).
  - The abducens nerve runs along the medial edge, whereas facial and auditory cranial nerves transverse the angle, to enter the internal auditory meatus.
Causes
  o Infection
    - Local meningeal involvement
    - Syphilis
    - Tuberculosis
  o Infiltration
    - Acoustic neuroma.
    - Meningioma
    - Cholesteatoma
    - Hemangioblastoma
    - Pontine glioma.
    - Medulloblastoma and astrocytoma of the cerebellum.
    - Carcinoma of the nasopharynx.
  o Vascular
    - Aneurysm of the basilar artery


Ear: cranial nerve VIII

Useful background:
- Rinne – normal: air > Bone conduction ([BC], conduction hearing loss)
- Weber - lateralizes to good ear – neurosensory loss; lateralizes to bad ear – conductive loss
- Vestibulo-ocular reflex (vestibular component)

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Middle ear</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rinne</td>
<td>AC&gt; BC</td>
</tr>
<tr>
<td>Weber</td>
<td>Normal ear louder</td>
</tr>
<tr>
<td></td>
<td>AC= AC; ACb, BCb</td>
</tr>
<tr>
<td></td>
<td>Abnormal ear louder</td>
</tr>
</tbody>
</table>

Abbreviations: AC, air conduction; BC, bone conduction; C, conductive; CN cranial nerves

Adapted from: Filate W., et al. Medical Society, Faculty of Medicine, University of Toronto, 2005, pages 153 to 154 and 157 to 158.
Weber test | Rinne test | Possible interpretations
---|---|---
- Midline | o AC> BC, bilateral | - Normal hearing, bilateral
- Neurosensory loss, bilateral
- Louder in left | o BC> AC, left | - Conductive loss, left
- o AC> BC, right
- Louder in left | o AC> BC, bilateral | - Normal hearing, bilateral
- Neurosensory loss, worse on right
- Louder in right | o BC> AC, bilateral | - Conductive loss, bilateral but worse on right
- Conductive loss on right and severe neurosensory loss on left


**Clinical Pearl**

Uvula – deviates to strong side; jaw and tongue – deviate to weak side.
Finding | PLR | NLR
--- | --- | ---
- **Hearing tests**
  - Abnormal whispered voice test | 6.0 | 0.03
- **Tuning fork tests (patients with unilateral hearing loss)**
  - Rinne test, detecting conductive hearing loss | 16.8 | 0.2
  - Weber test, lateralizes to good ear, detecting neurosensory loss | 2.7 | NS
  - Weber test lateralizes to bad ear, detecting conductive loss | NS | 0.5

NS, not significant; likelihood ratio (LR) if finding present= positive LR, LR if finding absent= negative LR.

<table>
<thead>
<tr>
<th>Probability Decrease</th>
<th>Probability Increase</th>
</tr>
</thead>
<tbody>
<tr>
<td>-45%</td>
<td>-30%</td>
</tr>
<tr>
<td>0.1</td>
<td>0.2</td>
</tr>
</tbody>
</table>

**Sen N out** – Sensitive test; when negative, rules out disease

**Sp P in** – Specific test; when positive, rules in disease

### Cranial nerves IX, X, XII

17. Perform a focused physical examination to determine the site of defect and the causes of dysarthria (disorder of articulation).

<table>
<thead>
<tr>
<th>Site of defect</th>
<th>Causes</th>
<th>Characteristics of speech</th>
</tr>
</thead>
</table>
| ➢ Supra-nuclear (pseudo-bulbar palsy) - UMN lesions of CN IX, X or XII | o CVA  
   o MND  
   o MS | - Monotonous  
   - High-pitched  
   - “hot potato” speech |
| ➢ Nuclear (bulbar palsy, LMN lesions of CN IX, X or XII) | o MND  
   o Guillain-Barré syndrome  
   o Tumour of medulla  
   o Bulbar polio  
   o Syringobulbia | |
| ➢ Basal ganglia | o Parkinsonism  
   o Wilson’s disease  
   o Choreoathetosis | - Slow  
   - Wuet  
   - Slurred  
   - Monotonous |
| ➢ Cerebellum | o MS  
   o Tumour  
   o Drugs and toxins (alcohol) | - Staccato  
   - Scanning |
| ➢ Muscle | o Myasthenia gravis  
   o Muscular dystrophy | |
| ➢ Mouth | o False teeth  
   o Cleft palate  
   o Stuttering | |

Abbreviations: CN, cranial nerve; CVA, cerebrovascular accident; LMN, lower motor neuron; MND, motor neuron disease; MS, multiple sclerosis; UMN, upper motor disease

Perform a focused physical examination for posterior inferior cerebellar artery thrombosis.

- V - Ipsilateral loss
- VI, VII, VIII – (Often transient)
- IX, X, XII – (Bulbar palsy)
- Cerebellum – ataxia, with nystagmus to the side of the lesion
- Lateral spinothalamic pathway – (Often transient)
- Homolateral Horner’s syndrome
- Bulbar palsy affects motor nuclei.

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 78.
19. Take a directed history and perform a focused physical examination for the jugular foramen syndrome.

➢ Definition
  - Symptoms/signs from impaired function of cranial nerves IX, X and XI.
  - The jugular foramen is beated between the lateral part of the occipital bone and the petrous portion of the temporal bones.

➢ History
  - Eye
    - Ptosis (due to Horner’s syndrome).
  - Ear
    - Pain in and around the ear (due to damage of the ninth and tenth cranial nerves which carry sensation to the external auditory meatus and behind the ear).
  - Head
    - Headache
  - Voice
    - Hoarseness of voice.
    - Nasal quality to the speech.
  - Throat
    - Nasal regurgitation and dysphagia.
    - Aspiration of food with choking attacks.
  - Tongue
    - Wasting of the tongue (often noticed by the dentist).
  - Neck
    - Weakness of the sternomastoids and trapezius.

➢ Physical Examination
  - Throat
    - Sluggish movement of the palate on the affected side when the patient says ‘aah’.
    - Absent gag reflex on the same side.
  - Chin
    - Weakness when the patient moves her chin to the opposite side.
  - Neck
    - Flattening of the shoulder on the same side.
    - Wasting of the sternomastoid.
    - Difficulty in shrugging the shoulder on the same side.

➢ Causes
  - Infection
    - Basal meningitis.
  - Infiltration
    - Carcinoma (of the pharynx is the commonest cause).
- Neurofibroma or any tumour.
  - Vascular
    - Thrombosis of jugular vein.
  - Trauma
    - Fractured base of the skull.
  - Metabolic
    - Paget’s disease.


**Nystagmus and vertigo**

Useful background: Nystagmus

- **Definition**
  - A series of involuntary, rhythmic oscillation of one or both eyes.
  - May be horizontal, vertical or rotary.
  - Slow drifting of eye, then rapid correcting movement.
  - The direction of the rapid correcting movement is the direction used to describe the nystagmus.
  - Ascentuate nystagmus by looking away from the straight-ahead or middle line.
  - Lesions of vestibular apparatus, cerebellum, toxins (alcohol).

- **Causes**

- **Physiological e.g. opto-kinetic**

- **Eye**
  - Errors of refraction and macular lesions
  - Weakness of ocular muscles (Lesion of CN III, IV or VI)

- **Lesions of vestibular apparatus, cerebellum, brain stem**

Vestibular nystagmus

<table>
<thead>
<tr>
<th></th>
<th>Central (vestibular nuclei)</th>
<th>Peripheral (labyrinth or vestibular nerve)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vertigo</td>
<td>Rare</td>
<td>Yes</td>
</tr>
<tr>
<td>Auditory symptoms</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Lying still, fixing eyes on bright objects helpful</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>

Where is the lesion in vestibular nystagmus?

- Central (affecting vestibular nuclei), as in
  - Cause
    - CVA
    - MS
    - Tumor
    - Alcoholism.
- Peripheral (labyrinth or vestibular nerve)
  - Meniere’s syndrome

- Acoustic neuroma
- Otitis media
- Head injury.


20. Perform a focused physical examination for the causes of nystagmus/vertigo.

- **Eyes (nystagmus)**
  - Physiological, e.g. opto-kinetic
  - Errors of refraction and macular lesions
  - Weakness of ocular muscles
  - Lesion of cranial nerves III, IV or VI
- **Brainstem lesions, cerebellum, temporal cortex**
- **Vestibular apparatus and nerves**

- **High cervical cord diseases**
  - Vestibular lesions
    - Physiological
    - Labrinthitis
    - Menière’s
    - Bleeding, in leukemia
    - Drugs, e.g. quinine, salicylates, alcohol
    - Otitis media
    - Motion sickness
  - Vestibular nerve lesions
    - Acoustic neuroma
    - Drugs, e.g. streptomycin
    - Vestibular neuronitis
  - Brain stem, cerebellar or temporal cortical lesions
    - Pontine infarction or hemorrhage
    - Vertebro-basilar insufficiency
    - Basilar artery migraine
    - Temporal lobe epilepsy
    - Disseminated sclerosis
    - Tumours
    - Benign post-traumatic positional vertigo

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 76.
Headache and facial pain

Useful background: Mechanisms of headache production

- **Muscle**
  - Skeletal muscle contraction (e.g. ‘tension’ headache)

- **ENT**
  - Referred pain, e.g. disease of eyes, ears, sinuses, teeth, cervical spine

- **Artery**
  - Systemic hypertension
  - Arterial dilatation
    - Intra-cranial
      - systemic infections
      - hypertension
      - nitrites
      - postictal
      - concussion
    - Extracranial (e.g. migraines)
  - Traction on arteries e.g. raised IC pressure

- **Vein**
  - Dilatation or traction on venous sinuses e.g. post lumbar puncture
  - Analgesics
  - Oral contraceptive pill

- **Inflammation**
  - Intra-cranial (e.g. meningitis)
  - Extra cranial (e.g. giant cell arteritis)

- **Psychogenic**

Abbreviation: IC, intracranial

Useful background: Headache and facial pain


Useful background: History taking for headache

- Likely many aspects of history taking, a combination of pretest probability estimation, a system of inquiry, and thoughtful reiteration and probing work best. For any pain or discomfort, the “PQRST” mnemonic described in DeGowan and DeGowan works well-provocative or palliating factors, quality, radiation, severity, and temporal relationships. Below is illustrated one approach. It is not intended to be exhaustive.

- Character of headache
  - Quality of pain: Is it steady/throbbing, constant/remittent, sharp/dull, superficial/deep?
Location of pain) regional or diffuse

Severity of pain: Try to quantify this if possible:
- Ask "on a scale of 1 to 10, 10 being “the worst pain you have ever had and 1 being pain free, what number would you give the pain?"

Temporal relationships
- Age of patient and age of onset of first episode, relationship to time of day, weekends, menses
- Clustering or chronicity
- Rapidity of onset and duration of episodes

Palliating and provocative factors: Changes with position, neck movement, chewing, foods, alcohol, menses, cough/straining, stressors, eyestrain, massage, sleep?

Associated symptoms: These include systemic illness/infection, nausea/vomiting, diarrhea, photophobia/photophobia, lacrimation/stuffy nose, facial flushing, scalp tenderness, jaw claudications/myalgias and stiffness/temporal tenderness, weight loss, palpitations, depression/change in mentation or personality, aura, scintillating scotomas, neurovisual disturbances

Medications: include BCP (efficacy or overuse of), analgesics, alcohol

Past Medical History: Commonly missed on history taking. It should include prior history of headache/ investigations/diagnosis/ treatment. Hypertension, seizures, sinusitis, head injury, glaucoma, problems with refractive error, temporal arteritis, dental or ENT problems.

Family history: Frequently missed and should include migraine, subarachnoid hemorrhage or stroke at an early age.

Ominous features
- Worst headache of patient’s life, especially if rapid onset
- Exacerbation of headache with coughing, sneezing, or bending down
- Headache with seizures, reduced level of consciousness, confusion focal neurological findings
- New or progressive headache persisting for days
- New-onset headache in middle age or older
- Change in frequency, severity, or clinical features of the usual headache pattern
- Presence of systemic symptoms including fever, myalgia, malaise, weight loss, scalp tenderness, or jaw claudication

Abbreviations: BCP, birth control pain

Useful background: Characteristics of headache

➢ Tension
  o Lasts 30 min – 7 days
  o Not pulsating, mild/moderate in intensity, bilateral
  o Not aggravated by exertion, not associated with nausea/vomiting, or sensitivity to light, sound, or smell
  o Episodic or chronic, bilateral frontal/occipital/frontal area, not awakening person at night, with no vomiting, no photophobia or stiff neck

➢ Migraine with aura
  o Unilateral headache preceded by flashing light or zig-zag lines, associated with photophobia.
  o Lasts 4-72 hrs
  o Throbbing, moderate/severe intensity, unilateral (not always the same side)
  o Worse with exertion
  o Associated with photophobia, phonophobia, nausea/vomiting
  o May be preceded by short prodromal period of depression, irritability, restlessness, or anorexia, 10-20% occurrences associated with an aura-transient, reversible neurologic visual, somatosensory, motor, and/or language deficit-usually precedes headache by no>1 h, can be concurrent
  o Diagnosis of migraine without aura plus neurological dysfunction sensed before or during an attack

➢ Migraine without aura
  o A diagnosis of migraines without aura requires each of the following:
    i. Minimum of 5 attacks
    ii. Duration of headache is 2-72 h (with or without therapy)
    iii. Two of the following are present: unilateral pain, pulsing or throbbing quality to pain, moderate-to-severe intensity preventing daily activities, or pain provoked by routine physical activity
    iv. One of the following is present: nausea, vomiting, photophobia, phonophobia, or osmophobia
    v. No evidence of other causes of headache

➢ Cluster
  o Usually with pain over one eye with tears, runny nose,
and flushing of the forehead lasting minutes to hours, in bouts lasting several weeks, and coming a few times a year.

- Lasts 15-180 min, occurs up to 8 times per day
- Severe, unilateral, located peri-orbitally and or temporally
- Associated with at least one of: tearing, red eye, stuffy nose, facial sweating, ptosis, miosis

- **Subarachnoid hemorrhage**
  - Acute, severe, ‘thunderclap’
  - May have neurologic deficits or changes in level of consciousness

- **Brain tumour**
  - Presents on wakening and improves during the day
  - Symptoms due to increased intracranial pressure
  - Generalized, may be more severe in occipital region, worse when lying down or with a Valsalva manoeuvre
  - May be associated with nausea/vomiting, blurring of vision, papilloedema, transient visual obscuration

- **Cervical spondylosis**
  - Occipital headache, neck stiffness

- **Meningitis**
  - Generalized headache, neck stiffness, photophobia, fever

- **Increased intracranial pressure**
  - Generalized headache, worse in the morning, with vomiting and drowsiness

- **Temporal arteritis**
  - Unilateral headache, blurred vision, tenderness over temporal artery

- **Sinusitis**
  - Generalized headache, pressure/fullness behind the eyes/cheek/forehead

- **Subarachnoid hemorrhage**
  - Localized generalized headache, sudden onset, neck stiffness

21. Take a directed history to determine the causes of facial pain.

- **Eye**
  - Glaucoma
  - Superior orbital fissure syndrome

- **TMJ**
  - Arthritis

- **Blood vessels**
  - Temporal arteritis
  - Cluster headache
  - Aneurysm of the internal carotid or posterior communicating artery
  - Risk factors for CVA
    - Hypertension
    - Smoking
    - Diabetes mellitus
    - Hyperlipidemia
    - Atrial fibrillation
    - Bacterial endocarditis
    - Myocardial infarction (emboli)
    - Hematological disease
    - Family history of stroke

- **Nerve**
  - Trigeminal neuralgia

- **Psychiatric disease**

**Abbreviation:** TMJ, temporomandibular joint

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 74.

"Be an advocate, seek something good for everyone"

Grandad
Brain stem

22. Perform a focused physical examination to distinguish between an intramedullary from an extramedullary cord lesion.

<table>
<thead>
<tr>
<th></th>
<th>Intramedullary</th>
<th>Extramedullary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Root pain</td>
<td>Rare</td>
<td>Common</td>
</tr>
<tr>
<td>Corticospinal signs</td>
<td>Late onset</td>
<td>Early onset</td>
</tr>
<tr>
<td>LMN signs</td>
<td>Extend for several segments</td>
<td>Localized</td>
</tr>
<tr>
<td>Sensory loss</td>
<td>Dissociated sensory loss (pain and temperature) may be present</td>
<td>Brown-Sequard syndrome if lateral cord compression</td>
</tr>
<tr>
<td>Sacral sparing</td>
<td>May have sacral sparing</td>
<td>No sacral sparing</td>
</tr>
<tr>
<td>CSF fluid</td>
<td>Normal or minimally altered</td>
<td>Early, marked abnormalities</td>
</tr>
</tbody>
</table>

Abbreviations: CSF, cerebrospinal fluid; LMN; lower motor neuron


Useful background: Lateral medullary infarction (Wallenberg’s syndrome)

- Physical Finding

  - Cranial nerves
    - Nystagmus
    - Ipsilateral Horner’s syndrome
    - Ipsilateral face analgesia (V)
    - Ipsilateral palate weakness (IX, X)
    - Diminished corneal reflex (V and VII)
    - Ipsilateral facial weakness (VII)

  - Sensory
    - Contralateral body analgesia

  - Coordination
    - Ipsilateral limb ataxia
    - Gait ataxia
Strictly speaking, Horner’s syndrome does not involve cranial nerves, although it is discovered during examination of the pupils and eyelids.


23. Take a directed history and perform a focused physical examination for lateral medullary syndrome (LMS).

-definition
- The LMS results from infarction of a wedge-shaped area of the lateral aspect of the medulla and inferior surface of the cerebellum.
- Due to occlusion of any of the following five vessels:
  - Posterior inferior cerebellar artery
  - Vertebral artery
  - Superior, middle or inferior lateral medullary arteries
- Caused by involvement of one side of the nucleus ambiguous, trigeminal nucleus, vestibular nuclei, cerebellar peduncle, spinothalamic tract and autonomic fibres.

-history
- Severe nausea, vomiting, nystagmus (involvement of the lower vestibular nuclei).
- Limb ataxia (involvement of the inferior cerebellar peduncle).
- Intractable hiccups, dysphagia (ninth and tenth cranial nerve involvement)

-physical examination
- Nystagmus.
- Ipsilateral involvement of CN V, VI, VII, VIII, IX, X
- Bulbar palsy: impaired gag, sluggish palatal movements.
- Ipsilateral Horner’s syndrome.
- Ipsilateral cerebellar signs
- Contralateral loss of pain and temperature (dissociated sensory loss)

24. Take a directed history and perform a focused physical examination to
distinguish between pseudobulbar and bulbar palsy.

<table>
<thead>
<tr>
<th></th>
<th>Pseudobulbar palsy (UMN)</th>
<th>Bulbar palsy (LMN)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prevalence</td>
<td>o Common</td>
<td>o Rare</td>
</tr>
<tr>
<td>Type of lesion</td>
<td>o UMN</td>
<td>o LMN, muscle</td>
</tr>
<tr>
<td>Site of lesion</td>
<td>o Bilateral, usually in the internal capsule</td>
<td>o Medulla oblongata</td>
</tr>
<tr>
<td>Tongue</td>
<td>o Small, stiff and spastic</td>
<td>o Flaccid, fasciculations</td>
</tr>
<tr>
<td>Speech</td>
<td>o Slow, thick and indistinct</td>
<td>o Nasal twang</td>
</tr>
<tr>
<td>Nasal regurgitation</td>
<td>o Not prominent</td>
<td>o Prominent</td>
</tr>
<tr>
<td>Jaw jerk</td>
<td>o Brisk</td>
<td>o Normal or absent</td>
</tr>
<tr>
<td>Other findings</td>
<td>o UMN lesions of the limbs</td>
<td>o LMN lesions of the limbs</td>
</tr>
<tr>
<td>Affect</td>
<td>o Emotionally labile</td>
<td>o Normal affect</td>
</tr>
<tr>
<td>Causes</td>
<td>o Strokes</td>
<td>o Motor neuron disease</td>
</tr>
<tr>
<td></td>
<td>o Multiple sclerosis</td>
<td>o Poliomyelitis</td>
</tr>
<tr>
<td></td>
<td>o Motor neuron disease</td>
<td>o Guillain-Barre syndrome</td>
</tr>
<tr>
<td></td>
<td>o Creutzfeld-Jakob disease</td>
<td>o Myasthenia gravis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>o Myopathy</td>
</tr>
</tbody>
</table>

Cerebellum

Useful background: Causes of cerebellar disorder

- **Congenital/ hereditary**
  - Friedreich’s ataxia and other hereditary ataxias
  - Congenital malformations at the level of the foramen magnum

- **Drugs/ toxins**
  - Phenytoin toxicity
  - Alcoholic cerebellar degeneration (there is atrophy of the anterior vermis of the cerebellum)

- **Tumor**
  - Space-occupying lesion in the posterior fossa including cerebellopontine angle tumour
  - Paraneoplastic manifestation of bronchogenic carcinoma

- **Vascular**
  - Brainstem vascular lesion

- **Demyelination**
  - Multiple sclerosis

- **Common**
  - Infectious
    - Viral infections
    - Prion disease (Creutzfeldt- Jakob disease)
  - Metabolic
    - Hepatic encephalopathy
    - Hypothyroidism
    - B12 deficiency
    - Thiamine deficiency
    - Hyperthermia
  - Cardiovascular
    - Anoxia
    - Infarction
    - Hemorrhage
  - Inherited
    - Friedreich’s ataxia
    - Ataxia telangiectasia
    - Ramsay- Hunt disease

- **Causes of ataxia**
  - Acute
    - Cerebellar hemorrhage or infarction
    - Trauma
    - Intoxication
    - Migraine
Chronic
- Alcoholic cerebellar degeneration
- Hypothyroidism
- Hydrocephalus
- Chronic infection (panencephalitis, rubella, prion disease)
- Vitamin E deficiency
- Paraneoplastic syndrome

➢ Alcoholic cerebellar degeneration
  - An ataxia that affects the trunk and gait (upper body ataxia and
dysarthria are less frequent).
  - Atrophy of anterior vermis in cerebellum
  - Gait is broad-based and is progressive, but partially reversible
    with abstinence.
  - May present as a complex called Wernicke’s encephalopathy
    (confusion, ataxia, ophthalmoplegia of CN VI).

Adapted from: Jugovic P.J., et al. Saunders/Elsevier 2004, page 166;

SO YOU STILL WANT TO BE A NEUROLOGIST!

Q: In addition to Friedreich’s ataxia, what are other syndromes with
spinocerebellar degeneration?
A: Roussy-Levy disease: hereditary spinocerebellar degeneration with
atrophy of lower limb muscles and loss of deep tendon reflexes.
Refsum’s disease
Machado-Joseph disease – dominant inheritance (first described in
families of Portuguese origin)
- Progressive ataxia, ophthalmoparesis, spasticity, dystonia,
  amyotrophy and parkinsonism.
Dentatorubral pallidoluysian atrophy, similar to Machado-Joseph
disease but maps on the short arm of chromosome 12 rather than 14.

25. **Perform a focused physical examination for disorders of the cerebellum.**

- **Nystagmus (75%)**
  - The cerebellum receives afferents from the vestibular nuclei, spinal cord, and cerebral cortex via the pontine nuclei.

- **Limb Ataxia**
  - Dysmetria (~75%)
  - Intention tremor (29%)
  - Hypotonia
  - Dysdiadochokinesia (~50%)
  - Arm drift (~50%)
  - Rebound

- **Finger-Nose Test**

- **Dysdiadochokinesia Rebound (50%)**
  - Rebound phenomenon - inability to arrest strong contraction on sudden removal resistance. This is known as Holmes' rebound phenomenon.

- **Intention Tremor**
  - Dysdiadochokinesia - impairment of rapid alternating movements (clumsy)

- **Heel-Shin Test**

- **Gross and Fine Motor Inco-ordination**
  - Lack of finger–nose coordination (past-pointing): movement is imprecise in force, direction and distance – dysmetria

- **Speech**
  - Dysarthria (say 'British Constipation')
  - Scanning dysarthria: halting, jerking dysarthria which is usually a feature of bilateral lesions

- **Intention Tremor (29%)**

- **Leg**
  - Tone

- **Knee Jerks**
  - Pendular
  - Reduced / absent

- **Gait & Balance (90%)**
  - Balance
    - Romberg's test, Pull test
  - Gait – Normal gait, Toe walking, Heel walking, Tandem gait, Ataxia
  - Dyssynergia – movements involving more than one joint are broken into parts
  - Lack of co-ordination of gait – patient tends to fall towards the side of the lesion

26. Perform a focused physical examination to distinguish between sensory ataxia and cerebellar ataxia.

<table>
<thead>
<tr>
<th>Clinical</th>
<th>Cerebellar ataxia</th>
<th>Sensory ataxia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site of lesion</td>
<td>Cerebellum</td>
<td>Posterior column or peripheral nerves</td>
</tr>
<tr>
<td>Deep tendon reflexes</td>
<td>Unchanged or pendular</td>
<td>Lost or diminished</td>
</tr>
<tr>
<td>Deep sensation</td>
<td>Normal</td>
<td>Decreased or lost</td>
</tr>
<tr>
<td>Sphincter disturbances</td>
<td>None</td>
<td>Decreased when posterior column involved, causing overflow incontinence</td>
</tr>
</tbody>
</table>


Useful background: Causes of spastic and ataxic paraparesis (upper motor neuron [UMN] and cerebellar signs combined)

- Congenital
  - Arnold-Chiari malformation, or other lesion at the craniospinal junction

- Infection
  - Syphilitic meningomyelitis

- Infiltration
  - Lesion at the craniospinal junction e.g. meningioma
Ischemic
  - Syringomyelia
  - Infarction (in upper pons or internal capsule on one side – ‘ataxic hemiparesis’)

Degeneration
  - Spinocerebellar degeneration e.g. Marie’s spastic ataxia
  - Multiple sclerosis
  - Spinocerebellar degeneration

NB: Unrelated diseases that are relatively common (e.g. cervical spondylosis and cerebellar degeneration from alcohol) may cause a similar clinical picture.


-----------------------------------------------
NEUROLOGY PROGRAM DIRECTOR: PLEASE GO AWAY AND LEAVE ME ALONE!!

Q1: What is Benedikt’s syndrome?
A1:  - Cerebellar signs on the side opposite the third nerve palsy (which is produced by damage to the nucleus itself or to the nerve fascicle).
  - Due to a midbrain vascular lesion causing damage to the red nucleus, interrupting the dentatorubrothalamic tract from the opposite cerebellum.

Q2: What is the medial medullary syndrome?
A2:  - Occlusion of the lower basilar artery of vertebral artery.
  - Ipsilateral lesions result in paralysis and wasting of the tongue.
  - Contralateral lesions result in hemiplegia and loss of vibration and joint position sense.
Spinal cord and nerve roots

Useful background: Spinal cord - Transverse section

Adapted from: Burton J.L.  *Churchill Livingstone* 1971, page 83.

Useful background: Spinal cord disorders

- Paraplegia or quadriplegia due to complete transverse lesions
- Effect depends on level (e.g. C1-C3: death from respiratory paralysis)

- Two stages:
  - Loss of all reflex activity below level of lesion
  - Atonic bladder/bowel with overflow incontinence
  - Gastric dilatation
  - Loss of vasomotor control
  - Hyperactive tendon reflex
  - Frequency and urgency of urination, automatic emptying of bladder
  - Hyperactive vasomotor and sweating reactions

- Central cord syndrome
  - Occurs more often in older people or in patients with cervical spondylosis
  - Weakened hands with impaired pain sensation (most prominent symptom)
- Relatively little long tract signs

- Anterior cord syndrome
  - Caused by infarction in anterior spinal artery territory or tumor invasion or inflammatory myelitis in similar region
  - Paraplegia or quadriplegia
  - Bilateral loss of pain and temperature sensation below the lesion
  - Sparing or posterior column (joint position and vibration) sense

- Conus Medullaris and Cauda Equina syndrome
  - Pain localized to the low back
  - Severe radicular pain in the legs
  - Loss of bladder and bowel control
  - Laxity of the anal sphincter
  - Erectile dysfunction
  - Loss of sensation in sacral segments (saddle parathesia)
  - Often asymmetric leg weakness with upper and lower motor neuron signs

- Spinal cord syndromes
  - Motor
    - Atrophy and areflexia of the arms
    - UMN lesion of legs
  - Sensory
    - Loss of pain and temperature over neck, shoulders and arms

- Subacute combined degeneration
  - Motor
    - UMN signs in both lower limbs
  - Sensory
    - Bilateral posterior column
    - Bilateral loss of position and vibration with ataxic gait
    - Rarely, peripheral sensory neuropathy
  - Reflexes
    - Ankle reflexes absent
    - Knee reflexes either absent, or exaggerated
  - Dementia
  - Optic atrophy

Abbreviations: UMN, upper motor neuron

27. Perform a focused physical examination to localize a spinal cord lesion to a specific lumbar or sacral nerve root level.

- **5th lumbar root level**
  - Muscular weakness: hamstring, peroneus longus, extensors of all the toes
  - Deep tendon reflexes affected: none
  - Radicular pain/paraesthesia: buttock, posteolateral thigh, anterolaeral leg, dorsum of foot
  - Superficial sensory deficit: dorsum of the foot and anterolateral aspect of the leg

- **1st sacral root level**
  - Muscular weakness: plantar flexors, extensor digitorum bevis, peroneus longus, hamstrings
  - Deep tendon reflexes affected: ankle jerk
  - Radicular pain/paraesthesia: buttock, back of thigh, calf and lateral border of the foot
  - Superficial sensory deficit: lateral border of the foot

- **Lower sacral root level**
  - Muscular weakness: none
  - Deep tendon reflexes affected: none (but anal reflex impaired)
  - Radicular pain/paraesthesia: buttock and back of thigh
  - Superficial sensory deficit: saddle and perianal areas

Useful background: Causes of dissociated sensory loss of only the spinothalamic tract or the dorsal column

<table>
<thead>
<tr>
<th>Only spinothalamic tract loss</th>
<th>Only dorsal column loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Brown-Séquard syndrome (contralateral leg)</td>
<td>o Brown-Séquard syndrome (ipsilateral leg)</td>
</tr>
<tr>
<td>➢ Syringomyelia</td>
<td>o Subacute combined degeneration</td>
</tr>
<tr>
<td>➢ Anterior spinal artery thrombosis</td>
<td>o Spino cerebellar degeneration (Friedreich’s ataxia)</td>
</tr>
<tr>
<td>➢ Diabetes</td>
<td>o Multiple sclerosis</td>
</tr>
<tr>
<td>➢ Amyloid</td>
<td>o Tabes dorsalis</td>
</tr>
<tr>
<td>➢ Lateral medullary syndrome (contra-lateral signs)</td>
<td>o Diabetes</td>
</tr>
<tr>
<td></td>
<td>o Hypothyroidism</td>
</tr>
<tr>
<td></td>
<td>o Dorsal root ganglion opathy (cancer, Sjögren’s syndrome, diabetes mellitus)</td>
</tr>
</tbody>
</table>


28. Perform a focused physical examination to distinguish between total spinal cord transection or incomplete cord compression.

<table>
<thead>
<tr>
<th>Physical finding</th>
<th>Total cord transection</th>
<th>Incomplete cord compression</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Paraplegia in flexion</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>➢ Paralysis</td>
<td>Symmetric</td>
<td>Asymmetrical</td>
</tr>
<tr>
<td>➢ Flexor – withdrawal reflex</td>
<td>+ without return (withdrawal phase only)</td>
<td>+ with return to original position</td>
</tr>
<tr>
<td>➢ Other</td>
<td>Vasomotor and sphincter changes</td>
<td>Variable area of anaesthesia which is not consistent with motor loss</td>
</tr>
</tbody>
</table>

Useful background: Cervical spine movements and their respective myotomes

<table>
<thead>
<tr>
<th>Movement</th>
<th>Myotome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neck flexion</td>
<td></td>
</tr>
<tr>
<td>- Forward</td>
<td>C1-C2</td>
</tr>
<tr>
<td>- Sideways</td>
<td>C3</td>
</tr>
<tr>
<td>Shoulder</td>
<td></td>
</tr>
<tr>
<td>- Elevation</td>
<td>C4</td>
</tr>
<tr>
<td>- Abduction</td>
<td>C5</td>
</tr>
<tr>
<td>Elbow</td>
<td></td>
</tr>
<tr>
<td>- Flexion and/or wrist extension</td>
<td>C5</td>
</tr>
<tr>
<td>- Extension and/or wrist flexion</td>
<td>C7</td>
</tr>
<tr>
<td>Thumb</td>
<td></td>
</tr>
<tr>
<td>- Extension and/or ulnar deviation</td>
<td>C8</td>
</tr>
<tr>
<td>- Abduction and/or adduction of hand intrinsics</td>
<td>T1</td>
</tr>
</tbody>
</table>

Adapted from: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto* 2005, page 138.

SO YOU WANT TO BE A NEUROLOGIST!

Q: In the context of spinal cord, what is paraplegia-in-flexion?
A:  
- Paraplegia-flexion is seen in partial transaction of the cord where the limbs are involuntarily flexed at the hips and knees because the extensors are more paralysed than the flexors.
- In complete transaction of the spinal cord, the extrapyramidal tracts are also affected and hence no voluntary movement of the limb is possible, resulting in paraplegia-in-extension.

Useful background: Spinal cord syndromes

29. Perform a focused physical examination to detect the following sensory syndromes.

- Complete spinal cord injury and anterior cord syndrome
- Brown-sequard syndrome
- Central cord syndrome (syringomyelia)
- Brainstem injury
- Thalamic or cerebral hemisphere injury

Adapted from: McGee S. R. Saunders/Elsevier 2007, Figure 58-2, pages 746 and 747.
30. Perform a focused physical examination for the causes and site of spinal cord compression.

- **Intra-medullary**
  - Neoplasm
  - Cyst
  - Hematomyelia

- **Intra-dural extra-medullary**
  - Arachnoiditis
  - Cyst

- **Meninges, dura**
  - Neoplasm (Meningioma, neurofibroma, metastatic Ca)
  - Hodgkin’s leukemic infiltrate
  - Abscess
  - Cyst
  - Epidural abscess
  - Arachnoiditis
  - Leptomeningitis
  - Meningioma
  - Neurofibroma
  - Lymphoma
  - Leukemia

- **Vertebral column**
  - Congenital bone anomaly
  - Trauma
  - Vertebral collapse
  - Disc prolapse, spondylolisthesis, spondylosis
  - Neoplasm (primary or secondary) – glioma, ependymoma, 2°
  - Infection – TB or pyogenic
  - Crush #
  - Disc protrusion
  - Tumour
  - Myeloma
  - TB
  - Cervical spondylosis
  - Paget’s disease

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 83.
31. Perform a focused physical examination for causes of spastic paraparesis.

- Inherited
  - Friedreich’s ataxia.
  - Familial spastic paraplegia

- Infection
  - HIV
  - Tabes dorsalis
  - Transverse myelitis (involves all tracts of spinal cord; spastic or flaccid paralysis; may be incomplete cord compression or total cord transaction)

- Infiltration
  - Spinal cord tumor (meningioma, neuroma)
  - Metastatic carcinoma
  - Parasagittal falx meningioma

- Degeneration
  - Motor neuron disease
  - Syringomyelia
  - Osteoarthritis of the cervical spine

- Nutritional
  - Vitamin deficiency
  - Subacute combined degeneration of the cord (associated peripheral neuropathy)

- Vascular
  - Anterior spinal artery thrombosis
  - Atherosclerosis of spinal cord vasculature

- Trauma

32. Take a directed history and perform a focused physical examination for tabes dorsalis.

- **History**
  - CNS
    - Ataxia
  - Eyes
    - Diplopia or ↓ vision
  - Voice – crisis: laryngeal
    - Visceral crises
      - Gastric
      - Rectal
  - GI
    - Loss of sphincter control
  - GU
    - Impotence
  - MSK
    - Paresthesia, especially in feet
      - Lightning pains
- **Physical examination**
  - Face
    - Tabetic facies
  - Eyes
    - Ptosis
    - Argyll Robertson pupils
    - Optic atrophy, etc.
  - Muscles
    - Hypotonia
  - Densation
    - ↓ proprioception and vibration sense, with Romberginsm and ataxic gait
    - ↓ superficial and deep pain sensation
  - Reflexes
    - ↓ tendon reflexes (ankles affected first)
  - MSK
    - Charcot joints
  - Skin
    - Neuropathic ulcers

Adapted from: Burton J.L. *Churchill Livingstone* 197, page 89.
Useful background: Sensory dermatomes

Adapted from: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto, 2005, Figure. 1, page 155.
Useful background: Nerve compression at the lumbosacral spine

33. Perform a focused physical examination to determine the nerve roots involved in spinal cord disease (sensory dermatomes).

- Dermatomes in the upper limb
- Causes of wasting of small muscles of hand
  - Cord lesions at C8, T1 level
  - Motor neurone disease
  - Tumor
  - Syringomyelia
  - Meningo-vascular
  - Syphilis
  - Cord compression
  - Root lesions
  - Cervical Spondylosis
  - Neurofibroma, etc
  - Brachial plexus lesions
  - Klumpke paralysis
  - Cervical rib, etc
  - Ulnar or median nerve lesions
  - Arthritis of hand or wrist, or disuse atrophy
Dermatomes in the lower limb

Type of carcinomatous neuropathy
- Dementia
- Encephalomyelitis
- Cerebellum xx
- Cord-bone, xx, cord itself
- Xx
- Neuropathy
- Myopathy, xx, xx
- Myasthenia syndrome


SO YOU WANT TO BE A NEUROLOGIST!

Q: What are the commonest causes of posterior root ganglial conditions?
A: Diabetes
   Tabes dorsalis
   Carcinomatous neuropathy
Useful background: Spinal cord disease

- Cognitive impairment
  - Subacute combined degeneration of the cord
    - Typical sequence
      - Peripheral neuropathy = peripheral paresthesia
      - Dorsal column loss = sensory ataxia
      - Cortico spinal tract damage = paraplegia
    - No ankle reflexes
  - Hematological abnormality
    - 75%
    - 25%
    - No hematological abnormalities

- Cervical myelopathy
  - Spastic tetraparesis progressive over several years
  - Sensory symptoms less common
  - Often asymmetrical
- Transverse myelitis
  - Acute onset
    - May relate to recent infection-‘para-infectious’
    - Commonly due to multiple sclerosis
    - Occasionally ‘band of pain’ at affected level, flaccid paralysis

- Anterior spinal artery thrombosis
  - Sensory level
  - Flaccid paralysis
  - Urine retention
  - Acute onset
    - Flaccid paraplegia
    - Normal dorsal column sensation
    - Spinal shock
    - Spasticity develops later
  - Causes
    - Emboli (e.g. atrial fibrillation)
    - ‘In-situ’ thrombosis (e.g. sickle cell disease, hypercoagulable states)
    - Decompression sickness ‘the bends’

Useful background: Motor and reflex changes of spinal cord compression

- **Upper cervical**
  - Upper motor neurone signs in the upper and lower limbs
  - **C5:**
    - LMN- weakness and wasting of rhomboids, deltoids, biceps and brachioradialis
    - UMN- signs affect the rest of the upper and all the lower limbs.
    - Reflexes- Biceps reflex is lost, brachioradialis is inverted.
  - **C8:**
    - LMN weakness and wasting of the intrinsic muscles of the hand.
    - Reflexes- UMN signs in the lower limbs.

- **Midthoracic**
  - Intercostal paralysis
  - UMN signs in the lower limbs
  - Reflexes-loss of upper abdominal reflexes at T7 and T8
  - **T10-T11:**
    - Loss of the lower abdominal reflexes and upward displacement of the umbilicus
    - UMN- signs in the lower limbs
  - **L1:**
    - Reflexes- Cremasteric is lost, normal abdominal reflexes
    - UMN signs in the lower limbs
  - **L4:**
    - LMN weakness and wasting of the quadriceps
    - Reflexes- Knee reflexes lost
    - Ankle reflexes may be hyperreflexic with extensor plantar response (up-going toes), but more often there is a lower motor neurone lesion.
  - **L5-S1**
    - LMN weakness of knee flexion and hip extension (S1), and abduction (L5) plus calf and foot muscles.
    - Knee present
    - No ankle reflexes or plantar responses
  - **S3-S4:**
    - Saddle sensory loss
    - Normal lower limbs
    - No anal reflex

*Lower motor neurone (LMN) signs may extend for several segments, and spastic paralysis occurs late, unlike the situation with extramedullary lesions.*
Causes of spinal cord compression
- Vertebral
  - Spondylosis
  - Trauma
  - Prolapse of a disc
  - Tumour
  - Infection
- Outside the dura
  - Lymphoma, metastases
  - Infection - e.g., abscess
- Within the dura but extramedullary
  - Tumour - e.g. meningioma, neurofibroma
- Intramedullary*
  - Tumour - e.g. glioma, ependymoma
  - Syringomyelia
  - Haematomyelia

Abbreviations: LMN, lower motor neuron; UMN, upper motor neuron


34. Perform a focused physical examination for closed spina bifida.

Definition
- Incomplete closure of the bony vertebral canal
- Is commonly associated with a similar anomaly of spinal cord
- The commonest site is the lumbosacral region but the cervical spine can be involved.
- May be associated with hydrocephalus

Skin
- Lumbosacral lipoma
- Hypertrichosis
- Sinus or dimple above the sacrum
- Nevus, or scarring

MSK
- Unilateral shortening of one leg and foot
- Loss of muscles below the knee
- Calcaneovalgus or equinovarus deformity
- Sensory loss in L5/ S1

GU
- Neuropathic bladder
- Enuresis

Radiculopathy

Useful background: Likelihood ratio for a focused physical examination for cervical radiculopathy

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Motor examination</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Weak elbow flexion (C5)</td>
<td>5.3</td>
<td>NS</td>
</tr>
<tr>
<td>o Weak wrist extension (C6)</td>
<td>2.3</td>
<td>NS</td>
</tr>
<tr>
<td>o Weak elbow extension (C7)</td>
<td>4.0</td>
<td>0.4</td>
</tr>
<tr>
<td>o Weak finger flexion (C8)</td>
<td>3.8</td>
<td>NS</td>
</tr>
<tr>
<td>➢ Sensory examination</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Sensory loss thumb (C6)</td>
<td>8.5</td>
<td>NS</td>
</tr>
<tr>
<td>o Sensory loss affecting middle finger (C7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Sensory loss affecting little finger (C8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>➢ Reflex examination</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o ↓ biceps or deep tendon reflex (C6)</td>
<td>14.2</td>
<td>0.5</td>
</tr>
<tr>
<td>o ↓ triceps reflex (C7)</td>
<td>3.0</td>
<td>NS</td>
</tr>
</tbody>
</table>
| NS, not significant; PLR, positive likelihood ratio; NLR, negative likelihood ratio

➢ Other tests
   o Straight leg raising manoeuvre            | 1.3 | 0.3 |
   o Crossed straight leg raising manoeuvre   | 3.4 | 0.8 |

Abbreviation: MCL, midclavicular line; NLR, negative likelihood ratio; NS, not significant; PLR, positive likelihood ratio

Adapted from: McGee S. R. Saunders/Elsevier 2007, Box 60-2, pages 779 and 780, and Box 60-4, page 786.

<table>
<thead>
<tr>
<th>NLR</th>
<th>Probability Decrease</th>
<th>PLR</th>
<th>Probability Increase</th>
</tr>
</thead>
<tbody>
<tr>
<td>-45%</td>
<td>0.1</td>
<td>+15%</td>
<td>0.2</td>
</tr>
<tr>
<td>-30%</td>
<td>0.2</td>
<td>+30%</td>
<td>0.5</td>
</tr>
<tr>
<td>-15%</td>
<td>0.5</td>
<td>+45%</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>1</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>0.1</td>
<td>10</td>
<td>10</td>
<td></td>
</tr>
</tbody>
</table>

LRs
Useful background: Perform characteristics of tests for lumbosacral radiculopathy

<table>
<thead>
<tr>
<th>Finding</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Motor examination</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Weak ankle extension [L3 or L4]</td>
<td>38-42</td>
<td>89</td>
<td>3.7</td>
<td>0.7</td>
</tr>
<tr>
<td>o Weak hallux extension, [L5]</td>
<td>12-61</td>
<td>54-91</td>
<td>1.6</td>
<td>NS</td>
</tr>
<tr>
<td>o Weak ankle dorsiflexion, [L5]</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Weak ankle plantarflexion, S1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Ipsilateral calf wasting, [S1]</td>
<td>43</td>
<td>82</td>
<td>2.4</td>
<td>0.7</td>
</tr>
<tr>
<td>o Weak ankle dorsiflexion</td>
<td>54</td>
<td>89</td>
<td>4.9</td>
<td>0.5</td>
</tr>
<tr>
<td>o Ipsilateral calf wasting</td>
<td>29</td>
<td>94</td>
<td>5.2</td>
<td>0.8</td>
</tr>
<tr>
<td><strong>Sensory examination</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Sensory loss L5</td>
<td>20-53</td>
<td>77-98</td>
<td>3.1</td>
<td>0.8</td>
</tr>
<tr>
<td>o Sensory loss S1 (leg sensation abnormal)</td>
<td>32-49</td>
<td>70-90</td>
<td>2.4</td>
<td>0.7</td>
</tr>
<tr>
<td><strong>Reflex examination</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Asymmetric quadriceps reflex [L3 or L4]</td>
<td>30-57</td>
<td>93-96</td>
<td>8.7</td>
<td>0.6</td>
</tr>
<tr>
<td>o Asymmetric Achilles reflex, [S1]</td>
<td>45-91</td>
<td>53-94</td>
<td>2.9</td>
<td>0.4</td>
</tr>
<tr>
<td>o Abnormal ankle jerk</td>
<td>14-48</td>
<td>89-93</td>
<td>2.7</td>
<td>NS</td>
</tr>
</tbody>
</table>

NS, not significant; likelihood ratio (LR) if finding present= positive LR, LR if finding absent= negative LR. Findings shown in round ( ) brackets were not associated with a significant PLR/- and the values of the sensitivity and specificity are not provided. The nerve involved in the radiculopathy is given in square [ ] brackets.

Useful background: Diagnosing lumbosacral radiculopathy in patients with sciatica*

<table>
<thead>
<tr>
<th>Finding</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor examination</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Weak ankle dorsiflexion</td>
<td>54</td>
<td>89</td>
<td>4.9</td>
<td>0.5</td>
</tr>
<tr>
<td>o Ipsilateral calf wasting</td>
<td>29</td>
<td>94</td>
<td>5.2</td>
<td>0.8</td>
</tr>
<tr>
<td>Sensory examination</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Leg sensation abnormal</td>
<td>16-50</td>
<td>62-86</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Reflex examination</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Abnormal ankle jerk</td>
<td>14-48</td>
<td>89-93</td>
<td>2.7</td>
<td>NS</td>
</tr>
<tr>
<td>Other tests</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Straight-leg raising manœuvre</td>
<td>64-98</td>
<td>11-61</td>
<td>1.3</td>
<td>0.3</td>
</tr>
<tr>
<td>o Crossed straight-leg raising manœuvre</td>
<td>22-43</td>
<td>88-98</td>
<td>3.4</td>
<td>0.8</td>
</tr>
</tbody>
</table>


What is “the best”? The “best” clinical tests for lumbosacral radiculopathy include:

- Weak ankle extension and dorsiflexion
- Ipsilateral calf wasting,
- Sensory loss in area of LSS
- Asymmetric quadriceps reflex
- Positive crossed straight leg-raising manœuvre
35. Perform a focused physical examination to determine if a person has a UMN or an LMN lesion.

<table>
<thead>
<tr>
<th>Loss</th>
<th>UMN lesion</th>
<th>LMN lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Muscle wasting</td>
<td>-</td>
<td>++</td>
</tr>
<tr>
<td>➢ Fasciculations</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>➢ Power</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Upper limbs</td>
<td>F &gt; E</td>
<td>↓</td>
</tr>
<tr>
<td>Lower limbs</td>
<td>E &gt; F</td>
<td>↓</td>
</tr>
<tr>
<td></td>
<td>Arms flexed, legs extended</td>
<td>Fasciculations</td>
</tr>
<tr>
<td>➢ Tone</td>
<td>↑/ spastic</td>
<td>↓</td>
</tr>
<tr>
<td>➢ Coordination impaired due to weakness</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>➢ Reflexes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Superficial (e.g. abdominal)</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>o Deep</td>
<td>Increased/ clonus</td>
<td>Decreased</td>
</tr>
<tr>
<td>o Barbinski</td>
<td>up-going (present)</td>
<td>Downgoing (absent)</td>
</tr>
</tbody>
</table>

Abbreviations: E, extensor muscles; F, flexor muscles; LMN, lower motor neuron; UMN, upper motor neuron

 Adapted from: Filate W., Et al. The Medical Society, Faculty of Medicine, University of Toronto, 2005, Table 9, page 163.

➢ In the upper limb, weakness is most marked in the abductors and extensors.
➢ In the lower limb, weakness is more marked in the flexor and abductor muscles.
➢ Spasticity
➢ Increased tone is present (may be clasp-knife) and often associated with clonus
➢ The reflexes are increased except for the superficial reflexes (e.g. abdominal), which are absent.
➢ There is an extensor (Babinski) plantar response (up-going toe)
➢ Weakness may be more obvious distally than proximally, and the flexor and extensor muscles are equally involved.

Useful background: Compare UMN versus LMN

<table>
<thead>
<tr>
<th>Site</th>
<th>Power</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper motor neuron</td>
<td>upper limbs - Flexors &gt; extensors</td>
</tr>
<tr>
<td></td>
<td>lower limbs - Extensors &gt; Flexors</td>
</tr>
<tr>
<td>Lower motor neuron</td>
<td>Reduced power in specific motor neuron (or nerve root) distributions</td>
</tr>
</tbody>
</table>

Reinforcement can involve teeth clenching, hand grips, etc. Remember to make side-to-side comparisons of tone, pattern of weakness and reflexes.

36. Perform a focused physical examination for loss of corticospinal inhibition.

- Hoffman finger flexion reflexes: sudden stretching of finger flexors causes the other finger flexors to contract involuntarily (finding of hyperrreflexia).
- Jaw Jerk: sudden stretching of the masseter muscle causes the jaw to move upwards briskly.
- Clonus: sudden stretching of a muscle with continued stretch force applied causes continued oscillation of the muscle brachioradialis reflex (aka “inverted supinator reflex”): tapping this muscle causes flexion of the fingers (UMN effect), not the elbow (loss of LMN effect), indicating C5-6 spinal cord disease.
- Inverted knee reflex: tapping knee causes knee flexion, not extension, indicating L2-4 spinal cord disease.
- Crossed adductor reflex: tapping on the medial femoral condyle, patella, or patellar tendon causes the contralateral adductor muscle to contract, moving the contralateral knee medially.\(^{42}\)


Tendon reflexes: root level

- Ankle S1, 2
- Knee L3, 4
- Biceps C5, 6
- Supinator C5, 6
- Triceps C6, 7

37. Perform a focused physical examination for hemisection of the spinal cord (Brown-Sequard syndrome).

- **Deficits at the level of the cord lesion:**
  - Ipsilateral LMN paralysis
  - Ipsilateral zone of cutaneous anaesthesia and zone of hyperaesthesia just below the anaesthetic zone
  - Segmental signs are usually unilateral
    - Muscular atrophy
    - Radicular pain
    - ↓ tendon reflexes

- **Deficits below the level of the cord lesion**
  - Ipsilateral monoplegia or hemiplegia
  - Ipsilateral loss of joint position and vibration sense
  - Contralateral loss of spinothalamic (pain and temperature) sensation.

- **Causes**
  - Cord tumour
  - Syringomyelia
  - Trauma
  - Degenerative disease of spine
  - Multiple myeloma
  - Hematomyelia

- **Differentiate from MS**

38. Perform a focused physical examination for a lesion in the spinal canal at any level below T10 (cauda equina syndrome).

- **Physical Examination**
  - Flaccid, asymmetrical paraparesis (LMN)
  - Knee and ankle jerks are diminished or absent.
  - Saddle distribution of sensory loss up to the L1 level.
  - Downgoing plantars.

- **Subtypes**
  - The lateral cauda equina syndrome:
    - Pain in the anterior thigh,
    - Wasting of the quadriceps muscle,
    - Weakness of the foot invertors (due to L4 root lesion)
    - Absent knee jerk
    - Causes include neurofibroma, a high disc lesion.
  - The midline cauda equina syndrome:
    - Bilateral lumbar and sacral root lesions.
    - Causes include disc lesion, primary sacral bone tumours (chordomas), metastatic bone disease (from prostate) and leukemia.

- **Causes**
  - Centrally placed lumbosacral disc or spondylolisthesis at the lumbosacral junction
  - Tumours of the cauda equina (ependymoma, neurofibroma)

- **Differentiate**
  - UMN lesion of conus medullaris (lowest part of spinal cord)
  - Other causes of crossed hemiplegia (lesion is in the midbrain)
    - Weber’s syndrome: contralateral hemiplegia (lesion is in the midbrain) with ipsilateral LMN lesion of the oculomotor nerve.
    - Millare-Gubler syndrome: contralateral hemiplegia (lesion is in the pons) with LMN lesion of the abducens nerve.
    - Foville’s syndrome: as Millard-Gubler syndrome, with gaze palsy


39. Perform a directed physical examination to establish the neurological cause of a brachial plexus lesion, and the cervical rib syndrome.

- **Brachial plexus lesions**
  - Complete lesion (rare)
    - Lower motor neurone signs affect the whole arm
    - Sensory loss (whole limb)
• Horner’s syndrome (an important clue)
  NB: this is often painful

➢ Upper lesion (C5, C6)
  o Loss of shoulder movement and elbow flexion – the hand is held in the waiter’s tip position
  o Sensory loss over the lateral aspect of the arm and forearm

➢ Lower lesion (C8, T1)
  o True claw hand with paralysis of all the intrinsic muscles
  o Sensory loss along the ulnar side of the hand and forearm
  o Horner’s syndrome

• Cervical rib syndrome
  ➢ Clinical features
    o Weakness and wasting of the small muscles of the hand (claw hand)
    o C8 and T1 sensory loss
    o Unequal radial pulses and blood pressure
    o Subclavian bruits on arm manoeuvring (may be present in normal persons)
    o Palpable cervical rib in the neck (uncommon)

Adapted from: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto page 139; Talley NJ et al. Maclennan & Petty Pty Limited 2003,. Table 10.18(6), page 407.

Useful background: Characteristics of abnormal tone

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Possible causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ ↓ tone</td>
<td></td>
</tr>
<tr>
<td>o Flaccid</td>
<td>o LMN lesion, cerebellar; rarely myopathies, ‘spinal shock’ (e.g. early response after a spinal cord trauma), chorea</td>
</tr>
</tbody>
</table>

➢ ↑ tone
  o spastic (‘clasp knife’)  o UMN lesion; corticospinal tract (commonly late or chronic stage after a stroke)
  o rigidity (‘lead pipe’, cog wheeling’)  o Extrapyrimidal tract lesion
    - Parkinsonism
    - phenothiazines

Adapted from: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto, 2005, page 164.
Harding’s criteria for Friedreich’s ataxia

- Essential criteria are onset before the age of 25 years
  - Ataxia of limbs and gait
  - Absent knee and ankle jerks
  - Extensor plantars
  - Autosomal recessive inheritance
  - Motor conduction velocity greater than 40 ms
  - Small or absent sensory nerve action potentials
  - Dysarthria within 5 years of onset
- Additional criteria (present in two thirds)
  - Scoliosis
  - Pyramidal weakness of lower limbs
  - Absent upper limb reflexes
  - Loss of vibration and joint position sense in the legs
  - Abnormal ECG
  - Pes cavus.
- Other features (present in less than 50% of cases)
  - Nystagmus
  - Optic atrophy
  - Deafness
  - Distal muscle wasting and diabetes


40. Perform a focused physical examination for syringomyelia.

Definition

- Formation of a cavity in the inner portion of the cord
- Destruction of the white and grey matter and an accompanying reactive gliosis
- The process generally begins in the cervical cord, and with expansion of the cavity the brainstem and distal cord also become affected

Physical Examination

- LMN, small muscles of the hands and forearm
  - ↓ tone
  - Weakness
  - Wasting
  - Fasciculation
  - ↓ DTR
- ↓ pain and temperature sensation
- Intact vibration, light touch and joint position sense
- Charcot’s joints of the shoulder and elbow
- At the level of the syrinx
  - LMN lesion, causing anterior horn cell involvement
- Involvement of the central decussating fibres of the spinothalamic tract producing
- Dissociated sensory loss
- Development of neuropathic arthropathy
- Trophic changes

- **Below the level of the syrinx:**
  - Involvement of pyramidal corticospinal tracts resulting in spastic paraparesis (sphincter function is usually well preserved)

- **Involvement of cervical sympathetic**
  - Horner's syndrome (miosis, enophthalmos, ptosis)

- **Ia main succulente**
  - Ugly hand
  - Cold
  - Cyanosed
  - Swollen fingers and palms.

- Differentiate from
  - Anterior spinal artery occlusion (affecting the dorsal horn and lateral spinothalamic tract).
  - Diabetic small-fibre polyneuropathy.
  - Hereditary amyloidotic polyneuropathy.
  - Leprosy (the latter three conditions affect small peripheral nerve axons).

- **Conditions with a similar picture**
  - Intramedullary tumours of the spinal cord.
  - Arachnoiditis around the foramen magnum obstructing the CSF pathway.
  - Hematomyelia.
  - Craniovertebral anomalies.
  - Late sequelae of spinal cord injuries (manifest as a painful ascending myelopathy).
  - Rarely patients may have hypertrophy in limbs hand and feet.
  - If fasciculation is seen, then the other diagnosis to consider is motor neuron disease

**Abbreviation:** DTR, deep tendon reflex

Useful background: Segmental Innervation of Muscles (Most muscles are innervated by nerves from more than one spinal root.)

<table>
<thead>
<tr>
<th>Spinal Level</th>
<th>Muscles</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Arm</strong></td>
<td></td>
</tr>
<tr>
<td>C5</td>
<td>Elbow flexors (biceps, brachialis)</td>
</tr>
<tr>
<td>C6</td>
<td>Wrist extensors (extensor carpi radialis longus and brevis)</td>
</tr>
<tr>
<td>C7</td>
<td>Elbow extensors (triceps)</td>
</tr>
<tr>
<td>C8</td>
<td>Finger flexors (flexor digitorum profundus of middle finger)</td>
</tr>
<tr>
<td>T1</td>
<td>Small finger abductors (abductor digiti minimi)</td>
</tr>
<tr>
<td><strong>Leg</strong></td>
<td></td>
</tr>
<tr>
<td>L2</td>
<td>Hip flexors (iliopsoas)</td>
</tr>
<tr>
<td>L3</td>
<td>Knee extensors (quadriceps)</td>
</tr>
<tr>
<td>L4</td>
<td>Ankle dorsiflexors (tibialis anterior)</td>
</tr>
<tr>
<td>L5</td>
<td>Long toe extensors (extensor hallucis longus)</td>
</tr>
<tr>
<td>S1</td>
<td>Ankle plantarflexors (gastrocnemius, soleus)</td>
</tr>
</tbody>
</table>


41. Perform a focused physical examination for muscle or UMN nerve root disease in the muscle groups of the upper and lower body.

<table>
<thead>
<tr>
<th>Joint</th>
<th>Movement</th>
<th>Muscle</th>
<th>Nerve roots</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Shoulder</td>
<td>o Abduction</td>
<td>- Deltoid, supraspinatus</td>
<td>C5, C6</td>
</tr>
<tr>
<td></td>
<td>o Adduction</td>
<td>- Pectoralis major, latissmus dorsi</td>
<td>C6, C7, C8</td>
</tr>
<tr>
<td>➢ Elbow</td>
<td>o Flexion</td>
<td>- Biceps, brachialis</td>
<td>C5, C6</td>
</tr>
<tr>
<td></td>
<td>o Extension</td>
<td>- Triceps brachii</td>
<td>C7, C8</td>
</tr>
<tr>
<td>➢ Wrist</td>
<td>o Flexion</td>
<td>- Flexor carpi ulnar, radialis</td>
<td>C6, C7</td>
</tr>
<tr>
<td></td>
<td>o Extension</td>
<td>- Extensor carpi</td>
<td>C7, C8</td>
</tr>
<tr>
<td>➢ Fingers</td>
<td>o Flexion</td>
<td>- Flexor digitorum profundus and sublimis</td>
<td>C7, C8</td>
</tr>
<tr>
<td></td>
<td>o Extension</td>
<td>- Extensor digitorum communis, extensor indicis, extensor digitii minimi</td>
<td>C7, C8</td>
</tr>
<tr>
<td></td>
<td>o Abduction</td>
<td>- Dorsal interossei</td>
<td>C8, T1</td>
</tr>
<tr>
<td></td>
<td>o Adduction</td>
<td>- Volar interossei</td>
<td>C8, T1</td>
</tr>
<tr>
<td>➢ Hamstrings</td>
<td>o Knee flexion</td>
<td>- Sciatic</td>
<td>L5, S1, 2</td>
</tr>
<tr>
<td>Joint</td>
<td>Movement</td>
<td>Muscle</td>
<td>Nerve roots</td>
</tr>
<tr>
<td>-------</td>
<td>----------</td>
<td>--------</td>
<td>-------------</td>
</tr>
<tr>
<td>➢ Tibialis anterior</td>
<td>o Ankle dorsiflexion</td>
<td>- Deep peroneal</td>
<td>L4, 5</td>
</tr>
<tr>
<td>➢ Gastrocnemius soleus</td>
<td>o Ankle plantar flexion</td>
<td>- Tibial</td>
<td>S1, 2</td>
</tr>
<tr>
<td>➢ Extensor hallucis longus</td>
<td>o Great toe dorsiflexion</td>
<td>- Deep peroneal</td>
<td>L5, S1</td>
</tr>
<tr>
<td>➢ Tibialis</td>
<td>o Posterior foot inversion</td>
<td>- Posterior tibial</td>
<td>L4, L5</td>
</tr>
<tr>
<td>➢ Peroneus longus, brevis</td>
<td>o Foot eversion</td>
<td>- Superficial peroneal</td>
<td>L5, S1</td>
</tr>
<tr>
<td>➢ Hip</td>
<td>o Flexion</td>
<td>- Psoas; iliacas</td>
<td>L2, L3</td>
</tr>
<tr>
<td></td>
<td>o Extension</td>
<td>- Gluteus maximus</td>
<td>L5, S1, S2</td>
</tr>
<tr>
<td></td>
<td>o Abduction</td>
<td>- Gluteus medius and minimus</td>
<td>L4, L5, S1</td>
</tr>
<tr>
<td></td>
<td>o Adduction</td>
<td>Sartorius, tensor fasciae latae</td>
<td>L2, L3, L4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Adductors longus, brevis, magnus</td>
<td></td>
</tr>
<tr>
<td>➢ Knee</td>
<td>o Flexion</td>
<td>“Hamstrings” (biceps femoris, semimembranosus, semitendinosus</td>
<td>L5, S1</td>
</tr>
<tr>
<td></td>
<td>o Extension</td>
<td>Quadriceps femoris</td>
<td>L3, L4</td>
</tr>
<tr>
<td>➢ Ankle</td>
<td>o Plantar flexion</td>
<td>Gastrocnemius, plantaris, soleus</td>
<td>S1, S2</td>
</tr>
<tr>
<td></td>
<td>o Dorsiflexion</td>
<td>Tibialis anterior, extensor digitorum longus, extensor hallucis longus</td>
<td>L4, L5</td>
</tr>
<tr>
<td>➢ Tarsal joint</td>
<td>o Eversion</td>
<td>Peroneus longus and brevis, extensor digitorum longus</td>
<td>L5, S1</td>
</tr>
<tr>
<td></td>
<td>o Inversion</td>
<td>Tibialis posterior, gastrocnemius, hallucis longus</td>
<td>L5, S1</td>
</tr>
</tbody>
</table>

Abbreviations: UMN, upper motor neuron

Adapted from: McGee S. R. Saunders/Elsevier 2007, Table 57-6 page 723, Table 10.15, page 404, Table 10.16, page 406.
Useful background: Common muscle stretch reflexes

<table>
<thead>
<tr>
<th>Name of reflex</th>
<th>Peripheral nerve</th>
<th>Spinal level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brachioradialis</td>
<td>Radial</td>
<td>C5-6</td>
</tr>
<tr>
<td>Biceps</td>
<td>Musculocutaneous</td>
<td>C5-6</td>
</tr>
<tr>
<td>Triceps</td>
<td>Radial</td>
<td>C7-8</td>
</tr>
<tr>
<td>Quadriceps (patellar)</td>
<td>Femoral</td>
<td>L2-L4</td>
</tr>
<tr>
<td>Archilles (ankle)</td>
<td>Tibial</td>
<td>S1</td>
</tr>
</tbody>
</table>


SO YOU WANT TO BE A NEUROLOGIST!

**Q1:** Distinguish ulnar lesions from T1 root lesions (abductor pollicis brevis).

The thumb is moved vertically against resistance, with the hand supine.

**Distinguish between median and ulnar nerve defects affecting the hands:**

**A2:** The Median nerve supplies

- Motor (mnemonic LOAF)
  - Lateral two lumbricals
  - Opponens pollicis
  - Abductor pollicis brevis
  - Flexor pollicis brevis

Sensory to the radial 3½ digits

Ulnar nerve supplies all the rest


SO YOU WANT TO BE A NEUROLOGIST!

**Q:** In the context of cervical radiculopathy. What is the “Spurling’s test” or “neck compression test.”?

**A:** In this test, the clinician turns and tilts the patient’s head and neck toward the painful side and then adds a compressive force to the top of the head. Aggravation of pain is a positive response!

Useful background: Nerve innervations of the muscles of the hand and forearm radial nerve (C5-C8): triceps, brachioradialis, extensor muscles of hand

- **Median nerve (C6-T1)**
  - Muscles on front of forearm, except flexor
  - Carpi ulnaris and ulnar half of flexor digitorium profundus
  - Short muscle of hands (“LOAF” muscles: the two lateral lumbricals, opponens pollicis, abductor pollicis brevis, flexor pollicis brevis [in some persons])

- **Ulnar nerve (C8-T1)**
  - Small muscles of the hand except for “LOAF” muscles, flexor carpi ulnaris, ulnar half of flexor digitorum profundus

Adapted from: Mangione S. *Hanley & Belfus* 2000, page 462.

SO YOU WANT, REALLY WANT TO BE A NEUROLOGIST!

**Q1:** What is the difference between hydromyelia (aka syringobulbia) and syringomyelia?

**A1:**
- Hydromyelia is the expansion of the ependyma-lined central canal of the spinal cord.
- Syringomyelia is the formation of a cleft-like cavity in the inner portion of the cord. Both these lesions are associated with

**Q2:** What are the clinical features of syringobulbia?

**A2:**
- Dissociated sensory loss of the face of the ‘onion-skin’ pattern (extending from behind forwards, converging on the nose and upper lip).
- Vertigo (common symptom).
- Wasting of the small muscles of the tongue (important physical sign).
- The process may be limited to the medullary region.
- The main cranial nerve nuclei involved are those of the fifth, seventh, ninth and tenth cranial nerves.

**Q3:** Do you wish to be really mean? What are the methods of eliciting deep pain?

**A3:**
- Abadie’s sign – the loss of pain sense in the Achilles tendon.
- Biernacki’s sign – the absence of pain on pressure on the ulnar nerve.
- Royal College Sign – too many questions on neurology.
- Pitres’ sign – loss of pain on pressure on the testes.
- Haenel’s sign – analgesia to pressure on the eyeballs.

---

*Achieving Excellence in the OSCE Part 2 © A.B.R Thomson*
Useful background: Sensory branches of peripheral nerves of the leg

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Sensory branches</th>
</tr>
</thead>
<tbody>
<tr>
<td>Femoral nerve</td>
<td>Anterior thigh</td>
</tr>
<tr>
<td></td>
<td>Medial calf</td>
</tr>
<tr>
<td>Obturator nerve</td>
<td>Medial thigh</td>
</tr>
<tr>
<td>Sciatic nerve trunk*</td>
<td>Posterior thigh</td>
</tr>
<tr>
<td>Peroneal nerve*</td>
<td>Lateral calf and dorsal foot</td>
</tr>
<tr>
<td>Tibial nerve*</td>
<td>Sole of foot</td>
</tr>
</tbody>
</table>

*The sciatic nerve trunk divides above the knee into the peroneal and tibial nerves. Therefore, lesions of the sciatic nerve trunk affect sensation from all three branches.


Useful background: Compression of lumbar discs and physical findings

<table>
<thead>
<tr>
<th>Disc</th>
<th>Root</th>
<th>Motor weakness</th>
<th>Sensory loss</th>
<th>Reflex affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>L4/5</td>
<td>L5</td>
<td>Doriflexors, EDL, EHL</td>
<td>Lateral calf and dorsum of foot</td>
<td>Medial hamstring</td>
</tr>
<tr>
<td>L5/6</td>
<td>S1</td>
<td>Plantar flexors</td>
<td>Lateral foot and sole</td>
<td>Ankle jerk</td>
</tr>
</tbody>
</table>

Legend: EDL=extensor digitorum longus, EHL= extensor hallucis longus

Source: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto 2005, page 175

SO YOU WANT TO BE A NEUROLOGIST OR A RHEUMATOLOGIST OR AN ORTHOPEDIC SURGEON!

Q: What are the causes of a claw hand (all fingers clawed)?
A:  
o Ulnar and median nerve lesion (ulnar nerve palsy alone causes a claw-like hand)
o Brachial plexus lesion (C8-T1)
o Other neurological disease – e.g. syringomyelia, polio
o Ischaemic contracture (late and severe)
o Rheumatoid arthritis (advanced, untreated disease)

42. Perform a focused physical examination of the sensory dermatomes of the peripheral nervous system, and give their signature zones.

<table>
<thead>
<tr>
<th>Spinal level</th>
<th>Signature zone</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cervical</strong></td>
<td></td>
</tr>
<tr>
<td>o C3</td>
<td>Supraclavicular fossa</td>
</tr>
<tr>
<td>o C4</td>
<td>Top of acromioclavicular joint</td>
</tr>
<tr>
<td>o C5</td>
<td>Lateral side of antecubital fossa</td>
</tr>
<tr>
<td>o C6</td>
<td>Thumb</td>
</tr>
<tr>
<td>o C7</td>
<td>Middle finger</td>
</tr>
<tr>
<td>o C8</td>
<td>Little finger</td>
</tr>
<tr>
<td><strong>Thoracic (selected levels)</strong></td>
<td></td>
</tr>
<tr>
<td>o T1</td>
<td>Medial (ulnar) side of the antecubital fossa</td>
</tr>
<tr>
<td>o T2</td>
<td>Apex of axilla</td>
</tr>
<tr>
<td>o T4</td>
<td>Fourth intercostals space (nipple line)</td>
</tr>
<tr>
<td>o T10</td>
<td>Tenth intercostals space (umbilicus)</td>
</tr>
<tr>
<td>o T12</td>
<td>Inguinal ligament at midpoint</td>
</tr>
<tr>
<td><strong>Lumbar</strong></td>
<td></td>
</tr>
<tr>
<td>o L1</td>
<td>Half the distance between the T12 and L2</td>
</tr>
<tr>
<td>o L2</td>
<td>Mid-anterior thigh</td>
</tr>
<tr>
<td>o L3</td>
<td>Medial femoral condyle, anterior knee</td>
</tr>
<tr>
<td>o L4</td>
<td>Medial aspect of leg medial malleolus</td>
</tr>
<tr>
<td>o L5</td>
<td>Lateral aspect of leg, medial aspect of feet</td>
</tr>
<tr>
<td><strong>Sacral</strong></td>
<td></td>
</tr>
<tr>
<td>o S1</td>
<td>Heel and most of the sole</td>
</tr>
<tr>
<td>o S2</td>
<td>Popliteal fossa in the mid-line (posterior thigh)</td>
</tr>
<tr>
<td>o S3</td>
<td>Ischial tuberosity</td>
</tr>
<tr>
<td>o S3-5</td>
<td>Perianal level, concentric rings</td>
</tr>
<tr>
<td>o T12</td>
<td>Inguinal ligament at midpoint</td>
</tr>
</tbody>
</table>

Peripheral nerves

Useful background: Testing thumb abduction

- Instructed patient to raise their his thumb perpendicular to the palm
- The examiner applies downward pressure on the distal phalanx.
- A defect in the thumb abduction test indicated weakness of the abductor pollicis brevis, which is innervated only by the median nerve.

Adapted from: Simel David L, et al. JAMA 2009, Figure 10-2, page 113.

43. Perform a focused physical examination to establish the neurological cause of wasting of the small muscles of the hand.

- Spinal cord lesions (C8, T1)*
  - Syringomyelia
  - Cervical spondylosis
  - Tumour
  - Trauma
  - Motor neuron disease
  - Syphilis

- Anterior horn cell disease
  - Motor neurone disease
  - Poliomyelitis
  - Spinal muscle atrophies

- Root lesion
  - Spondylosis
  - Neuro fibroma
  - Tumour

- Lower trunk brachial plexus lesion
  - Thoracic outlet syndromes
• Trauma, radiation, infiltration, inflammation
  • Pancoast syndrome
  • Cervical rib

➢ Peripheral nerve lesions
  • Median and ulnar nerve lesions
  • Peripheral motor neuropathy

➢ Myopathy
  • Dystrophia myotonica (forearms are more affected than the hands)
  • Distal myopathy

➢ Trophic disorders
  • Atrophy
  • Ischemia
  • Shoulder hand syndrome
  • Arthritis of hand or wrist

* Rarely diabetes, lead poisoning or a carcinoma-associated neuropathy may display a similar cord lesion


44. Perform a focused physical examination of the cutaneous sensory innervation of the hand.

Useful background: Causes of carpal tunnel syndrome

- **Joint/ bone**
  - Arthritis of wrist (esp. rheumatoid arthritis)
  - Previous scaphoid fracture
  - Intermittent trauma

- **Metabolic**
  - Myxedema
  - Acromegaly
  - Mucopolysaccharidosis V (Scheie’s syndrome)
  - Hyperparathyroidism

- **Sarcoidosis**

- **Amyloid** (such as in chronic renal failure or multiple myeloma)

- **Idiopathic**

- **Pregnancy, ‘Pill’, pre-menstrual**


45. Perform a focused physical examination for ulnar nerve palsy (C8, T1).

- **Physical examination**

  - **inspection**
    - Generalized wasting of the small muscles of the hand.
    - Ulnar claw hand, (hyperextension at the metacarpophalangeal joints and flexion at the interphalangeal joints of the fourth and fifth fingers).
    - Ulnar paradox - the higher the lesion in the upper limb, the lesser is the deformity
    - A lesion at or above the elbow causes paralysis of the ulnar half of the flexor digitorum profundus, interossei and lumbricals.

- **Motor**
  - Weakness of movement of the fingers, except that of the thenar eminence
  - *In the forearm* (lesions in the cubital fossa)
    - Flexor carpi ulnaris.
    - Medial half of the flexor digitorum profundus.
  - Ulnar claw hand.
Sensory
  o Sensory loss over the medial one and half fingers.
  o Movers of the little finger – abductor digiti minimi, flexor digiti minimi and opponens digiti minimi.
  o Adductor pollicis (oblique and transverse heads).
  o Dorsal and palmar interossei.
  o Third and fourth lumbricals.
  o Palmaris bevis.
  o Inner head of flexor pollicis brevis.


46. Perform a focused physical examination for the cause of a carcinomatous neuropathy.

CNS
  o Dementia
  o Encephalomyelitis

Cerebellum and corticospinal

Cord – bone, meninges, cord itself

Post root ganglion

Nerve
  o Neuropathy, mononeuritis multiplex

Muscle
  o Myopathy,
  o Myasthenic syndrome

47. Perform a focused physical examination for the causes of benign intracranial hypertension (pseudotumour cerebri).

- **Drugs**
  - Change in steroid dosage
  - Chlortetracycline, nalidixic acid, oral contraceptives

- **Head**
  - Head injury
  - Sagittal sinus thrombosis

- **Female**
  - Pregnancy, obesity, menarche

- **Hematology**
  - Anemia
  - Polycythemia

- **Metabolic**
  - Addison’s
  - Hypoparathyroidism

Useful background: Sensory branches of peripheral nerves of the arm

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Sensory branches</th>
</tr>
</thead>
<tbody>
<tr>
<td>Musculocutaneous nerve</td>
<td>Radial aspect forearm</td>
</tr>
<tr>
<td>Radial nerve</td>
<td>Dorsal arm and forearm</td>
</tr>
<tr>
<td></td>
<td>Radial aspect dorsal hand</td>
</tr>
<tr>
<td>Median nerve</td>
<td>Radial palm</td>
</tr>
<tr>
<td></td>
<td>First three digits and radial aspect</td>
</tr>
<tr>
<td></td>
<td>ring finger</td>
</tr>
<tr>
<td>Ulnar nerve</td>
<td>Ulnar aspect of hand and digits</td>
</tr>
</tbody>
</table>

Peripheral neuropathy

Useful background: Peripheral neuropathy

48. Perform a directed physical examination for the causes of peripheral neuropathy.

- **Definition:**
  - Bilateral symmetrical sensory loss for all modalities with or without motor weakness.
  - Motor or sensory component, one or more peripheral or cranial nerves, as well as mononeuritis multiplex.

- **Causes of peripheral neuropathy**
  - **Drugs** - e.g. isoniazid, vincristine, phenytoin, nitrofurantoin, cisplatinum, heavy metals (e.g. arsenic), amiodarone, thallium poisoning
  - Alcohol abuse (with or without vitamin B1 deficiency)
  - Metabolic - e.g. diabetes mellitus, chronic renal failure, porphyria, acromegaly
  - Infection – HIV, sarcoidosis, diphtheria, leprosy, Lyme disease
  - Guillain-Barre syndrome
  - Malignancy - e.g. carcinoma of the lung (paraneoplastic neuropathy), leukemia, lymphoma
  - Vitamin deficiency (e.g. B12) or excess (e.g. B6)
  - Connective tissue disease - e.g. PAN, SLE, rheumatoid arthritis, amyloid
  - Hereditary
  - Multifocal conduction block neuropathy (MCBN)
  - Compressive neuropathies
  - Idiopathic

- **Causes of a predominant motor neuropathy**
  - Guillain-Barre syndrome
  - Chronic inflammatory polyradiculoneuropathy
  - Perineal muscular atrophy
  - Give **DAD** some **Rum** (mnemonic)
    - Diabetes mellitus (diabetic chart, insulin injection sites, insulin pump)
    - Alcoholic liver disease (palmar erythema, spider naevi, tender liver)
    - Drug history
    - Rheumatoid arthritis
    - Uraemia
    - Malignancy
  - Hereditary motor and sensory neuropathy
  - Diabetes mellitus
  - Acute intermittent porphyria
  - Diphtheria
  - MCBN
  - Drugs/ toxins e.g.
- Lead
- Dapsone
- Organophosphorous poisoning

➤ Causes of a painful sensory peripheral neuropathy
  o Diabetes mellitus
  o Alcohol
  o Vitamin B1, or B12 deficiency
  o Carcinoma
  o Porphyria
  o Arsenic or thallium poisoning

➤ Mononeuritis multiplex *
  o Acute causes (usually vascular)
    - Polyarteritis nodosa
    - Diabetes mellitus
    - Connective tissue disease – e.g. rheumatoid arthritis, SLE
  o Chronic causes
    - Multiple compressive neuropathies
    - Sarcoidosis
    - Acromegaly
    - HIV infection
    - Leprosy
    - Lyme disease
    - Others- e.g. carcinoma (rare)

➤ In diabetes mellitus
  o Symmetrical, mainly sensory, polyneuropathy
  o Asymmetrical, mainly motor, polyneuropathy (diabetic amyotrophy)
  o Mononeuropathy
  o Autonomic neuropathy

*separate involvement of more than one peripheral (or less often cranial) nerve by a single disease

Adapted from: Talley N. J., et al. Maclellan & Petty Pty Limited 2003, Table 10.25, page 420; and AT.
Useful background: Causes of lower motor neuron (LMN) signs in the legs

- Peripheral neuropathy
- Prolapsed intervertebral disc
- Diabetic amyotrophy
- Poliomyelitis
- Cauda equine lesions
- Motor neuron disease


- Causes of Mononeuritis multiplex
  - Mnemonic: Go to the WARDS, PLeaCe
    - Wegener’s granulomatosis
    - Amyloidosis
    - Rheumatoid arthritis
    - Diabetes mellitus
    - SLE
    - Polyarteritis nodosa
    - Leprosy
    - Carcinomatosis, Churg-Strauss syndrome


SO YOU WANT TO BE A NEUROLOGIST!

Q1: What conditions demonstrate an up-going plantar reflex but absent knee reflexes?
A1:  
  - Friedreich’s ataxia
  - Multiple sclerosis
  - Peripheral neuropathy in a stroke patient.
  - Motor neuron disease.
  - Conus medullaris-cauda equina lesion.
  - Tabes dorsalis
  - Subacute combined degeneration of the spinal cord

Q2: What is the mechanism?
A2: A mixture of cerebellar, pyramidal and dorsal column signs with a combination of pyramidal weakness with peripheral neuropathy.

49. Perform a focused physical examination for Charcot-Marie-Tooth disease (features of hereditary motor and sensory neuropathy due to peripheral nerve degeneration which does not usually extend above the elbows or above the middle third of the thighs).

- Distal muscle atrophy
- Pes cavus (short arched feet)
- Sensation
  - Slight or no sensory loss in the limbs
- Reflexes
  - Absent
- Nerves
  - Thickened
- Eyes
  - Optic atrophy
  - Argyll Robertson pupils


50. Perform a focused physical examination for Charcot-Marie-Tooth (CMT) disease (hereditary peroneal muscular neuropathy).

- Motor
  - Calves/thighs
    - Wasting
    - Stops abruptly, usually in the lower third of the thigh
  - Toes
    - Pes cavus (clawing of toes)
    - Contractures of the Achilles tendon
    - Weakness of dorsiflexion
  - Ankle
    - Absent ankle jerks
    - Plantars are downing going or equivocal
Sensory
- Mild sensory impairment or no sensory loss (occasionally a response to pain in the stocking distribution).

Adapted from: Baliga R.R. *Saunders/Elsevier* 2007, pages 165 and 166.

51. Perform a focused physical examination for 7 common muscle stretch reflexes. Name the reflexes, as well as the peripheral nerve and spinal level of the nerve involved.

<table>
<thead>
<tr>
<th>Name of reflex</th>
<th>Peripheral nerve</th>
<th>Spinal level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brachioradialis</td>
<td>Radial</td>
<td>C5-6</td>
</tr>
<tr>
<td>Biceps</td>
<td>Musculocutaneous</td>
<td>C5-6</td>
</tr>
<tr>
<td>Triceps</td>
<td>Radial</td>
<td>C7-8</td>
</tr>
<tr>
<td>Quadriceps (patellar)</td>
<td>Femoral</td>
<td>L2-L4</td>
</tr>
<tr>
<td>Achilles (ankle)</td>
<td>Tibial</td>
<td>S1</td>
</tr>
<tr>
<td>Abdominal</td>
<td>Epigastric</td>
<td>T6-T9</td>
</tr>
<tr>
<td></td>
<td>Mid abdominal</td>
<td>T9-T11</td>
</tr>
<tr>
<td></td>
<td>Lower abdomen</td>
<td>T1-L1</td>
</tr>
<tr>
<td>Cremasteric reflexes</td>
<td></td>
<td>L1, L2</td>
</tr>
<tr>
<td>Saddle sensation</td>
<td></td>
<td>S3, S4, S5</td>
</tr>
<tr>
<td>Anal reflex</td>
<td></td>
<td>S3, S4, S5</td>
</tr>
</tbody>
</table>

Adapted from: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto*, 2005, Table 13, page 164; and McGee S. R. *Saunders/Elsevier* 2007, Table 59-1, page 756.

Useful background: Interpreting deep tendon reflexes (DTR)

<table>
<thead>
<tr>
<th>Characteristic of DTR</th>
<th>Possible causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased reflex or clonus</td>
<td>- UMN lesion above root at that level</td>
</tr>
<tr>
<td></td>
<td>- Generalized peripheral neuropathy</td>
</tr>
<tr>
<td></td>
<td>- Isolated-peripheral nerve or root lesion</td>
</tr>
<tr>
<td>Reduced (insensitive)</td>
<td>- Peripheral neuropathy</td>
</tr>
<tr>
<td></td>
<td>- Cerebellar syndrome</td>
</tr>
<tr>
<td>Inverted (reflex tested is absent e.g. biceps but there is spread)</td>
<td>- Spinal cord LMN involvement at the level of the absent reflex</td>
</tr>
</tbody>
</table>

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to lower or higher level e.g. produces a triceps response)

- Pendular (reflex continues to swing for several beats)
  - Cerebellar disease

- Slow relaxation (especially at ankle)
  - Hypothyroidism

Adapted from: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto*, 2005, page 66.

Useful background: Grading the power of deep tendon reflexes

<table>
<thead>
<tr>
<th>Grade</th>
<th>Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Absent</td>
</tr>
<tr>
<td>1</td>
<td>Trace</td>
</tr>
<tr>
<td>2</td>
<td>Weak</td>
</tr>
<tr>
<td>3</td>
<td>Fair</td>
</tr>
<tr>
<td>4</td>
<td>Good</td>
</tr>
<tr>
<td>5</td>
<td>Normal</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Grade</th>
<th>Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No contraction detected</td>
</tr>
<tr>
<td>1</td>
<td>Slight contraction detected but cannot move joint</td>
</tr>
<tr>
<td>2</td>
<td>Movement with gravity eliminated only</td>
</tr>
<tr>
<td>3</td>
<td>Movement against gravity only</td>
</tr>
<tr>
<td>4</td>
<td>Movement against gravity with some resistance</td>
</tr>
<tr>
<td>5</td>
<td>Movement against gravity with full resistance</td>
</tr>
</tbody>
</table>

Note: since rating scale is skewed towards weakness many clinicians further sub classify their findings by adding a (+) or a (-), e.g. 5- or 3+

Source: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto*, 2005, page 164.

- Conditions causing thickened nerve plus peripheral neuropathy or mononeuritis multiplex
  - Acromegaly
    - Neurofibromatosis
  - Amyloid
    - Chronic inflammatory demyelinating polyradiculo neuropathy (CIDP)
  - Sarcoid
    - Autosomal dominant hereditary motor and sensory neuropathy
  - Leprosy
  - Diabetes

Abbreviation: CIDP, chronic inflammatory demyelinating polyradiculo neuropathy

52. Perform a focused physical examination for inflammatory polyradiculoneuropathy.

- Guillain-Barré syndrome
  - Flaccid paralysis in lower limbs progressing to upper limbs one week after an infective illness
  - Sensory loss and wasting is minimal or absent
  - Cranial nerves rarely affected, but may be confined there
  - Sphincters never affected

- Transverse myelitis
  - As for Guillain-Barré syndrome (as above), with involvement of sphincters:
    - HIV

Abbreviation: HIV, human immunodeficiency virus


53. Take a directed history and perform a focused physical examination for common peroneal nerve palsy (aka: lateral popliteal nerve palsy [L4,5].

- Motor
  - Leg
    - Wasting of the muscles on the lateral aspect of the leg (namely the peronei and tibialis anterior muscle)
  - Foot
    - Weakness of dorsiflexion
    - Eversion of the foot
    - Foot-drop
  - Gait
    - High-stepping
    - Loss of sensation of the lateral aspect of the leg and dorsum of the foot.
    - If the deep peroneal branch is affected, the sensory loss may be limited to the dorsum of the web between the first and second toes.

- Differentiate from other causes of foot-drop
  - L4, L5 root lesion
  - Lumbosacral plexus lesion
  - Sciatic nerve palsy
  - Peripheral neuropathy
  - Motor neuron disease

Adapted from: Baliga R.R. Saunders/Elsevier 2007, pages 210 and 211.
Useful background: Distribution of muscle wasting or weakness

<table>
<thead>
<tr>
<th>Pattern</th>
<th>Possible causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Focal (one limb)</td>
<td>Nerve root or peripheral nerve pathology</td>
</tr>
<tr>
<td>Proimal (bilateral)</td>
<td>Myopathy (no sensory loss)</td>
</tr>
<tr>
<td>Distal (bilateral)</td>
<td>Peripheral neuropathy (distal sensory loss)</td>
</tr>
</tbody>
</table>

Source: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto*, 2005, page 163.

**Neuromuscular disease**

Useful background: Neuromuscular disease

<table>
<thead>
<tr>
<th>Clinical Features of motor neuron disease</th>
<th>Clinical Features of myasthenia gravis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predominant symptom is weak, wasted</td>
<td>Fatigue</td>
</tr>
<tr>
<td>Fasciculating muscles</td>
<td></td>
</tr>
<tr>
<td>No sensory symptoms</td>
<td>Intermitent ptosis, diplop</td>
</tr>
<tr>
<td>No bladder disturbance</td>
<td></td>
</tr>
<tr>
<td>Tongue fasciculation</td>
<td>Weak speech</td>
</tr>
<tr>
<td>May involve: Respiratory muscles</td>
<td>Difficult chew swallowing</td>
</tr>
<tr>
<td>Respiratory failure</td>
<td></td>
</tr>
<tr>
<td>May start as wasting of intrinsic hand</td>
<td>Usually variable symptoms (i.e. fatigueable)</td>
</tr>
<tr>
<td>muscles.</td>
<td>Improves with edrophonium test</td>
</tr>
<tr>
<td>Differential diagnosis:</td>
<td>(acetylcholinesterase inhibitor)</td>
</tr>
<tr>
<td>- Cervical rib</td>
<td>Worse with aminoglycosides (and some</td>
</tr>
<tr>
<td>- Pancoast tumor</td>
<td>other drugs)</td>
</tr>
<tr>
<td>- T1 root lesion</td>
<td></td>
</tr>
<tr>
<td>- Syngonomyelia</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Myotonic dystrophy</th>
<th>Familial hypokalemic paralysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wasted and weak muscles</td>
<td>Onset aged 10-20 yrs</td>
</tr>
<tr>
<td>Frontal balding</td>
<td>offset ≥ 36 yrs</td>
</tr>
<tr>
<td>Cataracts</td>
<td>Generalized weakness</td>
</tr>
<tr>
<td>Difficulty relaxing grip</td>
<td>Association:</td>
</tr>
<tr>
<td>Autosomal dominant</td>
<td>- Asian people</td>
</tr>
<tr>
<td>Diabetes</td>
<td>- Thyrotoxicosis</td>
</tr>
<tr>
<td>Cardiac disease</td>
<td>- Food provokes symptoms</td>
</tr>
<tr>
<td>Gynecomastia</td>
<td></td>
</tr>
<tr>
<td>Testicular atrophy</td>
<td></td>
</tr>
<tr>
<td>Absent or depressed reflexes</td>
<td></td>
</tr>
</tbody>
</table>

Useful background: Classification of muscle disease

- Primary
  - Muscular dystrophy
    - Duchenne’s (pseudohypertrophic)
      o Affects only males (sex linked recessive)
      o Calves and deltoids: hypertrophied early, weak later
      o Proximal weakness: early
      o Dilated cardiomyopathy
    - Becker
      o Affects only males (sex linked recessive)
      o Similar clinical features to Duchenne’s except for less heart disease, a later onset and less rapid progression
  - Limb girdle
    o Males or females (autosomal recessive), onset in the third decade
    o Shoulder or pelvic girdle affected
    o Face and heart usually spared
  - Facioscapulohumeral
    o Males or females (autosomal dominant)
    o Facial and pectoral weakness with hypertrophy of deltoids
  - Dystrophia myotonica (autosomal dominant)
  - Myasthenia
    o Gravis
    o Carcinomatosis myasthenic syndrome
  - Myositis
    o Infection
      - Staph. Aureus
      - Streptococcus
      - TB
      - Clostridium welchii
      - Granulomatous
      - Sarcoidosis
      - Trichiniasis
      - Cysticercosis
    o Collagen/ vascular
      - Polymyalgia rheumatica
      - Dermatomyositis
    o Idiopathic
- Myositis ossificans
- Progressive myositis fibrosa

Secondary myopathy
  o Inherited
    - Glycogen storage disease
    - Paroxysmal myoglobinuria
    - Mitochondrial disorders
  o Drugs/toxic
    - Chloroquine
    - Alcoholism
    - Corticosteroids
  o Endocrine/metabolic
    - Hyper and hypothyroidism
    - Diabetes mellitus
    - Cushing’s syndrome
    - Hyper and hypo kalemia (including familial periodic paralysis)
    - Osteomalacia
  o Infiltrative
    - Carcinomatous myopathy
    - Amyloidosis

Atrophy
  o Secondary to disuse, neurological deficit etc


Useful background: Distribution of muscle wasting or weakness

<table>
<thead>
<tr>
<th>Pattern</th>
<th>Possible causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Focal (one limb)</td>
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<tr>
<td>Distal (bilateral)</td>
<td>Peripheral neuropathy (distal sensory loss)</td>
</tr>
</tbody>
</table>

Source: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto*, 2005, page 163.

54. Take a directed history of the causes of muscle weakness.

Cerebral disease
  o Hemiparesis
  o Paraparesis-anterior cerebral artery
Spinal cord disease
- Transverse myelitis
- Epidural abcess
- Extradural tumor
- Epidural hematoma
- Herniated intervertebral disk
- Spinal cord tumor

Peripheral nerve disease
- Guillain Barre syndrome
- Acute intermittent porphyria
- Arsenic poisoning
- Toxic neuropathies
- Tick paralysis
- Neuromuscular junction disease
- Myasthenia gravis
- Botulism
- Organophosphate poisoning

Neuromuscular junction disease
- Myasthenia gravis
- Botulism
- Organophosphate poisoning

Muscle disease (no sensory loss; access tone, atrophy, fasciculations)
- Polymyosistis
- Rhabdomyolysis-myoglobinuria
- Acute alcoholic myopathy
- Electrolyte imbalances
- Endocrine disease
- Myopathy
- Non progressive or relatively non progressive congenital myopathies
- Inflammatory myopathies
  - Toxoplasmosis, trichinosis, polio
  - Idiopathic-polymyositis, dermatomyositis
  - Collagen vascular disease
- Metabolic myopathies
  - Glycogenoses

Reflexes

Other findings
- Ptosis
- Diplopia
- Myotonia

Proximal weakness
- Myopathy
- Neuromuscular junction disease e.g. myasthenia gravis
- Neurogenic eg motor neurone disease, polyradiculopathy

Proximal myopathy with peripheral neuropathy:
- Paraneoplastic syndrome
- Alcohol
- Hypothyroidism
- Connective tissue diseases

Abbreviations: CNS, central nervous system; IBM, inclusion body myositis; PNS, peripheral nervous system


Perform a focused physical examination for (Becker) muscular dystrophy (MD).

Demography
- Young adult male
- Family history of MD

Face
- No facial muscle weakness

Calves
- Pseudohypertrophy of calves.

Back
- Proximal weakness of the lower extremities
- In later stages more generalized muscle involvement
- Kyphoscoliosis

Useful background: Dystrophia myotonica

- Eye
  - Partial ptosis
  - Cataracts
  - Subcapsular fine deposits

- Proximal muscle
  - Westings
  - Weakness

- Breast
  - Gynecomastia

- Face
  - Baldness
  - Temporalis atrophy
  - Triangular faces

- Neck
  - Sternomastoid atrophy
  - Weak neck flexion

- Heart
  - Cardiac failure (cardiomyopathy)

- Muscle
  - Myotonia

- GU
  - Testicular atrophy

Adapted from: Talley NJ et al. Maclennan & Petty Pty Limited, 10.30, page 428; Table 10.59, page 429.

56. Perform a focused physical examination for myotonia dystrophia.

- Definition: Continued contraction of muscle after voluntary contraction ceases, followed by impaired relaxation

- Leg
  - Leg weakness (difficulty in kicking a ball)
  - “Pseudo-drop attacks” (weakness of quadriceps muscles).
    - pharyngeal dysphagia (esophageal involvement)

- GI

- GU
  - Gonadal atrophy (impotence)

- Lung
- Recurrent respiratory infection (weakness of muscles of bronchioles)

- **Eyes**
  - Ptosis, bilateral or unilateral. Differentiate bilateral ptosis if myotonia from
    - Myasthenia gravis
    - Congenital muscular dystrophies
    - Ocular myopathy
    - Syphilis
  - Cataracts
  - Difficulty in opening the eye after firm closure.

- **Face**
  - Wasting of temporalis, masseters and sternomastoid muscles
  - Frontal baldness (Is the patient may wearing a wig?).

- **Neck**
  - "swan neck”.

- **Hands**
  - Development of myotonia while shaking hands with the patient, note the myotonia (distal weakness)


57. Perform a focused physical examination for limb girdle dystrophy.

- **Definition**
  - Weakness and wasting of muscles of shoulder and/or pelvic girdle, but never the face occur
  - Shoulder girdle
    - Biceps
    - Brachioradialis
    - Wrist extensors
    - Deltoids
  - Pelvic girdle
    - In the early stages of the disease
      - Weak hip flexors and glutei
      - Wasting medial quadriceps and tibialis anterior
    - Hypertrophy of lateral quadriceps and calves

Myasthenia gravis

- Definition: Weakness of the eye and facial muscles that worsens with repeated contraction

Useful background:

- Likelihood ratio for clinical history and physical findings in a person with symptoms for myasthenia gravis (MG)

<table>
<thead>
<tr>
<th>Likelihood Ratio (PLR)</th>
<th>Factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>53</td>
<td>Abnormal sleep test</td>
</tr>
<tr>
<td>30</td>
<td>Peek sign</td>
</tr>
<tr>
<td>24</td>
<td>Abnormal ice test</td>
</tr>
<tr>
<td>15</td>
<td>Positive response to an anticholinesterase test</td>
</tr>
<tr>
<td>4.5</td>
<td>The history ‘speech becoming unintelligible during prolonged speaking’</td>
</tr>
</tbody>
</table>

Abbreviations: PLR, makes the diagnosis more likely


- Factors which increase the pretest probability of finding MG.
  - Patients with asymmetric fluctuating eyelid ptosis
  - Patients with extraocular dysmotility not referable to a single nerve
  - Patients with weakness of other specific muscles
  - Young women of child-bearing age, and men and women aged approximately 70 years


58. Perform a focused physical examination for myasthenia gravis (MG).

- Definition
  - Painless muscle weakness (fatigability) which increases with exercise and in the evening

- Physical examination

  - Eyes
    - Worsening of ptosis after sustained upward gaze for at least 45 seconds
    - Diplopia and variable squint

  - Face
    - Snarling face when the patient attempts to smile

  - Speech
    - Speech is nasal
Muscles
- Weakness without loss of reflexes, or alteration of sensation or coordination. The weakness may be generalized; it may affect the limb muscles, often proximal in distribution, as well as the diaphragm and neck extensors.
- Muscle wasting is rare, and presents late in the disease.

Associations

Endocrine
- Thyrotoxicosis
- Hypothyroidism
- Diabetes mellitus

MSK
- Rheumatoid arthritis
- Dermatomyositis
- SLE
- Sjogren’s disease
- Sarcoidosis

Skin
- Pemphigus

Autoimmune
- Pernicious anemia

Differentiate from
- Botulism
- Eaton-lambert syndrome
  - Myasthenic disorder, often associated with bronchial small cell carcinoma
  - Commonly, weakness of truncal and proximal limb muscles


This is for the Neurology Resident

Q1: What is myasthenic crisis?
A1: Exacerbation of MG, especially bulbar and respiratory involvement, leading to need for ventilation.

Q2: What is cholinergic crisis?
A2: Excessive sensitivity to cholinergics in MG, such as in myasthenic crisis, with excessive salivation, confusion, lacrimation, miosis, pallor and collapse.
**Motor neuron disease**

Useful background: Common etiologies of neuromuscular weakness

<table>
<thead>
<tr>
<th>Location of Lesion</th>
<th>Common Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ø Upper motor neuron</td>
<td>o Cerebrovascular disease</td>
</tr>
<tr>
<td></td>
<td>o Multiple sclerosis</td>
</tr>
<tr>
<td></td>
<td>o Brain tumor</td>
</tr>
<tr>
<td>Ø Lower motor neuron</td>
<td>o Polyneuropathy (diabetes, alcoholism)</td>
</tr>
<tr>
<td></td>
<td>o Entrapment neuropathy</td>
</tr>
<tr>
<td></td>
<td>o Trauma</td>
</tr>
<tr>
<td>Ø Neuromuscular junction/muscle</td>
<td>o Myasthenia gravis</td>
</tr>
<tr>
<td></td>
<td>o Drug-induced myopathy</td>
</tr>
<tr>
<td></td>
<td>o Thyroid disease</td>
</tr>
<tr>
<td></td>
<td>o Polymyositis</td>
</tr>
</tbody>
</table>

Source: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto*, 2005, page 719.

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**THIS IS FOR THE NEUROLOGY RESIDENT**

Q: What are the prognostic markers that predict more severe multiple sclerosis?

A:  
  o Progressive disease from the onset of symptoms.
  o Frequent relapses in the first two years.
  o Motor and cerebellar signs at presentation to neurologist.
  o Short interval between the first two relapses.
  o Male gender.
  o Poor recovery from relapse.
  o Multiple cranial lesions on T2-weighted MRI at presentation.

- Muscle stretch reflex scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Reflex absent</td>
</tr>
<tr>
<td>1</td>
<td>Reflex small, less than normal; includes a trace response or a response brought out only with reinforcement</td>
</tr>
<tr>
<td>2</td>
<td>Reflex in lower half of normal range</td>
</tr>
<tr>
<td>3</td>
<td>Reflex in upper half of normal range</td>
</tr>
<tr>
<td>4</td>
<td>Reflex enhanced, more than normal; includes clonus if present, which optionally can be noted in an added verbal description of the reflex</td>
</tr>
</tbody>
</table>


59. Perform a take focused physical examination for motor neuron disease (MND) in the adult.

- Definition
  - Bulbar or pseudobulbar palsy
  - Amyotrophic lateral sclerosis
    - Flaccid arms and spastic legs.
  - Progressive muscular atrophy
    - Lesion in the anterior horn cells
    - Retention of deep tendon reflexes
    - Severe muscular atrophy of distal muscles
  - Primary lateral sclerosis (rare): signs progress from an UMN to a LMN picture

- Upper limbs
  - Fasciculations
  - ↓ reflexes
  - Painless weakness

- Lower limbs
  - Spasticity
  - ↑ reflexes
  - Up-going plantars

- Cranial
  - Dysarthria and dysphagia.
  - Sluggish palatal movements, absent gag reflex, brisk jaw jerk.
  - Combination of the above signs
    - Presence of upper and lower motor neuron involvement of a single spinal segment, and motor dysfunction involving at least two limbs or one limb and bulbar muscles.
- Sensory symptoms or signs are not seen.
- Ocular movements are not affected.
- Cerebellar or extrapyramidal systems are not affected.
- Sphincters are involved late, if at all.
  - Emotional lability (if there is bulbar involvement).


- Power grading system for weakness of the limbs
  - 0 - Nil movement
  - 1 -Flicker of movement
  - 2 -Movement cannot overcome gravity
  - 3 -Movement cannot overcome any resistance
  - 4 - Movement against resistance is weaker than "normal"
  - 5 –Normal


**Gait, posture, movement disorder and Parkinsonism**

Useful background:

- The physiological components of gait
  - Antigravity support: Provided by reflexes located in the spinal cord and brainstem; anti-gravity reflexes are responsible for maintaining full extension of hips, knees, and neck.
  - Stepping: a basic patter of movement based on sensory input from soles and body (including inclination forward and from side to side) and integrated at the midbrain level.
  - Equilibrium: Responsible for maintaining balance and centre and gravity during shifting of weight from one foot to the other.
  - Propulsion: Involves leaning forward and slightly to one side, permitting the body to fall a certain distance before being checked by leg support.

Useful background:

- **Definitions**
  - **Dysmetria** – inability to control one’s range of motion
  - **Dysdiadochokinesia** – inability to perform rapid alternating movements
  - **Ataxia** – defective voluntary muscle coordination
  - **Dysarthria** – difficult or defective speech attributed to impairments of the tongue
  - **Nystagmus** – constant involuntary cyclical movements of the eyes

- **Structures Involved in Walking**
  - **Basal ganglia**
    - anatomic movements which accompany walking
  - **Midbrain, locomotor region**
    - Initiate walking
    - Anti-gravity reflexes
  - **Cerebellum**
    - Maintains posture, balance, characteristic of movement (trajectory, velocity, acceleration)
    - Sense and proprioception
    - Anti-gravity reflexes
  - **Spinal cord**
    - Anti-gravity reflexes


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**SO YOU WANT TO BE A NEUROLOGIST – OR A HEMATOLOGIST!**

**Q:** What hematological abnormalities may be associated with chorea?

**A:**
- Polycythemia vera
- Neuroacanthocytosis (chorea – acanthocystosis)
- Cerebellar Ataxia Gait
  - Clinical
    - Irregular rate, range, direction of gait
    - Tendency to fall in any direction
    - Wide-base gait
    - Standing- titubation, may lead to falls, worse when feet together, unaffected by opening or closing eyes
    - Steps- vary in length, swaying (looks like drunken swagger)
    - Other cerebellar signs present- limbs ataxia, nystagmus
  - Cause
    - Cerebellar disease
    - Chronic alcoholism
    - Demyelination
    - Infection
    - Inherited

- Sensory Ataxia Gait
  - Clinical
    - Standing wide stance
    - High stepping gait
    - Wide gait, worsen when opening/closing eyes; sway/fall only when eyes closed (difficult walking at night)
  - Cause
    - Impaired sensory and proprioception in lower limbs
    - Peroneal nerve palsy
    - Tertiary syphilis
High stepped gait (“foot drop”)
  o Clinical
    - No dorsiflexion of ankle while walking: foot is raised high and then brought down quickly, in a flopping manner
    - Asymmetrical wear on soles of shoes
    - Waddling gait if proximal girdle muscles are also affected (e.g. motor neuron disease, progressive muscular atrophy); known as the anserine (duct-like waddling) gait.
  o Cause
    - Motor neuron disease
    - Peripheral neuropathy
    - Peroneal neuropathy
    - Spinal muscle atrophy (C-M-T gait)

Charcot Marie-Tooth (CMT) Gait
  o Clinical
    - High steppage gait (even with high elevation of knees)
    - Pes cavus (equinovarus deformity)
    - Plantar flexion
    - Inversion and adduction of foot
    - Calluses/foot ulcers
    - Impaired touch, pain, proprioception sensation
    - Absent deep tender reflex
  o Causes
    - Progressive, hereditary degeneration of peripheral nerves and nerve roots (peroneal nerve paralysis)
    - Slow, progressive wasting of muscles of feet/legs (“stork legs”) and then hands/arms

Anserine gait
  o Clinical
    - Standing
      - legs spread wide, shoulder sloped forward
      - lumber lordosis, protruding abdomen
    - Walking
      - Getting up from chair: Gower’s manoeuvre – bend forward, hands on knees, slide hands up the thighs and pushing up to standing
      - Short steps, waddling from side-to-side (duck-like waddling)
Differentiate from with high stepped gait

- Cause
  - Dystrophy of girdle muscles
  - Progressive muscular atrophy

- Parkinsonian Gait
  - Clinical
    - Slow standing up and starting to walk
    - Standing head and chest bent forward, flexed arms at elbows and knees flexed hips
    - Slow small steps with no arm swinging (automatic movement)
    - Festination – accelerating of walking, once walking started
    - Propulsion- tendency to fall forward, calling festination
    - Walking – further forward bending to chest, non-swinging arms, legs bent at ankles, knees, hips
    - Poor balance, poor compensation of flexion/extension, resulting in frequent falls
    - Toes not always on ground

- Apraxic "magnetic gait"
  - Clinical
    - Standing –feet wide apart
Walking
- Flexion of upper trunk, arms, knees
- Decreased automatic arm swing
- Shuffling gait
- Normal sensation and reflexes; Babinski plantar reflex may be up-going (abnormal)

○ Cause
  - Frontal lobe disease
  - Normopressure hydrocephalus
  - Aging

Spastic Hemiplegia (circumduction gait)

○ Clinical
  - Standing: (affected side)
    - Adduction/flexion of fingers, wrist, elbows
    - Extension of ankle, knee, hip
    - Internal rotation of foot
  - Walking (affected side)
    - Upper body tilts to unaffected side
    - Foot/leg of affected side swing in a semi-circle
    - Slow, difficult walk

○ Cause
  - Internal capsule hemisphere CVA
Trendelenburg gait (abnormal gluteus medius and minimus). In the Trendelenburg gait (from ineffective or weak hip abductors), the opposite pelvis falls excessively (arrow), and the conspicuous but opposing swings of the upper body and pelvis create the impression of the hinge between the sacral and the lumbar spine.
  - The patient is bearing weight on the affected right hip, with and ineffective right hip abductors (Trendelenburg gait).

The trunk may lean over the abnormal leg during stance (arrow).
  - During walking, a slight dip of the opposite pelvis is normal during stance phase on one limb.
  - The finding of excessive drop of the opposite pelvis is the abnormal Trendelenburg gait.
  - When the abnormality is bilateral, the pelvis waddles like that of a duck.
  - Like patients with the coxalgic gait ["Colxalgic Gait"], patients with Trendelenburg gait may lean their trunk over the abnormal leg during stance, but the lean lacks the dramatic lurch seen in coxalgic gait, and the opposing sways of the ipsilateral shoulder and opposite pelvis make it

Causes
  - Occurs when the gluteus medius and menimus do not function properly.
  - These two muscles abduct the hip, an action that supports the opposite pelvis and prevents it from dropping excessive amounts during the normal single-limb stance.

Coxalgic gait
  - In both abnormal gaits, the trunk may lean over the abnormal leg during stance (arrow).
  - In patients with hip pain and coxalgic gait, the trunk lean and accompanying ipsilateral arm movement (arrow) is more dramatic ("lateral lurch"), and the opposite pelvis does not fall excessively.
Proximal myopathy

- Causes
  - Metabolic (K+, Ca\(^{2+}\) excess/deficiency)
  - Alcoholism
  - Steroids
  - Thyroid disease
  - Inherited disease
  - Inflammatory (myositis)
  - Myasthenia

- Clinical
  - Difficulty reaching up

Spasticity

- Causes
  - Spinal cord disease
  - Cervical myelopathy
  - Multiple sclerosis
  - Stroke (legs held in adduction at the hip, thighs rub together, knees slide over each other)

- Clinical
  - Easy tripping
  - Falls

SO YOU WANT TO BE A NEUROLOGIST!

Q: What is Hemiballismus?
A: Sudden onset of unilateral, involuntary, flinging movements of the proximal upper limbs
  - Cardiovascular disease (source of emboli)
    - Atrial fibrillation
    - Valvular heart disease
    - Severe left ventricular dysfunction, travelling to the ipsilateral subthalamic nucleus of lungs and causing an infarction

- Unilateral, involuntary, flinging movement of the proximal upper limbs

Useful background: Characteristic gait of weak muscles

- The shading indicates the limb with the weak muscle and the black arrows indicate the diagnostic movements.

Parkinsonism, extrapyramidal disease, tremor and involuntary movement

- Parkinson’s disease

  Bardykinesia
  Tremor
  Rigidity

  Asymmetric onset

  - Resting “cog wheel”
  - Greasy skin
  - No facial expression = hypomimia
  - Intellectual deterioration
  - On walking
    - No arm swinging
    - Small footsteps with ‘shuffling’ gait-festinant
    - Difficulty walking and turning-falls
    - Bent posture

Useful background: Causes of Parkinson’s disease

- True parkinsonism

  - Idiopathic (due to degeneration of the substantia nigra, aka Parkinson’s disease or “paralysis agitans”)
  - Familial
  - Drug/toxins
    - Antagonist of D2 receptors
      - Neuroleptics (haloperidol, risperidone, resperine, etc)
      - Anti-emetics (metaclopramide, prochlorperazine)
    - Other psychiatric drugs
      - Selective serotonin reuptake inhibitors
      - Tricyclics
      - Lithium
    - Cardiovascular drugs
      - Amiodarone
      - Calcium channel blockers (flunarizine)
      - Atorvastatin
    - Anticonvulsants
      - Valproate
    - Others
      - Cyclosporine
      - Metrodinazole
      - Caffeine & other methylxanthines
      - β-Adrenergic agonists
      - Thyroine
- Prednisone
  - Brain damage (e.g. cardiac arrest, exposure to manganese or carbon monoide)
  - Postencephalitic – as a result of encephalitis lethargic or von Economo’s disease
  - Multiple system atrophy
  - Progressive supranuclear atrophy
  - Post encephalitic
  - Syphilis
  - Midbrain compression
  - Post traumatic
  - Wilson’s disease (Hepato-lenticular degeneration)
  - Hypoparathyroidism
  - Kernicterus
  - Neurologic
    - Brain tumour
    - Spinal cord trauma
    - Sleep apnea
    - Porphyria
    - Progressive supranuclear palsy
    - Shy-drager syndrome

- Pseudoparkinsonism
  - Essential tremor

- Hemiparkinsonism (presenting feature of a progressive space-occupying lesion)


Practice suggestion: The “best” clinical tests for the diagnosis of the presence of unilateral cerebral hemispheric disease are: arm rolling test, Babinski response, pronator drift, finger tapping test and hyperflexia.
Useful background: Types of Parkinsonian syndrome

<table>
<thead>
<tr>
<th>If:</th>
<th>If:</th>
<th>If:</th>
<th>If:</th>
<th>If:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sudden onset</td>
<td>Symptomatic illness</td>
<td>Marked postural hypotension</td>
<td>Early progressive dementia</td>
<td>Axial rigidity</td>
</tr>
<tr>
<td>+ stuttering progressor</td>
<td>Younger patient</td>
<td>n 5 BP ≥ 30 mmHg fall</td>
<td>Nocturnal wandering + confusion</td>
<td>Failure of vertical gaze</td>
</tr>
<tr>
<td>+ minimal tremor</td>
<td>Taking dopamine</td>
<td>Sphincter disturbance</td>
<td>Consider dementia with Lewy bodies</td>
<td>Consider progressive Supranuclear palsy</td>
</tr>
<tr>
<td>+ lower limbs</td>
<td>Antagonists of lithium</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>much more affected than upper limb</td>
<td>Consider drug-induced parkinsonism</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Consider vascular parkinsonism</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>


60. Perform a directed physical examination for extrapyramidal disease.

- The most typical pathological hallmarks of Parkinson’s disease are:
  - Neuronal loss with depigmentation of the substantia nigra
  - Lewy bodies, which are eosinophilic cytoplasmic inclusions in neurons consisting of aggregates of normal filaments

- Face
  - Mask-like facies
  - Absent blinking
  - Titubation (tremor of head)
  - Dribbling

- Speech
  - Soft, faint, monotones (monotonous)
  - Repetition of the end of a word (phalilalia)

- General inspection
  - Tremor (at rest, pill rolling, 4-7 hz), increased by clenching fist
  - Rigid tone (especially wrist for cogwheel rigidity)
  - Akinesia/ dyskinesia
  - Postural instability (stooped)
  - Mask-like face (lack of blinking, dysarthria)
- Hypophnia
- Sialorrhea
- facial seborrhoea, depressed, tendency to protrude tongue/ tongue tremor (mask-like face)

- Coordination test
  - Coarse motor control
    - Heel from knee to ankle test
    - Finger to nose test
  - Fine motor control
    - Rapid alternating movements – tapping feet
    - Rapid alternating movements – Thumb-finger opposition
    - Rapid alternating movements – Pronate-supinate hands
  - Posture and gait
    - Regular, toe, heel, and tandem gait assessments (start hesitation, shuffling steps, loss of arm swing)
    - Examines rising from chair, and walking and turning
    - Looks for festinant gait, foot shuffling, loss of arm swing, postural instability (stooped), flexed posture, pro/retropulsion (attempt provocation with push), slow (en bloc) turning
    - Heel from knee to ankle test, finger to nose test
    - Looks for loss of spontaneous movements
    - Blank facies
    - Stare with decreased blinking and widened palpebral fissures
    - Writing test for micrographia

- Limbs
  - Bradykinesia
  - Kinesia paradoxical - ability to perform rapid but not slow movements
  - Resting tremor (may be accentuated by person concurrently performing subtraction of “serial 7’s”
  - Cogwheel rigidity
  - Chorea jerky, abrupt, involuntary movements

- Motor examination
  - Inspection
    - Muscle bulk
    - Fasciculation
    - Muscle tone
  - Tone (palpation)
    - Upper extremities
    - Lower extremities
    - Asymmetry
    - Graded
- Checks for rigidity with cogwheeling (intensified with clenching other hand into fist) (upper and lower extremities)
- Tests by flexion-extension of elbow or supination-pronation of wrist

Motor

- Upper body dyskinesia
  This must be present – it is a symptom complex containing many of the following features:
  - Slowness of movement (bradykinesia)
  - Poverty of movement (mask-like facies, diminished arm swing)
  - Difficulty in initiating movement
  - Diminished amplitude of repetitive alternative movement
  - Inordinate difficulty in accomplishing some simultaneous or sequential motor acts

- Rigidity
  This is usually but not always present:
  - Leadpipe rigidity, where the increase in tone is equal in flexors and extensors of all four limbs but slightly more in flexors, resulting in a part feel 'simian' posture
  - Cog-wheel rigidity is due to superimposed or underlying tremor

- Postural instability; usually a late feature; may cause frequent falls
  - Gait
  - Festinant gait, in a posture of slight flexion

- Tremor
  Absent in about one third of patients with Parkinson’s disease at presentation and throughout its course in some
  - Resting, pill, pronation and supination rolling tremor of the upper limb
  - Intermittent
  - Intensified by emotion or stress, and disappears during sleep
  - The legs, head and jaw may shake as well distressing; the teeth may pound together until they become unbearably painful

- Speech
  - Monotonous

- Writing
  - Micrographia

- Reflexes – positive globellar tap

- Sleep disorders

- Autonomic involvement
Neuropsychiatric
- Hallucinations
  - Medication effect (exclude RBD)
- Depression
  - Loss of serotonergic neurons
- Cognitive impairment
  - Badyphrenia
  - Dementia (consider DLB)
- Sensory symptoms
- Abnormal behavior

Anxiety
- Akathisia
- Stressors

Sleep disorders

Autonomic involvement

Sensory symptoms

Abnormal behavior

Useful background: Other manifestations in Parkinson disease

<table>
<thead>
<tr>
<th>Manifestation</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain</td>
<td>Early morning dystonia, Motor fluctuations, Mechanical</td>
</tr>
<tr>
<td>Arm paresthesia</td>
<td>May reflect insufficient levodopa treatment</td>
</tr>
<tr>
<td>Fatigue</td>
<td>Multifactorial</td>
</tr>
<tr>
<td>Diplopia</td>
<td>Medication effect, Poor convergence</td>
</tr>
<tr>
<td>Pathologic gambling</td>
<td>Activation of D3 receptors in limbic striatum</td>
</tr>
<tr>
<td>Hypersexuality</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviation: DLB, dementia with Lewy bodies; MAO, monoamine oxidase; RBD, rapid eye movement sleep behavior disorder; SSRI, selective serotonin reuptake inhibitor

Useful background: Glabella tap test


What is “the best”? The “best” clinical tests for the presence of Parkinson’s disease are: the presence of all three of tremor, bradykinesia, and rigidity. Also, useful are a positive glabella tap, soft voice and difficulty or inability to walk heel to toe.

Useful background: Likelihood ratios for suspected Parkinson’s disease

- **Diagnosing Parkinson’s disease**
  - Prominent rigidity on initial examination, tremor, tremor as initial symptom, tremor-dominant disease, signs are asymmetric
  - Bradykinesia; a combination of tremor, bradykinesia, rigidity; paralysis or weakness, impaired consciousness, asymmetric disease, brady kinesia (akinetic/ rigid disease)
  - Good response to levodopa, have a PLR < 2 for diagnosing Parkinson’s disease

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diagnosing Multiple System Atrophy</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rapid progression</td>
<td>2.5</td>
<td>0.6</td>
</tr>
<tr>
<td>Speech and/or bulbar signs</td>
<td>4.1</td>
<td>0.2</td>
</tr>
<tr>
<td>Autonomic dysfunction</td>
<td>4.3</td>
<td>0.3</td>
</tr>
</tbody>
</table>
Cerebellar signs | 9.5 | 0.7
Pyramidal tract signs | 4.0 | Ns
Downgaze palsy and postural instability within first year of symptoms | 60.0 | 0.5

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tremor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Arms or leg shake</td>
<td>1.4-17</td>
<td>0.24-0.25</td>
</tr>
<tr>
<td>o Tremor of head or limbs</td>
<td>11</td>
<td>0.26</td>
</tr>
<tr>
<td>Rigidity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o R rigidity and bradykinesia</td>
<td>4.5</td>
<td>0.12</td>
</tr>
<tr>
<td>o Muscle stiffness</td>
<td>2.3</td>
<td>0.73</td>
</tr>
<tr>
<td>Facies and general symptoms or historical findings</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Feet freeze</td>
<td>3.7</td>
<td>0.55</td>
</tr>
<tr>
<td>o Face less expressive</td>
<td>2.1</td>
<td>0.54</td>
</tr>
<tr>
<td>Bradykinesia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Difficulty rising from chair</td>
<td>1.9-5.2</td>
<td>0.39-0.58</td>
</tr>
<tr>
<td>Posture and motor tasks</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Loss of balance</td>
<td>1.6–6.6</td>
<td>0.29–0.35</td>
</tr>
<tr>
<td>o Shuffling gait</td>
<td>3.3–15</td>
<td>0.32–0.50</td>
</tr>
<tr>
<td>o Trouble turning in bed</td>
<td>13</td>
<td>0.56</td>
</tr>
<tr>
<td>o Trouble opening jars</td>
<td>6.1</td>
<td>0.26</td>
</tr>
<tr>
<td>o Trouble buttoning</td>
<td>3.0</td>
<td>0.33</td>
</tr>
<tr>
<td>o Micrographia (fine motor)</td>
<td>2.8–5.9</td>
<td>0.30–0.44</td>
</tr>
<tr>
<td>Tremor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Tremor with rigidity and bradykinesia</td>
<td>2.2</td>
<td>0.50</td>
</tr>
<tr>
<td>Rigidity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Rigidity</td>
<td>2.8</td>
<td>0.38</td>
</tr>
<tr>
<td>o Rigidity with bradykinesia</td>
<td>4.5</td>
<td>0.12</td>
</tr>
<tr>
<td>General findings</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Glabella tap</td>
<td>4.5</td>
<td>0.13</td>
</tr>
<tr>
<td>o Voice softer</td>
<td>3.7</td>
<td>0.25</td>
</tr>
<tr>
<td>o Change in speech</td>
<td>2.6</td>
<td>0.73</td>
</tr>
<tr>
<td>Bradykinesia</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Posture and motor tasks</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Difficulty or inability to walk heel to toe</td>
<td>2.9</td>
<td>0.32</td>
</tr>
</tbody>
</table>
Abbreviation: NLR, negative likelihood ratio; PLR, positive likelihood ratio
Note: several signs are not included because their PLR is less than 2. These are uncontrolled limbs, tremor without rigidity and bradykinesia, and asymmetric disease nd response to levodopa.

Useful background: Special tests for bradykinesia

A. Tapping the fingers  
B. Twiddling

C. Pinching and circling  
D. Tapping with the heel

Adapted from: Simel D. L., et al. McGraw-Hill Medical 2009, Figure 38-1, page 507.

"Harsh words are heavy and often fall with a big thud, but a kind word will bounce on and on…”
Anonymous
SO YOU WANT TO BE A NEUROLOGIST!

Q: What is the difference between rigidity, spasticity, gegenhalten and tardive dyskinesia?
A: o Rigidity indicates increased tone affecting opposing muscle groups equally, and is present throughout the range of passive movement. When smooth it is called ‘leadpipe’ rigidity, and when intermittent termed ‘cog-wheel’ rigidity. It is common in tetrapyrimal syndromes. Wilson’s disease and Creutzfeld-Jakob disease.
  o Spasticity of the clasp-knife type is characterized by increased tone which is maximal at the beginning of movement and suddenly decreases as passive movement is continued. It occurs chiefly in floors of the upper limb and extensors of the lower limb (antigravity muscle).
  o Gehenhalten, or paratonia, is where the increased muscle tone varies and becomes worse the more the patient tries to be relax.
  o Tardive dyskinesia is seen in patients taking neuroleptics. Its manifestations are orofacial dyskinesia such as smacking, chewing lip movements, discrete dystonia or choreiform movements and, rarely, rocking movements.


DO YOU STILL WANT TO BE A NEUROLOGIST AND HAVEN’T GIVEN UP YET?

Q: Some persons with Parkinson’s disease have other neurological deficits. These are called “Parkinson plus syndromes”, Give 4 examples.
A: o Steele – Richardson – Olszewski disease (akinesia, axial rigidity of the neck, bradyphrenia, supranuclear palsy)
  o Multiple system atrophy (MSA)
    - Olivopontocerebellar degeneration
    - Strionigral degeneration
    - Progressive autonomic failure (Shy – Drager syndrome)
  o Basal ganglia calcification
  o Give up and switch to something else

Useful background: Differential diagnosis of Parkinson – plus syndromes

<table>
<thead>
<tr>
<th>Manifestation</th>
<th>Suspect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poor response to levodopa</td>
<td>Any parkinson-plus syndrome (MSA &amp; PSP may respond)</td>
</tr>
<tr>
<td>Early falls</td>
<td>PSP &amp; MSA</td>
</tr>
<tr>
<td>Severe OH &amp; urologic Sx</td>
<td>MSA</td>
</tr>
<tr>
<td>Cerebellar signs</td>
<td>MSA or spinocerebellar degeneration</td>
</tr>
<tr>
<td>Vertical gaze</td>
<td>PSP</td>
</tr>
<tr>
<td>Asymmetric apraxia</td>
<td>Corticobasal degeneration</td>
</tr>
<tr>
<td>Early dementia</td>
<td>Dementia with Lewy bodies of Creutzfeldt-Jakob disease</td>
</tr>
</tbody>
</table>

Abbreviations: MSA, multiple system atrophy; OH, orthostatic hypotension; PSP, progressive supranuclear palsy; S, symptoms


Useful background: The differential diagnosis for Parkinson’s disease

- Ideopathic (degeneration of substantia nigra)
- Drugs
  - Neuroleptics
  - Metoclopimide
- Toxins
  - MPTP (drug abusers), manganese, carbon disulfide, CO

Abbreviations: CO, carbon monoxide

**Tremor**

Useful background:

- Tremor of Parkinson Disease
  - Slow frequency (4-6/s) tremor at rest
  - ‘Pill rolling quality’
  - Asymmetrical
  - Hands, legs, chin, jaw, but not the head
- Rigidity, bradykinesia
- ↓ during movement, sleep
- ↑ by emotional distress
- Family history in only 15%
- No consistent response to alcohol

Useful background: Tremors

61. Perform a focused physical examination of tremor.

- **Definition**
  - Tremors are involuntary movements that result from alternating contraction and relaxation of the group of muscles.
  - Rhythmic oscillations about a joint or a group of joints

- **Rest tremor**
  - As seen in Parkinsonism. The hands have characteristic motion of pill rolling, alternating flexion/extension of fingers or hands, alternating pronation/supination of forearms.

- **Intention tremor (cerebellar)**
  - **Clinical**
    - Worsens with alcohol.
    - Limbs or head tremor
    - ↑ by movement
    - contra-axial
    - Symmetrical
    - Often involves the head
    - Aggravated by voluntary movements
  - **Examples**
    - Chorea
    - Athetosis
    - Hemiballismus
    - Fasciculation
    - Torticollis
    - Clonus

- **Postural and action (kinetic) tremor**
  - **Clinical**
    - Fast tremor throughout movement seen best with arms and hands outstretched.
    - Exaggerated physiological tremor
  - **Causes**
    - Anxiety
    - Sleep deprivation
    - Fatigue
    - Cold
    - β-agonist drugs
    - withdrawal of alcohol, caffeine, or lithium
    - Hyperthyroidism or hypoglycemia.
    - Brain damage seen in Wilson’s disease, syphilis

- **Essential tremor**
  - Autosomal dominant inheritance
- Shaking when carrying a teacup, putting a glass to the mouth, or trying to eat soup. It may affect handwriting and voice.
  - Bilateral, usually symmetric postural or kinetic tremor
  - ↓ by action, postural change, alcohol
  - Hands, head, neck voice
  - Tremor due to neuropathy (postural tremor; arms more than legs)
  - No rigidity or bradykinesis
  - Family history in 60%
  - Family history of tremor is common

➢ Physiologic tremor
  - Variable
  - Enhanced form is visible, postural, and has a high frequency (8-12/s)
  - No underlying neurologic disease
  - Cause is usually reversible (e.g. caffeine)


62. Take a directed history and perform a focused physical examination for chorea.

- Causes of Chorea

➢ Neurological disorder
  - Post CVA
  - Huntington’s disease (autosomal dominant)

➢ Drugs: e.g. excess levodopa, phenothiazines, the contraceptive pill, phenytoin

➢ Metabolic
  - Wilson’s disease
  - Kernicterus (rare)
  - Thyrotoxicosis (very rare)
  - Hypoparathyroidism

➢ Infection
  - Viral encephalitis (very rare)
  - Sydenham’s chorea (rheumatic fever) and other postinfectious states (both rare)

➢ Hematological
  - Polycythemia or other hyperviscosity syndromes (very rare)

➢ Vascular and vasculitis
Dementia

- History for differential diagnosis ("Dementia")
  - Drugs (alcohol, barbiturates, bromides)
  - Emotion (depression, schizophrenia)
  - Metabolic (Wernicke-Korsakoff syndrome, B12/folate deficiency, hyper/hypothyroid)
  - Eye and ear (severe visual and auditory impairment)
  - Neurodegenerative (Huntington’s, Parkinson’s, Alzheimer’s disease)
  - Trauma (head injury, dementia pugilistica), tumour (subfrontal menigioma)
  - Infection (HIV, syphilis, viral encephalitis, Creutzfeld-Jacob disease)
  - Arteriosclerotic and vascular (multi infarct dementia, vasculitis, cerebral hemorrhage)

Abbreviations: ADL, activities of daily living

Useful background: Determine site of cause

- Cortical coma (brainstem functions are present)
  - Brainstem coma
    - Response to pain
    - Decerebrate- mild
      - Upper extremity flexion (hands point towards heart)
      - Lower extremity extension and internal rotation
    - Moderate
      - Upper and lower extremities – extension and internal rotation
    - Severity
  - Midbrain
    - Fixed pupils
o Pons
  - Doll’s eye reflex eyes - remain fixed in the midline when head is turned (CNI; normally, touching cornea on one side causes a wink response on both sides with a unilateral disturbance of V, wink response does not occur on either side when affected side is stimulated)
  - Loss of corneal reflex
  - Loss of jaw reflex (CN) jaw will deviate towards the side of the lesion

o Medulla
  - Dysfunction of cardiopulmonary centers

o Corticopontine

o Impaired conjugate eye movement (cerebral or basilar artery thrombosis)
  Death
  - Global absence of brainstem function on two neurological examinations 12 hours apart, and exclusion of toxic-metabolic cause


63. Take a directed history and perform a focused physical examination for the causes of dementia.

➢ History
  o Initial screen
    - Assesses hearing/vision
    - Assesses orientation (person, place, time)
    - Elicits chief complaint
  o Description of symptoms
    - Onset, duration, and course of current complaint(s)
    - Palliating/provoking factors
    - Limitations in functioning (ADLs, IADLs)
  o Depression symptoms
    - Assesses depression symptoms (low mood, anhedonia, sleep disturbance, etc)
    - Assesses suicidality and homicidality
  o Anxiety symptoms
    - Anxiety symptoms (phobias, obsessions, compulsions, etc)
  o Perception disturbances
    - Psychotic symptoms (hallucinations, delusions, ideas of reference etc)
  o Personality and behavioral disturbances
- Changes in personality
- Behavioral abnormalities (apathy, agitation, odd behaviors, etc)
  o Past and family medical history
    - Hx of alcohol/drug abuse
    - Medications and Hx of adverse drug reactions
    - Hx of psychiatric illness
    - Hx of other metabolic or systemic illness(s)
  o Collateral history from family member
    - Elicits concerns
    - Confirms history
    - Inquiries about safety, home fire risks, driving, wandering

➢ Physical examination
  o Inspection
    - Dress and grooming
    - Speech
    - Attitude and behavior in office
  o Folstein mini mental status exam
    - Orientation (place, time: 5pt for each)
    - Registration (name 3 objects: 1 pt for each)
    - Attention and concentration (serial 7’s, world, months: 5 pt total)
    - Recall (recall 3 objects: 1 pt for each)
    - Language:
      ▪ identify 2 objects pointed to: 2 pt total
      ▪ ask no ifs ands or buts: 1 pt total
      ▪ perform 3 stage command: 3 pt total
      ▪ read and obey written command: 1 pt total
      ▪ write a sentence: 1 pt total
      ▪ draw intersecting pentagons: 1 pt total
  o Additional cognitive tests
    - Perseveration (ask patient to copy a series of loops)
    - Construction ability (draw hands of clock for different times)
    - Concrete thinking (compare word similarities)
    - Abstract thinking (describe meaning of proverb)

Abbreviation: ADL, activities of daily living; IADL, instrumental activities of daily living

Useful background: Differential diagnosis of dementia

- **Structural lesions**
  - Normal-pressure hydrocephalus
  - Subdural hematoma
  - Neoplasm
  - Vascular dementia

- **Infections**
  - Chronic meningitis
  - Neurosyphilis
  - HIV dementia
  - Encephalitis
  - Meningitis
  - Abscess
  - Creutzfeldt – Jakob disease
  - Cryptococcal meningitis

- **Inflammatory/immune disorders**

- **Vasculitis**

- **Hashimoto/autoimmune encephalopathy**

- **Tumor**
  - Intracranial (especially frontal)

- **Degenerative dementia**
  - Alzheimer disease
  - Diffuse Lewy body disease
  - Frontotemporal dementia (including Pick disease)
  - Huntington disease
  - Progressive supranuclear palsy
  - Multiple sclerosis

Abbreviation: HIV, human immunodeficiency virus

64. Take a directed history to differentiate between delirium and dementia.

<table>
<thead>
<tr>
<th>Delirium</th>
<th>Dementia</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Onset</td>
<td>➢ Rapid</td>
</tr>
<tr>
<td>➢ Course</td>
<td>➢ Fluctuates over time</td>
</tr>
<tr>
<td>➢ Orientation</td>
<td>➢ Disoriented to time and place</td>
</tr>
<tr>
<td>➢ Psychosis</td>
<td>➢ More likely present</td>
</tr>
<tr>
<td>➢ Other</td>
<td>➢ Perceptual disturbances, sleep wake cycles disturbed, ↑ or ↓ psychomotor activity</td>
</tr>
<tr>
<td>➢ Reversible</td>
<td>➢ Often</td>
</tr>
</tbody>
</table>


Useful background: Performance characteristics for dementia and delirium*

<table>
<thead>
<tr>
<th>Finding</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Dementia</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Abnormal clock drawing test</td>
<td>36-75</td>
<td>72-98</td>
<td>5.3</td>
<td>0.5</td>
</tr>
<tr>
<td>➢ Mini mental status examination: 3 levels</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o &lt;20</td>
<td>39-69</td>
<td>93-99</td>
<td>14.5</td>
<td>...</td>
</tr>
<tr>
<td>o 21 to 25</td>
<td>26-51</td>
<td>...</td>
<td>2.2</td>
<td>...</td>
</tr>
<tr>
<td>o &lt; 23</td>
<td>69-100</td>
<td>78-99</td>
<td>8.1</td>
<td>0.2</td>
</tr>
<tr>
<td>o &gt; 26</td>
<td>4-10</td>
<td>14-27</td>
<td>0.1</td>
<td>...</td>
</tr>
<tr>
<td>➢ Delirium</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Positive test using “Confusion Assessment Method”</td>
<td>46-94</td>
<td>83-98</td>
<td>10.3</td>
<td>0.2</td>
</tr>
</tbody>
</table>

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Probability

<table>
<thead>
<tr>
<th>Decrease</th>
<th>Probability</th>
<th>Increase</th>
</tr>
</thead>
<tbody>
<tr>
<td>-45%</td>
<td>-30%</td>
<td>-15%</td>
</tr>
<tr>
<td>+15%</td>
<td>+30%</td>
<td>+45%</td>
</tr>
</tbody>
</table>

NLR

<table>
<thead>
<tr>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1</td>
</tr>
<tr>
<td>0.2</td>
</tr>
<tr>
<td>0.5</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>5</td>
</tr>
<tr>
<td>10</td>
</tr>
</tbody>
</table>

Sen N out – Sensitive test; when negative, rules out disease

Sp P in – Specific test; when positive, rules in disease

Seizures

Epilepsy - localized (or partial) seizures
- Simple: normal conscious level
- Complex: altered conscious level

Focal motor seizures
- Jacksonian seizures
- Jerking of affected muscle
- Neighbouring muscle groups jerk as electrical discharge spreads ('marches') over motor cortex
- Post-ictal loss of motor function ('paralysis') for a few hours/day ('Todd's paresis')

Temporal lobe seizures
- Often relate to structural abnormality e.g. scarring from (prolonged) childhood febrile convulsions

Aura
- Over – under-familiarity with surroundings (déjà vu and jamais vu)
- Unpleasant taste or smell
- Epigastric discomfort

Seizure
- Facial grimacing
- Complex motor actions e.g. undressing
- Bizarre behaviour

Post-ictal
- Usually rapid recovery
- Amnesia of seizure events

Typical epileptic seizure

<table>
<thead>
<tr>
<th>Aura</th>
<th>Seizure</th>
<th>Post seizure phenomena</th>
</tr>
</thead>
<tbody>
<tr>
<td>Usually &lt; 1 min</td>
<td>Lasts &lt; few minutes</td>
<td>Post ictal</td>
</tr>
<tr>
<td>Depends on site</td>
<td>Rarely continues for prolonged</td>
<td>If generalized, very sleepy &lt; few hours</td>
</tr>
</tbody>
</table>

Focal sensory seizures
- Unpleasant tingling 'marching' over body in < few seconds
- Differential diagnosis includes migraine sensory symptoms here 'march' over body in 10-15 min

Generalized seizures
- Often involve diencephalic structures
- Typical childhood absences ('petit mal')
- Occur in childhood, very rare to continue in adulthood common
- Myoclonic epilepsy
- Akinetic epilepsy \(\rightarrow\) sudden complete loss of postural tone \(\rightarrow\) sudden collapse. Rare.
- Grand mal seizures (see text)

Useful background: Causes of seizure

- **Idiopathic**

- **Congenital**
  - Cerebral malformation
  - Lipidoses (Tay-Sachs disease)

- **Trauma**
  - Tumour
  - Scar
  - Birth injury

- **Infection**
  - Encephalitis
  - Meningitis
  - Abscess
  - Cysticercosis
  - GPI
  - Pyrexia (especially in children)

- **Metabolic**
  - Anoxia
  - Hypoglycemia
  - Hypocalcemia
  - Alkalosis
  - Water intoxication
  - Uremia
  - Hepatic coma

- **Drugs/toxins**
  - Nikethamide
  - Lead poisoning
  - Cocaine
  - Ether
  - Barbiturate withdrawal

- **Tumor**

- **Vascular**
  - CVA
  - Hypertension

- **Degenerative**
  - Presenile dementias

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 70.
65. Take a directed history for seizures.

- **Age (at onset)**
  - Onset, off set, duration, fluctuation

- **Type** - “phase out”, myoclonic, tonic, tonic clonk, atonic (“drop seizures”), partial (affecting only a part of the brain rather than a generalized seizure affecting all the brain; partial seizures may include OR not include awareness of events)
  - Simple partial seizures
    - Motor, sensory or psychomotor phenomena without loss of consciousness
    - Seizures can begin in one part of the body and spread to other parts
  - Complex partial seizures
    - May be preceded by an aura (sensory or psychic manifestations that represent seizure onset)
    - Staring, performing of automatic purposeless movements, uttering of unintelligible sounds, resisting aid
    - Motor, sensory or psychomotor phenomena
    - Post-ictal confusion
  - Tonic-clonic seizures (formerly known as grand-mal)
    - Tonic phase – stiffening of limbs
    - Clonic phase – jerking of limbs
    - Respiration may decrease during tonic phase but usually returns during clonic phase, although it may be irregular
    - Incontinence may occur
    - Post-ictal confusion
  - Atonic seizures
    - Brief, primarily generalized seizures in children
    - Complete loss of muscle tone, resulting in falling or pitching to the ground
    - Risk of serious trauma, particularly head injury
  - Absence seizures
    - Brief, primarily generalized attacks manifested by a 10 to 30-second loss of consciousness
    - Eyelid flutterings at a rate of 3 Hz
    - No loss of axial muscle tone
    - No falling or convulsing
    - No post-ictal symptoms
  - Status epilepticus – a medical emergency!
    - Repeated seizures with no intervening periods of normal neurologic function
    - Generalized convulsive status epilepticus may be fatal
    - With complex partial or absence seizures, an EEG may be needed to diagnose seizure activity
- **Jacksonian epilepsy**
  - Clonic movements
  - Always start at same site
  - Always show same order of speed
  - Early on, may be followed by transient paralysis
  - Later on, may be followed by later paralysis
  - Sometimes, no causative lesion is found

- **Generalized seizures**
  - Generalized tonic-clonic seizures.
  - Petit mal and atypical absences
  - Myoclonus
  - Akinetic seizures. Petit mal describes only 3 Hz seizures, rather than clinically similar absence attacks which are partial seizures

- **Partial or focal seizures** (a partial seizure is epileptic activity confined to one area of cortex with a recognizable clinical pattern)
  - Simple partial seizures (no impairment of consciousness)
  - Jacksonian epilepsy: it is a simple partial seizure which usually originates in one portion of the prefrontal motor cortex so that fits begin in one part of the body (e.g. thumb) and then proceed to involve that side of the body and then the whole body. It suggests a space-occupying lesion.
  - Complex partial seizures
  - Partial seizures evolving to tonic-clonic

- **Todd’s paralysis**
  - Paresis of a limb or hemiplegia occurring after an epileptic attack, which may last up to 3 days.

- **No precipitating factor identified** (e.g., sleep deprivation, alcohol use)

- **Associations** - aura, salivation, tongue biting, incontinence, chewing, lip smacking, Jacksonian march, onset during sleep or with fever

- **Other factors**
  - Family history of seizures (in first degree relative)
  - History of febrile seizures or birth trauma
  - Postictal Todd paralysis

- **Abnormal electroencephalogram** (spikes or non-specific)

- **Abnormal imaging study**

- **Causes**
  - Congenital – malformations – birth injury
  - Ideopathic
- Trauma
- Tumor
- Infection
  - Meningitis
  - Encephalitis
  - Abscess
  - Syphilis
  - Cysticercosis
- Vascular
  - CVA
  - NTN
  - Hypertermia, especially in children
  - Hypothermia
- Degeneration – presenile dementia
- Metabolic*
  - Anoxia
  - Hypoglycemia
  - Hypocalcemia
  - Renal failure
  - Hyponatremia
  - Alkalosis
- Motor intoxication
- Drugs
  - Lead
  - Cocaine
  - Barbiturate, alcohol withdrawal

- Abnormal neurologic examination - CVA, trauma, Meningism

*Metabolic causes may cause delirium.
Abbreviation: CVA, cerebral vascular accident; NTN

66. Take a directed history and perform a physical examination for a cerebral vascular accident (CVA).

- **History**
  - **Weakness**
    - Location and extent
    - Time course (onset, duration, change with time)
    - Previous episodes (TIAs)
    - Quality of deficit (sensory, movement, power)
  - **Associated symptoms**
    - Paresthesia
    - Pain
    - Dizziness
    - Level of consciousness
    - Amaurosis Fugax
    - Slurred speech
    - Skin changes (colour, swelling, warmth)
    - Injury or trauma
    - Infection (fever, chills, sweating)
  - **Risk factors**
    - Family Hx of neurological Disease
    - Hx of stroke
    - Hx of MI, Murmur, Palpitations, Rheumatic heart disease
    - Atherosclerosis RF (hypertension, DM, FHx of CAD, hypercholesterolemia, smoking)
  - **Impact on ADLs**
    - Is the patient R or L handed?
    - Gross motor (reaching shelves, opening doors)
    - Fine motor (buttoning shirt, using keys, writing)
    - Impact on personal and family life

- **Physical examination**
  - **Inspection**
    - Compares right arm to left arm for:
      - Atrophy
      - Fasciculation
      - Abnormal position
      - Abnormal movements
  - **Tone**
    - Compares right arm to left arm for:
      - Rigidity
      - Spasticity (velocity dependant)
  - **Power**
    - Compares and grades right arm to left arm power for:
      - Shoulder extension and abduction
      - Elbow flexion, extension, pronation and supination
Achieving Excellence in the OSCE Part 2

- Wrist flexion, extension, ulnar and radial deviation
- Digit abduction, adduction, thumb extension and thumb opposition
- Pronator Drift Test
  - Reflexes
    - Compares and grades right arm to left arm reflexes for: Biceps (C5-6), Brachioradialis (C5,6) and triceps (C7,8)
  - Coordination
    - Finger to nose test
    - Rapid alternating movement

Abbreviations: ADL, activities of daily living; AF, atrial fibrillation; CAD, coronary artery disease; CVA, cerebral vascular accident; HBP, hypertension; MI, myocardial infarction; TIA, transient ischemic attack;


67. Perform a focused physical examination for the causes of paraplegia.

- Congenital (cerebral palsy)
  - Hereditary ataxia

- Infiltration
  - Abdominal
  - Anterior cerebral artery occlusions
  - Cord compression
  - Tumourous myelitis

- Ischemia
  - Superior sagittal sinus thrombosis
  - Spinal artery occlusion (Erb's paraplegia)

- Infection
  - Poliomyelitis

- Degeneration
  - Motor neuron disease
  - Multiple sclerosis
  - Syringomyelia

- Metabolic
  - Subacute combined degeneration

Adapted from: Burton J.L. Churchill Livingstone 1971, page 84.
Useful background: Common stroke syndromes

<table>
<thead>
<tr>
<th>Location of artery occlusion</th>
<th>Clinical significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left sided</td>
<td>Aphasia</td>
</tr>
<tr>
<td>Right sided</td>
<td>Neglect of left space, lack of awareness of deficit, apathy, impersistence</td>
</tr>
<tr>
<td>Anterior cerebral artery (ACA)</td>
<td>Contralateral weakness of the lower limb and shoulder shrug</td>
</tr>
<tr>
<td>Middle cerebral artery (MCA)</td>
<td>Contralateral motor, sensory and visual loss</td>
</tr>
<tr>
<td>Posterior cerebral artery (PCA)</td>
<td>Contralateral hemianopia and hemisensory loss</td>
</tr>
<tr>
<td>Internal carotid artery (ICA)</td>
<td>Contralateral MCA and ACA signs</td>
</tr>
<tr>
<td></td>
<td>May also have ipsilateral transient monocular blindness (amaurosis)</td>
</tr>
<tr>
<td>Basilar artery</td>
<td>Bilateral motor weakness, ophthalmoplegia and diplopia</td>
</tr>
</tbody>
</table>

Source: Filate W., et al. Medical Society, Faculty of Medicine, University of Toronto 2005, page 171.

SO YOU WANT TO BE A NEUROLOGIST!

Q. In the context of blindness, what is amaurosis fugax?

A. A transient monocular blindness due to episodic retinal ischemia, usually associated with ipsilateral carotid artery stenosis or embolism of the retinal arteries resulting in a sudden, and frequently complete, loss of vision in one eye.

Useful background: Amaurosis fugax – definition

- Transient monocular blindness due to episodic retinal ischemia, usually associated with ipsilateral carotid artery stenosis or embolism of the retinal arteries resulting in a sudden and frequently complete loss of vision in one eye.

- For 45 sec stretch out both arms, with palms upright and eyes closed.
- Positive test: the arm on the side opposite to the cerebellar lesion drifts downward and pronates.

- Bend elbows, place forearms parallel to each other, and rotate the forearms about each other in a rapid rolling motion for 10 seconds in each direction. Positive test: the arm on the side opposite to the cerebellar lesion is stay still, while the other arm "orbits" around it.

- Tap the thumb and index finger together repeatedly at about two taps per second. Positive test: the fingers on the side opposite to the cerebellar lesion tap more slowly, and may even look like the two fingers are sticking together.

Useful background: Carotid artery stenosis

- In patients with TIA, a carotid bruit indicates the presence of a > 50% stenosis of the carotid artery (confirmed by carotid angiography) with 29% sensitivity and 88% specificity.

Adapted from Sauve JS et al., JAMA 1993; 270: 2843-5.

Useful background: Strokes

- 80% Ischemic thrombosis
  - Embolus
  - Systemic hypoperfusion

- 20% Hemorrhagic intracerebral
  - Sub-arachnoid
  - Subdural/extradural

Source: Filate W., Et al. The Medical Society, Faculty of Medicine, University of Toronto, 2005, page 191.

- The presence of a carotid bruit (CB) may increases the likelihood of a 70-99% carotid stenosis:

<table>
<thead>
<tr>
<th>Patient</th>
<th>Ipsilateral bruit</th>
<th>PPV</th>
</tr>
</thead>
<tbody>
<tr>
<td>o Asymptomatic</td>
<td>Yes</td>
<td>4.0-10.0</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>Uncertain</td>
</tr>
<tr>
<td>o Symptomatic*</td>
<td>Yes</td>
<td>3.0 (1.3-7.1)</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>0.49 (0.36-0.67)</td>
</tr>
</tbody>
</table>

*Carotid-territory cerebrovascular symptoms.

- The presence of a carotid bruit cannot be used to rule it in, nor can its absence be used to rule it out (JAMA, Chapter 9, page 109).

Abbreviations: CB, carotid bruit; PPV, positive predictive value; TIA, transient ischemic attack

68. Perform a focused neurological examination to determine the location of an arterial cerebral occlusion.

<table>
<thead>
<tr>
<th>Middle cerebral artery (MCA)</th>
<th>Posterior cerebral artery (PCA)</th>
<th>Anterior cerebral artery (ACA)</th>
<th>Internal carotid artery (ICA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Infarction middle third of hemisphere: UMN face, arm&gt; leg</td>
<td>o Infarction of thalamus and occipital cortex</td>
<td>o Cortical sensory loss leg only</td>
<td>o Contralateral HCA and MCA signs</td>
</tr>
<tr>
<td>➢ Homonymous hemianopia; aphasia or non-dominant hemisphere signs (depends on side)</td>
<td>o Contralateral sensory loss</td>
<td>o Contralateral weakness of leg and shoulder shrug</td>
<td>o Ipsilateral transient monocular blindness (amaurosis fugax)</td>
</tr>
<tr>
<td>➢ Cortical sensory loss</td>
<td>o Contralateral hemianopia</td>
<td>o Urinary incontinence</td>
<td></td>
</tr>
</tbody>
</table>

➢ Perforating artery

➢ Internal capsule infarction: UMN face UMN arm> leg

➢ Left-sided
  o Aphasia

➢ Right-sided
  o Neglect of left space
  o Lack of awareness of deficit
  o Apathy
  o Impersistence

➢ Basilar artery
  o Bilateral motor weakness
  o Diplopia
  o Ophthalmoplegia

Abbreviation: ACA, anterior cerebral artery; ICA, internal carotid artery; MCA, middle cerebral artery; PCA, posterior cerebral artery; UMN, upper motor neuron lesion

Useful background: Unilateral cerebral hemispheric disease

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arm rolling test</td>
<td>21.7</td>
<td>NS</td>
</tr>
<tr>
<td>Pronator drift</td>
<td>10.3</td>
<td>0.1</td>
</tr>
<tr>
<td>Finger tapping test</td>
<td>6.6</td>
<td>0.3</td>
</tr>
<tr>
<td>Babinski response</td>
<td>19.0</td>
<td>0.6</td>
</tr>
<tr>
<td>Hyperreflexia</td>
<td>5.8</td>
<td>0.4</td>
</tr>
</tbody>
</table>

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

Note that hemianopia and hemisensory disturbance are not included since had a PLR < 2.

Adapted from: McGee S. R. *Saunders/Elsevier* 2007, page 726

Useful background: Cardiac risks for cerebral infarction (CVA) or transient ischemic attack (TIA)

- Proven possible
  - Rhythm
    - Atrial fibrillation
    - Paroxysmal atrial fibrillation
    - Sustained atrial fibrillation
  - Valve defect
    - Mechanical valve
    - Rheumatic valve disease
  - Valve infection
    - Infectious endocarditis
    - Nonbacterial thrombotic endocarditis
  - Lumen
    - Intracardiac thrombus
  - Wall
    - Intracardiac mass (eg. atrial myxoma, papillary fibroelastoma)
    - Dilated cardiomyopathy
- Vessels
Recent (within 1 month) myocardial infarction

Possible cardiac risks

- **Rhythm**
  - Sick sinus syndrome
  - Spontaneous echocardiographic contrast

- **Valve defect**
  - Calcification of mitral annulus

- **Valve infection**

- **Lumen**

- **Wall**
  - Patent foramen ovale with or without atrial septal aneurysm
  - Hypokinetic or a kinetic left ventricular segment

- **Vessels**
  - Atherosclerotic debris in the thoracic aorta
  - Myocardial infarction 2-6 month earlier


Useful background: Subarachnoid hemorrhage

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neck stiffness</td>
<td>10.3</td>
<td>0.4</td>
</tr>
<tr>
<td>Neurological findings not focal</td>
<td>5.9</td>
<td>0.4</td>
</tr>
<tr>
<td>Seizures</td>
<td>2.2</td>
<td>NS</td>
</tr>
</tbody>
</table>

Abbreviation: NS, not significant; NLR, negative likelihood ratio; PLR, positive likelihood ratio

Useful background: The National Institutes of Health Stroke Scale

<table>
<thead>
<tr>
<th>Item</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>1a. Level of consciousness</td>
<td>0=Alert</td>
</tr>
<tr>
<td></td>
<td>1= Not alert</td>
</tr>
<tr>
<td></td>
<td>2= Obtunded</td>
</tr>
<tr>
<td></td>
<td>3= Unresponsive</td>
</tr>
<tr>
<td>1b. Level of consciousness questions</td>
<td>0= Answers both correctly</td>
</tr>
<tr>
<td></td>
<td>1= Answers 1 correctly</td>
</tr>
<tr>
<td></td>
<td>2= Answers neither correctly</td>
</tr>
<tr>
<td>1c. Level of consciousness commands</td>
<td>0= performs both tasks correctly</td>
</tr>
<tr>
<td></td>
<td>1= Performs 1 task correctly</td>
</tr>
<tr>
<td></td>
<td>2= Performs neither task</td>
</tr>
<tr>
<td>2. Gaze</td>
<td>0= Normal</td>
</tr>
<tr>
<td></td>
<td>1= Partial gaze palsy</td>
</tr>
<tr>
<td></td>
<td>2= Total gaze palsy</td>
</tr>
<tr>
<td>3. Visual fields</td>
<td>0= No visual loss</td>
</tr>
<tr>
<td></td>
<td>1= Partial hemianopsia</td>
</tr>
<tr>
<td></td>
<td>2= Complete hemianopsia</td>
</tr>
<tr>
<td></td>
<td>3= Bilateral hemianopsia</td>
</tr>
<tr>
<td>4. Facial palsy</td>
<td>0= Normal</td>
</tr>
<tr>
<td></td>
<td>1= Minor paralysis</td>
</tr>
<tr>
<td></td>
<td>2= Partial paralysis</td>
</tr>
<tr>
<td></td>
<td>3= Complete paralysis</td>
</tr>
<tr>
<td>5. Motor arm</td>
<td>0= No drift</td>
</tr>
<tr>
<td>a. Left</td>
<td>1= Drift before 5 s</td>
</tr>
<tr>
<td>b. Right</td>
<td>2= Falls before 10 s</td>
</tr>
<tr>
<td></td>
<td>3= No effort against gravity</td>
</tr>
<tr>
<td></td>
<td>4= No movement</td>
</tr>
<tr>
<td>6. Motor leg</td>
<td>0= No drift</td>
</tr>
<tr>
<td>a. Left</td>
<td>1= Drift before 5 s</td>
</tr>
<tr>
<td>b. Right</td>
<td>2= Falls before 5 s</td>
</tr>
<tr>
<td></td>
<td>3= No effort against gravity</td>
</tr>
<tr>
<td></td>
<td>4= No movement</td>
</tr>
<tr>
<td>7. Ataxia</td>
<td>0= Absent</td>
</tr>
<tr>
<td></td>
<td>1= One limb</td>
</tr>
<tr>
<td></td>
<td>2= Two limbs</td>
</tr>
<tr>
<td>8. Sensory</td>
<td>0= Normal</td>
</tr>
<tr>
<td></td>
<td>1= Mild loss</td>
</tr>
<tr>
<td></td>
<td>2= Severe loss</td>
</tr>
<tr>
<td>Item</td>
<td>Response</td>
</tr>
<tr>
<td>-----------------------------</td>
<td>-----------------------------------</td>
</tr>
<tr>
<td>9. Language</td>
<td>0= Normal</td>
</tr>
<tr>
<td></td>
<td>1= Mild aphasia</td>
</tr>
<tr>
<td></td>
<td>2= Severe aphasia</td>
</tr>
<tr>
<td></td>
<td>3= Mute or global aphasia</td>
</tr>
<tr>
<td>10. Dysarthria</td>
<td>0= Normal</td>
</tr>
<tr>
<td></td>
<td>1= Mild</td>
</tr>
<tr>
<td></td>
<td>2= Severe</td>
</tr>
<tr>
<td>11. Extinction/inattention</td>
<td>0= Normal</td>
</tr>
<tr>
<td></td>
<td>1= Mild</td>
</tr>
<tr>
<td></td>
<td>2= Severe</td>
</tr>
</tbody>
</table>

*aThe actual form for recording the data contains detailed instructions for the use of the scale. This is available at www.ninds.nih.gov/doctors/NIH_stroke_Scale.pdf.
An online course for provider education is available at www.ninds.nih.gov/doctors/stroke_scale_training.htm.

b Score = sum of scores from each item.


Useful background: Bamford clinical classification of stroke*

- **Total anterior circulation syndrome**
  - Unilateral motor deficit of face, arm and leg
  - Homonymous hemianopia
  - Higher cerebral dysfunction (e.g. aphasia, neglect)

- **Parietal anterior circulation syndrome**
  - Any two of the following features:
    - Unilateral motor and/or sensory deficit
    - Ipsilateral hemianopia or higher cerebra dysfunction
    - Higher cerebral dysfunction alone, or isolated motor and/or sensory deficit restricted to one limb or to the face

- **Posterior circulation syndrome**
  - One or more of the following features:
    - Bilateral motor or sensory signs not secondary to brainstem compression by a large supratentorial lesion
    - Cerebellar signs, unless accompanied by ipsilateral motor deficit (see ataxic hemiparesis)
    - Unequivocal diplopia with or without external ocular muscle palsy
Crossed signs, for example left facial and right limb weakness
- Hemianopia alone or with any of the four items above

- Lacunar syndrome
  - Pure motor stroke:
    - Unilateral, pure motor deficit
    - Clearly involving two of three areas (face, arm and leg)
    - With the whole of any limb being involved
  - Pure sensory stroke:
    - Unilateral pure sensory symptoms (with or without signs)
    - Involving at least two of three areas (face arm and leg)
    - With the whole of any limb being involved
  - Ataxic hemiparesis
    - Ipsilateral cerebellar and corticospinal tract signs
    - With or without dysarthria
    - In the absence of higher cerebral dysfunction or a visual field defect
  - Sensorimotor stroke:
    - Pure motor and pure sensory stroke combined (i.e. unilateral motor or sensory signs and symptoms)
    - In the absence of higher cerebral dysfunction or a visual field defect

Stroke is characterized by rapidly progressive clinical symptoms and signs of focal, and at times global, loss of cerebral function lasting more than 24 hours or leading to death, with no apparent cause other than that of vascular origin.


69. Perform a focused physical examination to determine if a lesion affects functions of the dominant cerebral hemisphere.

- Dominant hemisphere
  - Right-left orientation
  - Finger identification
  - Calculation

- Non-dominant hemisphere
  - Facial recognition
  - Awareness of body and space
  - Drawing ability
  - Topographic ability
  - Construction
  - Dressing
  - Motor persistence

70. Perform a focused physical examination to determine the presence of parietal lobe dysfunction.

- Loss of accurate localization (of touch, position, joint sense and temperature appreciation)
- Loss of two-point discrimination
- Astereognosis
- Dysgraphethesia
- Sensory inattention
- Attention hemianopia, homonymous hemianopia, or lower quadrantic hemianopia


71. Take a directed history to differentiate between a carotid or vertebrobasilar transient ischemic attack (TIA).

- Carotid TIA
  - Hemiparesis
  - Aphasia or transient loss of vision in only one eye (amaurosis fugax)

- Vertebrobasilar TIA
  - Vertigo, dysphagia, ataxia, drop attacks (at least two of these should occur together)
  - Bilateral or alternating weakness or sensory symptoms
  - Sudden bilateral blindness in patients aged over 40 years

### Clinical gem: Transient Ischemic Attack (TIA)

- A TIA is a stroke syndrome with neurological symptoms lasting from a few minutes to as long as 24 hours followed by complete functional recovery. A RIND (reversible ischemic neurological deficit) is a condition in which a person has neurological abnormalities similar to acute completed stroke, but the deficit disappears after 14 to 36 hours, leaving few or no detectable neurological sequelae.

- The symptoms include:
  - Intention tremor (29%)
  - Hypotonia
  - Dysdiadochokinesia (47-69%)
  - Arm drift (44-69%)
  - Rebound
  - Balance
    - Rhomberg’s test, Pull test
  - Gait- Normal gait, Toe walking, Heel walking, Tandem gait, Ataxia
  - Reflexes reduced

### Useful background: Oxfordshire classification of subtypes of cerebral infarction

- **Total anterior circulation infarction syndrome (TACS)**
- **IC/MCA-** A combination of new higher cerebral dysfunction (ie dysphasia, dyscalculia, visuospatial disorder); homonymous visual field defect; and ipsilateral motor or sensory deficit of at least 2 areas of the face, arm and leg.
- **Partial anterior circulation infarction syndrome (PACS)**
- **MCA-** Only 2 of the 3 components of the TACS syndrome are present with higher cerebral dysfunction alone or with a motor/sensory deficit more restricted than those classified as LACS (ie confined to 1 limb or to face and hand, but not to the whole arm).
- **Lacunar infarction syndrome (LACS)**
- **Penetids-** Pure motor stroke, pure sensory stroke, sensorimotor stroke, or ataxic hemiparesis.
- **Posterior circulation infarction syndrome (POCS)**
Ventrilosis basilar- Any of the following; ipsilateral cranial nerve palsy with contralateral motor or sensory deficit; bilateral motor or sensory deficit; disorder of conjugate eye movement; cerebellar dysfunction without ipsilateral long tract deficit (ie ataxic hemiparesis); or isolated homonymous visual field defect.

Oxfordshire classification, as published in Simel David L, Et al. JAMA 2009, Box 48-1, page 634.

Useful background: Likelihood ratios for stroke (in the absence of head trauma) from summing combinations of findings (Cincinnati prehospital stroke scale)\(^a\)

<table>
<thead>
<tr>
<th>Combination of findings</th>
<th>Findings present</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Facial paresis</td>
<td>3 present</td>
<td>14</td>
</tr>
<tr>
<td>➢ Arm drift</td>
<td>2 present</td>
<td>4.2</td>
</tr>
<tr>
<td>➢ Abnormal speech</td>
<td>1 present</td>
<td>5.2</td>
</tr>
<tr>
<td></td>
<td>0 present</td>
<td>0.4</td>
</tr>
</tbody>
</table>

Hospital evaluation

| ➢ Persistent neurologic deficit | 4 present    | 40  |
| ➢ Focal neurologic deficit     | 1-3 present  | Uncertain LR, but probability of stroke \(\geq10\%\) |
| ➢ Acute onset of symptoms during the previous week | 0 present | 0.14 |

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

# Useful background: Diagnostic approach to upper motor neuron weakness

<table>
<thead>
<tr>
<th>Distribution of Weakness</th>
<th>Diagnostic Possibilities</th>
<th>Additional Finding</th>
<th>Location of lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Left monoparesis</td>
<td>➢ Right cerebral hemisphere o Right brainstem o Left spinal cord</td>
<td>➢ New seizures</td>
<td>➢ Right cerebral hemisphere</td>
</tr>
<tr>
<td>➢ Right hemiparesis</td>
<td>➢ Left cerebral hemisphere o Left brainstem o Right spinal cord</td>
<td>➢ Aphasia - Right homonymous hemianopia - Left sixth nerve palsy - Loss of sensation left arm and leg; face spared</td>
<td>➢ Left cerebral hemisphere - Left cerebral hemisphere - Left brainstem - Right spinal cord</td>
</tr>
<tr>
<td>➢ Paraparesis</td>
<td>➢ Bilateral lesion of thoracic cord or above</td>
<td>➢ Sensory level at midchest; normal arm strength and reflexes o Spine tenderness between scapulae</td>
<td>➢ Bilateral lesion, thoracic cord</td>
</tr>
<tr>
<td>➢ Tetraparesis</td>
<td>➢ Bilateral lesion of cervical cord or above</td>
<td>➢ Hyperactive jaw jerk o Dementia o Sensory level upper chest o Absent biceps reflexes but hyperactive triceps reflexes</td>
<td>➢ Bilateral lesion, cerebral hemispheres - Bilateral lesion, cervical cord</td>
</tr>
</tbody>
</table>

Useful background: Localizing signs in upper motor neuron weakness

<table>
<thead>
<tr>
<th>Anatomic location</th>
<th>Associated finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Cerebral hemisphere</td>
<td>o Seizures</td>
</tr>
<tr>
<td></td>
<td>o Hemianopia</td>
</tr>
<tr>
<td></td>
<td>o Aphasia (right hemiparesis)</td>
</tr>
<tr>
<td></td>
<td>o Inattention to left body, apraxia (left hemiparesis)</td>
</tr>
<tr>
<td></td>
<td>o Cortical sensory loss*</td>
</tr>
<tr>
<td></td>
<td>o Hyperactive jaw jerk</td>
</tr>
<tr>
<td>➢ Brainstem</td>
<td>o Crossed motor findings†</td>
</tr>
<tr>
<td></td>
<td>o Contralateral third nerve palsy (midbrain)</td>
</tr>
<tr>
<td></td>
<td>o Contralateral sixth nerve palsy (pons)</td>
</tr>
<tr>
<td></td>
<td>o Sensory loss on contralateral face*</td>
</tr>
<tr>
<td>➢ Spinal cord</td>
<td>o Sensory level†</td>
</tr>
<tr>
<td></td>
<td>o Pain and temperature sensory loss on contralateral arm and leg†</td>
</tr>
<tr>
<td></td>
<td>o No sensory or motor findings in face</td>
</tr>
<tr>
<td></td>
<td>o Additional lower motor neuron findings (atrophy, fasciculations)</td>
</tr>
</tbody>
</table>

† crossed motor findings refers to unilateral cranial nerve palsy opposite the side of limb weakness.


72. Perform a focused physical examination to determine the location of lesions causing sensory loss.

<table>
<thead>
<tr>
<th>Location of lesion</th>
<th>Distribution of sensory loss</th>
<th>Examples of causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Cortical (parietal)</td>
<td>o Able to recognize all primary modalities but localizes them poorly</td>
<td>- Stroke, cerebral tumour, trauma</td>
</tr>
<tr>
<td></td>
<td>o Loss of secondary modalities</td>
<td></td>
</tr>
<tr>
<td>➢ Brainstem</td>
<td>o Pain and temperature: ipsilateral face, contralateral body</td>
<td>- Demyelination (young), brainstem stroke (older)</td>
</tr>
</tbody>
</table>
- Thalamic sensory loss
  - All modalities; contralateral hemisensory loss (face, body) and pain –dysesthesia (e.g. burning feeling)
  - Stroke, cerebral tumour, MS, trauma

- Spinal cord
  - Depends on level of lesion and complete vs. partial lesion
  - Trauma, spinal cord compression by tumour, cervical spondylitis, MS

- Medulla involving descending nucleus of spinal tract of the fifth nerve and ascending spinothalamic tract (lateral medullary lesion)

- Root or roots
  - Confined to single root or roots in close proximity; commonly C5, 6,7 in arm and L4, 5, S1 in leg
  - Compression by disc prolapse

- Peripheral nerve
  - Distal glove and stocking deficit
  - Diabetes mellitus, alcohol related B12 deficiency, drugs

- Single nerve
  - Within distribution of single nerve; commonly median, ulnar, peroneal, lateral cutaneous nerve to the thigh
  - Entrapment, most commonly in diabetes mellitus, carpal tunnel syndrome, rheumatoid arthritis, and hypothyroidism; multiple (mononeuritis multiplex) ; vasculitis

- Multiple nerves
  - Mononeuritis multiplex, from involvement of multiple nerves
  - Vasculitis

Abbreviations: MS, multiple sclerosis

Adapted from: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto 2005, page 168.
**Coma, delirium, dementia, confusion**

Useful background: Assessment of confusion

- Acute onset with fluctuating course
- Inattention
- Disorganized thinking
- Altered level of consciousness

<table>
<thead>
<tr>
<th>Function</th>
<th>Normal</th>
<th>Diencephalon</th>
<th>Midbrain</th>
<th>Pons</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiration</td>
<td>Normal</td>
<td>Cheyne-Stokes</td>
<td>Regular, hyperventilation</td>
<td>Irregular, erratic</td>
</tr>
<tr>
<td>Pupil size and response to light</td>
<td>Normal</td>
<td>Small, Reactive</td>
<td>Mid Position, fixed</td>
<td>Pinpoint Reactive</td>
</tr>
<tr>
<td>Oculo-vestibular reflex</td>
<td>Suppressed</td>
<td>Constant (tonic) deviation</td>
<td>Dysconjugate gaze</td>
<td>No response</td>
</tr>
<tr>
<td>Motor response to pain</td>
<td>Appropriate</td>
<td>Decorticate (arms flexor response)</td>
<td>Decerebrate (all extensor response)</td>
<td>No response</td>
</tr>
</tbody>
</table>

Reproduced with the permission of Dr. B. Fisher, (U of Alberta)
Useful background: Mini – mental test for confusion

<table>
<thead>
<tr>
<th>Acute confusional states and coma</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Altered/ fluctuating conscious level</td>
<td>Confusion +/-</td>
</tr>
</tbody>
</table>

**Mini mental test score – continuum**

**Test for confusion**

- **Orientation**
  - Person (1)
  - Time (2)
  - Place (3)

- **Memory**
  - Short term
    - Recent events (4)
    - Memory test (5)
  - Long term
    - Prime minister or king/queen (6)
    - Dates of World war II (7)

- **Attention**
  - Subtract from 100 (8)

Marks out of 10


What is “the best”? The two “best” clinical tests for the presence of a subarachnoid hemorrhage are: neck stiffness, and neurological findings which are not focal. Recall that neck stiffness is also common in persons with meningitis, and they will much more often have fever.
73. Perform a focused physical examination for coma.

- Determine Glasgow coma scale

<table>
<thead>
<tr>
<th>Response</th>
<th>Score</th>
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</thead>
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<tr>
<td><strong>Eye opening</strong></td>
<td></td>
</tr>
<tr>
<td>o Spontaneous</td>
<td>4</td>
</tr>
<tr>
<td>o On your verbal command (‘open your eyes’)</td>
<td>3</td>
</tr>
<tr>
<td>o In response to painful stimulus</td>
<td>2</td>
</tr>
<tr>
<td>o No response</td>
<td>1</td>
</tr>
<tr>
<td><strong>Motor response</strong></td>
<td></td>
</tr>
<tr>
<td>o Correct response to ‘show me two fingers’</td>
<td>6</td>
</tr>
<tr>
<td>o Localises painful stimulus and tries to stop it</td>
<td>5</td>
</tr>
<tr>
<td>o Withdraws from painful stimulus to fingernail</td>
<td>4</td>
</tr>
<tr>
<td>o Abnormal flexor response of forearms, wrists and fingers</td>
<td>3</td>
</tr>
<tr>
<td>o Abnormal extensor response of arms and legs</td>
<td>1</td>
</tr>
<tr>
<td>o No response</td>
<td></td>
</tr>
<tr>
<td><strong>Verbal response to the question: What year is this?</strong></td>
<td></td>
</tr>
<tr>
<td>o Correct year</td>
<td>4</td>
</tr>
<tr>
<td>o Wrong year</td>
<td>3</td>
</tr>
<tr>
<td>o Words but no year</td>
<td>2</td>
</tr>
<tr>
<td>o Incomprehensible sounds</td>
<td>1</td>
</tr>
<tr>
<td>o No response</td>
<td></td>
</tr>
<tr>
<td><strong>Total Points</strong></td>
<td></td>
</tr>
<tr>
<td>Glasgow Coma Scale points</td>
<td></td>
</tr>
<tr>
<td>14 - 15 = 5</td>
<td></td>
</tr>
<tr>
<td>11 - 13 = 4</td>
<td></td>
</tr>
<tr>
<td>8 - 10 = 3</td>
<td></td>
</tr>
<tr>
<td>5 - 7 = 2</td>
<td></td>
</tr>
<tr>
<td>3 - 4 = 1</td>
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74. Take a directed history to detect disease of the frontal, parietal or temporal lobe, or the motor cortex.

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<th>Motor cortex</th>
<th>Parietal lobe</th>
<th>Temporal lobe</th>
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<td>➢ Forethought</td>
<td>o UMN hemiplegia</td>
<td>o Spatial disorientation</td>
<td>o Hallucinations</td>
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<tr>
<td>➢ Consequences</td>
<td></td>
<td></td>
<td>o Illusions</td>
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<tr>
<td>➢ Apathy</td>
<td>o Jacksonian epilepsy</td>
<td>o Aproxia</td>
<td>o Receptive dysphasia</td>
</tr>
<tr>
<td>➢ Dementia</td>
<td></td>
<td>o Agnesia</td>
<td>o Altered memory, coma,</td>
</tr>
<tr>
<td>➢ Grasp reflex</td>
<td>o Expressive dysphasia</td>
<td>o Perceptual rivalry</td>
<td>o Upper temporal quadrantanopia</td>
</tr>
<tr>
<td>➢ Ataxia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>➢ Akinesia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>➢ Aspasia</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviation: UMN, upper motor neuron


75. Take a directed history for delirium.

➢ **Definition**
  - Acute onset, with fluctuating course
  - Inattention
  - Disorganised thinking
  - Altered level of consciousness
  - Diagnosis of delirium requires the presence of 1 and 2 and either 3 or 4.

➢ **Clinical**
  - Initial screen
    - Assesses hearing/vision
    - Assesses orientation (person, place, time)
    - Elicits chief complaint
  - Description of symptoms
    - Onset, duration, and course of current complaint(s)
    - Palliating/provoking factors
    - Limitations in functioning (ADLs, IADLs)
  - Depression symptoms
    - Assesses depression symptoms (low mood, anhedonia, sleep disturbance, etc)
    - Assesses suicidality and homicidality
- Anxiety symptoms
  - Anxiety symptoms (phobias, obsessions, compulsions, etc)
- Perception disturbances
  - Psychotic symptoms (hallucinations, delusions, ideas of reference etc)
- Personality and behavioral disturbances
  - Changes in personality
  - Behavioral abnormalities (apathy, agitation, odd behaviors, etc)
- Past and family medical history
  - Hx of alcohol/drug abuse
  - Medications and Hx of adverse drug reactions
  - Hx of psychiatric illness
  - Hx of other metabolic or systemic illness(s)
- Collateral history from family member
  - Elicits concerns
  - Confirms history
  - Inquiries about safety, home fire risks, driving, wandering

- Causes
  - Drugs
    - Sedative- hypnotics
    - Anticholinergic agents
    - NSAIDs (nonsteroidal anti inflammatory drug)
    - Adrenergic blockers
    - Antipsychotic agents
  - Metabolic disturbances
    - Hyperglycemia
    - Hypoglycemia
    - Hypercalcemia
  - Hypoxia
  - Hypotension

76. Perform a focused physical examination to distinguish between meningeal irritation, versus a lesion of the sciatic nerve or its spinal roots (Lasègue’s sign).

- To detect meningeal irritation
  - Kernig’s sign
    - Straightening leg with hip flexed produces pain and spasms of hamstrings

- To detect lesions of sciatic nerve or its spinal roots
  - Straight-leg raising test produces pain below the normal full excursion

Meningitis and subarachnoid hemorrhage

77. Perform a focused physical examination for meningitis (the numbers in brackets represent valves for sensitivity)

➢ General
  o Jolt accentuation of headache
  o Myalgia
  o Fever (87%)
  o Altered mental status (69%)

➢ Eye
  o Papilledema

➢ CNS
  o Focal neurological signs (9%) (eg, cranial nerve palsy; 21%)
  o Seizures (13%)
  o Subarachnoid hemorrhage
  o Acute bacterial meningitis
  o Cervical fusion
  o Spondylitis
  o Parkinson’s disease
  o Increased intracranial pressure (with impending tonsillar herniation)

➢ Meningeal signs
  o Stiff neck with passive motion (80%)
  o Chin toward chest
  o Kernig’s sign, Brudzinski’s sign (61%)

➢ Skin
  o Petechial rash (13%)

➢ CVS
  o ↓ PR
  o ↑ SBP

➢ Differential

Note: the sensitivity is 46% for the classic triad (fever, neck stiffness, headache), also note that fever and altered mental status are less frequent with subarachnoid hemorrhage than with acute bacterial meningitis.

Abbreviation: PR, pulse rate; SBP, systolic blood pressure

Differentiate between four similar signs

1. Kernig’s sign. To detect meningeal irritation

   ![Diagram of Kernig's sign]

   Straightening leg with hip flexed produces pain and spasm of hamstrings

2. Brudzinski’s sign. To detect meningeal irritation

   ![Diagram of Brudzinski's sign]

   Flexing neck produces flexion of lower limbs

3. Thomas’s test. To detect fixed flexion deformity of the hip – joint

   ![Diagram of Thomas's test]

   Eliminating lumbar lordosis produces flexion of the affected hip

4. Straight-leg raising test. To detect lesions of sciatic nerve or its spinal roots

   ![Diagram of Straight-leg raising test]

   Straight-leg raising produces pain below the normal full excursion


---

SO YOU WANT TO BE A NEUROLOGIST!

Q: In the person with neurofibromatosis, what is a Lisch nodule?
A: Lisch nodules are melanocytic hamartomas, well-defined, dome-shaped elevations projecting from the surface of the iris and are clear to yellow and brown.
**Syncope and dizziness**

Useful background: Major causes of syncope

- **Definition:** Syncope is a transient loss of consciousness with spontaneous recovery.

- **Cardiogenic syncope**
  - Structural heart disease
  - Coronary artery disease
  - Rhythm disturbance
  - Vasovagal
  - Carotid sinus hypersensitivity
  - Dysrhythmia e.g. AF, BBB
  - Orthostatic hypotension
  - After exercise
  - Coronary artery disease, previous myocardial infarction
  - Structural heart disease
  - Left ventricular dysfunction
  - Congestive heart failure

- **Neurologic**
  - In patients who present with a prodrome (e.g. nausea, diaphoresis), a neurocardiogenic mechanism is likely.
  - Patients who experience rapid recovery (less than 5-10 minutes) rarely have neurologic cause for syncope and are most unlikely to have syncope due to seizure or 'brain hypoperfusion' because recovery in such circumstances takes hours.
  - For cases in which recovery from syncope is rapid and no residual neurologic signs or symptoms are present, detailed (and expensive) neurologic evaluation should be avoided.

- **Metabolic**
- **Psychiatric**
- **Situational**
  - **Lung**
    - Tussive
    - Valsalva manoeuvre
    - Sneeze
  - **GI**
    - Deglutition
    - Defecation
    - Glossopharyngeal neuralgia
    - Postprandial
  - **GU**
    - Micurition
Miscellaneous
  o Oculovagal
  o Instrumentation
  o Diving


78. Take a directed history to determine the cause of a patient’s dizziness.

Physiological
  o Motion sickness
  o Space sickness
  o Height vertigo

Psychological
  o Acute anxiety
  o Agoraphobia (fear & avoidance of being in public places)
  o Chronic anxiety

Eye
  o High magnification & lens implant
  o Imbalance in extraocular muscles
  o Oscillopsia

Balance
  o Brain stem, cerebellar or temporal cortical lesions
    - Pontine infraction or haemorrhage
    - Vertebro-basilar insufficiency
    - Basilar artery migraine
    - Temporal lobe epilepsy
    - Disseminated sclerosis
    - Tumours
    - ‘Benign post traumatic positional vertigo’, etc
  o Vestibular lesions
    - Physiological
    - Labyrinthitis
    - Meniere’s
    - Drugs eg quinine, salicylates, alcohol
    - Otitis media
    - Motion sickness
  o Vestibular nerve lesions
    - Acoustic neuroma
    - Drugs eg streptomycin
    - Vestibular neuritis
- Disequilibrium
  - Lesions of basal ganglia, frontal lobes, & white matter
  - Hydrocephalus
  - Cerebellar dysfunction
- Ear
  - Vertigo
    - Peripheral
    - Central

- CVS
  - Orthostatic hypotension
  - Vasovagal attacks
  - Impaired cardiac output
  - Hyperventilation

- Multisensory dizziness

Multiple sclerosis

Useful Background: Multiple sclerosis

- Intellectual loss ("dementia") in long-standing MS
- **Eye**
  - **Optic neuritis**
    - Acute phase
      - Central visual field defect
      - "Scotoma" – ‘like cotton wool’
      - Discomfort – worse on eye movement
      - Often normal fundoscopy
      - Usually recovers in 10-20 days
    - Chronic phase
      - Fundoscopy shows optic atrophy i.e. very pale disc
      - Visual loss often minor i.e. colour vision
- **Homer syndrome**

- **Motor weakness**
  - Due to pyramidal tract damage (in spinal cord or higher)
  - Arm extension
  - Leg flexion
  - Spasticity i.e. "clasp knife" pattern
  - Increased reflexes ± clonus
  - Upgoing plantar
  - Wasting

- **Cerebellar signs**
  - Axial FLAIR showing MS lesions

- **Brain stem involvement**
  - Dysconjugate eye gaze due to internuclear ophthalmoplegia
  - Trigeminal neuralgia – like syndrome
  - Recurrent facial nerve palsy

- **Spinal cord**
  - Gradual onset spastic para – or tetraparesis
  - Acute 'transverse myelitis' – leads to flaccid paralysis in acute phase, spasticity in chronic phase
  - 'Sensation'
    - Joint position
    - Pain/temperature
  - Dorsal column damage → abnormal gait (sensory ataxia) due to loss of position sense
  - Lhermitte phenomena bending neck forward → electric shock passing along spine

- **Sensory loss**
  - Difficult to describe – anesthesia or paresthesia (i.e. altered sensation)
  - If isolated symptom, differential diagnosis is hyperventilation, or peripheral neuropathy
  - Can occur anywhere in the body


- **Definition**
  - Demyelination causing remissions and relapses of: weakness, incoordination, pain, paraesthesias, urinary urgency, impotence
  - Steinberg's triad: history of incontinence of bladder, impotence and constipation
Diagnostic criteria
- Poser’s criteria: a history of two episodes of neurological deficit and objective clinical signs of lesions at more than one site within the central nervous system establishes the diagnosis of definite multiple sclerosis
- In the presence of only one clinical sign, the demonstration of an additional lesion by laboratory tests – such as evoked potentials, MRI, CT or urological studies – also fulfills the criteria
- A diagnosis of probable multiple sclerosis is defined as either two attacks with clinical evidence of one lesion, or one attack with clinical evidence of two lesions

Clinical course
- Relapsing-remitting: episodes of acute worsening with recovery and a stable course between relapses.
- Secondary progressive: gradual neurological deterioration with or without superimposed acute relapse in a patient who previously had relapsing-remitting multiple sclerosis.
- Primary progressive: gradual, almost continuous neurological deterioration from the onset of symptoms
- Progressive relapsing: gradual neurological deterioration from the onset symptoms but with subsequent superimposed relapses
- Variation of MS
  - Optic neuritis alone
  - Optic neuritis plus a single episode of transverse myelitis


**Neurofibromatosis**

Useful background: Causes of benign intracranial hypertension (Pseudotumor celebri)

- **CNS**
  - Addison’s
  - Head injury
  - Sagital sinus thrombosis

- **Endocrine**
  - Hypoparathyroidism
  - Obesity

- **Blood**
  - Anemia
  - Polycythemia
Drugs
- Chlortetracycline, nalixide acid, oral contraceptive agents
- Change in steroid dosage

GU
- Pregnancy
- Menarche


**Temporal arteritis**

Useful background: Temporal arteritis

- Temporal artery
  - Tender, non-pulsatile temporal arteries

- Eyes
  - Blindness
  - Diplopia (3.5)

- Jaw
  - Jaw claudication (4.3)

- Sometimes proximal muscle tenderness

- General
  - Malaise
  - Fever
  - Appears "depressed"

- Muscle
  - Beaded temporal artery (4.6)
  - Prominent or enlarged temporal artery (4.3)
  - Absent temporal artery pulse (2.7)
  - Tender temporal artery (2.6)
  - Any temporal artery abnormal (2.0)

Note: the numbers show in brackets represent the values of the positive likelihood ratios (PLR). Note that value of the PLR is ≤ 2 for scalp tenderness, optic atrophy, ischemic neuropathy, signs of anemia, or any valve of ↑ ESR.

CT of head

- Circumstances when computed tomography (CT) or magnetic resonance imaging (MRI) are preferred for neurologic imaging

<table>
<thead>
<tr>
<th>CT</th>
<th>MRI</th>
</tr>
</thead>
<tbody>
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<td>Suspected acute hemorrhage</td>
<td>Subacute &amp; chronic hemorrhage</td>
</tr>
<tr>
<td>Skull fractures</td>
<td>Ischemic stroke</td>
</tr>
<tr>
<td>Meningiomas</td>
<td>Posterior fossa &amp; brainstem tumor &amp; lesion</td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td>Diagnosis of multiple sclerosis</td>
</tr>
<tr>
<td></td>
<td>Evaluation of spinal cord</td>
</tr>
</tbody>
</table>

Abbreviation: SSRI, selective serotonin reuptake inhibitor


Miscellaneous

Useful background: Neurological complications of diabetes mellitus

- Cerebellum
  - Cerebral disturbance due to hyper- or hypo glycaemia
- Cranial nerves
  - Isolated cranial nerve lesions
- Spinal column
  - Diabetic pseudotabes
  - Diabetic amyotrophy
- Peripheral nerves
  - Asymptomatic loss of ankle jerks and vibration sense, with decreased motor conduction-velocity
  - Painful subacute neuritis, usually in lower limbs
  - Mononeuritis multiplex
  - ‘Insulin neuritis’ during stabilization
- Autonomic
  - Visceral disturbances due to autonomic involvement
- Miscellaneous/ mixed
  - Mixed syndromes

Adapted from: Burton J.L. *Churchill Livingstone* 1971.
Useful background: Characteristics of paraneoplastic neurological degeneration associated tumours.

- Tumours are often difficult to detect.
- Tumours are histologically identical to tumours that develop in patients without paraneoplastic neurological degeneration (PND), except that many tumours have evidence of immune infiltration. (immunogenic tumors)
- Patients often have improved prognosis relative to those with comparable but non-immunogenic tumours (anti-Hu paraneoplastic syndrome, Lambert-Eaton myasthenic syndrome and some paraneoplastic cerebellar degeneration).
- Spontaneous regression is rare.
- Presence of antitumour immune response predicts improved prognosis (Hu syndrome)
- Tumours are associated with circulating PND-antigen-specific killer T-cells (paraneoplastic cerebellar degeneration).


---

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

**Q.** A patient is thought to possibly have Parkinsonism, but striatonigral degeneration, Shy-Drager syndrome, and olivopontocerebellar atrophy needs to be excluded. Perform a focused physical examination to diagnose the patient’s type of multisystem atrophy.

**A.**

- **Striatonigral degeneration**
  - Resembles Parkinson’s disease, but without tremor
  - Does not respond to anti-Parkinson medications

- **Shy-Drager syndrome:**
  - Parkinsons disease plus autonomic neuropathy (especially postural hypertension)
  - Impotence
  - Bladder disturbances

- **Olivopontocerebellar atrophy**
  - Extrapyramidal signs plus cerebellar ataxia
  - Autonomic neuropathy plus anterior horn cell degeneration

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OSCE Questions in Respirology Chapter

1. Perform a directed physical examination for asymmetry in the expansion of the chest
2. Perform a directed physical examination of the pulmonary system for tracheal deviation
3. Take a directed history for cough.
4. Take a directed history of hemoptysis
5. Perform a directed physical examination of the pulmonary system for consolidation, collapse, effusion, or fibrosis.
6. Perform a focused physical examination to distinguish between the major causes of dullness at a lung base.
7. Perform a directed physical examination for clubbing.
8. Perform a directed physical examination for sarcoidosis.
9. Causes of slow resolution or recurrence of pneumonia
10. Take a directed history for asthma.
11. Perform a focused physical examination for asthma.
12. Take a focused history and perform a directed physical examination for chronic bronchitis.
13. Take a directed history to differentiate between bronchial asthma, chronic bronchitis, and emphysema.
14. Take a directed history for the harmful effects of cigarette smoking
15. Take a focused history and perform a directed physical examination for bronchiectasis.
16. Perform a directed physical examination of the pulmonary system in the patient with suspected mediastinal compression (e.g. carcinoma of the lung).
17. Perform a directed physical examination for Pancoast’s (superior pulmonary sulcus tumor) syndrome (often from cancer [often non-small cell] of the apex of the lung, infiltrating C8, T1, 2; may also occur with lymphoma, or by spread of lymph node metastases in breast or lung cancer).
18. Take a directed history and perform a focused physical examination for pulmonary hypertension.
19. Perform a focused physical examination for acute respiratory distress syndrome (ARDS).
20. Take a directed history and perform a focused physical examination to determine the possible presence of a deep vein thrombosis (DVT).

21. Take a focused history for the causes of pneumothorax.

22. Take a focused history for the causes of lung abscess.

23. Take a directed history and perform a focused physical examination for fibrosing alveolitis.
Commonly used terms

- **Pectus carinatum**
  - Pigeon chest

- **Pectus excavatum**
  - Funnel test

- **Adventitial sounds**
  - Continuous “wheeze”; discontinuous “crackles”

- **Inspiratory wheeze**
  - Severe airway narrowing

- **Stridor**
  - Foreign body tumour
    - Gas inhalation
    - Anaphylaxis
    - Epiglottitis
    - Bilateral vocal cord palsy

- **Bronchial breath sounds**
  - Normal
  - Pneumonia, fibrosis, effusion, collapse

- **Broadbent’s sign**
  - Systolic retraction of intercostals space, especially seen at the post auxiliary line just below the angle of the scapula, with cardiac hypertrophy or fibrous pericarditis

- **Pulsus alternans**
  - Strong/weak pulse in CHF

- **Sinus arrhythmia**
  - ↑HR with inspiration

- **Pulsus paradoxicus**
  - The normal ↑height of pulse wave with inspiration is lost

- **Kröning’s isthmus**
  - A posterior band of resonance 2 inches wide across the shoulder from the apex of the lung, which extends 1 inch above the clavicles.

- **Distinguish pulmonary rub versus rales**
Present in both phases of respiration, but rub is enhanced by increasing the pressure on the stethoscope (closer to ear), and a rub is not affected by coughing

- **Pectoriloquy**
  - ↑Intensity of spoken voice
  - Bronchophony - ↑clarity of the spoken voice

- **Pursed-lip breathing**
  - Pursed lips increase positive pressure in the weakened bronchial airways in emphysema and COPD, thereby helping to prevent the airway trapping

- **Kussmaul breathing**
  - Rapid, deep breathing due to metabolic-diabetic ketoacidosis, lactic acidosis, uremia
  - Drugs and poisons – methanol, ethylene glycol, ASA, paraldehyde

- **When is orthopnea due to lung disease**
  - Dyspnea relieved by sitting up is usually due to L-CHF, but may rarely occur with bilateral bullous apical disease, when sitting up improves the ventilation/perfusion matching and gas exchange in the normal lower lung

- **Horner’s syndrome** - apical lung tumour compressing sympathetic nerves in neck, causing myosis, ptosis and anhydrosis

- **What are you looking for when you are examining accessory muscles?**
  - Motion in scalene muscles (earliest affected), sternocleidomastoid muscles, neck muscles, indrawing of intercostal spaces and supraclavicular fossa
  - Abdominal motion when person inspires.

  - **Paradoxical respiration.**
    - The abdomen draws inward on inspiration when it normally should move outward due to diaphragm descent

  - **Position of trachea**
    - Palpate the trachea in the suprasternal notch to determine if it is midline
    - Trachea is deviated to *ipsilateral* side in atelectasis, fibrosis, lung collapse
Chest inspection

Useful background: Abnormalities in rhythm of respiration.

- Abnormal rhythms of respiration usually result from lesions in the neurogenic control of the respiratory pump.
- They help to localize the site of neurologic lesions. Abnormalities of respiratory rhythm are found in the following sequence from the uppermost to the lower-most neurologic center.
  - Cheyne-Stokes respiration
  - Biot’s respiration
  - Apneustic breathing
  - Central hyperventilation
  - Ataxic (agonal) respiration

Adapted from: Mangione S. Hanley & Belfus 2000, page 279.

SO YOU WANT TO BE A RESPIROLOGIST!

Q: What is respiratory alternans (aka paradoxical respiration, or abdominal paradox)?
A: Normally with inspiration both chest and abdominal wall rises. With muscular weakness and fatigue, the abdominal wall does not rise.
**Inspection: chest asymmetry**

1. Perform a directed physical examination for asymmetry in the expansion of the chest.

   - **Inspection**
     - Instruct patient to inhale deeply while watching for slow expansion of one hemithorax
     - Pigeon chest, funnel chest
     - Lordosis, kyphosis, gibbus (extreme kyphosis, aka hunchback)

   - **Palpation**

   - **Causes**
     - Atelectasis
     - Pleural effusion
     - Severe pneumonia

   - **Complications**
     - Cyanosis, chibbing
     - Asterixis (hypercapnea)

Adapted from: Mangione S. *Hanley & Belfus* 2000, pages 281-282.

**Useful background:**

- **Causes of Platypnea** (preference for the breathing in a lying position; caused by a bilateral pulmonary process)
  - Recurrent pulmonary emboli
  - Pleural effusion
  - Bibasilar pneumonia
  - Bibasilar AV shunts
  - ASD

- **Tachypnea**, increased rate of breathing (>20/min)
  - If present – suggests cardiopulmonary disease (CPD)
  - If absent – argues strongly against CPD

- **Hyperpnea** (increase rate and depth [tidal volume] of breathing)
Finding in anion – gap metabolic acidosis ( "MAKE UP a List")

- Methanol
- Uremia
- Aspirin
- Paraldehyde
- Ketoacidosis
- Ethylene Glycol ingested
- Lactoacidosis


Useful background: Types of positional dyspnea (SoB)

<table>
<thead>
<tr>
<th>Type</th>
<th>Possible causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orthopnea (SoB when lying horizontal)</td>
<td>o Congestive heart failure</td>
</tr>
<tr>
<td></td>
<td>o Mitral valvular disease</td>
</tr>
<tr>
<td></td>
<td>o Severe asthma (rarely)</td>
</tr>
<tr>
<td></td>
<td>o COPD (rarely)</td>
</tr>
<tr>
<td></td>
<td>o Neurological diseases (rarely)</td>
</tr>
<tr>
<td>Trepopnea (SoB when lying on one side)</td>
<td>o Congestive heart failure</td>
</tr>
<tr>
<td>Platypnea (SoB when seated)</td>
<td>o Status post pneumonectomy</td>
</tr>
<tr>
<td></td>
<td>o Neurological diseases</td>
</tr>
<tr>
<td></td>
<td>o Cirrhosis (intrapulmonary shunts)</td>
</tr>
<tr>
<td></td>
<td>o Hypovolemia</td>
</tr>
</tbody>
</table>

Source: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto 2005, page 282.

SO YOU WANT TO BE A RESPIROLOGIST!

Q1: What is Behçet's syndrome?
A1: Aphthous ulcers in mouth and genitals, associated with arthritis, uvertis and various neurological disorders

SO YOU WANT TO BE A RESPIROLOGIST!

Q1: In the persons with smoker’s face and nicotine staining of fingers, pursed lips and using the accessory muscles of expiration (intercostals muscles), what is Dahl’s sign?

A1: Patches of hyperpigmented cullses above both knees from chronic pressure of the elbows on the skin of the legs resulting from sitting up and leaning forward to breath better (orthopnea), placing the elbows near the knees and fixing the position of the shoulder and the neck muscles to improve the contractility of the accessory muscles and improving basilar perfusion and lung mechanics.


**Breath sounds**

Useful background: normal auscultatory breath sounds

<table>
<thead>
<tr>
<th>Tubular (tracheal/bronchial) muffled</th>
<th>Bronchovesicular</th>
<th>Vesicular (soft/muffled)</th>
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<tr>
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<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Tracheal</th>
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<th>Vesicular</th>
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<tbody>
<tr>
<td>Description</td>
<td>Harsh</td>
<td>Air rushing through tube</td>
<td>Rustling, but tubular</td>
<td>Gentle rustling</td>
</tr>
<tr>
<td>Intensity</td>
<td>Very loud</td>
<td>Loud</td>
<td>Moderate</td>
<td>Soft</td>
</tr>
<tr>
<td>Pitch</td>
<td>Very high</td>
<td>High</td>
<td>Moderate</td>
<td>Low</td>
</tr>
<tr>
<td>Insp./exp. Ratio</td>
<td>1:1</td>
<td>1:3</td>
<td>1:1</td>
<td>3:1</td>
</tr>
<tr>
<td>Normal Location</td>
<td>Extrathoracic trachea</td>
<td>Manubrium sterni</td>
<td>Mainstem bronchi</td>
<td>Peripheral lung fields</td>
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</tbody>
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*SO YOU WANT TO BE A RESPIROLOGIST!*

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<td>Peripheral lung fields</td>
</tr>
<tr>
<td>Respiratory sound</td>
<td>Mechanisms</td>
<td>Origin</td>
<td>Acoustics</td>
<td>Relevance</td>
</tr>
<tr>
<td>------------------</td>
<td>------------</td>
<td>--------</td>
<td>-----------</td>
<td>-----------</td>
</tr>
<tr>
<td>o Wheeze</td>
<td>- Airway wall flutter (vortex shedding)</td>
<td>- Central and lower airways</td>
<td>- Sinusoid (range 100 to &gt; 1,000 Hz; duration, typically &gt; 80 ms)</td>
<td>- Airway obstruction, flow limitation</td>
</tr>
<tr>
<td>o Rhonchus</td>
<td>- Rupture of fluid films</td>
<td>- Large airways</td>
<td>- Series of rapidly dampened sinusoids (typically &lt; 300 Hz and duration &gt; 100 ms)</td>
<td>- Secretions, abnormal airway collapsibility</td>
</tr>
<tr>
<td>o Crackle</td>
<td>- Airway wall stress-relaxation</td>
<td>- Central and lower airways</td>
<td>- Rapidly dampened wave deflection (duration typically &lt; 20 ms)</td>
<td>- Airway closure, secretions</td>
</tr>
</tbody>
</table>

Adapted from: Mangione S. Hanley & Belfus 2000, page 279 and 297.; and Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto 2005, page 286.

Useful background: Performance characteristics for pulmonary auscultation for breath sounds and vocal resonance

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>o Breath sound score</td>
<td></td>
</tr>
<tr>
<td>Detecting chronic airflow obstruction</td>
<td></td>
</tr>
<tr>
<td>&lt;9</td>
<td>10.2</td>
</tr>
<tr>
<td>10-12</td>
<td>3.6</td>
</tr>
<tr>
<td>o Diminished breath sounds</td>
<td></td>
</tr>
<tr>
<td>Detecting underlying pleural effusion in mechanically ventilated patient</td>
<td>4.3</td>
</tr>
<tr>
<td>Detecting asthma during methacholine challenge</td>
<td>4.2</td>
</tr>
</tbody>
</table>
Finding                  | PLR
---|---
Detecting pneumonia in patients with cough and fever | 2.3

- Bronchial breath sounds
  Detecting pneumonia in patients with cough and fever | 3.3

- Egophony
  Detecting pneumonia in patients with cough and fever | 4.1

Abbreviation: PLR, positive likelihood ratio


Useful background: Characteristics of crackles in various disorders

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Mean number of crackles per inspiration</th>
<th>Timing of crackle</th>
<th>Type of crackle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary fibrosis</td>
<td>6-14</td>
<td>Late inspiratory (0.5→0.9)</td>
<td>Fine</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>3-7</td>
<td>Paninspiratory (0.3→0.7)</td>
<td>Coarse</td>
</tr>
<tr>
<td>Chronic airflow obstruction</td>
<td>1-4</td>
<td>Early inspiratory (0.3→0.5)</td>
<td>Coarse or fine</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>4-9</td>
<td>Late or paninspiratory (0.4→0.8)</td>
<td>Coarse or fine</td>
</tr>
</tbody>
</table>

Useful background: Effect of pulmonary disease on lung sounds

<table>
<thead>
<tr>
<th>Respiratory sound</th>
<th>Mechanisms</th>
<th>Origin</th>
<th>Acoustics</th>
<th>Relevance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basic sounds</td>
<td>- Turbulent flow vortices</td>
<td>- Central airways (expiration), lobar to segmental airway (inspiration)</td>
<td>- Low-pass filtered noise (range &lt; 100 to 1,000 Hz)</td>
<td>- Regional ventilation, airway caliber</td>
</tr>
<tr>
<td>Normal lung sound</td>
<td>- Turbulent flow impinging on airway walls</td>
<td>- Pharynx, larynx, trachea, large airways</td>
<td>- Noise with resonances (range &lt; 100 to &gt; 3,000 Hz)</td>
<td>- Upper airway configuration</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lung disease</th>
<th>Breath sounds</th>
<th>Adventitious lung sound</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumonia</td>
<td>o Bronchial or absent</td>
<td>o Inspiratory crackles</td>
</tr>
<tr>
<td></td>
<td>o Harsh/ bronchial</td>
<td>o Late inspiratory crackles</td>
</tr>
<tr>
<td>Atelectasis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>o Absent</td>
<td>o None</td>
</tr>
<tr>
<td>Emphysema</td>
<td>o Diminished</td>
<td>o Early inspiratory crackles</td>
</tr>
<tr>
<td>Chronic bronchitis</td>
<td>o Normal</td>
<td>o Wheezes and crackles</td>
</tr>
<tr>
<td>Pulmonary fibrosis</td>
<td>o Harsh</td>
<td>o Inspiratory crackles</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>o Diminished</td>
<td>o Inspiratory crackles</td>
</tr>
<tr>
<td>Pleural effusion</td>
<td>o Diminished</td>
<td>o None</td>
</tr>
<tr>
<td>Asthma</td>
<td>o Diminished</td>
<td>o Wheezes</td>
</tr>
</tbody>
</table>

Useful background: Abnormal (adventitious) breath sounds

<table>
<thead>
<tr>
<th>Recommended ATS Nomenclature</th>
<th>Characteristics</th>
<th>Wave form</th>
</tr>
</thead>
</table>
| **Course crackle**
  (aka “course rale”) | o Acoustic
  - Short, discontinuous interrupted explosive non-musical sounds
  - Heard best on inspiration
  o Mechanism
  - Excess airway secretions
  o Causes
  - Bronchitis
  - Respiratory infections
  - Pulmonary edema
  - Atelectasis fibrosis
  - CHF | ![Course Crackles](image) |
| **Fine crackle**
  (aka “fine rale crepitation”) | o Acoustic
  - Less loud and shorter duration than course rale
  - Higher in pitch than coarse rales
  o Mechanism | ![Fine Crackles](image) |
| **Wheeze**
  (aka “sibilant rhonchus”) | o Acoustic
  - Continuous musical sounds
  - Longer than 250 ms
  - High pitched
  - Heard best on expiration
  o Mechanism
  - Rapid airflow through obstructed airway
  o Causes
  - Asthma
  - Pulmonary edema
  - Bronchitis | ![Wheeze](image) |
- CHF
- Secretions
- Tumor
- Foreign body

- Rhonchus
  (aka “sonorous rhonchus”)
  - Acoustic
  - Continuous sounds
  - Longer than 250 ms
  - Low pitch
  - Snoring sound
  - May disappear after coughing
  - Mechanism
  - Transient larger airway plugging by mucus
  - Cause
  - Bronchitis

- Stridor
  - Acoustic
  - Inspiratory musical sounds
  - Best heard over trachea during inspiration
  - Mechanism
  - Upper airway extra-thoracic obstruction
  - Causes
  - Partial obstruction of larynx or trachea

- Pleural rub
  - Acoustic
  - Grating or creaking sounds
  - Best heard at end of inspiration and beginning of expiration
  - Mechanism
  - Inflammation of the pleura
  - Cause
  - Pneumonia pulmonary infarction

Abbreviation: C, Crackles

Normal crackles are usually end-inspiratory and high-pitched, and resemble the late inspiratory crackles of interstitial fibrosis. They usually resolve after a few deep inspirations.

In healthy persons, crackles represent the reinflation of atelectatic lung units. The greater number of collapsed units, the greater the number of crackles generated. These crackles are generally limited to the posterior lung bases. They occur frequently in people who have been breathing close to their functional residual capacity, and then are suddenly asked to take a deep breath. Because a mild degree of basilar collapse is common in healthy persons breathing shallowly below closing capacity, many basilar airways are collapsed. This collapse leads to the reabsorption of oxygen and further atelectasis. The sudden reopening of these airways on inspiration generates the crackles.


Useful background: The role of various techniques of chest examination in the diagnosis of disease - Chest examination findings and disease processes

<table>
<thead>
<tr>
<th>Disease</th>
<th>Trachea</th>
<th>Fremitus</th>
<th>Percussion note</th>
<th>Breath sounds</th>
<th>Advential breath sounds</th>
<th>Transmitted Breath sounds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal lung</td>
<td>Midline</td>
<td>Normal</td>
<td>Resonant</td>
<td>Vesicular</td>
<td>Late-inspiratory crackles at bases (resolve with deep breaths)</td>
<td>Absent</td>
</tr>
<tr>
<td>Consolidation (pneumonia, hemorrhage)</td>
<td>Midline</td>
<td>↑ Dull</td>
<td>Bronchial</td>
<td>Late-inspiratory crackles</td>
<td><em>+</em></td>
<td></td>
</tr>
<tr>
<td>Pulmonary fibrosis</td>
<td>Midline</td>
<td>Normal ↑</td>
<td>Resonant</td>
<td>Broncho-vesicular</td>
<td>Late-inspiratory crackles</td>
<td>-</td>
</tr>
<tr>
<td>Bronch-ectasis</td>
<td>Midline</td>
<td>Normal</td>
<td>Resonant</td>
<td>Vesicular</td>
<td>Mid-inspiratory</td>
<td>-</td>
</tr>
<tr>
<td>Condition</td>
<td>Location</td>
<td>Phonation</td>
<td>Breathing</td>
<td>Lung Sounds</td>
<td>Crackles</td>
<td></td>
</tr>
<tr>
<td>---------------------------------</td>
<td>----------------</td>
<td>-----------</td>
<td>-----------------</td>
<td>-------------</td>
<td>----------</td>
<td></td>
</tr>
<tr>
<td>Bronchitis</td>
<td>Midline</td>
<td>Normal</td>
<td>Normal to hyper-resonant</td>
<td>Vesicular</td>
<td>Early-inspiratory crackles</td>
<td></td>
</tr>
<tr>
<td>Emphysema</td>
<td>Midline</td>
<td>↓</td>
<td>Hyper-resonant</td>
<td>Diminished vesicular</td>
<td>Usually absent</td>
<td></td>
</tr>
<tr>
<td>Large pleural effusion</td>
<td>Shifted to opposite side</td>
<td>↓/0</td>
<td>Flat</td>
<td>Bronchial immediately above effusion Absent over effusion</td>
<td>? Rub above effusion May be present above effusion Absent over effusion</td>
<td></td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>Shifting to opposite side</td>
<td>↓/0</td>
<td>Tympanic</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Atelectasis (patent bronchi)</td>
<td>Shifted to same side</td>
<td>↑</td>
<td>Dull</td>
<td>Bronchial</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Atelectasis (plugged bronchi)</td>
<td>Shifted to same side</td>
<td>↓/0</td>
<td>Dull</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Status asthmaticus</td>
<td>Midline</td>
<td>↓</td>
<td>Hyper-resonant</td>
<td>Vesicular</td>
<td>Inspiratory/ expiratory wheezes</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviation: advent, adventitial; trans, transmitted

Sweet Nothings:
- Posture-induced crackles (PIC) are associated with elevated values of the pulmonary venous compliance. PIC is an independent variable for risk assessment after the number of disease coronary vessels, and increased PCWP (pulmonary capillary wedge pressure).
- A localized persistent rhonchus may indicate underlying lung cancer.


SO YOU WANT TO BE A RESPIROLOGIST!

Q1: What is the effect of coughing on expiratory crackles?
A1: Obstructive disease, decreased course expiratory crackles restrictive disease, no change with coughing.

Q2: Are late inspiratory crackles common in all types of intestinal lung disease?
A2: Common in Ideoathic Pulmonary Fibrosis (IPF) or Asbestosis (60%), but uncommon in sarcoidosis (18%; upper lobe and peribronchial fibrosis, vs lower lobe and subpleural fibrosis in IPF).

SO YOU WANT TO BE A RESPIROLOGIST!

Q: Under what conditions does the auscultation of vesicular breath sounds not signify reduced air flow (eg. in COPD)?

A:  
- Normal thickness of the chest wall
- Normal pleura (no fluid or air)
- Normal function of respiratory muscles
- Reduced/distant breath sounds suggest COPD, as also do vesicular breath sounds. Breath sounds of normal intensity mean that the FEV1 is normal or near normal.
- Auscultation of the breath sound intensity (BSI) at the bedside (reduced intensity) correlates with FEV1, FEV1/FVC, and distribution of ventilation – a poor person’s pulmonary function test – because of airtrapping and distruction of lung parenchyma in COPD
- In COPD (asthma, chronic bronchitis), the intensity of breath sounds heard at the mouth without the use of a strethoscope increases: with airway obstruction, intensity at mouth increases, over the chest diseases.


Useful background:

- Cause of late inspiration crackles (produced by reopening small airways in inspiration):
  - Interstitial fibrosis
  - Interstitial edema
    - Pneumonia
    - Pulmonary hemorrhage
    - CHF

- Mid inspiration crackles
  - Usually pathogenomic of bronchiectasis
Useful background: Bronchial breath sounds

- Higher and higher-pitch than vesicular breath sounds.
- Present in areas of airless lungs and patent bronchi (alveolar pneumonia) loading to collapse of alveolar lung tissue, or filling of alveoli with pus, blood or edema fluid, or rarely branchial breath sounds may be heard over areas of severe fibrosis.
- Chest X-ray may show air bronchogram (air-filled bronchi against the background of disease, airless, consolidated alveoli (lung parenchyma).


SO YOU WANT TO BE A RESPIROLOGIST!

Q1: Are breath sounds reduced when auscultated over a pleural effusion?
A1: It depends.
   - Above the effusion-normal
   - At the margin of the effusion-increased
   - Over the rest of the effusion-reduced


Q2: Is Campbell’s sign specific for COPD?
A2: Tracheal descent with inspiration ("tracheal tug", aka Campbell’s sign) is caused by any cause of chronic airflow obstruction, and not just COPD.


Q3: Does pneumonia increase or decrease tactile vocal fremitus (TVF)?
A3: It depends in bronchopneumonia (involving bronchi and alveoli, often from H. Influenza, with bronchial mucus plugs), ↑ in alveolar pneumonia (infection in alveoli but bronchial tree leaves bronchi patern; the infectious fluid in the alveoli plus the air in the bronchi make the TVF increase.

SO YOU WANT TO BE A RESPIROLOGIST!

BREATH SOUNDS

Q1: How can you differentiate between chronic bronchitis or asthma versus emphysema by listening to the patient’s breath sounds heard at the mouth (BSM) with the unaided ear, ie without using a stethoscope?

A1: In chronic bronchitis and asthma, there is a positive relation between the loudness of BSM and the FEV₁, or PEFR (peaked expiratory flow rate), whereas in asthma, BSM, becomes softer as airflow obstruction worsens. Thus, the intensity of BSM is not increased in all persons with COPD.


Q2: During auscultation of the chest, if you hear crackles or rhonchi, do you ask the patient to cough?

A2: Why not! Coughing clears the crackles and rhonchi of airflow obstruction caused by extra sounds at air-fluid interfaces of medium-to-large airways.

Useful background: Performance characteristics of pulmonary auscultation crackles and wheezes

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Crackles</td>
<td></td>
</tr>
<tr>
<td>o Detecting pulmonary fibrosis in asbestos workers</td>
<td>5.9</td>
</tr>
<tr>
<td>o Detecting elevated left atrial pressure in patients with cardiomyopathy</td>
<td>3.4</td>
</tr>
<tr>
<td>o Detecting myocardial infarction in patients with chest pain</td>
<td>2.1</td>
</tr>
<tr>
<td>o Detecting pneumonia in patients with cough and fever</td>
<td>1.8</td>
</tr>
<tr>
<td>➢ Early inspiratory crackles</td>
<td></td>
</tr>
<tr>
<td>o Detecting chronic airflow obstruction in patients with crackles</td>
<td>14.6</td>
</tr>
<tr>
<td>o Detecting severe disease in patients with chronic airflow obstruction</td>
<td>20.8</td>
</tr>
</tbody>
</table>
Unforced wheezing
  o Detecting chronic airflow obstruction  2.8

Wheezing during methacholine challenge testing
  o Detecting asthma  6.0

Crackles from pulmonary edema disappear on coughing, while pleural rub does not. Crackles from fibrosing alveolitis do not disappear on coughing, and lessen on leaning forward.

Abbreviation: PLR, positive likelihood ratio


SO YOU WANT TO BE A RESPIROLOGIST!
Q: Can you distinguish a pleural rub from a crackle, a wheeze and a pericardial rub?
A:
  A pleural rub
  Present during both inspiration and expiration (never present only in expiration)
  Does not change with coughing
  Long, louder, lower-pitched than crackle
  May be palpable
  A wheeze
  Usually occur in expiration only, whereas rubs are usually heard in both inspiration and expiration, or just in inspiration, but never only in expiration.
  A pericardial rub
  If the rub persists when the breath is held, then (dah!) it is more likely a pericardial than a pleural rub.

Tracheal deviation

2. Perform a directed physical examination of the pulmonary system for tracheal deviation.

- Lung findings in the affected side
  - ↓tactile vocal fremitus
  - Dullness
  - ↓breath sounds

- Tracheal deviation to the same side as the above lung findings, which is due to the pull effect of atelectasis

- Tracheal deviation to the normal side of the lung, due to the push effect of pleural effusion


Cough

Useful background: Description of “cough”

<table>
<thead>
<tr>
<th>Cough</th>
<th>Some causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sound</td>
<td>Viral interstitial lung disease,</td>
</tr>
<tr>
<td></td>
<td>- Tumour</td>
</tr>
<tr>
<td></td>
<td>- Allergies,</td>
</tr>
<tr>
<td></td>
<td>- Anxiety</td>
</tr>
<tr>
<td>o Dry, hacking</td>
<td>COPD</td>
</tr>
<tr>
<td>o Chronic, productive</td>
<td>Bronchiectasis</td>
</tr>
<tr>
<td></td>
<td>Abscess</td>
</tr>
<tr>
<td></td>
<td>Pneumonia</td>
</tr>
<tr>
<td></td>
<td>TB</td>
</tr>
<tr>
<td>o Wheezing</td>
<td>Bronchospasm (Asthma, Allergies)</td>
</tr>
<tr>
<td></td>
<td>Congestive heart failure</td>
</tr>
<tr>
<td>o Barking</td>
<td>Epiglottal disease (e.g. “croup”)</td>
</tr>
<tr>
<td>o Stridor</td>
<td>Tracheal obstruction</td>
</tr>
</tbody>
</table>
Timing

- Morning - Smoking
- Nocturnal - Post-nasal drip
  - Congestive heart failure
  - Asthma
- Upon eating/ drinking - Neuromuscular disease of the upper esophagus (aspiration)

Adapted from: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto 2005, page 281.

Useful background: Differential diagnosis for cough

Lung

- Airway irritants
  - Inhaled smoke, dusts, fumes, smoking
  - Aspiration of gastric contents, oral and nasal secretions, foreign body
- Airway disease
  - Acute/ chronic bronchitis
  - Bronchiectasis
  - Neoplasm
  - Asthma
  - COPD
- Parenchymal disease
  - Pneumonia
  - Lung abscess
  - Interstitial lung disease

Heart

- Congestive heart failure

Drugs

- ACE inhibitors
3. Take a directed history for cough.

- **History**
  - **Cough**
    - Acute/chronic (duration)
    - Change
    - Frequency
    - Onset/offset
    - Dry or wet
    - Sputum
      - Onset/ duration
      - Frequency
      - Progression
      - Quantity
      - Colour
      - Consistency
      - Odour
      - Hemoptysis
      - Mucoid (uninfected) sputum is odourless, transparent, and whitish-gray; small volumes suggest presence of asthma.
      - Purulent (infected) sputum contains pus, is often coloured, and large daily volume (50-1000 cc’s) suggests bronchiectasis
      - Foul-smelling sputum is suggestive of a lung abscess
  - **Complications**
    - Fever, chills
    - Anorexia, weight loss
    - Pleuritic chest pain
    - On/off
    - Dyspnea (SOB), dyspnea on exertion, (SOBOE), paroxysmal nocturnal dyspnea (PND)
  - **Causes**
    - Smoking (pack years)
    - Lung cancer in family
    - Inhalation work
    - TB exposure
    - Travel
    - Sexual orientation
    - Allergies
    - Drugs
    - COPD, CF, asthma
    - CHF, MI, AF
    - Puffers, Rx
    - Sleep
    - Past history
- Co-morbid illness (CaL, CF, CRF, cirrhosis)

Abbreviations: AF, atrial fibrillation; CaL, cancer of lung; CF, Cystic fibrosis; CHF, congestive heart failure; MI, myocardial infarctions; CRF, chronic renal failure; COPD, chronic obstructive pulmonary disease; PND, paroxysmal nocturnal dyspnea; Rx, medications; SOB, shortness of breath; SOBOE, shortness of breath on exertion.


**Hemoptysis**

4. Take a directed history of hemoptysis.

- Nose  
  - Bleeding

- GI  
  - Upper GI bleeding

- CVS  
  - Acute L-CHF, severe mitral stenosis

- Bleeding diathesis

- Respiratory  
  - Infection – bronchitis, bronchiectasis, pneumonia, abscess, TB  
  - Infarction  
  - Infiltration  
  - Ischemia – ruptured blood vessel from coughing; Good pasture’s syndrome  
  - Cystic fibrosis  
  - Foreign body

Abbreviation: L-CHF, left-sided congestive heart failure

SO YOU WANT TO BE A RESPIROLOGIST!

➢ In the setting of the patient with a lung collapse, what is Brock’s syndrome?
  o Brock’s syndrome is lung collapse due to compression of the right middle lobe bronchus by an enlarged lymph node, often from TB.


Consolidation, collapse, effusion, fibrosis

5. Perform a directed physical examination of the pulmonary system for consolidation, collapse, effusion, or fibrosis.

➢ General inspection
  o Contents of sputum cup (blood, pus etc.)
  o Type of cough
  o Rate and depth of respiration, and breathing pattern at rest and after exercise
  o Accessory muscles of respiration
  o Cheyne-Stokes breathing
  o Kussmaul hyperventilation
  o Temperature chart
  o Anemia
  o Obesity (sleep apnea)
  o Weight loss
  o Wasting, infraclavicular region
  o Mental status change (especially in the elderly)

➢ Face
  o Eyes – Horner’s syndrome (apical lung cancer)
  o Mouth - central cyanosis of tongue
  o Voice – hoarseness (recurrent laryngeal nerve palsy)
  o Skin – pallor

➢ Neck
  o Nodes
  o Thyromegaly
  o Trachea
  o Jugular venous pressure (CCF, SVC obstruction)
  o Use of accessory muscles

➢ Hands
  o Nicotine staining (actually from tobacco tar)
- Clubbing
- Cyanosis (peripheral)
- Wasting, weakness – finger abduction and adduction (lung cancer involving the brachial plexus)
- Wrist tenderness (hypertrophic pulmonary osteoarthritis)
- Pulse (tachycardia; pulsus paradoxus)
- Flapping tremor (CO$_2$ retention)
- Warms palms and rapid bounding pulse (CO$_2$ retention)

➢ Trachea shift towards lesion
  - Atelectasis, fibrosis, pneumonectomy

➢ Chest
  - Inspect
    - Shape of chest and spine
    - Scars
    - Prominent veins (determine direction of flow)
    - Movement of R/L side of chest
    - Barrel-chest shaped
    - Pemberton’s sign (SVC obstruction)
    - Radiotherapy marks
  - Palpate
    - Rib tenderness
    - Expansion
    - Position of trachea
    - Tactile vocal fremitus (TVF) (‘ninety nine’)
    - Pemberton’s sign (superior vena cava obstruction)
  - Percuss
    - Supraclavicular region
    - Dullness or hyperresonance
    - Upper, middle and lower chest on each side, front and back
  - Auscultate
    - Breath sounds (vesicular or bronchial)
    - Adventitial sounds (wheeze, crackles, pleural rub; do crackles disappear on coughing? Rub does not).
    - Vocal resonance (“one, one, one”; better than TVF)
    - Murmur of TR
    - Early diastolic Graham Steell murmur
    - P$_2$, loud ejection click
    - Forced expiratory time (FET, full inspiration to full expiration, over trachea, < 6 seconds is normal)

➢ Other
  - Breasts
  - Liver
  - Spleen
o Lower limbs - edema, cyanosis

*Remember to inspect, palpate, percuss, auscultate in right (R) axilla for R middle lobe disease.

Abbreviations: FET, forced expiratory time; SVO, superior vena cava; TR, tricuspid regurgitation; TVF, tactile vocal fremitus


Useful background: The causes of lung collapse

- Tumor
  - Bronchogenic carcinoma
  - Other intrabronchial tumours (eg bronchial adenoma)

- Plugs
  - Asthma
  - Allergic bronchopulmonary aspergillosis

- Infection
  - Extrinsic compression from hilar adenopathy (e.g. primary TB)
  - Tuberculosis


6. Perform a focused physical examination to distinguish between the major causes of dullness at a lung base.

- Pleural effusion: stony, dull note; trachea may be deviated to the opposite side in large effusions

- Pleural thickening: trachea not deviated; breath sounds will be heard

- Consolidation: vocal resonance increased; bronchial breath sounds and associated crackles

- Collapse; trachea deviated to the affected side; absent breath sounds

Useful background: Physical findings – tactile vocal fremitus (TVF)

<table>
<thead>
<tr>
<th>Transmission</th>
<th>Possible pathologies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased</td>
<td>o Consolidation (e.g. pneumonia)</td>
</tr>
<tr>
<td>Decreased-unilateral</td>
<td>o Atelectasis, bronchial obstruction, pleural effusion, pneumothorax, pleura thickening</td>
</tr>
<tr>
<td>Bilateral</td>
<td>o Chest wall thickening (muscle, fat), COPD, Bilateral pleural effusion</td>
</tr>
</tbody>
</table>

Useful background: Surface anatomy for underlying lobes of the lung

![Diagram of lung anatomy](image)

A, anterior
B, posterior
C, lobes of the right lung;
D, lobes of the left lung

Useful background: Types of percussion notes and pathologic examples

<table>
<thead>
<tr>
<th>Percussion note</th>
<th>Pathologic example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dullness</td>
<td>o lobar pneumonia</td>
</tr>
<tr>
<td></td>
<td>o pleural effusion</td>
</tr>
<tr>
<td></td>
<td>o hemothorax</td>
</tr>
<tr>
<td></td>
<td>o empyema</td>
</tr>
<tr>
<td></td>
<td>o atelectasis</td>
</tr>
<tr>
<td></td>
<td>o tumour</td>
</tr>
<tr>
<td>Resonance</td>
<td>o chronic bronchitis</td>
</tr>
<tr>
<td>Hyperresonance</td>
<td>o emphysema, pneumothorax</td>
</tr>
<tr>
<td></td>
<td>o asthma</td>
</tr>
</tbody>
</table>

Useful background: The physical findings in the chest for consolidation, collapse, effusion and fibrosis

<table>
<thead>
<tr>
<th></th>
<th>Consolidation</th>
<th>Collapse</th>
<th>Effusion</th>
<th>Fibrosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inspection</td>
<td>^</td>
<td>^</td>
<td>^</td>
<td>^</td>
</tr>
<tr>
<td>o Chest wall movement</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td></td>
<td>Apical, flattening of chest on the affected side</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Palpation</td>
<td>^ alveolar pneumonia (bronchi patent)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Tactile vocal fremitus (TVF; “E”, “1-2-3”, “99”) (Same as vocal resonance, VR)</td>
<td>Alveolar ↑ TVF</td>
<td>↑ above effusion; absent over effusion</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>↓ bronchopneumonia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Displaced trachea</td>
<td>-</td>
<td>Towards collapse</td>
<td>Away from effusion</td>
<td>Towards the fibrosis</td>
</tr>
<tr>
<td>Percussion dullness</td>
<td>+</td>
<td>+</td>
<td>*</td>
<td>+</td>
</tr>
<tr>
<td>Auscultation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Breath sounds</td>
<td>Bronchial</td>
<td>Bronchovesicular</td>
<td>Bronchial above fluid, absent over fluid</td>
<td>↓ Bronchial</td>
</tr>
</tbody>
</table>
o Adventitial sounds

o Auscultatory vocal resonance (E-E-E, Same as TVF)
  - Crackles
  - Crackles
  - Crackles

Abbreviations: TVF, tactile vocal fremitus; VR (Auscultatory) vocal resonance.


Useful background: Lung disease caused by occupational exposure

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Lung disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grain dust, wood dust, tobacco, pollens,</td>
<td>o Asthma</td>
</tr>
<tr>
<td>many others</td>
<td></td>
</tr>
<tr>
<td>Asbestos</td>
<td>o Pleural mesothelioma</td>
</tr>
<tr>
<td>Coal</td>
<td>o Pneumoconiosis</td>
</tr>
<tr>
<td>Sandblasting and quarries</td>
<td>o Silicosis</td>
</tr>
<tr>
<td>Industrial dusts</td>
<td>o Chronic bronchitis</td>
</tr>
<tr>
<td>Birds</td>
<td>o Psittacosis</td>
</tr>
<tr>
<td>Cotton</td>
<td>o Byssinosis</td>
</tr>
</tbody>
</table>

Source: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto* 2005, page 283.

Useful background: Causes of pleural effusion

- Lung
  o Infections (usually an exudates)
    - Parapneumonic (bacterial) effusions
    - Bacterial empyema
    - Tuberculosis
    - Fungi
    - Parasites
    - Viruses & mycoplasma
  o Neoplasms
    - Primary metastatic lung tumors
    - Lymphoma and leukaemia
    - Benign and malignant tumors of pleura
    - Intra-abdominal tumors with ascites
  o Vascular disease
- Pulmonary embolism
- Wegener granulomatosis

○ Trauma
- Hemothorax
- Chylothorax

○ Miscellaneous
- Drug induced effusions

➢ Heart
- Congestive heart failure
- Superior vena caval obstruction
- Constrictive pericarditis

➢ Liver
- Cirrhosis with ascites
- Hypoalbuminemia
- Salt retaining syndromes

➢ Kidney
- Peritoneal dialysis
- Hydronephrosis
- Nephrotic syndrome
- Uremic pleuritis

➢ GI
- Intra abdominal diseases
  - Pancreatitis and pancreatic pseudocyst
  - Subdiaphragmatic abscess
  - Malignancy with ascites
  - Esophageal rupture
  - Intra abdominal surgery

➢ Miscellaneous
- Meigs Syndrome
- Myxedema
- Familial Mediterranean fever

Clubbing

7. Perform a directed physical examination for clubbing.

- Clubbing is a painless focal, usually symmetric enlargement of the connective tissue in the terminal phalanges of the digits of the fingers more than the toes.

- Signs
  - Interphalangeal depth ratio
  - Appearance: Parrot’s beak, watchglass, drumstick
  - Schamroth’s sign

- CVS
  - Cyanotic congenital heart disease
  - Infective endocarditis
  - Axillary artery aneurysm

- Lung
  - Lung carcinoma (usually not small cell carcinoma)
  - Bronchial arteriovenous aneurysm
  - Chronic suppuration
    - Bronchiectasis
    - Lung abscess
    - Empyema
  - Idiopathic pulmonary fibrosis
    - Cystic fibrosis
    - Asbestosis
    - Pleural mesothelioma (benign fibrous type) or pleural fibroma

- Gastrointestinal
  - Cirrhosis
  - Inflammatory bowel disease (Crohn, ulcerative colitis)
  - Celiac disease

- Endocrine
  - Thyrotoxicosis
  - Secondary hyperparathyroidism

- Rare
  - Neurogenic diaphragmatic tumours
  - Pregnancy


- Interphalangeal depth ratio: Measurement of the interphalangeal depth ratio is described in the Figure below. If this ratio exceeds 1, clubbing is present, a conclusion supported by tow observations:
The interphalangeal depth ratio of normal persons is 0.895 ± 0.041, making the threshold of 1.0 more than 2.5 standard deviations above the normal. A ratio of 1.0 distinguishes digits of healthy persons from those of patients with disorders traditionally associated with clubbing (such as cyanotic heart disease and cystic fibrosis). Special equipment is required for precise measurements.

- A: normal digit
- B: Clubbed digit

The distal interphalangeal joint is denoted by “a”. The junction of the nail and skin at the midline is denoted by “b”. The interphalangeal depth ratio is the ratio of the digit's depth measured at “b” divided by that at “a”. The hyponychial angle is the angle “wxy”. In the Figure, the depth ratio is 0.9 for the normal digit and 1.2 for the clubbed digit (a ratio >1 indicates clubbing) and the hyponychial angle is 185 degrees for the normal digit and 200 degrees for the clubbed digit (a hyponychial angle > 190 degrees indicates clubbing).

A – Parrot’s beak-accumulation of connective tissue in proximal portion of distal digit
B – Watchglass-connnective tissue at base of nail
C – Drumstick-connnective tissue at base of nail
Schamroth’s sign?
- Disappearance of the diamond-shaped window normally present when the terminal phalanges of paired digits are juxtaposed.
- Accumulation of connective tissue may occur quickly (<10 days)


SO YOU WANT TO BE A PULMONARY RESIDENT!

Q: What is hypertrophic pulmonary osteoarthropathy?
A:  
- Hypertrophic pulmonary osteoarthropathy is digital clubbing with periostosis (Marie-Bamberger syndrome)
  - A systemic disorder of bones, joints, and soft tissues most commonly associated with an intrathoracic neoplasm (usually bronchogenic carcinoma but also lymphomas and metastatic cancers).
  - Periosteal new-bone proliferation that accompanies digital clubbing, especially prominent in the long bones of the extremities.
  - Other features of symmetric arthritis-like changes in one or more joints (ankles, knees, wrists, and elbows); coarsening of the subcutaneous tissue in the distal portions of arms and legs (and occasionally the face); neurovascular changes in hands and feet (with chronic erythema, paresthesias, and increased sweating).
  - Associations may be seen in
    - Cystic fibrosis,
    - Bronchiectasis,
    - Chronic empyema,
    - Lung abscesses (all typically associated with clubbing),
    - Pulmonary interstitial fibrosis

Granulomatous lung disease

8. Perform a directed physical examination for sarcoidosis.

- Lung  
  - Hilar lymph adenopathy

- Skin  
  - Erythema nodosum
  - Lupus pernio

- Ophthalmologic  
  - Uveitis
  - Keratoconjunctivitis
  - Sjogrens syndrome

- Cardiac  
  - Conduction defects
  - Valvular insufficiency
  - Cardiomyopathy
  - Cor pulmonale,

- Arthritic  
  - In 10 to 15% most common in ankles and knees. Axial skeleton spared

- Renal  
  - Nephrolithiasis
  - Hypercalcaemia
  - Nephrocalcinosis
  - Memb nephropathy

- Liver  
  - Granuloma (in 86%)
  - Cholestasis
  - Post- necrotic cirrhosis

- Neurolgic  
  - Peripheral neuropathy
  - Lymphocytic meningitis
  - Diabetes insipidus
  - Mononeuritis multiplex
  - Cranial nerve palsies

- Endocrine  
  - Thyroid nodules
  - Infertility
  - Hypogonadism

Useful background: Grading of sarcoidosis

<table>
<thead>
<tr>
<th>Grade</th>
<th>Abnormality (% cases)</th>
<th>% Resolution</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal</td>
<td>-</td>
</tr>
<tr>
<td>1</td>
<td>BHL (65%)</td>
<td>80%</td>
</tr>
<tr>
<td>2</td>
<td>BHL and pulmonary infiltrate (22%)</td>
<td>50%</td>
</tr>
<tr>
<td>3</td>
<td>Pulmonary infiltrate without BHL (13%)</td>
<td>25%</td>
</tr>
</tbody>
</table>

Abbreviations: BHL, Bilateral hilar lymphadenopathy


SO YOU WANT TO BE A RESPIROLOGIST!

Q1: What are the skin manifestations of sarcoidosis?
A1:  
   ➢ Small, non-scaling, skin-coloured, dome-shaped papules, usually on face and neck  
   ➢ If lesions coalesce, nodules and plaque form on trunk  
   ➢ Non-caseating granulomas on biopsy


PTFs
Q2: How can you estimate the value of FEV$_1$/FVC with your stethoscope?
A2: Auscultate over sternal notch and time how long it takes the patient to take a deep breath and blow out hard. This gives the forced-expiratory time (FET$_0$).

<table>
<thead>
<tr>
<th>FET$_0$</th>
<th>FEV$_1$/FVC</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;6 sec</td>
<td>≤ 40%</td>
</tr>
<tr>
<td>&lt;5 sec</td>
<td>&gt; 60%</td>
</tr>
</tbody>
</table>


Pulmonary fibrosis

Useful background: Causes of pulmonary fibrosis (diffuse lung disease)

➢ Idiopathic  
   o Idiopathic pulmonary fibrosis- usual interstitial pneumonia (IPF-UIP)
- Nonspecific interstitial pneumonia (NSIP)
- Bronchiolitis obliterans with organizing pneumonia/cryptogenic organizing pneumonia (BOOP/COP)
- Eosinophilic lung disease
- Lymphocytic interstitial pneumonia (LIP)
- Alveolar microlithiasis
- Lymphangioleiomyomatosis (LAM)
- Langerhans cell histiocytosis/eosinophilic granulomatosis
- Pulmonary alveolar proteinosis
- Acute respiratory distress syndrome/acute lung injury

- Infections
  - TB
  - Mycosis
  - Varicella
  - Psittacosis

- Pneumoconiosis
  - Coal dust
  - Silicon
  - Asbestos
  - Benylum
  - Iron
  - Kaolin
  - Cadmium
  - Nickel
  - Chromium
  - Talc
  - Byssinosis
  - Bagassosis
  - China-clay
  - Aluminium

- Hypersensitivity
  - Farmers lung
  - Bird fancier’s lung
  - Pituitary snuff-taker’s lung
  - Mushroom workers lung
  - Paprika splitters lung
  - Silo fillers disease
  - Workers in gas works

- Fibrosing Alveolitis
  - Rheumatoid lung
  - Desquamative interstitial pneumonitis

- Aspiration

- Cardiac
  - Pulmonary edema
Mitral stenosis ossificiation
Multiple pulmonary infarcts
Uramic lung

Neoplastic
Alveolar cell carcinoma
Lymphangitis carcinomatosa

Miscellaneous
Sarcoidosis
Systemic sclerosis
SLE
Radiation
Histiocytosis X
Xanthomatosis
Biliary cirrhosis
Paraquat poisoning
Prolonged busulphan therapy

Useful background: Poor prognostic factors in patients with community-acquired pneumonia (CAP)

Age over 65 years
Coexisting conditions such as cardiac failure, renal failure, chronic obstructive pulmonary disease, malignancy
Clinical features: respiratory rate > 30 per min, hypotension (systolic blood pressure < 90 mmHg or diastolic pressure < 60 mmHg), temperature > 38.3°C, impaired mental status (stupor, lethargy, disorientation or coma), extrapulmonary infection (e.g. septic arthritis, meningitis)
Investigations: hematocrit < 30%, white cell count < 4000 or > 30 000 per mm³, azotemia, arterial blood gas < 60 mmHg while breathing room air, chest radiograph showing multiple lobe involvement, rapid spread or pleural effusion.
Microbial pathogens: Staph. Aureus, Legionella, Strep. pneumoniae

Pneumonia

Useful background: Complications of pneumonia

- **General**
  - Septicemia
  - Multi-organ failure
  - Hemolytic syndrome
  - Death

- **Lung**
  - Lung abscess
  - Empyema
  - Adult respiratory distress syndrome

- **Kidney**
  - Renal failure
  - Glomerulonephritis

- **Atypical pneumonia**
  - Typical pneumonia is caused by pneumococcus (Streptococcus pneumonia)
  - Atypical pneumonia is caused by Mycoplasma, Legionella, Chlamydia, Coxiella, etc.
  - The clinical picture in atypical pneumonia is dominated by constitutional symptoms, such as fever and headache, rather than by respiratory symptoms

- **CNS**
  - Central and peripheral nervous systems

- **Heart**
  - Pericarditis, myocarditis

- **Liver**
  - Hepatitis

- **Blood**
  - Disseminated intravascular coagulation (DIC)
  - Autoimmune hemolytic anemia

- **MSK**
  - Arthralgia and arthritis

- **Skin**
  - Non-specific rash
  - Erythema multiforme
  - Stevens-Johnson syndrome

Useful background: Performance characteristics of physical examination for pneumonia

The PLRs for crackles and wheezes for the diagnosis of pneumonia are each < 2.0

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>General appearance</td>
<td></td>
</tr>
<tr>
<td>- Cachexia</td>
<td>4.0</td>
</tr>
<tr>
<td>Vital signs</td>
<td></td>
</tr>
<tr>
<td>- Temperature &gt; 37.8°C</td>
<td>2.0</td>
</tr>
<tr>
<td>- Respiratory rate &gt; 28/min</td>
<td>2.0</td>
</tr>
<tr>
<td>Lung findings</td>
<td></td>
</tr>
<tr>
<td>- Percussion dullness</td>
<td>3.0</td>
</tr>
<tr>
<td>- Diminished breath sounds</td>
<td>2.3</td>
</tr>
<tr>
<td>- Bronchial breath sounds</td>
<td>3.3</td>
</tr>
<tr>
<td>- Egophony</td>
<td>4.1</td>
</tr>
</tbody>
</table>

Abbreviation: PLR, positive likelihood ratio


Useful background: Predictors of hospital mortality of pneumonia

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>General appearance</td>
<td></td>
</tr>
<tr>
<td>- Abnormal mental status</td>
<td>2.8</td>
</tr>
<tr>
<td>Vital signs</td>
<td></td>
</tr>
<tr>
<td>- Respiratory rate &gt; 30/min</td>
<td>2.1</td>
</tr>
<tr>
<td>- Systolic blood pressure &lt; 90 mmHg</td>
<td>10.0</td>
</tr>
<tr>
<td>- Heart rate &gt; 100/min</td>
<td>2.1</td>
</tr>
<tr>
<td>- Hypothermia</td>
<td>3.5</td>
</tr>
</tbody>
</table>

Abbreviation: PLR, positive likelihood ratio

Useful background: Multivariate findings for adult pneumonia

Add points for the presence of findings as follows:
- Rhinorrhea = 2 points
- Sore throat = -1
- Night sweats = 1
- Myalgias = 1
- Sputum all day = 1
- Respiratory rate > 25/min = 2
- Temperature ≥37.8°C (100°F) = 2

<table>
<thead>
<tr>
<th>Threshold score</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥3</td>
<td>14</td>
</tr>
<tr>
<td>≥1</td>
<td>5.0</td>
</tr>
<tr>
<td>≥ -1</td>
<td>1.5</td>
</tr>
<tr>
<td>&lt; -1</td>
<td>0.22</td>
</tr>
</tbody>
</table>

Abbreviation: PLR, positive likelihood ratio


Useful background: As the number of findings increases, the probability of pneumonia increases

Count the number of findings present; absence of asthma; temperature ≥37.8°C (100°F); heart rate > 100/min; decreased breath sounds; crackles

<table>
<thead>
<tr>
<th>Number of Findings</th>
<th>Probability, % (baseline prevalence 5%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>50</td>
</tr>
<tr>
<td>4</td>
<td>25</td>
</tr>
<tr>
<td>3</td>
<td>20</td>
</tr>
<tr>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>0</td>
<td>&lt;1</td>
</tr>
</tbody>
</table>


Useful background: Criteria to determine hospital admission in the patient with pneumonia

- Any of the below should result in admission:
  - Respiratory rate >28/min
  - Systolic BP <90 mmHg or 30 mmHg below baseline
  - Delirium
  - Hypoxia: Oxygen sat <90% or pO2 of <60 mmHg on room air
  - UnsTable co-morbid illness e.g.: renal failure, CHF
- Lobar multi pneumonia
- Pleural effusion >1cm, and has features of a complicated parapneumonic effusion

- The British Thoracic Society severity score is based on four criteria.
  - CURB
    - Confusion
    - Urea > 7mmol/L
    - Respiratory rate > 30/min* (without underlying lung disease)
    - Blood pressure <60 mmHg diastolic or systolic <90 mmHg

Mortality rate with no CURB features is 2.4%, with one feature, 8%; with two, 23%; with three, 33% and with all four CRUB features, 83%.


9. List the causes of slow resolution or recurrence of pneumonia.

- Bronchial obstruction
  - Neoplasm
  - Aspiration of foreign body

- General
  - Antibiotic
  - Decreased host resistance
  - Cachexia
  - Agranulocytosis
  - Immunoglobulin defects etc

- Lung
  - Formation of abscess, empyema or serous effusion
  - Other causes of pulmonary fibrosis

- Pharynx
  - Pharyngeal pouch with ‘spilling’


**Airflow obstruction and asthma**

Useful background: Wheezing

- Wheezing on maximal forced exhalation
  - A sensitivity of only 57% and a specificity of only 37% for airflow obstruction.
  - A forced expiratory manoeuvre aimed at “unmasking silent bronchospasm” should not be relied on for the clinical diagnosis of airflow obstruction.
Wheezing that occurs only in exhalation
  o Not as severe as wheezing that occurs both in exhalation and inspiration.
  o Longer expiratory wheezes reflect worse obstruction than shorter expiratory wheezes.

Wheezes are not perfect diagnostic findings and are less valuable than crackles.


Useful background: Differential diagnosis of wheezing

Larynx
  o Laryngeal edema
  o Laryngo-, tracheo-, or bronchomalacia
  o Vocal cord dysfunction

Trachea
  o Stenosis or compression
  o Foreign body
  o Central airway tumors
  o Aspiration
  o Vascular ring affecting trachea

Heart
  o Cardiac failure
  o Asthma
  o Chronic obstructive pulmonary disease
  o Bronchorrhoeal states (such as chronic bronchitis, cystic fibrosis, bronchiectasis)
  o Hypersensitivity pneumonitis
  o Pulmonary edema
  o Forced expiration in normal subjects

Lung
  o Pulmonary embolism
  o Carcinoid syndrome
  o Löffler syndrome
  o Bronchiectasis
  o Tropical eosinophilia
  o α-1 Antiprotease deficiency
  o Immotile cilia syndrome
  o Bronchopulmonary dysplasia
  o Bronchiolitis (including bronchiolitis obliterans), croup
  o Cystic fibrosis
Infections (croup, whooping cough, laryngitis, tracheobronchitis)

- Ribs
  - Chondromalacia/ polychondritis

- CNS
  - Hyperventilation syndrome
  - Facitious (including psychophysiological vocal cord adduction)


10. Take a directed history for asthma.

- Wheezing
  - Acute/chronic (duration)
  - Onset/offset
  - Cough, sputum
  - Fever, chills
  - Aspiration
  - Cardiac disease
  - Personal history of eczema, hay fever
  - Immunizations
  - Family history of asthma, allergies, eczema, hay fever

- Complications
  - ER visits
  - Hospital admissions, ICU, intubation
  - Use of beta-blockers
  - Medications, including Tablets, inhalers, ASA, steroids

- Causes
  - Seasonal allergies – pollens, foods, animals, medications
  - Upper respiratory infection
  - Medications
  - Family history of other pulmonary conditions
  - Exercise
  - Cold weather
  - Smoke - first/second hand
  - Stress
  - Gastroesophageal reflux disease

11. Perform a focused physical examination for asthma.

- **Physical examination**
  - **General**
    - Ability to speak (inability to complete a sentence in one breath)*
    - Restlessness
    - Altered mental status, confusion, coma*
    - Fatigue, exhaustion*
    - Vital signs (RR, HR, BP, O$_2$ saturation)
    - Pulsus paradoxus
    - Cyanosis
  - **Cardiology**
    - PR > 110 bpm*
    - ↓ PR, ↓ BP*
  - **Respiratory**
    - RR > 25/min*
    - ↓ RR (poor respiratory effort)*
    - Cyanosis*
    - Barrel chest
    - Cough
    - Accessory muscle use
    - Air entry
    - Wheezes and location
    - Crackles and location
    - Percussion – hypertympanic
    - Prolonged expiration
    - Clubbing
    - Peak expiratory flow rate less than 50% of predicted or best*
  - **Focus of infection**
    - Rhinorrhea, coryza
    - Pharynx
    - Tympanic membrane
    - Cardiovascular examination
    - Abdominal examination
    - Skin examination – rash

*denotes severe asthmatic attack (risk stratification)

Useful background: Definitions

- Chronic bronchitis is cough with mucoid expectoration for at least 3 months in a year for 2 successive years.

- Emphysema is the abnormal permanent enlargement of the airway distal to the terminal respiratory bronchioles with destruction of their walls. (Clinical, radiological and lung function tests give an imprecise picture in an individual case, but a combination of all these features gives a reasonable picture)

- The term COPD encompasses chronic obstructive bronchitis (with obstruction of small airways) and emphysema (with destruction of lung parenchyma, loss of lung elasticity, and closure of small airways). Most patients also have mucus plugging


12. Take a focused history and perform a directed physical examination for chronic bronchitis.

- **General**
  - Sputum pot full
  - Pursed lip
  - O₂ mask/ respirator
  - Cyanosis of lips/ tongue

- **Neck**
  - Inspection
    - Use of accessory muscles of respiration (sternocleidomastoids, scaleni and trapezi)
    - ↑ JVP
  - Palpation
    - Tracheal deviation
    - ↓ distance (< 3 fingers’ breadth) between the cricoid cartilage and suprasternal notch

- **Lungs**
  - Inspection
    - Barrel-shaped
    - ↑ RR
    - Dyspnea
    - Chest expansion
  - Palpation
    - Apex beat
    - ↓ Chest expansion
    - Tactile vocal fremitus
- Percussion
  - Hyper-resonance
- Auscultation
  - ↓ breath sounds
  - Vocal resonance
  - Forced expiratory time > 6 seconds indicates airflow obstruction

- CVS
  - ↑ PR
  - Bounding pulse
  - ↓ heart dullness
  - Displaced apex beat
  - ↑ P₂

- Abdomen
  - ↓ liver dullness
  - Palpable liver (not necessarily enlarged)

- Hands
  - Warm palms
  - Tar staining
  - No clubbing

Abbreviations: RR, respiratory rate; PR, pulse rate


Sweet Nothing: Not all that wheezes is “asthma”.
13. Take a directed history to differentiate between bronchial asthma, chronic bronchitis, and emphysema.

<table>
<thead>
<tr>
<th>Differential feature</th>
<th>Bronchial asthma</th>
<th>Chronic bronchitis (“blue bloater”)</th>
<th>Emphysema (“pink puffer”)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Onset</td>
<td>70% &lt; 30 y</td>
<td>&gt;50 y</td>
<td>≤ 60 y</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td>0</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Pattern</td>
<td>Paroxysmal</td>
<td>Chronic, progressive</td>
<td>Chronic, progressive</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>0 to +++</td>
<td>+ to +++</td>
<td>+++ to +++</td>
</tr>
<tr>
<td>Cough</td>
<td>0 to +++</td>
<td>++ to +++</td>
<td>+ to +++</td>
</tr>
<tr>
<td>Sputum</td>
<td>0 to ++</td>
<td>Profuse, mucopurulent</td>
<td>scanty</td>
</tr>
<tr>
<td>Atopy</td>
<td>50% (adult)</td>
<td>15%</td>
<td>15%</td>
</tr>
<tr>
<td>Infections</td>
<td>↑ Symptoms</td>
<td>↑↑↑ Symptoms</td>
<td>↑ Symptoms</td>
</tr>
<tr>
<td>Cyanosis</td>
<td>---</td>
<td>++</td>
<td>--</td>
</tr>
<tr>
<td>Hyperinflation</td>
<td>---</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>Cor Pulmonale</td>
<td>---</td>
<td>+++ frequent, remittent</td>
<td>+ (pre-terminal)</td>
</tr>
<tr>
<td>Respiratory drive</td>
<td>---</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Polycythemia</td>
<td>---</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Chest x ray, vessels</td>
<td>---</td>
<td>---</td>
<td>↓</td>
</tr>
<tr>
<td>Arterial PCO₂</td>
<td>---</td>
<td>↑</td>
<td></td>
</tr>
<tr>
<td>Alveolar gas transfer</td>
<td>---</td>
<td>---</td>
<td>↓</td>
</tr>
</tbody>
</table>

Chronic obstructive pulmonary disease (COPD)

Useful background: Operating characteristics of clinical history for COPD

<table>
<thead>
<tr>
<th>Item</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking history</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o ≥70 vs. &lt;70 pack yrs</td>
<td>8.0</td>
<td>0.63</td>
</tr>
<tr>
<td>Sputum production ≥ ¼ cup</td>
<td>4</td>
<td>0.84</td>
</tr>
<tr>
<td>Symptoms of chronic bronchitis</td>
<td>3.0</td>
<td>0.78</td>
</tr>
<tr>
<td>Wheezing</td>
<td>3.8</td>
<td>0.66</td>
</tr>
<tr>
<td>Exertional dyspnea</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Grade 4 vs. 3 or less</td>
<td>3.0</td>
<td>0.98</td>
</tr>
<tr>
<td>o Any vs. none</td>
<td>2.2</td>
<td>0.83</td>
</tr>
<tr>
<td>Wheezing</td>
<td>36</td>
<td>0.85</td>
</tr>
<tr>
<td>Barrel chest</td>
<td>10</td>
<td>0.90</td>
</tr>
<tr>
<td>Decreased cardiac dullness</td>
<td>10</td>
<td>0.88</td>
</tr>
<tr>
<td>Match test</td>
<td>7.1</td>
<td>0.43</td>
</tr>
<tr>
<td>Rhonchi</td>
<td>5.9</td>
<td>0.95</td>
</tr>
<tr>
<td>Hyperresonance</td>
<td>4.8</td>
<td>0.73</td>
</tr>
<tr>
<td>Forced expiratory time, sec</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;9</td>
<td>4.8</td>
<td></td>
</tr>
<tr>
<td>6-9</td>
<td>2.7</td>
<td></td>
</tr>
<tr>
<td>&lt;6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subxiphoid cardiac apical impulse</td>
<td>4.6</td>
<td>0.94</td>
</tr>
<tr>
<td>Pulsus paradoxus (&gt;15 mm Hg)</td>
<td>3.7</td>
<td>0.62</td>
</tr>
<tr>
<td>Decreased breath sounds</td>
<td>3.7</td>
<td>0.70</td>
</tr>
</tbody>
</table>

PLR, positive likelihood ratio; NLR negative likelihood ratio

Note: A forced expiratory time of < 6 seconds is not included because the value of its PLR is < 2.

Useful background: Characteristic results of pulmonary function tests in obstructive and restrictive lung diseases

<table>
<thead>
<tr>
<th></th>
<th>Obstructive</th>
<th>Restrictive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lung volumes</td>
<td>VC ↓</td>
<td>↓</td>
</tr>
<tr>
<td></td>
<td>FRC ↑</td>
<td>↓</td>
</tr>
<tr>
<td></td>
<td>RV ↑</td>
<td>↓</td>
</tr>
<tr>
<td></td>
<td>TLC ↑ or N</td>
<td>↓</td>
</tr>
<tr>
<td>Flow rates</td>
<td>FEV 1.0 ↓</td>
<td>↓ or N</td>
</tr>
<tr>
<td></td>
<td>FEV 1.0/FVC ↓</td>
<td>↑ or N</td>
</tr>
<tr>
<td></td>
<td>FEF 50% VC ↓</td>
<td>↑ or N</td>
</tr>
<tr>
<td></td>
<td>FEF 25% VC ↓</td>
<td>↑ or N</td>
</tr>
<tr>
<td>Diffusion capacity</td>
<td>DCco ↓ or N</td>
<td>↓ or N</td>
</tr>
</tbody>
</table>

Abbreviations: DCCO, diffusion capacity of carbon monoxide; N, normal


14. Take a directed history for the harmful effects of cigarette smoking.

- **CNS**
  - Autonomic: transient stimulation, followed by depression of all ganglia
  - CNS: stimulation, especially respiratory, vasomotor and emetic centres
  - Antidiuretic: (due to ADH release)
  - Tobacco amblyopia

- **CVS**
  - Rise in BP, tachycardia, cutaneous vasoconstriction
  - Tobacco angina
  - Atrial extrasystoles
  - Myocardial ischemia
  - Buerger’s disease

- **Lung**
  - Post-operative pneumonia
  - Bronchitis
  - Bronchus (increased carcinoma incidence)

- **GI**
  - Esophagus (increased carcinoma incidence)
Cirrhosis incidence increased (Probably due to associated alcoholism)

- Kidney
  - Prostate (increased carcinoma incidence)
  - Bladder (increased carcinoma incidence)

- Endocrine
  - Adrenal: discharges adrenaline
  - Hypoglycemia

- Fetus (pregnant mother smoking)
  - ↓fetal growth
  - ↑perinatal mortality rate

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 27.

15. Take a focused history and perform a directed physical examination for bronchiectasis.

- **History**
  - **General**
    - Intermittent fever and night sweats
    - Weight loss
  - **Lungs**
    - Cough with copious purulent sputum
    - Recurrent hemoptysis
    - History of recurrent chest infections
    - History of associated lung disease

- **Physical examination**
  - **Lungs**
    - Copious purulent expectoration
    - Collapse
    - Fibrosis
    - Pneumonia
    - Bilateral coarse, late, inspiratory crackles
  - **Spine**
    - Possible kyphoscoliosis
  - **Hands**
    - Finger clubbing
Spleen
  o Possible splenomegaly (amyloidosis)

Adapted from: Baliga R.R. Saunders/Elsevier 2007, page 266.

Carcinoma of the lung

16. Perform a directed physical examination of the pulmonary system in the patient with suspected mediastinal compression (e.g. carcinoma of the lung).

Many patients have no signs

General
  o Anorexia
  o Weight loss
  o Cachexia
  o Fever
  o Fatigue
  o Night sweats

CNS
  o Mediastinal nerve compression (sympathetic, left recurrent laryngeal, phrenic nerve)
  o Cortex – dementia
  o Peripheral neuropathy (polyneuropathy, subacute sensory neuropathy)
  o Polymyositis; progressive muscle weakness (Eaton-Lambert syndrome)
  o Retinal blindness (small cell carcinoma)
  o Subacute cerebellar degeneration
  o Polymyositis
  o Cortical degeneration.

Eyes
  o Exophthalmos
  o Conjunctival redness, venous dilation in the fundi

Face
  o Plethora
  o Cyanosis
  o Periorbital edema

Neck
  o JVP is raised but not pulsatile
Thyroid (large retrosternal goiter) may be supraclavicular lymphadenopathy

Metastases

Chest
- Superior vena caval obstruction
  - The face is plethoric and cyanosed with periorbital edema
  - The eyes may show exophthalmos, conjunctival infection, and venous dilatation in the fundi
- Metastases
  - Effusion
  - Hemoptyses
  - Lobar collapse or volume loss
  - Pneumonia
  - Fixed inspiratory wheeze
  - Tender ribs (secondary deposits of tumour in the ribs)

Mediastinal compression
- Trachea
  - Stridor – respiratory distress
- Nerve compression
  - Sympathetic nerves - myosis, ptosis, anhydrosis, (Horner’s syndrome)
  - Left recurrent laryngeal nerve compression– hoarseness
  - Phrenic nerve-unilateral absent breath sounds; percussion dullness at affected based, with no inspiratory changes (paralysis of diaphragm)
- Superior Vena Cava (SVC)
  - Pemberton’s sign for SVC obstruction – lift arms, wait 1 minute for facial plethora, cyanosis, inspiratory stridor, non-pulsative elevation of JVP
  - Also eyes, face, neck as above

Skin
- Wasting of small muscles of hands
- Clubbing
- Tar staining of fingers
- Radiation marks
- Membranous glomerulonephritis
- Acanthosis nigricans
- Dermatomyositis (rare)

Liver metastases

Bone metastases
Endocrine
- Adrenal metastases
- Hypercalcaemia, due to secretion of parathyroid hormone like substances, occurs in squamous cell carcinoma
- Hyponatraemia-antidiuretic hormone is released by small (oat) cell carcinomas
- Ecopic ACTH syndrome (small cell carcinoma)
- Carcinoid syndrome (small cell carcinoma)
- Gynecomastic (gonadotrophins)
- Hypoglycaemia (insulin like peptide from squamous cell carcinoma).
- Dermatomyositis
- Acanthosis nigrans
- Herpes zoster
- Atrial fibrillation
- Pericarditis
- Non-bacterial thrombotic endocarditis
- Aortic aneurysm (rare)
- Thyrotoxicosis
- ↑ADH (↓Na)
- ↑PTH (↑Ca²⁺)
  - ↑ACTH (hypokalemic alkalosis, Cushing’s syndrome)
  - ↑Gonadotropins (gynecomastia)
  - ↑serotonin, carcinoid syndrome

Hematological features
- Migrating venous thrombophlebitis
- Disseminated intravascular coagulaton
- Anemia
- TTP

Kidney
- Nephritic syndrome (membranous glomerulonephritis)

Abbreviations: DIC, disseminated intravascular coagulopathy; TTP, thrombotic thrombocytopenic purpura

17. Perform a directed physical examination for Pancoast’s (superior pulmonary sulcus tumor) syndrome (often from cancer [often non-small cell] of the apex of the lung, infiltrating C8, T1, 2; may also occur with lymphoma, or by spread of lymph node metastases in breast or lung cancer).

- Shoulder pain radiating in the ulnar distribution in the arm
- Numbness of digits #4, 5
- Weakness of hand muscles innervated by ulnar nerve
- Radiologic destruction of ribs #1, 2
- Horner’s syndrome


Useful background: Malignant tumors which metastasize to lung or bone

![Diagram showing lung and bone metastases]

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 34.

**Pulmonary hypertension and Cor pulmonale**

Useful background: Cor Pulmonale

- Definition: Cor pulmonale is right ventricular enlargement due to the increase in afterload that occurs in diseases of the lung, chest wall or pulmonary circulation
- Causes
  - Obstructive lung disease
    - COPD
- Chronic asthma
  - Restrictive lung disease
    - Intrinsic
      - interstitial fibrosis
      - lung resection
    - Extrinsic
      - Obesity
      - muscle weakness
      - kyphoscoliosis
      - high altitude
  - Vascular disorders
    - Pulmonary emboli
    - Vasculitis (small pulmonary arteries)
    - ARDS
    - Primary pulmonary hypertension


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

- **Definition**
  - Systolic pulmonary artery (PA) pressure (SPAP) > 30 mm Hg
  - When SPAP > 50 mm Hg, symptoms occur

- **History**
  - SOBOE, fatigue, chest pain (in 50%, from RV ischemia)

- **Physical**
  - **General**
    - ↑RR (respiratory rate)
    - Peripheral cyanosis (↓ cardiac output [CO])
    - Cold extremities (↓ CO)
    - Hoarseness (PA compression of L. recurrent laryngeal nerve)
    - Dyspnea at rest, centrally cyanosis
    - Tar staining of the fingers
  - **Heart**
    - ↓ pulse volume (↓CO)
    - ↑ JVP both ‘a’ and ‘v’ waves are seen, ‘v’ waves being prominent if there is associated tricuspid regurgitation
    - RV heave, gallop
    - Palpable P₂, loud P₂ (forceful valve closure due to ↑ SPAP)
    - S₁
    - Pulmonary ejection murmur (PA dilation with turbulent flow)
- Pulmonary regurgitation (PA dilation)
  - Lung
    - R-CHF (cor pulmonale)
    - Bilateral wheeze, and other signs of COPD
    - Pansystolic murmur of tricuspid regurgitation
    - Early diastolic Graham Steell murmur in the pulmonary area
  - Abdomen
    - Look for signs of hepatomegaly
  - Legs
    - Pedal edema
  - Causes
    - Lung
      - Obstruction
      - Pulmonary emboli, blood clots, fat globules, tumor particles
    - COPD
    - Obstructive sleep apnea
    - Pulmonary fibrosis (interstitial lung disease)
    - Vasoconstriction secondary to hypoxia
      - High altitude
      - Chronic bronchitis
      - Kyphoscoliosis
      - Upper respiratory tract obstruction
    - Heart
      - L-CHF
      - Pulmonary venous hypertension (MS, atrial myxoma, cor triatriatum)
      - Congenital heart disease, L-R shunt (ASD, VSD, PPA)
    - Spine
      - Kyphoscoliosis
    - Ideopathic (primary)

Abbreviations: L-CHF, Left-sided congestive heart failure; R-CHF, Right-sided congestive heart failure; CO, Cardiac output; PA, pulmonary artery; SPAP, systolic pulmonary artery pressure; COPD, Chronic obstructive pulmonary disease; RR, Respiratory rate; PA, Pulmonary artery

**Acute respiratory distress syndrome (ARDS)**

19. Perform a focused physical examination for acute respiratory distress syndrome (ARDS).

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shock</td>
<td>o Any cause</td>
</tr>
<tr>
<td>Sepsis</td>
<td>o Lung infections, other bacteremic or endotoxic states</td>
</tr>
<tr>
<td>Trauma</td>
<td>o Hypotension, especially if prolonged, or with trauma</td>
</tr>
<tr>
<td></td>
<td>o The systemic inflammatory response syndrome (SIRS)</td>
</tr>
<tr>
<td></td>
<td>o Obstetric causes: amniotic fluid embolism, pre eclampsia</td>
</tr>
<tr>
<td></td>
<td>o High altitude related lung injury</td>
</tr>
<tr>
<td></td>
<td>o Head injury, lung contusion, fat embolism</td>
</tr>
<tr>
<td></td>
<td>o Gastric, near-drowning, tube feeding, Inhaled-O2, smoke</td>
</tr>
<tr>
<td>Aspiration</td>
<td>o Blood transfusions (especially if massive), leukoagglutinin, DIC (disseminated intravascular coagulation), thrombotic thrombocytopenic purpura</td>
</tr>
<tr>
<td>Hematologic</td>
<td>o Pancreatitis, uremia</td>
</tr>
<tr>
<td>Metabolic</td>
<td>o Narcotics, barbiturates, aspirin, “street drugs”</td>
</tr>
<tr>
<td>Drugs</td>
<td>o Chemicals-paraquat</td>
</tr>
<tr>
<td>Toxic</td>
<td>o Irritant gases- NO2, Cl2, SO2, NH3</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>Radiation, air embolism, altitude</td>
</tr>
</tbody>
</table>

> Appropriate setting
  - Pulmonary injury, shock, trauma
  - Acute event
  - Clinical respiratory distress, tachypnea

> Diffuse pulmonary infiltrates on chest radiography
  - Interstitial or alveolar pattern (or both)

> Hypoxemia
  - PaO2/F102 ratio <150
Exclude
- Chronic pulmonary disease accounting for the clinical features
- Left ventricular failure (most series require pulmonary artery wedge pressure <18 mm Hg)

Abbreviations: ARDS, Acute respiratory distress syndrome; BOOP, Bronchiolitis obliterans organizing pneumonia; COP, cryptogenic organizing pneumonia; F102, fraction of inspired oxygen


Useful background: Causes of hypoventilation and hypercapnic respiratory failure

- Central nervous system
  - Drugs
  - Hyperthyroidism
  - Ondine curse
  - Brainstem injury
  - Metabolic alkalosis

- Chest wall disorders
  - Kyphoscoliosis
  - Rib fractures
  - Pain
  - Flail chest
  - Respiratory muscle disease

- Diaphragm disorders
  - Rupture, myopathy

- Spinal cord & peripheral nervous system
  - Lesion at C3 to C5
  - Neuropathy
  - Trauma

- Muscular dysfunction
  - Muscular dystrophies
  - Guillain-Barre syndrome
  - Myasthenia gravis
  - Amyotrophic lateral sclerosis
  - Malnutrition
  - Acidosis
  - Hypoxemia
  - Anemia
  - Low cardiac output
  - Steroids
  - Aminoglycosides
  - Calcium channel blockers
  - Post paralytic condition
  - Detraining, atrophy, overuse fatigue
  - Increased workload
Respiratory failure

Useful background: Perform a focused physical examination for causes of disorder of ventilation

- Hypoventilation (hypercapnic respiratory failure)
- Hyperventilation

➢ CNS/Cord
  o Respiratory centre depression
  o Anxiety
  o Raised intracranial pressure
  o Hypercapnia
  o Brainstem injury
  o Spinal cord and peripheral nervous system
  o Lesion at C3 to C5
  o Neuropathy
  o Amyotrophic lateral sclerosis
  o Post-paralytic condition
  o Atrophy
  o Overuse fatigue

➢ Drugs
  o Causing ↓ cardiac output
  o Steroids
  o Aminoglycosides

➢ Lung
  o Respiratory muscle disease
  o Limited thoracic movement
  o Kyphoscoliosis
  o Elevated diaphragm, emphysema
  o Limited lung movement
  o Pleural effusion
  o Pneumothorax
  o Lung disease
  o Obstruction in upper or lower respiratory tract
  o Atelectasis, pneumonia
  o Trauma

➢ CNS
  o Anxiety, hysteria, pain
  o CNS lesions: meningitis, encephalitis, trauma, CVA

➢ Drugs
  o Salicylates, analeptics, adrenaline

➢ Lung
  o Pulmonary reflexes: irritant gases, atelectasis, pneumothorax
  o Artificial ventilation
- Metabolic
  - Hypothyroidism
  - Metabolic alkalosis
  - Acidosis
  - Malnutrition

- Muscle disease
  - Muscular dysfunction
  - Muscular dystrophies
  - Guillain-Barre syndrome
  - Myasthenia gravis


Useful background: Categorization of hypercapnia (↑PaO$_2$)

<table>
<thead>
<tr>
<th>Cause</th>
<th>Example</th>
<th>VE</th>
<th>VD/VT</th>
<th>A-a gradient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Defective central control of breathing</td>
<td>Drug overdose</td>
<td>↓</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Most causes of coma</td>
<td></td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td>Neuro-muscular disease</td>
<td>ALS</td>
<td>↓</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Spinal cord lesions</td>
<td></td>
<td>or ↑</td>
<td>or ↑</td>
</tr>
<tr>
<td></td>
<td>Myasthenia gravis</td>
<td></td>
<td>or ↑</td>
<td>or ↑</td>
</tr>
<tr>
<td></td>
<td>Guillian-Barre</td>
<td></td>
<td>or ↑</td>
<td>or ↑</td>
</tr>
<tr>
<td>Chest wall disease</td>
<td>Kyphoscoliosis</td>
<td>↓</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td>Large effusions</td>
<td></td>
<td>or ↑</td>
<td>or ↑</td>
</tr>
<tr>
<td>Primary lung disease</td>
<td>COPD</td>
<td>Normal or ↑</td>
<td>↑</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: ALS, amyotrophic lateral sclerosis; COPD, chronic obstructive pulmonary disease; VE, minute ventilation; VD, dead space; VT, tidal volume

Deep vein thrombosis

20. Take a directed history and perform a focused physical examination to determine the possible presence of a deep vein thrombosis (DVT).

Wells Scoring Scheme for pretest probability of deep vein thrombosis

<table>
<thead>
<tr>
<th>Clinical Feature</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Risk factors</td>
<td></td>
</tr>
<tr>
<td>o Active cancer</td>
<td>1</td>
</tr>
<tr>
<td>o Paralysis, paresis or recent plaster immobilization of the lower extremities</td>
<td>1</td>
</tr>
<tr>
<td>o Recently bedridden &gt; 3 days or major surgery within 4 weeks</td>
<td>1</td>
</tr>
<tr>
<td>➢ Signs</td>
<td></td>
</tr>
<tr>
<td>o Localized tenderness along the distribution of the deep venous system</td>
<td>1</td>
</tr>
<tr>
<td>o Entire leg swollen</td>
<td>1</td>
</tr>
<tr>
<td>o Asymmetric calf swelling (&gt;3 cm difference, 10 cm below tibial tuberosity)</td>
<td>1</td>
</tr>
<tr>
<td>o Asymmetric pitting edema</td>
<td></td>
</tr>
<tr>
<td>o Collateral superficial veins (nonvaricose)</td>
<td></td>
</tr>
<tr>
<td>➢ Alternative diagnosis</td>
<td></td>
</tr>
<tr>
<td>o Alternative diagnosis as likely or more likely than deep venous thrombosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>-2</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
</tr>
</tbody>
</table>

*Interpretation of score: High probability if 3 points or more, moderate probability if 1 or 2 points and low probability if 0 points or less.


Note that a number of historical features are not given points for the calculation of the wells score for DVT; these include:

- Stasis – immobilization, right-sided heart failure, obstruction, shock
- Hypercoagulability, estrogen use, pregnancy, neoplasms, tissue trauma, nephritic syndrome, deficiency of antithrombin III, protein C or S
- Endothelial damage – venulitis, trauma
- Symptoms suggesting pulmonary emboli (dyspnea, pleuritic chest pain, and hemoptysis)
<table>
<thead>
<tr>
<th>Pretest probability</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Positive LR</th>
<th>Probability of DVT (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low (0)</td>
<td>2-21</td>
<td>36-77</td>
<td>0.2</td>
<td>5</td>
</tr>
<tr>
<td>Moderate (1-2)</td>
<td>13-39</td>
<td>...</td>
<td>NS</td>
<td>17</td>
</tr>
<tr>
<td>High (≥ 3)</td>
<td>38-87</td>
<td>71-96</td>
<td>5.2</td>
<td>53</td>
</tr>
</tbody>
</table>

Abbreviation: NS, not significant.


Useful background: Likelihood ratios for pulmonary embolus for the combination of clinical probability estimate with the D-dimer result

<table>
<thead>
<tr>
<th>Clinical probability</th>
<th>D-dimer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any probability</td>
<td>Abnormal</td>
</tr>
<tr>
<td>Low (&lt;15%) to moderate (15%-35%)</td>
<td>Normal</td>
</tr>
</tbody>
</table>

Abbreviation: CI, confidence interval; LR, likelihood ratio


Useful background: Simplified Wells Scoring System

<table>
<thead>
<tr>
<th>Findings in the simplified Wells Scoring system</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical signs/symptoms of DVT of the leg (minimum of leg swelling and pain with palpation of the deep veins)</td>
<td>3.0</td>
</tr>
<tr>
<td>No alternate diagnosis that is as likely as or more likely than a pulmonary embolus</td>
<td>3.0</td>
</tr>
<tr>
<td>Heart rate &gt; 100/min</td>
<td>1.5</td>
</tr>
<tr>
<td>Immobilization or surgery in the last 4 weeks</td>
<td>1.5</td>
</tr>
<tr>
<td>History of DVT or PE</td>
<td>1.5</td>
</tr>
<tr>
<td>Hemoptysis</td>
<td>1.0</td>
</tr>
<tr>
<td>Cancer actively treated in the past 6 months</td>
<td>1.0</td>
</tr>
</tbody>
</table>

Abbreviations: DVT, Deep Vein thrombosis; PE, pulmonary embolism
Category scores determined by the sum of the individual scores: low, <2; moderate, 2-6; high, >6.


Useful background: Probability of deep vein thrombosis after first determining the clinical probability and then obtaining the D dimer result

<table>
<thead>
<tr>
<th>Clinical probability estimates(^a)</th>
<th>High probability (~50%)</th>
<th>Moderate Probability (~20%)</th>
<th>Low probability (~5%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>High sensitivity D-dimer</td>
<td>Positive 63</td>
<td>25</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>Negative 8.6</td>
<td>1</td>
<td>0.5</td>
</tr>
<tr>
<td>Moderate sensitivity D-dimer</td>
<td>Positive 67</td>
<td>34</td>
<td>17</td>
</tr>
<tr>
<td></td>
<td>Negative 19</td>
<td>4.4</td>
<td>0.9</td>
</tr>
</tbody>
</table>

Abbreviation: DVT, Deep vein thrombosis

\(^a\) Values in the Table use the exact summary pretest probability estimates, but a clinician might simplify by remembering that a high probability is about 50%, moderate probability 20% and low probability 5%.


**Pulmonary embolism**

Useful background: ECG changes in pulmonary embolism. These include:

- Sinus tachycardia
- Tall R wave in lead VI
- S1, S2, S3 syndrome (S waves in limb leads I, II and III)
- S1, Q3, T3 syndrome (S in limb lead 1 and Q wave and inverted T wave in limb lead III)

Useful background: Modified Wells criteria for pre-test probability of pulmonary embolism

<table>
<thead>
<tr>
<th>Clinical feature</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Clinical symptoms of DVT</td>
<td>3.0</td>
</tr>
<tr>
<td>➢ Other diagnosis less likely than PE</td>
<td>3.0</td>
</tr>
<tr>
<td>➢ Heart rate &gt; 100</td>
<td>1.5</td>
</tr>
<tr>
<td>➢ Immobilization &gt; 3 days or surgery in past 4 weeks</td>
<td>1.5</td>
</tr>
<tr>
<td>➢ Previous PE or DVT</td>
<td>1.5</td>
</tr>
<tr>
<td>➢ Hemoptysis</td>
<td>1.0</td>
</tr>
<tr>
<td>➢ Malignancy (current or recent)</td>
<td>1.0</td>
</tr>
</tbody>
</table>

Interpretation: Score

<table>
<thead>
<tr>
<th>Score</th>
<th>Probability of PE</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;6.0</td>
<td>High</td>
</tr>
<tr>
<td>2-6</td>
<td>Moderate</td>
</tr>
<tr>
<td>&lt;2.0</td>
<td>Low</td>
</tr>
</tbody>
</table>

Source: Likelihood of Pulmonary embolism according to scan category and clinical probability in PIOPED study JAMA 1990;263:2753

Simplified clinical model

- Active cancer (treatment ongoing or within previous 6 months or palliative) 1
- Paralysis, paresis, or recent plantar immobilization of lower extremity 1
- Recently bedridden for >3 days or major surgery within 4 weeks 1
- Localized tenderness along the distribution of the deep venous system 1
- Entire leg swelling -2
- Calf swelling >3cm, compared to other calf (10cm below tibial tuberosity) 1
- Pitting edema (greater in symptomatic leg) 1
- Collateral superficial veins (nonvaricose) 1
- Alternate diagnosis as likely or greater than DVT 1

Total
Useful background: EBM- carotid artery stenosis and deep vein thrombosis

- Gold standard for diagnosing DVT is venography, however it is invasive. Compression ultrasonography is highly sensitive, specific for detecting proximal DVTs and is less invasive.
- A simplified clinical model was generated to stratify individuals into pretest categories of low (score <0), moderate (score 1-2), or high risk (score >3) for DVT. Combining this with compression ultrasonography determined positive likelihood ratios.
- In patients with TIA, a carotid bruit indicated the presence of a >50% stenosis of the carotid artery (confirmed by carotid angiography) with 29% sensitivity and 88% specificity.

Source: Likelihood of Pulmonary embolism according to scan category and clinical probability in PIOPED study *JAMA* 1990;263:2753.

**Pneumothorax**

21. Take a focused history for the causes of pneumothorax.

- Traumatic
- Iatrogenic
  - Thoracentesis, thoracic surgery
  - Artificial pneumothorax
  - Cervical surgery, stellate block etc
- Spontaneous
  - *Localized air space disorder*
    - Congenital bullae
    - Localized emphysema
    - Acquired cysts, etc
  - *Generalized emphysema*
  - *Secondary to specific lung disease*
    - Congenital
    - COPD (emphysematous bulla)
    - Diffuse cystic disease (CF)
    - Bronchiectasis; eosinophilic granuloma; tuberose sclerosis,
      - Silicosis
  - Infection
    - TB
    - Lung abscess
    - Malignancy
    - Hydatid cysts
Secondary to spontaneous mediastinal emphysema
- Asthma
- Labour
- Straining at stool
- Rapid decompression of divers

Associated with menstruation
- Endometriosis


**Lung abscess**

22. Take a focused history for the causes of lung abscess.

- **Aspiration**
  - CNS
    - Coma, anesthesia, alcoholic debauch
  - Mouth
    - Oral or pharyngeal sepsis
    - Pharyngeal pouch
    - Esophageal obstruction, trachesophageal fistula
    - Drowning
    - Foreign body

- **Lung disease**
  - Infection
    - TB
    - Straphylococcal
    - Friedlander’s (klebsiella)
    - Actinomycosis
    - Entameba histolytica, and other fungi
    - Secondary infection of pulmonary infarct
    - Septic emboli due to pyemia
    - Parasites, eg schistosomiasis
  - Infiltration
    - Necrotic bronchial carcinoma
    - Benign tumor
  - Ideopathic
    - Pulmonary fibrosis (interstitial lung disease)
  - Inherited/ acquired cysts
  - Miscellaneous
    - Arteritis
    - Mucoviscidosis

**Bronchiectasis**

Useful background: Diseases associated with bronchiectasis

<table>
<thead>
<tr>
<th>General clinical features</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cystic fibrosis</strong></td>
</tr>
<tr>
<td>o Due to malfunction of the gene coding for the CF transmembrane conductance regulator (CFTR) protein. Usually diagnosed at a young age</td>
</tr>
<tr>
<td>o Lung disease often dominates the clinical pictures.</td>
</tr>
<tr>
<td>o Malabsorption very common; cirrhosis, azoospermia, etc</td>
</tr>
<tr>
<td><strong>Kartegeners syndrome</strong></td>
</tr>
<tr>
<td>o Due to mutation in gene coding for dynein protein</td>
</tr>
<tr>
<td>o Ciliary dysmotility, resulting in sinusitis, situs inversus, infertility in men</td>
</tr>
<tr>
<td><strong>Young’s syndrome</strong></td>
</tr>
<tr>
<td>o Triad of bronchiectasis, rhinosinusitis and decreased fertility (obstructive azoospermia) due to abnormally viscous mucus</td>
</tr>
<tr>
<td><strong>Immune defects</strong></td>
</tr>
<tr>
<td>o IgA deficiency: repeated respiratory tract infections, 25% develop autoimmune conditions (eg RA, SLE, celiac disease) IgM deficiency</td>
</tr>
<tr>
<td>o Recurrent infancy/childhood infections with encapsulated organisms. Later on, autoimmune illnesses and malignancy</td>
</tr>
<tr>
<td><strong>Allergic bronchopulmonary aspergillosis (ABPA)</strong></td>
</tr>
<tr>
<td>o Usually complicates long standing asthma, leading to worsening of asthma symptoms</td>
</tr>
<tr>
<td>o Transient pulmonary infiltrates on CXR</td>
</tr>
<tr>
<td>o Often with eosinophilia</td>
</tr>
<tr>
<td><strong>Rheumatoid arthritis</strong></td>
</tr>
<tr>
<td>o Bronchiectasis can occur before overt arthritis, though more common during overt RA</td>
</tr>
<tr>
<td><strong>Alpha-1 antitrypsin deficiency</strong></td>
</tr>
<tr>
<td>o Chest disease, especially in smokers, at a young age.</td>
</tr>
<tr>
<td>o Symptomatic liver disease (cirrhosis) occurs at a young age</td>
</tr>
</tbody>
</table>
Marfans syndrome

- Family history of premature sudden cardiac death, due to aortic dissection
- Aortic root enlargement; dissection
- Tall, joint hypermobility, lens dislocation

Extrinsic allergic alveolitis (hypersensitivity pneumonitis)

Useful background: Causes of pulmonary eosinophilia and vasculitis

- Lung
  - Asthma
  - ABPA
    - Chronic eosinophilic pneumonia
- Drugs
- Infection
  - Parasites
    - Loffler’s Syndrome (eosinophilia transient CXR infiltrate lasting 4-6 weeks, often related to bugs, parasites or worms)
- Connective tissue disorders

Abbreviations: ABPA, Allergic bronchopulmonary aspergillosis; CXR, Chest x-ray

Adapted from: Davey P. Wiley-Blackwell 2006, page 204.

Useful background:

- Distinguishing between acute and chronic extrinsic allergic alveolitis (EAA)

<table>
<thead>
<tr>
<th></th>
<th>Acute</th>
<th>Chronic</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC, ESR</td>
<td>Ground glass</td>
<td>May be normal</td>
</tr>
<tr>
<td>Chest x ray</td>
<td>Multiple nodules</td>
<td>Fibrosis</td>
</tr>
<tr>
<td>CT chest</td>
<td></td>
<td>Fibrosis</td>
</tr>
<tr>
<td>ABG</td>
<td>Type 1 respiratory</td>
<td></td>
</tr>
<tr>
<td>Lung function tests</td>
<td>failure</td>
<td>50-60%</td>
</tr>
<tr>
<td>Steroid response</td>
<td>80-90%</td>
<td>Very poor</td>
</tr>
<tr>
<td>Prognosis</td>
<td>Very good</td>
<td>Poor</td>
</tr>
</tbody>
</table>

Presence of precipitins implies exposure but not necessarily disease

Abbreviations: ABG, Arterial blood gases; EAA, Extrinsic allergic alveolitis
Pulmonary eosinophilic disorders

Löffler’s syndromes

Transient pulmonary infiltrates
  o Peripheral eosinophilia
  o Associated with parasitic infections, drug allergies and exposure to inorganic chemicals (such as nickel carbonyl)

Course is benign and respiratory failure almost unknown

Eosinophilia in asthmatics
  o The most common cause is allergic bronchopulmonary aspergillosis

Tropical eosinophilia which is secondary to filarial infection (Wuchereria bancrofti or W. malayi Brug)

Churg Strauss syndrome
  o Diagnosis requires four of the following features
    - Asthma
    - Eosinophilia greater than 10%
    - Mononeuropathy or polyneuropathy
    - Paranasal sinus abnormality
    - Non-fixed pulmonary infiltrates visible on chest x-ray
    - Blood vessels with extravascular eosinophils found on biopsy

Chronic eosinophilia pneumonia
  o Chronic debilitating illness characteristic malaise, fever, weight loss and dyspnea. The chest radiograph shows a peripheral alveolar filling infiltrate predominantly in the upper lobes (the graphic negative of pulmonary edema)


Useful background: Causes of fibrosing alveolitis

Primary

Secondary
  o Rheumatoid arthritis
  o Systemic lupus erythematosus
  o Scleroderma
  o Dermatomyositis
  o Chronic extrinsic allergic alveolitis

Useful background: Investigations to distinguish between acute and chronic fibrosing alveolitis

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<thead>
<tr>
<th></th>
<th>Acute</th>
<th>Chronic</th>
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<tr>
<td>Lung function test</td>
<td>- Restrictive defect</td>
<td>- Restrictive defect</td>
</tr>
<tr>
<td>defect</td>
<td></td>
<td></td>
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<td>Chest x ray</td>
<td>- Ground glass</td>
<td>- Honey comb</td>
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<tr>
<td>CT scan</td>
<td>- ‘Alveolitis’</td>
<td>- ‘fibrosis’</td>
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<tr>
<td>ABG</td>
<td>- Type 1 failure</td>
<td>- Early: normal at rest, ↓ pO2 on exercise</td>
</tr>
<tr>
<td>RF/ANA</td>
<td></td>
<td>- Late: type I failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- 30-50%</td>
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Abbreviations: RF/ANA, Rheumatoid factor/antinuclear antibody; ABG, arterial blood gases


23. Take a directed history and perform a focused physical examination for fibrosing alveolitis.

- **History**
  - Progressive exertional dyspnea (90%)
  - Chronic cough (74%)
  - Arthralgia/ arthritis (19%)
  - Obtain a drug history (amiodarone, nitrfurantoin and busulfan)

- **Physical examination**
  - **Chest**
    - Bilateral, basal, fine, end-inspiratory crackles which disappear or become quieter on leaning forwards
    - The “velco-like” crackles do not disappear on coughing (unlike those of pulmonary edema)
    - Tachypnea (in advanced cases)
  - **Hands** (for rheumatoid arthritis, systemic sclerosis)
    - Clubbing
  - **Face** (for typical rash of SLE, heliotropic rash of dermatomyositis, typical facies of systemic sclerosis, lupus pernio of sarcoid)
    - Central cyanosis
  - **Mouth** (for aphthous ulcers of Crohn’s disease, dry mouth of Sjögren’s syndrome)
  - **CVS**
    - Signs of pulmonary hypertension: ‘a’ wave in the JVP, left parasternal heave and $P_2$
Examine patient for conditions which have similar pulmonary changes:
- Rheumatoid arthritis, SLE, dermatomyositis, chronic active hepatitis, ulcerative colitis, systemic sclerosis
- Pneumoconiosis
- Granulomatous disease; sarcoid, TB
- Chronic pulmonary edema
- Radiotherapy
- Lymphangitis carcinomatosa
- Extrinsic allergic alveolitis: farmer’s lung, bird fancier’s lung


Useful background: Causes of pulmonary eosinophilia

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<td>Segmental bilateral peripheral ground glass infiltrates on CT</td>
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<td>Restrictive lung function</td>
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<tr>
<td>Hypereosinophilic syndrome</td>
<td>Eosinophilia &lt;20 × 10⁹/L</td>
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<td>Pulmonary infiltrates and effusions</td>
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<td></td>
<td>Myocardial infiltration and CHF</td>
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<tr>
<td>Churg-Strauss syndrome</td>
<td>↑ IgE</td>
</tr>
<tr>
<td></td>
<td>pANCA (positive in 50%)</td>
</tr>
<tr>
<td></td>
<td>Pulmonary infiltrates</td>
</tr>
<tr>
<td></td>
<td>Pleural effusion</td>
</tr>
<tr>
<td></td>
<td>Vasculitis on biopsy (multisystem disease)</td>
</tr>
</tbody>
</table>


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

Q: In which condition is clubbing associated with pulmonary crackles?
A:  
- Bronchogenic carcinoma (crackles are localized)
- Bronchiectasis (coarse crackles)
- Asbestosis
- Fibrosing alveolitis (fine, end-expiratory, not disappearing on coughing, but disappear on leaning forward)

Chest X-ray

Useful background: Chest X-ray

- Features that differentiate between the left and right hemidiaphragms in a lateral film
  - The right hemidiaphragm is usually higher than the left
  - The left hemidiaphragm is silhouetted out by the heart
  - The gastric air bubble is below the left hemidiaphragm
  - The right ribs are usually magnified since they are farther away from the film than the left ribs, so the right hemidiaphragm is the hemidiaphragm that meets the right ribs.

- An apical loidotic view is useful when the right clavicle and first rib hinder visualization on the PA film
  - Air-space disease
    - A pathological process affecting primarily the alveoli.
    - The radiological findings are Acinar shadows; air bronchograms; silhouette sign
  - Fluid (e.g., pulmonary edema); pus (e.g., pneumonia); cells (e.g., lung cancer); blood (e.g., hemorrhage); proteins (e.g., alveolar proteinosis)
  - Interstitial lung disease
    - A pathological process affecting primarily the interstitium of the lung. The radiological findings include a reticular pattern (net-like), a nodular pattern (nodules), or both
    - A differential diagnosis includes Pulmonary edema; military tuberculosis; pneumoconiosis; sarcoidosis


- Silhouette sign (actually, the "loss of silhouette")
  - The loss of normally appearing interfaces
  - Causes
    - RML consolidation – loss of right heart border
    - Lingula consolidation – loss of left heart border
    - Anterior segment of left upper lobe – loss of aortic arch

Useful background: Systematic approach to reading a PA and lateral chest x-ray

- **General**
  - Date, name, age, sex
  - State types of studies PA and lateral
  - Obtain previous films for comparison
  - Quality of film
  - Rotation
  - Exposure
  - Inspiration
  - Any obvious abnormalities

- **Bones and joints** (fractures, arthritis)
  - Anterior and posterior ribs
  - Vertebral column
  - Clavicles
  - Scapulae

- **Soft tissues** (calcifications, subcutaneous emphysema)
  - Axillae
  - Breast shadows (e.g., mastectomy)
  - Pleura
  - Major and minor fissures
  - Costovertebral angles

- **Diaphragm**
  - Level
  - Right and left hemidiaphragm
  - Abdominal free air

- **Heart**
  - Size (shouldn’t be > 50% the size of the cardiothoracic ratio)
  - Calcifications
  - Atrial/ventricular enlargement

- ** Mediastinum**
  - Position of trachea, aortic arch, right heart border

- **Hila**
  - Size
  - Compare right and left hilum
  - Upward/downward displacement

- **Lung parenchyma**
  - Nodules
  - Parenchymal density
  - Vascular abnormalities (e.g., redistribution)

- **Abdomen**
  - Free air
  - Gastric air bubble

Clinical Anatomy

Provided through the courtesy of: Dr. A. Leung
Useful background: An example of a normal chest x-ray (CHR)

- Male patient. CXR.
- PA and lateral views.
- No date or name available.
- The film is technically adequate.
- Cardiac-thoracic ratio approximately 30%.
- No obvious cardiac chamber enlargement. Right and left hilar contours preserved.
- No obvious lymphadenopathy, vessel enlargement, or masses.
- Normal AP window.
- Contour of descending aorta normal.
- Sharp costophrenic angles.
- Lung volumes preserved with normal lung markings.
- No obvious lung lesions or consolidative changes.
- Soft tissue and boney structures normal.
- Right hemidiaphragm slightly elevated.
- Gastric bubble noted inferior to left hemidiaphragm.
- Final interpretation: normal CXR.
Useful background: lung fields on inspiration, lobes and fissures.

- Adequate inspiration - should be able to see the lung fields well: 6-8 anterior, 9-11 posterior; penetration: should be able to see the spine behind the heart.
- Pay careful attention to the pulmonary lobes and fissures.

Provided through the courtesy of: Dr. A. Leung
Case studies

Case one: Please describe the findings, give a differential, and state your most likely diagnosis.

- 75 year old man in ER with dyspnea

- Interstitial and airspace changes
- Cardiomegaly
- Ill-defined bronchovascular markings
- Peribronchial cuffing;
- Small pleural effusions
- Fissural fluid and thickening

Final diagnosis: pulmonary interstitial edema
Case two: Please describe the findings, give a differential, and state your most likely diagnosis.

- Chest x-ray finding in the patient with congestive heart failure (CHF)
  - Kerley B & A septal lines
  - Fissure lines
  - Pleural effusions
  - Peribronchial cuffing
  - Consolidative changes (bat-wings appearance)
  - Vascular redistribution (cephalization)
  - Cardiomegaly
  - DDx pulmonary edema:
    - HF, sepsis, renal failure, ARDS, lymphangitis carcinomatosis, CHF

Case three: Please describe the findings, give a differential, and state your most likely diagnosis.

- 29 year old with asthma presenting with dry cough

  ![Chest X-Ray](image)

- Differential diagnosis of lymphadenopathy seen on chest x-ray
  - Sarcoidosis
  - TB
  - Cancer (mets and lymphoma)
  - Silicosis
  - Pulmonary hypertension
Case four: Please describe the findings, give a differential, and state your most likely diagnosis.

- 60 year old with worsening exertional dyspnea

- Chest x-ray findings suggestive of pleural effusions
  - Blunted costophrenic angle
  - Opacification of diaphragm
  - Usually dependent, mobile, “meniscus” sign
  - Lateral view more sensitive
  - Supine: hazy ‘veiling’

- Differential diagnosis
  - Volume overload states (CHF, cirrhosis, renal failure)
  - Parapneumonic effusion from pneumonia
  - Infections (empyema)
  - Malignancy (especially with isolated left-sided effusions)
  - Pancreatitis
  - Hypothyroidism
Case five: Please describe the findings, give a differential, and state your most likely diagnosis.

- 17 year old with asthma presenting with sudden dyspnea

- Chest x-ray findings suggestive of pneumothorax
  - Beware the normal CHX:
    - Always suspect a pneumothorax when you’re given an x-ray on an exam that initially looks normal. With your finger, draw a very careful line around the pleural edges. Look for the following:
      - Pleural reflection line, no lung markings beyond
      - Sign of tension: mediastinal shift
  - Pneumothoraces “pop-up” commonly on exams:
    - Young, thin & slim men with asthma...
    - Older men with a history of emphysema...
    - Mechanically ventilated patients with acute hypoxia...
Case six: Please describe the findings, give a differential, and state your most likely diagnosis.

- Asymptomatic 40 year old 25 pack year smoker

- Lung nodules’ seen on chest x-ray
  - Size & doubling time ( < 30 days or > 2 years = good)
  - Borders (round & smooth = likely good; spiculated = likely bad)
  - Cavitation (necrosis)
  - Calcification (central/complete = TB/histoplasma; popcorn = benign)
  - Vascular markings (AVM)
  - Associated lesions/lymphadenopathy/collapse
  - Nodules < 3 cm; masses > 3 cm in diameter
Case seven: Please describe the findings, give a differential, and state your most likely diagnosis.

- Multiple lung nodules seen on chest x-ray
  - Neoplastic
    - Benign (hamartomas, cysts)
    - Malignant (mets, lymphoma, Kaposi’s sarcoma, bronchoalveolar cancer)
  - Infectious
    - Granulomas (TB, histoplasmosis)
    - Septic emboli
    - Viral pneumonias (measles, chickenpox)
  - Non-infectious, non-malignant
    - Rheumatoid nodules
    - Sarcoid
    - Wegener’s granulomatosis
    - Infarcts
    - Round atelectasis
Case eight: Please describe the findings, give a differential, and state your most likely diagnosis.

- 50 year old with 3 day history of fever and chills, with productive cough

- Chest x-ray findings suggestive of right middle lobar consolidation
  - RML: abutment of major/minor fissures, silhouettes right heart border; easiest to identify on lateral view

- Differential diagnosis:
  - Infection
  - Aspiration
  - Tumor-mass
  - Tuberculosis
  - Interstitial pneumonia
  - Consolidation does not usually cause volume loss, unlike atelectasis; look at the fissure lines and surrounding structures for help (diaphragm and mediastinal structures)
  - Look for air bronchograms

- Chest x-ray findings suggestive of RML collapse
  - Right-sided volume loss
  - Hazy mid thoracic density obscuring right heart border
  - Wedge-shaped opacity on lateral film often not seen on frontal view except for volume loss with inferior displacement of minor fissure
...and worsens

- Chest x-ray findings suggestive of RLL collapse and effusion
  - Lower and posterior zone opacity
  - Posterior displacement of major fissure
  - The right heart border is not obscured
  - The medial diaphragm is obscured
Case nine: Please describe the findings, give a differential, and state your most likely diagnosis.

- 70 year old with bronchiectasis presents with cough, dyspnea and pleuretic chest pain

- Chest x-ray findings suggestive of right upper lobar disease collapse
  - Collapse
    - Elevation of minor fissure and right hilum
    - Wedge-shaped opacity at right lung apex
  - Sometimes, you’ll see the “Golden S sign” if there is a mass causing the RUL collapse
  - Consolidation
    - Normal position of minor fissure
Case ten: Please describe the findings, give a differential, and state your most likely diagnosis.

- 30 year old with a 4 day history of cough and dyspnea

- Things to remember about right lobar consolidation
  - RML
    - Silhouettes right heart border
    - Easiest to see on lateral view
  - RLL
    - Difficult to differentiate from RML consolidation on PA film
    - Cardiac silhouette preserved
  - RUL
    - Look for triangle in right apex
Case eleven: Please describe the findings, give a differential, and state your most likely diagnosis.

- 65 year old with a 10 day history of productive cough, dyspnea and pleuritic chest pain
Pay careful attention to the pulmonary lobes and fissures

This patient has lingular consolidation
- Lingular consolidation
- Small left pleural effusion
- Azygos lobe fissure
Case twelve: Please describe the findings, give a differential, and state your most likely diagnosis.

- 80 year old with a 50 year pack history presenting with fever, night sweats and weight loss

- Chest x-ray findings suggestive of LUL collapse
  - The toughest one to diagnose.
  - **PA**
    - Volume loss (elevation of L hemidiaphragm)
    - Luftsichel sign (LUL has retracted medically and superiorly. The hyperinflated LLL produces a crescent of lucency along the mediastinum and aortic knuckle)
  - **Lateral**
    - Anterior displacement of major fissure (/\ to sternum)
  - **Golden S sign**
    - Obstructing hilar mass (RUL or LUL)

(This man most likely has lung cancer with secondary LUL collapse)
Case thirteen: Please describe the findings, give a differential, and state your most likely diagnosis.

- 55 year involved in a MVA accident one year ago, when his CHX was normal

- Chest x-ray findings suggestive of elevation of a hemidiaphragm
  - Normal R hemidiaphragm is ½ ICS higher than L in 90%
  - DDX of elevated hemidiaphragm: The right hemidiaphragm is elevated

- Differential diagnosis
  - Diaphragmatic paralysis
  - Eventration of the diaphragm (R:L = 5.1)
  - Atelectasis
  - Subpulmonic effusion
  - Abdo disease (subphrenic abscess, liver mass, hernia)
  - Diaphragmatic rupture
Case fourteen: Please describe the findings, give a differential, and state your most likely diagnosis.

- “Routine” chest x-ray in a 55 year old asymptomatic woman with treated myasthenia gravis

- Chest x-ray findings suggestive of anterior mediastinal masses

- If you are given an x-ray showing an anterior mediastinal mass, comment on:
  - Shape
  - Associated lung lesions (nodules, masses)
  - Surrounding structures (obscures ascending aorta, retrosternal airspace)

- Differential diagnosis (6 T’s)
  - Thymoma
  - Thymic hyperplasia
  - Thyroid
  - Teratoma
  - Terrible lymphoma
  - Tumor

This lady also has a posterior mediastinal mass: final Dx turned out to be metastatic malignant thymoma.
Case fifteen: Please describe the findings, give a differential, and state your most likely diagnosis.

- 50 year old homeless man with productive cough, decreased LOC and wasting

- Chest x-ray findings suggestive of apical fibrosis
  - Right sided volume loss with apical fibrosis
  - Right sided lung nodule with central cavitation
  - Right sided mediastinal lymphadenopathy

- Differential diagnosis
  - Active TB
  - Talcosis
  - Sarcoidosis
  - Fibrosis secondary to recurrent pneumonias
# Suggested practice case scenarios for OSCE examinations

<table>
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<tr>
<th>Primary Stem</th>
<th>Secondary Stem</th>
<th>Diagnosis</th>
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<td>Dyspnea</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Post operative</td>
<td></td>
<td>Pulmonary embolism</td>
</tr>
<tr>
<td>o Six week post acute MI</td>
<td></td>
<td>CHF</td>
</tr>
<tr>
<td>o With fever &amp; productive cough in young person</td>
<td></td>
<td>Community acquire pneumonia</td>
</tr>
<tr>
<td>o With fever &amp; productive cough in demented person</td>
<td></td>
<td>Aspiration Pneumonia</td>
</tr>
<tr>
<td>o With fever in MHSM</td>
<td></td>
<td>Pneumocystis</td>
</tr>
<tr>
<td>o With purulent sputum and clubbing</td>
<td></td>
<td>Bronchiectasis/Cystic fibrosis</td>
</tr>
<tr>
<td>o Acute onset in elderly person with palpitations</td>
<td></td>
<td>Atrial fibrillation</td>
</tr>
<tr>
<td>o Fever and pleural effusion</td>
<td></td>
<td>Parapneumonic effusion/empyema</td>
</tr>
<tr>
<td>o Advanced COPD</td>
<td></td>
<td>Cor Pulmonale</td>
</tr>
<tr>
<td>o Young person</td>
<td></td>
<td>Primary pulmonary HTN</td>
</tr>
<tr>
<td>o Exertional in young person</td>
<td></td>
<td>Myocarditis</td>
</tr>
<tr>
<td>Hemoptysis</td>
<td></td>
<td></td>
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<tr>
<td>o With fever in Asian immigrant</td>
<td></td>
<td>TB</td>
</tr>
<tr>
<td>o Chronic smoker</td>
<td></td>
<td>Ca Lung</td>
</tr>
<tr>
<td>o Chronic Smoker</td>
<td></td>
<td>Bronchitis</td>
</tr>
<tr>
<td>o With fever, epistaxis &amp; renal failure</td>
<td></td>
<td>Wegeners</td>
</tr>
<tr>
<td>Abnormal CXR</td>
<td></td>
<td></td>
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<tr>
<td>o Young female with hot nodules in legs</td>
<td></td>
<td>Sarcoidosis</td>
</tr>
<tr>
<td>Chest X-ray</td>
<td></td>
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<tr>
<td>o Solitary pulmonary nodule in 68 yr old</td>
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<td>Ca Lung</td>
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Source: Kindly provided by Dr. P Hamilton (U of Alberta)
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OSCE Questions in Rheumatology Chapter

1. Take a directed history for a musculoskeletal (MSK) disorder.
2. Perform a focused physical examination of the joints of the upper and lower body and from the abnormal articular findings, give the most likely diagnosis.
3. Take a directed history for the common side effects of nonsteroidal anti-inflammatory drugs.
4. Perform a focused physical examination of the elbow.
5. Take a directed history and a focused physical examination for features differentiating diseases affecting the elbow.
6. Perform a focused physical examination of the shoulder.
7. Perform a focused physical examination for causes of shoulder pain.
8. Take a directed history of back pain.
9. Perform a focused physical examination of back pain.
10. Perform a focused physical examination of the hip.
11. Perform a directed physical examination of the knee.
12. Perform a focused physical examination of the ankle.
13. Perform a focused physical examination of the feet.
14. Perform a focused physical examination for complications of rheumatoid arthritis (RA), and its complications.
15. Take a directed history and perform focused physical examination to distinguish rheumatoid arthritis from osteoarthritis.
16. Perform a focused physical examination for diseases that may have positive rheumatoid factor.
17. Perform a focused physical examination for the causes of spondyloarthritis.
18. Take a directed history for ankylosing spondylitis.
19. Perform a focused physical examination for ankylosing spondylitis.
20. Perform a focused physical examination for primary vs secondary osteoarthritis.
21. Perform a focused physical examination for psoriatic arthritis.
22. Perform a focused physical examination for systemic lupus erythematosus (SLE) and its complications.
23. Perform a focused physical examination for scleroderma and its complications.

24. Perform a directed physical examination for Raynaud’s phenomenon (white->blue->red fingers/toes in response to cold temperature).

25. Take a directed history and perform a focused physical examination for systemic vasculitis.

26. Take a directed history and perform a focused physical examination for the causes of vasculitis.

27. Take a directed history and perform a focused physical examination for causes of polymyalgia rheumatica-like syndromes.

28. Take a directed history of the cause of aseptic necrosis of the bone. (acronym: ASEPTIC)

29. Perform a directed physical examination for Charcot’s joint (neuroarthropathy).

30. Perform a focused physical examination for polymyositis/dermatomyositis.

31. Perform a focused physical examination for Marfan’s syndrome.
**Introduction**

1. Take a directed history for a musculoskeletal (MSK) disorder.

- **Joints**
  - Pain and stiffness
  - Weight-bearing, activity, time of day
  - Swelling and deformity
  - Motor
    - Weakness, instability, falls
  - Sensation
    - Functional assessment
  - Gait

- **Extra-articular**
  - Dry mouth
  - Dry, red eyes
  - Ulcers
  - Raynaud’s phenomenon
  - Rash
  - Fatigue, weight loss, fever
  - Diarrhea

*hip pain may be referred to knee or lower thigh*


**Useful background: Definition of abnormal physical findings in MSK system**

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<th>Definition of Abnormal Finding</th>
</tr>
</thead>
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<td></td>
</tr>
<tr>
<td>- Weak thumb abduction</td>
<td>weakness of resisted abduction, ie, movement of the thumb at right angles to the palm</td>
</tr>
<tr>
<td>- Thenar atrophy</td>
<td>a concavity of the thenar muscles when observed from the side</td>
</tr>
<tr>
<td><strong>SENSORY EXAMINATION</strong></td>
<td></td>
</tr>
<tr>
<td>- Hypalgesia</td>
<td>diminished ability to perceive painful stimuli applied along the palmar aspect of the index finger when compared with the ipsilateral little finger</td>
</tr>
<tr>
<td>Physical finding</td>
<td>Definition of Abnormal Finding</td>
</tr>
<tr>
<td>----------------------------------------</td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>➢ Diminished 2 point discrimination</td>
<td>o Diminished ability to identify correctly the number of points using callipers whose points are set 4-6 mm apart, comparing the index with little finger</td>
</tr>
<tr>
<td>➢ Abnormal vibratory sensation</td>
<td>o Diminished ability to perceive vibratory sensations using a standard vibrating tuning fork (128 of 256 Hz), comparing the distal interphalangeal joint of the index finger to the ipsilateral fifth finger</td>
</tr>
<tr>
<td>➢ Abnormal monofilament testing</td>
<td>o Using a Semmes Weinstein monofilament applied to the pulp of the index finger, the patients threshold is greater than the 2.83 monofilament</td>
</tr>
<tr>
<td>➢ Square wrist sign</td>
<td>o The anteroposterior dimension of the wrist divided by the mediolateral dimension equals a ratio of greater than 0.70 when measured with calipers at the distal wrist crease</td>
</tr>
<tr>
<td>➢ Closed fist sign</td>
<td>o Paresthesias in the distribution of the median nerve when the patient actively flexes the fingers into a closed fist for 60 s</td>
</tr>
<tr>
<td>➢ Flick sign</td>
<td>o When asking the patient, “what do you actually do with your hand(s) when the symptoms are at their worst?” the patient demonstrates a flicking movement of the wrist and hand, similar to that used in shaking down a thermometer</td>
</tr>
<tr>
<td>➢ Tinel sign</td>
<td>o Paresthesias in the distribution of the median nerve when the clinician taps on the distal wrist crease over the median nerve</td>
</tr>
<tr>
<td>➢ Phalen sign</td>
<td>o Paresthesias in the distribution of the median nerve when the patient flexes both wrists 90° for 60 seconds</td>
</tr>
<tr>
<td>➢ Pressure provocation test</td>
<td>o Paresthesias in the distribution of the median nerve when the examiner presses with his/her thumb on the palmar aspect of the patients wrist at the level of the carpal tunnel for 60 s</td>
</tr>
</tbody>
</table>
- **Tourniquet test**  
  Paresthesias in the distribution of the median nerve when a blood pressure cuff around the patient's arm is inflated above systolic pressure for 60 seconds

Adapted from: Simel David L, et al. *JAMA* 2009 page 112; and RCE, Table 10.1, page 112.

2. Perform a focused physical examination of the joints of the upper and lower body and from the abnormal articular findings, give the most likely diagnosis.

<table>
<thead>
<tr>
<th>Finding</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Shoulder</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>
  o Inspection                 |                                                     |
  - Flattening of rounded lateral aspect of shoulder | Anterior dislocation                              |
| **Elbow**                     |                                                     |
|  
  o Inspection                 |                                                     |
  - Swelling over anterior elbow | Glenohumeral synovitis; synovial cyst               |
  - Localized cystic swelling over olecranon          | Olecranon bursitis                                 |
  - Swelling obscures paraolecranon grooves          | Elbow synovitis                                    |
  - Nodules over extensor surface of ulna             | Gouty tophi; rheumatoid nodules                    |
  o Palpation                   |                                                     |
  - Elbow pain and tenderness over lateral epicondyle| Lateral epicondylitis (‘tennis elbow’)             |
  - Elbow pain and tenderness over medial epicondyle | Medial epicondylitis (‘golfers elbow’)             |
| **Wrists and carpal joints**  |                                                     |
|  
  o Inspection                 |                                                     |
  - Firm, painless cystic swelling, often located over volar or dorsal wrist | Ganglion (synovial cyst)                          |
  - Thickening of palmar aponeurosis, causing flexion | Dupuytren’s contracture                           |
Fingers

- Abnormal prominence of distal ulna
- Non-pitting swelling proximal to wrist joint, sparing joint itself; associated clubbing of digits
- Subluxation of ulna (from chronic inflammatory arthritis, especially rheumatoid arthritis)
- Hypertrophic osteoarthropathy

**Inspection**

- Loss of normal knuckle wrinkles
- Loss of ‘hills and valleys’ between metacarpal heads
- Ulnar deviation at metacarpophalangeal joints
- Swan neck deformity (flexion contracture at MCP joint, hyperextension of PIP joint, flexion of DIP joint)
- Boutonniere deformity (flexion of PIP, hyperextension of DIP)
- Osteophytes: Heberden’s nodes at DIP, Bouchards nodes at PIP
- Mallet finger: flexion deformity of DIP
- ‘Telescoping’ or ‘opera glass hand’; shortening of digits and destruction of IP joints

**Palpation**

- Flexion and extension of digits causes snapping or catching sensation in palm
- Finkelstein’s test: pain when patients makes fist with fingers over thumb and bends the wrist in an ulnar direction
- Trigger finger (flexor tenosynovitis)
- Tenosynovitis of long abductor and short extensor of thumb (‘De Quervain’s stenosing tenosynovitis’)

**Hip**

- Trauma, hip externally rotated
- Femoral neck fracture; anterior
- Trauma, hip internally rotated dislocation
- Pelvic tilt (imaginary line through the anterior iliac spines is not horizontal) - Posterior dislocation
- Scoliosis; anatomic leg length discrepancy; hip disease

- Palpation
  - Hip pain, tenderness localized over greater trochanter - Trochanteric bursitis
  - Hip pain, tenderness localized over middle third of inguinal ligament, lateral to femoral pulse - Iliopsoas bursitis
  - Hip pain and tenderness localized over ischial tuberosit - Ischiogluteal bursitis (‘Weaver’s bottom’)

- Knee
  - Inspection
    - Localized tenderness and swelling over patella - Prepatellar bursitis (‘Housemaid’s knees’)
    - Generalized swelling of popliteal space - Baker’s cyst (enlarged semimembranosus bursa, which communicates with knee joint)

  - Genu varum and genu valgum

  - Palpation
    - Knee pain and tenderness localized over medical aspect of upper tibia - Anserine bursitis
    - Distressed reaction if patella moved laterally (‘apprehension test’) - Recurrent patellar dislocation

- Ankle and feet
  - Inspection
    - Flattening of longitudinal arch - Pes planus
    - Abnormal elevation of medical longitudinal arch - Pes cavus
    - Outward angulation of great toe with prominence over medial 1st MTP joint (bunion) - Hallux valgus
    - Hyperextension of MTP joints and flexion of PIP joints - Hammer toes
Finding | Diagnosis
--- | ---
- Palpation
  - Nodules with Achilles tendon | Tendon xanthoma
  - Foot pain, localized tenderness over calcaneal origin of plantar fascia | Plantar fasciitis
  - Foot pain, localized tenderness over plantar surface of MT heads | Metatarsalgia
  - Forefoot pain, tenderness between 2nd or 3rd toes or between 3rd and 4th toes | Morton’s interdigital neuroma
  - Ankle pain, dysesthesias of sole, aggravated by forced dorsiflexion and eversion of foot | Tarsal tunnel syndrome

Abbreviations: DIP, distal interphalangeal; MCP, metacarpophalangeal; MT, metatarsal; MTP, metatarsophalangeal; PIP, proximal interphalangeal


3. Take a directed history for the common side effects of nonsteroidal anti-inflammatory drugs.

- CNS
  - Delirium/ confusion
  - Headache
  - Dizziness
  - Blurred vision
  - Mood swings
  - Aseptic meningitis

- Pulmonary
  - Pulmonary infiltrates
  - Noncardiac pulmonary edema (aspirin toxicity)
  - Anaphylaxis
  - Bronchospasm
  - Nasal polyps

- GI
  - Nausea, vomiting
  - Abdominal pain
  - ↑/ ↓ Bowel movement
- Iron deficiency anemia
- Peptic ulcer disease
- Colitis
- Hemorrhage from diverticulae

> Kidney
- ↓ renal blood flow
- ↓ glomerular filtration rate
- ↑ creatinine clearance
- Purpura
- Interstitial nephritis
- Papillary necrosis
- Nephrotic syndrome
- Hyperkalemia
- Type IV renal tubular acidosis
- Fluid retention

> Blood
- Bone marrow suppression
  - Agranulocytosis
  - Aplastic anemia
- Platelet-aggregating defect

> Skin
- Dermatitis
- Urticaria
- Erythema multiforme
- Exfoliative syndromes (toxic epidermal necrolysis)
- Oral ulcers

> Drug interactions
- ↑ hemostatic effect of warfarin
- ↑ antihypertensive effect of diuretics, beta-blockers, angiotensin-converting enzyme inhibitors
- Influence drug metabolism
  - Methotrexate (high doses only)
  - Lithium
  - Oral hypoglycemic agents

Useful background: Activities if daily living (ADL) and instrumental activities of daily living (IADL)

<table>
<thead>
<tr>
<th>ADL</th>
<th>IADL</th>
</tr>
</thead>
<tbody>
<tr>
<td>o Bathing</td>
<td>o Use of telephone</td>
</tr>
<tr>
<td>o Dressing</td>
<td>o Shopping</td>
</tr>
<tr>
<td>o Use of toilet</td>
<td>o Meal preparation</td>
</tr>
<tr>
<td>o Mobility</td>
<td>o Housekeeping</td>
</tr>
<tr>
<td>o Continence</td>
<td>o Laundry</td>
</tr>
<tr>
<td>o Feeding self</td>
<td>o Transportation</td>
</tr>
<tr>
<td></td>
<td>o Taking medicine</td>
</tr>
<tr>
<td></td>
<td>o Money management</td>
</tr>
</tbody>
</table>


Useful background: Abnormal articular findings and implied diagnosis

<table>
<thead>
<tr>
<th>Finding</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Fingers</td>
<td></td>
</tr>
<tr>
<td>o Inspection</td>
<td></td>
</tr>
<tr>
<td>- Loss of normal knuckle wrinkles</td>
<td></td>
</tr>
<tr>
<td>- Loss of “hills and valleys” between metacarpal heads</td>
<td></td>
</tr>
<tr>
<td>- Ulnar deviation at metacarpophalangeal joints</td>
<td>Chronic inflammatory arthritis</td>
</tr>
<tr>
<td>- Swan neck deformity (flexion contracture at MCP joint, hyperextension of PIP joint, flexion at DIP joint)</td>
<td>Chronic inflammatory arthritis, especially rheumatoid arthritis</td>
</tr>
<tr>
<td>- Boutenniere deformity (flexion of PIP, hyperextension of DIP)</td>
<td>Detachment of central slip of extension tendon to PIP, common in rheumatoid arthritis</td>
</tr>
<tr>
<td>- Osteophytes: Heberden’s nodes at DIP, Bouchard’s nodes at PIP</td>
<td>Osteoarthritis</td>
</tr>
<tr>
<td>- Mallet fingers: flexion deformity of DIP</td>
<td>Detachment of extensor tendon from base of distal phalanx or fracture</td>
</tr>
</tbody>
</table>
- “Telescoping” or “opera glass hand” shortening of digits and destruction of IP joint
- Arthritis mutilans, in rheumatoid or psoriatic arthritis

➢ Wrist
   o Inspection
     - Firm, painless cystic swelling, often located over volar or dorsal wrist
     - Thickening of palmar aponeurosis, causing flexion deformity of MCP joints (4th finger > 5th finger > 3rd finger)
     - Abnormal prominence of distal ulna
     - Nonpitting swelling proximal to wrist joint sparing joint itself, associated clubbing of digits
     - Ganglion (synovial cyst)
     - Dupuytren’s contracture
     - Subluxation of ulna (from chronic inflammatory arthritis, especially rheumatoid arthritis)
     - Hypertrophic osteoarthropathy

   o Special tests
     - Flexion and extension of digits causes snapping or catching sensation in palm
     - Finkelstein’s test: pain when patient makes fist with fingers over thumb and bends the wrist in an ulnar direction
     - Trigger finger (flexor tenosynovitis)
     - Tenosynovitis of long abductor and short extensor of thumb, or “De Quervain’s stenosing tenosynovitis”

➢ Elbows
   o Inspection
     - Localized cystic swelling over olecranon
     - Swelling obscures pata-olecranon grooves
     - Nodules over extensor surface of ulna
     - Olecranon bursitis
     - Elbow synovitis
     - Gouty tophi: rheumatoid nodules
Palpation
- Elbow pain and tenderness over lateral epicondyle - Lateral epicondylitis ("tennis elbow")
- Elbow pain and tenderness over medial epicondyle - Medial epicondylitis ("golfer’s elbow")

Shoulder
- Inspection
  - Flattening of rounded lateral aspects of shoulder - Anterior dislocation
  - Swelling over anterior aspect - Glenohumeral synovitis; synovial cyst

Hip
- Inspection
  - Trauma, hip externally rotated - Femoral neck fracture; anterior dislocation
  - Trauma, hip internally rotated - Posterior dislocation
  - Pelvic tilt (imaginary line through the anterior iliac spine is not horizontal) - Scoliosis; anatomic leg-length discrepancy; hip disease
- Palpation
  - Hip pain, tenderness localized over greater trochanter - Trochanteric bursitis
  - Hip pain, tenderness localized over middle third of inguinal ligament, lateral to femoral pulse - Iliopsoas bursitis
  - Hip pain and tenderness localized over ischial tuberosity - Ischiogluteal bursitis ("Weaver’s bottom")

Knee
- Inspection
  - Localized tenderness and swelling over patella - Prepatellar bursitis ("housemaid’s knees")
  - Generalized swelling of popliteal space - Baker’s cyst (enlarged semimembranosus bursa, which communicates with knee joint)
- Genu varum and genu valgum - Anserine bursitis
  
  o Palpation
  
  - Knee pain and tenderness localized over medial aspect of upper tibia
  - Distressed reaction if patella moved laterally ("apprehension test")

- Recurrent patellar dislocation

➢ Ankle and feet
  
  o Inspection
  
  - Flattening of longitudinal arch - Pes planus
  - Abnormal elevation of medial longitudinal arch - Pes cavus
  
  - Outward angulation of great toe with prominence over medial 1st MTP joints (bunion) - Hallux valgus
  - Hyperextension of MTP joints and flexion of PIP joints - Hammer toes

  o Palpation
  
  - Nodules within Achilles tendon - Tendon xanthoma
  - Foot pain, localized tenderness over calcaneal origin if plantar fascia - Plantar fasciitis
  
  - Foot pain, localized tenderness over plantar surface of MT heads - Metatarsalgia

# Hands and wrists

Useful background: Normal range of motion of joints

<table>
<thead>
<tr>
<th>Joint</th>
<th>Flexion/ extension (degrees)</th>
<th>Abduction/ adduction (degrees)</th>
<th>Rotation (degrees)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shoulder</td>
<td>180</td>
<td>180 (abduction)</td>
<td>90 (internal rotation)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>45 (adduction across body)</td>
<td>90 (external rotation)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>90 (internal rotation)</td>
<td>180 (radiohumeral)</td>
</tr>
<tr>
<td>Elbow</td>
<td>150 (humero-ulnar)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wrist and carpal joints</td>
<td>70 (wrist extension)</td>
<td>50 (ulnar deviation)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>80-90 (palmar flexion)</td>
<td>20-30 (radial deviation)</td>
<td></td>
</tr>
<tr>
<td>Fingers (MCP, PIP and DIP joints)</td>
<td>90 (MCP)</td>
<td>30-40 (MCP combined abduction/ adduction)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>120 (PIP)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>80 (DIP)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hip</td>
<td>10-20 (extension)</td>
<td>40 (abduction)</td>
<td>40 (internal rotation)</td>
</tr>
<tr>
<td></td>
<td>120 (flexion, knee flexed)</td>
<td>25 (adduction)</td>
<td>45 (external rotation)</td>
</tr>
<tr>
<td>Knee</td>
<td>130</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ankle and feet</td>
<td>45 (plantar flexion)</td>
<td></td>
<td>30 (inversion)</td>
</tr>
<tr>
<td></td>
<td>20 (dorsiflexion)</td>
<td></td>
<td>20 (eversion)</td>
</tr>
</tbody>
</table>


Useful background: Causes of wasting of small muscles of hand

- **CNS/ PNS**
  - Cord (C8, T1)
    - Motor neurone disease
    - Tumour
    - Syringomyelia
    - Meningovascular disease
    - Cord compression
  - Roots
    - Cervical spondylosis
    - Neurofibroma etc
- Brachial plexus
  - Klumpke paralysis
  - Cervical rib etc
- Ulnar or median nerve lesions

- MSK
  - Arthritis of hand or wrist
  - Disuse atrophy
  - Muscle diseases

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 86.

Useful background: Common deformities of the hand

<table>
<thead>
<tr>
<th>Name of deformity</th>
<th>Associations</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Mallet finger/thumb</td>
<td>A flexed DIP caused by damage to the extensor tendon - Trauma or RA</td>
</tr>
<tr>
<td>➢ Swan neck deformity</td>
<td>A flexed DIP and hyperextended PIP - RA but has many other causes</td>
</tr>
<tr>
<td>➢ Boutonniere deformity</td>
<td>A hyperextended DIP and flexed PIP - Trauma or RA</td>
</tr>
<tr>
<td></td>
<td>Occurs when the central slip of the extensor tendon detaches from the middle phalanx</td>
</tr>
<tr>
<td>➢ Dupuytren's contracture</td>
<td>Flexion deformity of the fingers at the MCP and IPs associated with nodular thickening in the palm and fingers - Diabetes, epilepsy, alcoholism and hereditary tendencies</td>
</tr>
<tr>
<td>➢ Heberden’s nodes</td>
<td>Hard dorsolateral nodules of DIPs, often associated with a deviation of the distal phalanx - OA</td>
</tr>
<tr>
<td>➢ Bouchard’s nodes</td>
<td>Similar to Heberden’s nodes, but affects the PIPs - OA</td>
</tr>
</tbody>
</table>
Abbreviation: DIP, distal interphalangeal; MCP, meta carpophalangeal; OA, osteoarthritis; PIP, proximal interphalangeal; RA, rheumatoid arthritis.

Adapted from: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto 2005, page 135.

- A flexed DIP caused by damage to the extensor tendon
- Interpretation of trauma or RA

- A flexed DIP and hyperextended PIP
- Interpretation of RA, but has many other causes

- Hyperextended DIP and flexed PIP
- Occurs when the central slip of the extensor tendon detaches from the middle phalanx
- Trauma or RA
- Ulnar deviation
- Deformity

Mallet finger Mallet thumb

Swan – neck deformity

Boutonniere deformity
Useful background: More deformities

<table>
<thead>
<tr>
<th>Site</th>
<th>Location of MSK disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Finger</td>
<td></td>
</tr>
<tr>
<td>o   Loss of normal knuckle wrinkles</td>
<td>PIP or DIP synovitis</td>
</tr>
<tr>
<td>o   Loss of “hills and valleys” between metacarpal heads</td>
<td>MCP synovitis</td>
</tr>
<tr>
<td>o   Ulnar deviation at metacarpophalangeal joints</td>
<td>Chronic inflammatory arthritis</td>
</tr>
<tr>
<td>o   Swan-neck deformity (flexion contracture at MCP joint, hyperextension of PIP joint, flexion of DIP joint)</td>
<td>Chronic inflammatory arthritis, especially rheumatoid arthritis</td>
</tr>
<tr>
<td>o   Boutonniere deformity (flexion of PIP, hyperextension of DIP)</td>
<td>Detachment of central slip of extensor tendon to PIP, common in rheumatoid arthritis</td>
</tr>
<tr>
<td>o   Mallet finger: flexion deformity of DIP</td>
<td>Detachment of extensor tendon from base of distal phalanx or fracture</td>
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<tr>
<td>o   “Telescoping” or “opera glass hand”: shortening of digits and destruction of IP joints</td>
<td>“Arthritis mutilans”, in rheumatoid or psoriatic arthritis</td>
</tr>
</tbody>
</table>
Proximal and distal interphalangeal joints
- Spindle-shaped deformity of finger (tensynovites, especially in psoriatric arthritis)
- Tophi (gout)
- Wasting of small muscles of the hand
- Heberden’s nodes – bony nodules at DIP joints (OA)
- Bouchard’s nodes – long nodules at PIP joints (OA)
- Deformity of thumb
- Ulnar deviation of thumb

Wrist and carpal joints
- Ganglion (synovial cyst): Firm, painless cystic swelling, often located over volar or dorsal wrist
- Dupuytren’s contracture: Thickening of palmar aponeurosis, causing flexion deformity of MCP joints (4th finger > 5th finger > 3rd finger)
- Subluxation of ulna (from chronic inflammatory arthritis, especially rheumatoid arthritis): Abnormal prominence of distal ulna
- Hypertrophic osteoarthropathy: Nonpitting swelling proximal to wrist joint, sparing joint itself; associated clubbing of digits

Special tests
- Trigger finger (flexor tenosynovitis): Flexion and extension of digits causes snapping or catching sensation in palm

<table>
<thead>
<tr>
<th>Site</th>
<th>Location of MSK disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tenosynovitis of long abductor and short extensor of thumb, or “De Quervain’s stenosing tenosynovitis”:</td>
<td>Finkelstein’s test: pain when patient makes fist with fingers over thumb and bends the wrist in an ulnar direction</td>
</tr>
</tbody>
</table>

Elbows
- Subcutaneous nodules
- Psoriatric rash

Palpation – (Feel and move passively and actively)

Hands
- Tenderness or pain
o Synovitis
o Effusions
o Range of movement
o Crepitus
o Subluxation
- Hand function
  - Grip strength
  - Key grip (abduction of thumb)
  - Opposition strength
  - Practical ability
    ▪ Button and unbutton clothes
    ▪ Pincer movement
    ▪ Writing

➢ Palms
  o Scars, palmar erythema, pale palmar creases (anemia
  o Wasting of thenar and hyperthenar eminence
  o Palmar tendon crepitus
  o Erythema
  o Thickening of palmar fascia (Dupuytren’s contracture)

➢ Wrists
  o Synovitis
    - Effusions
    - Range of movement
    - Crepitus
  o Carpal tunnel syndrome tests

“We are inherently critical as scientists,
and inherently kind as physicians.”

Grandad
Useful background: Thumb movements


“Trustworthiness is a gating mechanism for social interactions.”

Grandad
Useful background: Testing the superficial and profundus flexor tendons

Flexor profundus  Flexor superficialis

The key grip


“Science, like good diagnosis, represents incremental progress of small steps taken slowly on solid ground.”

Grandad
Useful background: MCP and IP joint movements

Flexion   Extension

Abbreviations: DIP, distal interphalangeal joint; IP, interphalangeal joint; MCP, metacarpophalangeal joint; MT, metatarsal; OA, osteoarthritis; PIP, proximal interphalangeal joint; RA, rheumatoid arthritis.


Useful background: Normal ranges of wrist motion

<table>
<thead>
<tr>
<th>Movement</th>
<th>Normal range of motion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flexion</td>
<td>$75^\circ$</td>
</tr>
<tr>
<td>Extension</td>
<td>$75^\circ$</td>
</tr>
<tr>
<td>Radial deviation</td>
<td>$20^\circ$</td>
</tr>
<tr>
<td>Ulnar deviation</td>
<td>$35^\circ$</td>
</tr>
<tr>
<td>Supination</td>
<td>$80^\circ$ from vertical (with pencil grasped in hand)</td>
</tr>
<tr>
<td>Pronation</td>
<td>$75^\circ$ from vertical (with pencil grasped in hand)</td>
</tr>
</tbody>
</table>

Source: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto* 2005, page 133.
Useful background: Nerve supply of the hand

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Sensory</th>
<th>Motor</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Motor and sensory</td>
<td></td>
<td></td>
</tr>
<tr>
<td>➢ Radial</td>
<td>o Dorsum of first webspace</td>
<td>o Extension of fingers, thumb, and wrist</td>
</tr>
<tr>
<td>➢ Ulnar</td>
<td>o Dorsal tip of small finger</td>
<td>o Finger abduction and adduction of ring and small finger</td>
</tr>
<tr>
<td></td>
<td>o Pulmar surface of small finger/ medial ring finger</td>
<td>o DIP flexion of fingers</td>
</tr>
<tr>
<td></td>
<td></td>
<td>o Opposition of small finger</td>
</tr>
<tr>
<td></td>
<td></td>
<td>o Wrist flexion</td>
</tr>
<tr>
<td>➢ Median</td>
<td>o Dorsal tip of index/ middle/ lateral half of ring finger</td>
<td>o Thumb IP flexion</td>
</tr>
<tr>
<td></td>
<td>o Palmar surface of index/ middle/ lateral half of ring finger</td>
<td>o Index/ middle finger flexion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>o Wrist flexion</td>
</tr>
<tr>
<td>• Motor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>➢ Posterior interosseous branch</td>
<td></td>
<td>o Extension of thumb</td>
</tr>
<tr>
<td>➢ Anterior interosseous branch</td>
<td></td>
<td>o Flexion of index/ middle finger</td>
</tr>
<tr>
<td>➢ Lateral terminal branch</td>
<td></td>
<td>o Opposition of thumb</td>
</tr>
</tbody>
</table>

Adapted from: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto* 2005, page 136.

"The meaning of life is to fill your three score and ten with love, respect and compassion for others."

Grandad
Useful background: Normal ranges of hand motion

<table>
<thead>
<tr>
<th>Movement</th>
<th>Normal range of motion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flexion</td>
<td>145°</td>
</tr>
<tr>
<td>Extension</td>
<td>0°</td>
</tr>
<tr>
<td>Supination</td>
<td>80° from vertical (with pencil grasped in hand)</td>
</tr>
<tr>
<td>Pronation</td>
<td>75° from vertical (with pencil grasped in hand)</td>
</tr>
</tbody>
</table>

Source: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto* 2005, page 132.

Useful background: Wrist movements

Useful background: Normal ranges of motion

<table>
<thead>
<tr>
<th>Digit</th>
<th>Joint</th>
<th>Range of motion</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Fingers</td>
<td>MCPs</td>
<td>0-90°</td>
</tr>
<tr>
<td></td>
<td>PIPs</td>
<td>0-100°</td>
</tr>
<tr>
<td></td>
<td>DIPs</td>
<td>0-80°</td>
</tr>
<tr>
<td>➢ Thumb</td>
<td>MCP</td>
<td>5° extension; 55° flexion</td>
</tr>
<tr>
<td></td>
<td>IP</td>
<td>20° extension; 80° flexion</td>
</tr>
<tr>
<td>➢ Wrist and carpal joints</td>
<td>70° (wrist extension)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>80°-90° (palmar flexion)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>50° (ulnar deviation)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>20°-30° (radical deviation)</td>
</tr>
<tr>
<td>➢ Fingers (MCP, PIP, and DIP joints)</td>
<td>90° (MCP)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>120° (PIP)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>80° (DIP)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>30°-40° (MCP, combined abduction/adduction)</td>
</tr>
</tbody>
</table>

Abbreviations: DIP, distal interphalangeal joint; IP, interphalangeal joint; MCP, metacarpophalangeal joint; MT, metatarsal; OA, osteoarthritis; PIP, proximal interphalangeal joint; RA, rheumatoid arthritis.

Special tests

➢ For intact flexor digitorum superficialis: restrict motion of 3 out of 4 fingers by holding down distal phalanges with the dorsum of the patients hand (palm up) rested on a table; ask the patient to flex the free finger and look for PIP flexion

➢ For intact flexor digitorum profundus; hold down both the proximal and middle phalanges and ask the patient to flex fingers; look for DIP flexion

Adapted from: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto 2005, pages 133 and 136.
Useful background: Distribution of arthritis in the hand and wrist

<table>
<thead>
<tr>
<th>Joint</th>
<th>Osteoarthritis</th>
<th>Rheumatoid arthritis</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ DIP</td>
<td>Very common</td>
<td>Rare</td>
</tr>
<tr>
<td>➢ PIP</td>
<td>Common</td>
<td>Very common</td>
</tr>
<tr>
<td>➢ MCP</td>
<td>Rare</td>
<td>Very common</td>
</tr>
<tr>
<td>➢ Wrist</td>
<td>Rare</td>
<td>Very common</td>
</tr>
</tbody>
</table>

*Osteoarthritis will sometimes affect only the carpometacarpal joint of the thumb

Abbreviations: DIP, distal interphalangeal; MCP, metacarpophalangeal; PIP, proximal interphalangeal

Source: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto 2005, Table 10, page 136.

SO YOU WANT TO BE A RHEUMATOLOGIST!

Q1: De Quervain’s disease
A1: o Tenosynovitis involving abductor pollicis longus and extensor pollicis brevis
     o Patient complains of weakness of grip and pain at the base of the thumb which is aggravated by certain movements of the wrist

Q2: Finkelstein test
A2: o Ask the patient to flex thumb and close the fingers over it then attempt to move the hand into ulnar deviation
     o Excruciating pain with this manoeuvre occurs in De Quervain’s tenosynovitis

Adapted from: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto 2005, page 137.
Elbows

4. Perform a focused physical examination of the elbow.

- Inspection
  - Olecranon bursitis:
  - Localized cystic swelling over olecranon
  - Ellebow synovitis:
  - Swelling obscures para-olecranon grooves
  - Gouty tophi; rheumatoid nodules:
  - Nodules over extensor surface of ulna

- Palpation
  - Lateral epicondylitis ("tennis elbow"): - Elbow pain and tenderness over lateral epicondyle
  - Medial epicondylitis ("golfer’s elbow"): - Elbow pain and tenderness over medial epicondyle

- Active Movement
  - Normal range of movement (ROM) - 145°
    - Flexion
    - Extension - 0°
    - Supination - 80° from vertical (with pencil grasped in hand)
    - Pronation - 75° from vertical (with pencil grasped in hand)
    - Rotation - 180° (radio humeral)

- Elbow
  - Elbow flexion and/or wrist extension - C5
  - Elbow extension and/or wrist flexion - C7

“The meaning of life is to fill your three score and ten with love, respect and compassion for others.”

Grandad
5. Perform a focused physical examination for features differentiating diseases affecting the elbow.

<table>
<thead>
<tr>
<th>Clinical feature</th>
<th>Rheumatoid arthritis</th>
<th>Psoriatic arthritis</th>
<th>Acute gout</th>
<th>Osteo-arthritis</th>
<th>Lateral epicondylitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Redness</td>
<td>Absent</td>
<td>Uncommon</td>
<td>Common</td>
<td>Common</td>
<td>Absent</td>
</tr>
<tr>
<td>Deformity</td>
<td>Flexion contractures, usually bilaterally</td>
<td>Flexion contractures, usually bilaterally</td>
<td>Flexion contractures, only in chronic state</td>
<td>Flexion contractures</td>
<td>None</td>
</tr>
<tr>
<td>Skin</td>
<td>Subcutaneous nodules</td>
<td>Psoriatic nails</td>
<td>Gout tophi</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- **Remember:**
  - Stiffness is absent with acute gout
  - Swelling is absent with lateral epicondylitis
  - Redness is absent with rheumatoid arthritis and very uncommon with psoriatic arthritis

Adapted from: Filate W., Et al. *The Medical Society, Faculty of Medicine, University of Toronto*, 2005, Table 4, page 132.

What is “the best”? There are no “best” clinical tests of physical examination for carpal tunnel syndrome, since the likelihood of each of the above tests is < 2.
Shoulder

Useful background: Anatomy of the shoulder

- Shulder
  - Elevation
  - Abduction


Useful background: Common conditions that affect the shoulder

- Rotator cuff tendon tear
- Rotator cuff tendinitis
- Frozen shoulder
- Biceps tendinitis
- Impingement syndrome
- Subscapular bursitis
- Glenohumeral osteoarthritis
- Acromioclavicular strain

Useful background: Shoulder instability or labrum lesion

- **Instability**
  - The relocation test and the anterior release test are the most useful in diagnosing anterior instability.
  - **Relocation test:** PLR, 6.5
  - **Anterior release test:** PLR, 8.3.
  - The sulcus sign for inferior instability has a sensitivity of 31% and a specificity of 89%.
  - The apprehension test is of limited value due to low specificity.

Useful background: The shoulder

- The impingement syndrome is the impingement of the supraspinatus tendon between the greater tuberosity of the head of the humerus and the undersurface of the acromion and acromioclavicular joint. There is a painful arc felt between 90° and 130°, and tenderness with palpation of the rotator cuff. The impingement syndrome is often due to osteophytes under the acromion.

- The apprehension test is used to identify anterior shoulder dislocations.
  - The patient’s affected arm is abducted and externally rotated until a look of apprehension is noted if the shoulder is dislocatable.
  - The shoulder has a “squared off” appearance, with reduction of internal rotation and possible loss of sensation and contraction over the lateral deltoid muscle.
  - The causes of posterior shoulder dislocation include:
    - Epileptic seizures
    - Ethanol intoxication,
    - Electrulation/electroshock therapy
    - Encephalitis.

6. Perform a focused physical examination of the shoulder.

- **Inspection**
  - **Swelling**
    - Flattening aspect of shoulder; anterior dislocation
  - **Erythema**
    - Swelling over anterior aspect; glenohumeral synovitis; synovial cyst
  - **Assymetry/atrophy**
  - **Deformity**
  - **Skin changes**

- **Palpation**
  - **Tenderness**
  - **Temperature**
- Edema
- Crepitus
- Biceps groove
- Subdeltoid bursa

- Passive and active movement

- Normal ranges of motion (ROM)

<table>
<thead>
<tr>
<th>Movement</th>
<th>Normal ROM</th>
</tr>
</thead>
<tbody>
<tr>
<td>✓ Forward flexion</td>
<td>165°</td>
</tr>
<tr>
<td>✓ Backward flexion</td>
<td>60°</td>
</tr>
<tr>
<td>✓ Abduction</td>
<td>170°</td>
</tr>
<tr>
<td>✓ Adduction</td>
<td>50°</td>
</tr>
<tr>
<td>✓ External rotation (with elbows at sides)</td>
<td>70°</td>
</tr>
<tr>
<td>✓ Internal rotation (with shoulder abducted to 90° &amp; elbow flexed)</td>
<td>70°</td>
</tr>
</tbody>
</table>

One way to test for limitations of passive motion is to ask the patient to bend over and try to touch his or her toes. In those with normal shoulder passive motion, the arms dangle toward the floor.

Abbreviation: ACJ, acromioclavicular joint; ROM, ranges of motion


---

**Impress the staff Rheumatologist!**

- Shoulder pain (radiating down the arm to the elbow) when combing one’s hair, putting on a coat or reaching into a back pocket, indicates supraspinatus inflammation.
- Diffuse shoulder pain upon moving the humerus posteriorly (without radiation to the arm) indicates infraspinatus inflammation.

Source: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto*, 2005, page 130.
Useful background:

- Active movements: Shoulder

Passive movements
- Neer impingement sign
- Hawkins impingement sign

Yergason’s sign
- “turn your forearm out”

Suprasinatus test
- “Don’t let me push down your arms”

The “supraspinatus test” is also known as the “empty can” or “Jobe” test.


Special tests

“push your arm apart”

- Acromion
- Sulcus
- Torn supraspinatus
- Humerus (extended)


Range of motion
- Active and passive ROM for
  - Flexion/extension
  - Abduction/adduction
  - Internal/external rotation

Special manoeuvres
- Apprehension test

Source: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto* 2005, page 129.

What’s “the best”? The “best” clinical test for the presence of rotator cuff tendonitis is a positive Yergason’s sign; The “best” clinical tests for the presence of a rotator cuff tear are a palpable tear, a positive dropped arm test, and age ≥ 60 years.
Useful background: Common clinical conditions of the shoulder

<table>
<thead>
<tr>
<th>Condition</th>
<th>Clinical features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rotator cuff tendinitis</td>
<td>o Shoulder pain on activity</td>
</tr>
<tr>
<td></td>
<td>o Sharp pain on elevation of arm into overhead position</td>
</tr>
<tr>
<td></td>
<td>o History of chronic usage (e.g. throwing, swimming) or trauma</td>
</tr>
<tr>
<td>Rotator cuff tear/rupture</td>
<td>o Sharp pain after trauma</td>
</tr>
<tr>
<td></td>
<td>o Pain over greater tuberosity</td>
</tr>
<tr>
<td></td>
<td>o Characteristic shoulder shrug</td>
</tr>
<tr>
<td></td>
<td>o Pain on attempted abduction</td>
</tr>
<tr>
<td></td>
<td>o Weakness on external rotation</td>
</tr>
<tr>
<td>Bicipital tendinitis</td>
<td>o Generalized anterior tenderness over long head of biceps</td>
</tr>
<tr>
<td></td>
<td>o Pain, especially at night</td>
</tr>
<tr>
<td></td>
<td>o Reproduction of anterior shoulder pain during resistance to forearm supination</td>
</tr>
</tbody>
</table>

Adapted from: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto* 2005, page 131

7. Perform a focused physical examination for causes of shoulder pain.

Useful background:

- Tests for anterior shoulder instability
  - Anterior apprehension test – limited valve due to low specificity
  - Relocation test
    - With patient supine, patient’s arm is passively abducted to 90°, elbow is flexed to 90° and arm is externally rotated 90°. Examiner applies downward (posterior) pressure to humeral head. Relief of symptoms of apprehension or pain is a positive result.
    - PLR, 6.5
  - Anterior release test
    - The relocation test is performed, and the examiner’s hand is suddenly removed from the proximal humerus. Expression of apprehension or pain is a positive result.
    - PLR, 8.3

- Test for inferior shoulder instability (Sulcus sign)
The patient stands or sits with the arm by the side and shoulder muscles relaxed
o The arm is pulled vertically downward.
o The presence of a sulcus sign (indentation between acromion and humeral head) is suggestive of interior shoulder instability
o Sensitivity, 31%; specificity, 89%

Tests for anterior shoulder instability
o With the patient supine, the arm is abducted to 90° and the humerus is maximally internally rotated.
o Examiner applies downward (posterior) pressure to humeral head
o Apprehension by the patient is a positive result and indicates posterior instability

Labrum lesion: sensitivities of ≥ 83%, specificities of ≥ 90%
- The biceps load I and II tests
- The pain provocation test
- The internal rotation resistance strength test

Adapted from: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto 2005, pages 129-130.

Useful background: Performance characteristics for detecting rotator cuff tendinitis and tear

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Detecting rotator cuff tendinitis</td>
<td></td>
</tr>
<tr>
<td>Yergason’s sign</td>
<td>2.8</td>
</tr>
<tr>
<td>Detecting rotator cuff tear-individual findings</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td></td>
</tr>
<tr>
<td>≥ 60 years</td>
<td>3.2</td>
</tr>
<tr>
<td>Neer’s impingement sign, Hawkin’s impingement sign, Supraspinatus testing causing pain</td>
<td></td>
</tr>
<tr>
<td>Supraspinatus atrophy</td>
<td>2.0</td>
</tr>
<tr>
<td>Infraspinatus atrophy</td>
<td>2.0</td>
</tr>
<tr>
<td>Supraspinatus weakness</td>
<td>2.0</td>
</tr>
<tr>
<td>Infraspinatus weakness</td>
<td></td>
</tr>
<tr>
<td>Painful arc</td>
<td></td>
</tr>
<tr>
<td>Dropped arm test</td>
<td>5.0</td>
</tr>
<tr>
<td>Palpable tear</td>
<td>10.2</td>
</tr>
</tbody>
</table>
Detecting rotator cuff tear – Combined findings

- 3 findings 48.0
- 2 findings 4.9

Note: Several signs have not been included because the value of their PLR was less than 2. These include Neer’s impingement sign, Hawkin’s impingement sign, Supraspinatus testing causing pain, Infraspinatus weakness and painful arc.


**Spine**

8. Take a directed history of back pain.

- **History**
  - Case – pain
    - How, when, where, why, what is quality of life
  - Complications
    - Fever, chills, night sweats
    - Anorexia, weight loss
    - Fatigue
    - Bowel bladder symptoms (retention, incontinence)
    - Nerve compression (sensory, motor, erectile dysfunction)
    - Scanda equine syndrome

- **Causes**
  - Joint
    - Inflammation, seropositive, seronegative
  - Bone
    - Infection, osteomyelitis, TB, immunosuppression
  - Inflammation
    - Pancreatitis, prostatitis, endometriosis, pyelonephritis
  - Malignancy
    - 1°, 2° (pancreas, prostate)
  - Metabolic
    - Osteoporosis, Paget’s, fracture
  - Blood vessels
    - AAA rupture

Useful background: Red flags that may indicate potential serious etiology of low back pain

- Age > 50 years
- Recent history of
  - bacterial infections
  - malignancies
  - trauma
  - inflammatory disease
- Bowel or bladder dysfunction
- Saddle anesthesia
- IV drug use
- Chronic disease
- Neurological deficits


9. Perform a focused physical examination of back pain.

- Inspection
  - Symmetry; deformity
    - Lordosis
    - Kyphosis
    - Scoliosis
  - Trauma, scars
  - Inflammation
  - Edema

- Palpation
  - Tenderness
    - Spinous processes
    - Paraspinal
  - Range of motion
    - Flexion, extension, lateral flexion, rotation
    - Chest expansion

- Nerve compression
  - Straight leg raising
    - (L4,5; S1,2,3)
  - L4 medial calf, knee jerk, squat and rise
  - L5 first web space, heel walk
o S1 lateral foot, ankle jerk, toe walk

- Pulses/bruits
  o Femoral, popliteal, dorsalis pedis


Useful background: Cervical spine movements and their respective myotomes

<table>
<thead>
<tr>
<th>Movement</th>
<th>Myotome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neck flexion</td>
<td>C1-C2</td>
</tr>
<tr>
<td>Neck side flexion</td>
<td>C3</td>
</tr>
<tr>
<td>Shoulder elevation</td>
<td>C4</td>
</tr>
<tr>
<td>Shoulder abduction</td>
<td>C5</td>
</tr>
<tr>
<td>Elbow flexion and/or wrist extension</td>
<td>C5</td>
</tr>
<tr>
<td>Elbow extension and/or wrist flexion</td>
<td>C7</td>
</tr>
<tr>
<td>Thumb extension and/or ulnar deviation</td>
<td>C8</td>
</tr>
<tr>
<td>Abduction and/or adduction of hand intrinsics</td>
<td>T1</td>
</tr>
</tbody>
</table>

Source: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto* 2005, page 138.

"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: Active movements of the thoracolumbar spine


Useful background:

- Active movements of the cervical spine and their normal range of motion.

<table>
<thead>
<tr>
<th>Maneuvre</th>
<th>Normal ROM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flexion (&quot;touch your chin to your chest&quot;)</td>
<td>80-90°</td>
</tr>
<tr>
<td>Extension (&quot;put your head back&quot;)</td>
<td>70°</td>
</tr>
<tr>
<td>Side flexion* (&quot;touch each shoulder with your ear without raising your shoulders&quot;)</td>
<td>20-45°</td>
</tr>
<tr>
<td>Rotation* (&quot;turn your head to the left and right&quot;; look for symmetrical movements)</td>
<td>70-90°</td>
</tr>
</tbody>
</table>

Abbreviation: ROM, range of motion.

Source: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto* 2005, page 138.
Common causes of kyphoscoliosis

- Ideopathic
  - Rib cage
    - Thoracoplasty
    - Empyema
  - Connective tissue
    - Marfan syndrome
    - Ehlers-Danlos syndrome
    - Morquio syndrome
  - Spine
    - Osteoporosis
    - Osteomalacia
    - Vitamin D-resistant rickets
    - Tuberculous spondylitis
    - Neurofibromatosis
  - Neuromuscular
    - Muscular dystrophy
    - Poliomyelitis
    - Cerebral palsy
    - Friedreich ataxia

Adapted from: Mangione S. *Hanley & Belfus* 2000, page 283.

Useful background: Special tests of back movement

- Restriction of spinal movement
<table>
<thead>
<tr>
<th>Movement and instructions</th>
<th>ROM</th>
</tr>
</thead>
<tbody>
<tr>
<td>▶ Forward flexion: (&quot;Bend forward and touch your toes&quot;)</td>
<td>20-45°</td>
</tr>
<tr>
<td>▶ Extension: (Arch your back&quot;)</td>
<td>25-45°</td>
</tr>
<tr>
<td>▶ Side flexion: (&quot;Slide your hand down your leg&quot;)</td>
<td>20-40°</td>
</tr>
<tr>
<td>▶ Rotation: (twist toward each side&quot;)</td>
<td>35-50°</td>
</tr>
<tr>
<td>▶ Chest expansion: (with a tape measure)</td>
<td>&gt; 5 cm</td>
</tr>
</tbody>
</table>

- **Schober’s test**
  - In health an increase from 15 to 22 cm is seen on forward flexion measured above (10 cm standing) and below (5 cm) a line drawn between the dimples of Venus.
  - In those with decreased spinal flexibility the distance measured increases to < 22 cm.

- **Lumbar vertebral fractures**
  - A rib-pelvis distance value < 2 fingerbreadths had a good sensitivity (87%) and moderate specificity (47%) for lumbar vertebral fracture

- **Straight leg raising test for lumbar disc herniation**
  - Diagnostic value of this straight-leg raising test in detecting lumbar disc herniation may lie primarily in ruling out its presence, because sensitivity (0.8) is far greater than specificity (0.4).
However, the crossed **straight** leg raise test (positive result = reproduction of contralateral pain with elevation and abduction of unaffected leg) identified lumbar disc herniation with a sensitivity of 25% and a specificity of 90% in patients with sciatica.

Adapted from: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto* 2005, page 140, page 142 and 143.

- **Causes of lower back symptoms**
  - Degenerative
    - Disk herniation
  - Infiltrative
    - Primary, metastatic
  - Inflammatory
    - Seronegative/spondyloarthropathies
    - Prostatitis, endometriosis, pyelonephritis, pancreatitis
  - Infectious
    - Osteomyelitis, TB
  - Metabolic
    - Osteoporosis with fractures
    - Osteomalacia
    - Paget’s disease
  - Compression
    - Cauda equine syndrome
    - Abdominal aortic aneurysm
  - Neurological deficits of cauda equine syndrome
    - Saddle anesthesia
    - Decreased anal tone or perianal sensory loss
    - Fecal incontinence
    - Urinary retention
    - Severe or progressive neurological deficit

Adapted from: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto* 2005, page 137; and Jugovic P.J., et al. *Saunders/Elsevier* 2004, page 110

“Our lives begin to end the day we become silent about the things that matter.”

Martin Luther King
Hips

Useful background: Pain in the hip

- Structures giving rise to pain around the hip and buttocks
  1. Sacro-iliac joint
  2. Hip joint (OA, RA, sepsis)
  3. Trochanteric bursa (overuse, mechanical imbalance)
  4. Ischiogluteal bursa (posterior)
  5. Insertion of adductor tendon

- Patterns of pain around the hip
  1. Intrinsic hip or knee joint pain
  2. Trochanteric bursitis
  3. Adductor tendinitis

  Compensatory postures that might be seen in an examination of the hip if there is:
  - A scoliotic deformity - flexion of the longer leg
  - An abduction deformity - flexion of ipsilateral knee
  - An adduction deformity - flexion of contralateral knee
  - A flexion deformity - exaggerated lordosis.

Useful background:

- **Manoeuvres for the hip and normal range of motion**

<table>
<thead>
<tr>
<th>Maneuvre</th>
<th>Normal ROM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flexion – with patient lying supine, have patient pull knee to chest; knee is also flexed</td>
<td>- 120°</td>
</tr>
<tr>
<td>Extension – with patient lying on side, palpate the ASIS and PSIS and have patient fully extend the leg until pelvis shifts</td>
<td>- 15°</td>
</tr>
<tr>
<td>Abduction – place one hand on the contralateral ASIS and with the other hand, grasp the heel and abduct the patient’s leg until the pelvis shifts</td>
<td>- 40°</td>
</tr>
<tr>
<td>Adduction – place one hand on the ipsilateral ASIS and with the other hand, grasp the heel and adduct the patient’s leg until the pelvis shifts</td>
<td>- 25°</td>
</tr>
<tr>
<td>Rotation – flex knee and hip to 90°, grasp the lower leg and move medially (external rotation) and laterally (internal rotation)</td>
<td>- External rotation in ext -35° - External rotation at 90° - Flex -45° - Internal rotation in ext: -45° - Internal rotation at 90° - Flex -45°</td>
</tr>
<tr>
<td>Or, with patient lying supine with the leg fully extended, roll the leg medially and laterally</td>
<td>-</td>
</tr>
</tbody>
</table>

Abbreviation: ROM; range of motion

Source: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto* 2005, page 145.
Useful background:

- **Radiation of pain** (Where is the pain felt in the following conditions?)
  - Osteoarthritis → to groin
  - Bursitis → superior margin of the greater trochanter
  - Sacroilitis → sacroiliac joint

- **Compensatory postures** of the lower leg, knee or spine if there are associated deformities
  - A scoliotic deformity - flexion of the longer leg
  - An abduction deformity - flexion of ipsilateral knee
  - An adduction deformity - flexion of contralateral knee
  - A flexion deformity - exaggerated lordosis.


Useful background:

- **Normal ROM of hip**

![Diagram](image)

Useful background: Internal and external rotation of the hip

![Internal rotation](image1)
![External rotation](image2)


Useful background: Lower limb movements and their respective myotomes

<table>
<thead>
<tr>
<th>Lower limb movement</th>
<th>Myotome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip flexion</td>
<td>L2</td>
</tr>
<tr>
<td>Knee extension</td>
<td>L3</td>
</tr>
<tr>
<td>Ankle dorsiflexion</td>
<td>L4</td>
</tr>
<tr>
<td>Great toe extension</td>
<td>L5</td>
</tr>
<tr>
<td>Ankle plantar flexion, ankle eversion, hip extension</td>
<td>S1</td>
</tr>
<tr>
<td>Knee flexion</td>
<td>S2</td>
</tr>
</tbody>
</table>

Source: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto 2005, page 141.

**Knees**

Useful background: Common causes of a painful knee joint

- Musculoskeletal
  - Rheumatoid arthritis
  - Osteoarthritis
  - Gout
  - Pseudogout
- Infection
- Viral infection
- Septic arthritis

- Metabolic

- Hematology
  - Hemophilia

- Trauma


10. Perform a directed physical examination of the knee.

- Inspection
  - For symmetry, deformity, genu valgum or varum, rubor, swelling, quadriceps, atrophy (assess with tape)
  - Skin bruising and any abnormal movements used to compensate for pain/stiffness in knee joint
  - Assess gait
  - Assess standing, feet together (hip, knee, ankle in straight line)

- Palpation – Flex knee for best assessment
  - Joint line and along course of medial and lateral collateral ligaments, tibial tubercle and infrapatellar tendon, bursal areas including anserine, prepatellar and infrapatellar popliteal fossa (for cyst, etc)
    - Tenderness at 90°, 180°
    - Flexion, 135°; extension, 0°
    - Warmth
    - Popliteal fossa Baker's cyst
    - Swelling of patella
    - Crepitation
  - Knee should be flexed for best assessment
  - Effusions
    - Temperature
    - Wipe test
    - Ballotment
    - Fluid displacement sign
    - Patellar tap
  - Bulge sign/fluid displacement sign
  - Balloon sign/fluctuation test

- Active and passive ROM: flexion/extension

- Stability (ligaments)
o Anterior and posterior cruciate draw test
o Collateral and medial collateral ligament stability

➢ Provocative tests
  o Meniscal tests (McMurray’s/Apley’s), as well as apprehension test and femoral-patellar grind test
  o Anterior drawer test (anterior cruciate ligament)
  o Pivot shift test
  o Posterior drawer test (posterior cruciate ligament)
  o Stability of lateral and medial collateral ligaments

➢ Menisci
  o Crouch compression test


What is “the best”? The three “best” clinical tests for osteoarthritis of the knee in a person with chronic pain are bony enlargement, varus (not valgus)

o Posterior anterior drawer sign

With the patient positioned as for the anterior drawer sign, the clinician pushes posteriorly on the patient’s upper calf. In the PCL-deficient knee, this force reveals an abnormal posterior tibial movement (arrow) with a soft endpoint
- Anterior drawer sign


- Lachman’s sign


**Clinical pearl:** pain referred to groin and thigh is **HIP** pain, but pain referred to buttocks is **BACK** pain.
o Pivot shift sign


o The McMurray test

Adapted from: McGee S. R. *Saunders/Elsevier* 2007, Figure 53.2, page 647.
What is “the best”? The “best “clinical test for the presence of meniscal injury of the knee is McMurray sign.

Useful background: Performance characteristics of physical examination for detecting anterior cruciate ligament rupture or tear

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Detecting anterior cruciate ligament rupture or tear</td>
<td></td>
</tr>
<tr>
<td>○ Lachman’s sign</td>
<td>17.0</td>
</tr>
<tr>
<td>○ Anterior drawer sign</td>
<td>11.5</td>
</tr>
<tr>
<td>○ Pivot shift sign</td>
<td>8.0</td>
</tr>
<tr>
<td>➢ Detecting meniscal injury</td>
<td></td>
</tr>
<tr>
<td>○ McMurray sign</td>
<td>8.2</td>
</tr>
<tr>
<td>○ Block to full extension</td>
<td>3.2</td>
</tr>
</tbody>
</table>

Abbreviation: PLR, positive likelihood ratio

Note that joint line tenderness does not have significant values for PLR/ NLR.


What is “the best”? The three “best” tests of physical examination for clinically significant knee fracture are an inability to flex the knee beyond 90°, inability to bear weight, and tenderness at the head of the fibula.

Useful background: Performance characteristics of physical examination for clinically significant knee fracture

<table>
<thead>
<tr>
<th>Finding</th>
<th>LR+</th>
</tr>
</thead>
<tbody>
<tr>
<td>○ Age ≥ 55 years</td>
<td>3.0</td>
</tr>
<tr>
<td>○ Joint effusion</td>
<td>2.5</td>
</tr>
<tr>
<td>- Limitation of knee flexion;</td>
<td></td>
</tr>
<tr>
<td>▪ Not able to flex beyond 90 degrees</td>
<td>2.9</td>
</tr>
<tr>
<td>▪ Not able to flex beyond 60 degrees</td>
<td>4.7</td>
</tr>
<tr>
<td>○ Isolated tenderness of patella</td>
<td>2.2</td>
</tr>
<tr>
<td>○ Tenderness at head of fibula</td>
<td>3.4</td>
</tr>
</tbody>
</table>

*Achieving Excellence in the OSCE Part 2* © A.B.R Thomson
Inability to bear weight, immediately and in emergency department  3.6

Abbreviation: PLR, positive likelihood ratio


Useful background: Performance characteristics of tests for osteoarthritis of knee in patients with chronic pain

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Individual findings</strong></td>
<td></td>
</tr>
<tr>
<td>o Morning stiffness &lt;30 minutes</td>
<td>3.0</td>
</tr>
<tr>
<td>o Bony enlargement</td>
<td>11.8</td>
</tr>
<tr>
<td>o Varus deformity</td>
<td>3.4</td>
</tr>
<tr>
<td><strong>Combined findings</strong></td>
<td></td>
</tr>
<tr>
<td>At least 3 out of 6:</td>
<td>3.1</td>
</tr>
<tr>
<td>o Age &gt; 50 years</td>
<td></td>
</tr>
<tr>
<td>o Stiffness &lt;30 minutes</td>
<td></td>
</tr>
<tr>
<td>o Crepitus</td>
<td></td>
</tr>
<tr>
<td>o Bony tenderness along margins of joint</td>
<td></td>
</tr>
<tr>
<td>o Bone enlargement</td>
<td></td>
</tr>
<tr>
<td>o No palpable warmth</td>
<td></td>
</tr>
</tbody>
</table>

Valgus deformity does not have a significant PLR values for osteoarthritis of the knee, nor does crepitus on passive movement (PLR, 2.1) or a palpable increase in the temperature of the knee.


What is “the best”? The three “best” clinical tests for the presence of an anterior cruciate ligament tear are a positive Lachman’s sign, anterior drawer sign, and pivot shift sign.


Ankles

- Active movement: Normal ROM

<table>
<thead>
<tr>
<th>Joint</th>
<th>Flexion/extension</th>
<th>Rotation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle and feet</td>
<td>45° (plantar flexion)</td>
<td>30° (inversion)</td>
</tr>
<tr>
<td></td>
<td>20° (dorsiflexion)</td>
<td>20° (eversion)</td>
</tr>
</tbody>
</table>

Abbreviations: DIP, distal interphalangeal; MCP, metacarpophalangeal; MT, metatarsal; PIP, proximal interphalangeal


11. Perform a focused physical examination of the ankle.

<table>
<thead>
<tr>
<th>Finding</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inspection</td>
<td></td>
</tr>
<tr>
<td>- Flattening of longitudinal arch</td>
<td>o Pes planus</td>
</tr>
<tr>
<td>- Abnormal elevation of medial longitudinal arch</td>
<td>o Pes cavus</td>
</tr>
<tr>
<td>- Outward angulation of great toe with prominence over medial 1st MTP</td>
<td>o Hallux valgus</td>
</tr>
<tr>
<td>joint (bunion)</td>
<td></td>
</tr>
<tr>
<td>- Hyperextension of MTP joints and flexion of PIP joints</td>
<td>o Hammer toes</td>
</tr>
<tr>
<td>Palpation</td>
<td></td>
</tr>
<tr>
<td>- Nodules within Achilles tendon</td>
<td>o Tendon xanthoma</td>
</tr>
<tr>
<td>- Foot pain, localized tenderness over calcaneal origin of plantar</td>
<td>o Plantar fasciitis</td>
</tr>
<tr>
<td>fascia</td>
<td></td>
</tr>
<tr>
<td>- Foot pain, localized tenderness over plantar surface of MT heads</td>
<td>o Metarsalgia</td>
</tr>
<tr>
<td>- Forefoot pain, tenderness between 2nd or 3rd toes or between 3rd</td>
<td>o Morton’s interdigital</td>
</tr>
<tr>
<td>and 4th toes</td>
<td>neuroma</td>
</tr>
<tr>
<td>- Ankle pain, dysesthesias of sole, aggravated by forced dorsiflexion</td>
<td>o Tarsal tunnel syndrome</td>
</tr>
<tr>
<td>and eversion of foot</td>
<td></td>
</tr>
</tbody>
</table>

Feet

12. Perform a focused physical examination of the feet.

➢ Inspection
  o For skin rash, scars
  o At the nails for changes of psoriasis
  o At the forefoot for hallux valgus, clawing and crowding of the toes (rheumatoid arthritis)
  o At the callus over the metatarsal heads which may occur in subluxation
  o At both the arches of the foot, in particular medial and longitudinal (flat foot, pes cavus)

➢ Palpate
  o Ankles for synovitis, effusion, passive movements at the subtalar joints (inversion and eversion) and talar joint (dorsiflexion and plantar flexion); remember that tenderness on movement is more important that the range of movement
  o Metatarsophalangeal joints for tenderness
  o Individual digits, for synovial thickening
  o Bottom of heel, for tenderness (plantar fasciitis), and Achilles tendon for nodules.

Adapted from: Baliga R.R.. Saunders/ Elsevier 2007, pages 334-335; Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto 2005, page 151.

What is “the best”? The “best” clinical test of physical examination for ankle and midfoot fracture is tenderness over the posterior medial malleolus.
Gout and pseudo-gout

Useful background:

- Distinguishing between gout and pseudo-gout

<table>
<thead>
<tr>
<th>Crystalline arthritis</th>
<th>Sex distribution</th>
<th>Joint involvement</th>
<th>Crystal</th>
<th>Crystal characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gout*</td>
<td>Male &gt; female</td>
<td>Asymmetrical distal joints, especially great toe</td>
<td>Uric acid</td>
<td>Long, needle-shaped negative birefringence</td>
</tr>
<tr>
<td>Calcium pyrophosphate deposition disease (pseudo-gout)</td>
<td>Female &gt; male</td>
<td>Proximal joints, especially knee and wrist</td>
<td>Calcium pyrophosphate</td>
<td>Rectangular, positive birefringence</td>
</tr>
</tbody>
</table>

* Examine ears, olecranon bursae and Achilles tendons for tophi.


- Causes of hyperuricemia
  - Primary gout
  - Chronic renal failure
  - ↑ Production
  - ↓ Excretion
    - Chronic renal failure
    - Hyperparathyroidism
    - Ketosis and lactic acidosis
  - Increased cell turnover
    - Polycythaemia
    - Leukaemia
    - Reticulosis
    - Myelosclerosis
    - Psoriasis
  - Drugs
    - Salicylates (in low doses)
    - Thiazides and furosemide
    - Pyrazinamide
  - High purine diet and alcohol
  - Down’s syndrome
  - Metabolic
    - Obese
    - Hypertensive patients
  - Congenital
- Lesch-Nyhan syndrome (congenital mental deficiency, choreoathetosis and lip chewing)


- **Causes of radiological erosions in hands**
  - Musculoskeletal
    - Gout
    - Rheumatoid arthritis
    - Chondromata
  - Infection
    - Sarcoidosis
  - Trauma
    - Traumatic cysts
  - Ideopathic
    - Localised osteitis fibrosa cystica


- **Causes of a migratory arthralgia**
  - Musculoskeletal
    - Rheumatic fever
    - SLE
  - Infection
    - Subacute bacterial endocarditis
    - Whipple’s disease
    - Sarcoidosis
    - Brucellosis
    - Gonorrhoea
  - Immune
    - Serum sickness
    - Reiters disease
    - Stevens-Johnson syndrome

- **Clinical features of Reiters Syndrome**
  - Eye
    - Recurrent conjunctivitis or uveitis
  - Mouth
    - Buccal ulcers
  - Musculoskeletal
    - Symmetrical subacute arthritis, tenosynovitis, periostitis
      - Ankles and all joints of feet
      - Knees
      - Hips, sacroiliac pints and spine
  - GU
    - Non-specific urethritis, hematuria, sterile pyuria
- Circinate balanitis
  o Skin
    - Keratoderma blenorrhagica, nail dystrophy


- Clinical features of Stevens-Johnson syndrome
  o Eye
    - Conjunctivitis, corneal ulcers, uveitis
  o Mouth
    - Oral bullae and hemorrhagic crusting
  o Skin
    - Maculopapular or bullous erythema multiforme
  o GI
    - Diarrhea
  o Lung
    - Bronchitis, pneumonitis
  o Kidney/GU
    - Urethritis, balanitis, vulvovaginitis
    - Renal lesions, diarrhea, polyarthritis, otitis media
  o General
    - Constitutional symptoms and high fever

- Clinical features of Behçet’s syndrome
  o CNS
    - Meningoencephalitis
      - brain-stem syndromes
  o Eyes
    - Conjunctivitis or uveitis
  o Mouth
    - Buccal ulcers with a red areola
  o Skin
    - Cutaneous pustules, dermal nodules
  o Kidney/GU
    - Genital ulcers
  o Veins
    - Thrombophlebitis
  o CVS/lung
    - Rarely cardiac and pulmonary lesions

Rheumatoid arthritis

Useful background:

- American Rheumatism Association (ARA) criteria for rheumatoid arthritis (RA)
  - Morning stiffness for at least 1 hour for duration of 6 weeks or more
  - Swelling of at least three joints for 6 weeks or more
  - Swelling of wrist, metacarpophalangeal or proximal interphalangeal 6 weeks or more
  - Symmetry of swollen joint areas for 6 weeks or more
  - Subcutaneous nodules
  - Positive rheumatoid factor
  - Radiographic features typical of rheumatoid arthritis, i.e. erosions periarticular osteopenia.

*When four or more of the above criteria are met, there is 93% sensitivity and 90% specificity for the diagnosis of rheumatoid arthritis.*


- Causes of deforming polyarthopathy
  - Rheumatoid arthritis
  - Psoriatic arthritis
  - Ankylosing spondylitis
  - Reiter’s disease
  - Chronic tophaceous gout
  - Osteoarthritis
  - Lyme arthritis)

- Causes of arthropathy plus nodules
  - Rheumatoid arthritis
  - Systemic lupus erythematosus
  - Rheumatic fever
  - Sarcoidosis

SO YOU WANT TO BE A RHEUMATOLOGIST!

Q: What causes arthritis plus nodules?
A:  
  - Rheumatoid arthritis
  - Systemic lupus erythematosus (rare)
  - Rheumatic fever (Jaccoud’s arthritis) (very rare)
  - Granulomas - e.g. sarcoid (very rare)


Q: What are the poor prognostic factors for RA?
A:  
  - Systemic features: weight loss, extra-articular manifestations
  - Insidious onset
  - Rheumatoid nodules
  - Presence of rheumatoid factor more than 1 in 512


13. Perform a focused physical examination for rheumatoid arthritis (RA), and its complications.

- Poor general health
  - Weight loss
  - Pale
  - Depression and social problems

- Joints
  - Metacarpophalangeal (MCP) joints
    - Synovitis
    - Effusions
    - Low range of movement
    - Crepitus
    - Subluxation, deformity
    - Boutonniere deformity (fixed flexion of PIP and extension of DIP, due to protrusion of the PIJ through ruptured extensor tendon)
    - Z deformity (thumb IPJ hypertension and fixed flexion and of subluxation of MCJ)
    - Tendon rupture
  - Arms
    - Entrapment neuropathy (e.g. carpal tunnel)
    - Subcutaneous nodules
  - Elbow, shoulder joint
- Axillary nodes
- Baker’s synovial cyst
  - Back, hips, knees

➢ Lower limbs
  - Ulceration (vasculitis)
  - Calf swelling (ruptured synovial cyst)
  - Peripheral neuropathy
  - Mononeuritis multiplex
  - Pressure sores
  - Infected ulcers (from nodules)
  - Cord compression

➢ Joint complications
  - Deformity, subluxation
  - Pyoarthrosis
  - Tendon rupture (due to attrition or nodules)
  - Nerve compression (due to tenosynovial swelling)
  - Cord or root compression (due to cervical subluxation)
  - Baker’s synovial cyst
  - Acute rupture of synovial sac (especially in knee)
  - Hoarseness, due to crico-arytenoid arthritis
  - Deafness, due to arthritis of auditory ossicles

➢ MSK
  - Osteoporosis
  - Muscle atrophy

➢ Eye
  - Episcleritis
  - Scleritis
  - Scleromalacia perforans, scleromalacia
  - Uveitis
  - Sjogren’s syndrome
  - Pyoderma gangrenosum

➢ Face
  - Eyes – dry eyes (Sjögren’s), scleritis, episcleritis, scleromalacia perforans, uveitis, Sjogren’s syndrome, anemia, cataracts (steroids, chloroquine)
  - Fundi – hyperviscosity
  - Face – parotid enlargement (Sjögren’s),
  - Mouth – dryness, ulcers, dental caries
  - Temporomandibular joint (crepitus)
  - Hoarseness (crico-arytenoid arthritis)
  - Ears – deafness (arthritis of auditory ossicles)
Neck
- Cervical nodes
- Swan neck (hyperextension at PIJ [subluxation], and fixed flexion deformity of DIJ [tendon shortening])
- Thyroiditis

Skin
- Pressure sores and infected ulcers (due to nodules)

Heart/CVS
- Pericardial effusion
- Pericarditis
- Arteritis
- Aortic regurgitation
- Rheumatoid granuloma of heart
- Murmurs from rheumatic heart disease

Lung
- Pleuritis, pleural effusion
- Nodules in lung or pleura
- Fibrosing alveolitis
- Caplan’s syndrome in pneumoconiosis
- Fibrosis
- Infarction
- Infection
- Arteritis

Arteritis
- Digital ischemia
- Raynaud’s syndrome
- Leg ulcers
- Mesenteric ischemia
- Arteritis of lungs, kidneys, liver
- Peripheral and autonomic neuropathy
- Amyloidosis
- May develop renal vein thrombosis

Abdomen
- Mesenteric ischemia
- Arteritis of kidneys, liver
- Splenomegaly
- Pernicious anemia
- Subfertility
- Renal/ vein thrombosis
- Character and distribution of deformities
- Contractures, hyperextension, ulnar deviation (late finding), abnormal posture, nodules, muscular atrophy
- Bony swelling
- Soft tissue swelling, redness or rash, palmar erythema, fingernail or finger tuft abnormalities, Dupuytren’s contractures
- Range of motion (ROM)
  o Active
  o Passive, to include:
    - Making fist
    - Grip strength
    - Wrist flexion/extension
    - Finger flexion/extension
    - Opposition of thumb and 5\textsuperscript{th} finger/thumb and index finger
  o Abdomen
    - Splenomegaly (e.g. Felty’s syndrome)
    - Inguinal nodes

- Functional assessment in rheumatoid arthritis
  o Class 1: Normal functional ability
  o Class 2: Ability to carry out normal activities, despite discomfort or limited mobility of one or more joints
  o Class 3: Ability to perform only a few of the tasks of the normal occupation or of self-care
  o Complete or almost complete incapacity with the patient confined to wheelchair or to bed
  o Other
    - Urine: protein, blood (drugs, vasculitis, amyloidosis)
    - Rectal examination (blood)
  o Complications of therapy

- Felty’s syndrome (Splenomegaly, RA and leucopenia)

- Complications of therapy

- Associated auto-immune disease
  o PA
  o Thyroiditis
  o Hemolytic anemia

- Subfertility (prior to development of arthritis)
  o Amyloidosis
  o SLE
  o Chronic brucellosis
  o CNS-posterior and anterior neuropathy
    - Nerve entrapment
  o Eye-keratoconjunctivitis sicca
- Uveitis
- Corneal ulceration
- Cytoid bodies
- Thyroiditis
- Skin-infection
  - Ulcers
- Bone marrow-normochromic normocytic anemia
  - Pernicious anemia
  - Hemolytic anemia
  - Lymphadenopathy
  - Splenomegaly
- Lung-fibrosis
  - Pleural effusion
  - Nodules
  - Caplans
- Kidney-proteinuria
  - Amyloid
- Decreased fertility

Note: In both RA and OA, spread is slow usually symetrically additive, progressing to multiple points, although OA may involve only one joint. The onset of each is usually insidious, with remissions and exacerbations.

14. Perform a focused physical examination to distinguish rheumatoid arthritis (RA) from osteoarthritis (OA).

<table>
<thead>
<tr>
<th>Rheumatoid arthritis</th>
<th>Osteoarthritis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Process</strong></td>
<td><strong>Osteoarthritis</strong></td>
</tr>
<tr>
<td>o Chronic inflammation of synovial membrane</td>
<td>o Degeneration and progressive loss of cartilage within the joints</td>
</tr>
<tr>
<td>o Secondary erosion of adjacent cartilage and bone</td>
<td>o Damage to underlying bone</td>
</tr>
<tr>
<td>o Damage to ligaments and tendons</td>
<td>o Formation of new bone at the margins of the cartilage</td>
</tr>
<tr>
<td><strong>Common locations</strong></td>
<td><strong>Common locations</strong></td>
</tr>
<tr>
<td>o Hands (PIP, MCP), feet (MCP), wrists, knees, elbows, ankles</td>
<td>o Knees, hips, hands (DIP, sometimes PIP), cervical and lumbar spine, wrists, and joints that were previously injured or diseased</td>
</tr>
<tr>
<td><strong>Weakness, fatigue, weight loss and low fever</strong></td>
<td><strong>Weakness, fatigue, weight loss and low fever</strong></td>
</tr>
<tr>
<td>o Common</td>
<td>o Very uncommon</td>
</tr>
<tr>
<td><strong>Flexion contractures</strong></td>
<td><strong>Flexion contractures</strong></td>
</tr>
<tr>
<td>o Usually bilaterally</td>
<td>o Uncommon</td>
</tr>
<tr>
<td><strong>Swelling</strong></td>
<td><strong>Swelling</strong></td>
</tr>
<tr>
<td>o Swelling of synovial tissue in joints or tendon sheaths’ subcutaneous nodules</td>
<td>o Small effusions in the joints</td>
</tr>
<tr>
<td>o Small effusions in the joints</td>
<td>o Bony enlargement</td>
</tr>
<tr>
<td><strong>Joint inflammation</strong></td>
<td><strong>Joint inflammation</strong></td>
</tr>
<tr>
<td>o Common</td>
<td>o Uncommon</td>
</tr>
<tr>
<td><strong>Stiffness</strong></td>
<td><strong>Stiffness</strong></td>
</tr>
<tr>
<td>o Prominent</td>
<td>o Frequent</td>
</tr>
<tr>
<td>o Often for an hour or more in the mornings</td>
<td>o Brief (usually 5-10 min) in the morning</td>
</tr>
<tr>
<td>o After inactivity</td>
<td>o After inactivity</td>
</tr>
<tr>
<td><strong>Limitation of motion</strong></td>
<td><strong>Limitation of motion</strong></td>
</tr>
<tr>
<td>o Often</td>
<td>o Often</td>
</tr>
<tr>
<td><strong>Radio-graphic changes</strong></td>
<td><strong>Radio-graphic changes</strong></td>
</tr>
<tr>
<td>o Symmetric joint space narrowing</td>
<td>o Asymmetric joint space narrowing, osteophytes, subchondral sclerosis, cystic changes</td>
</tr>
</tbody>
</table>

Abbreviations: DIP, distal interphalangeal; MCP, metacarpophalangeal; OA, osteoarthritis; PIP, proximal interphalangeal; RA, rheumatoid arthritis
Useful background: Causes of pyoderma gangrenosum

- **MSK**
  - Rheumatoid arthritis
  - Seronegative arthritis associated with gastrointestinal symptoms
- **GI**
  - IBD (Ulcerative colitis, Crohn’s disease)
  - Chronic active hepatitis
- **Hematology**
  - Acute and chronic myeloid leukemia
  - Myelocytic leukemia
  - Hairy cell leukemia
  - Polycythemia rubra vera
  - Multiple myeloma
  - IgA monoclonal gammopathy


**Sjögren’s syndrome**

Useful background:

This syndrome occurs in rheumatoid arthritis, and also with other connective tissue diseases.

- **Classic triad**
  - Arthritis (typically episodic polyarthritis)
  - Dry eyes
  - Dry mouth

- **Physical examination**
  - Dry eyes: conjunctivitis, keratitis, corneal ulcers (rarely vascularisation of the cornea)
  - Dry mouth
  - Arthritis
  - Chest: infection secondary to reduced mucus secretion, pleurisy or interstitial pneumonitis
  - Kidneys; renal tubular acidosis or nephrogenic diabetes insipidus
  - Genital tract: atrophic vaginitis
  - Pseudolymphoma: lymphadenopathy and splenomegaly, which may rarely progress to a true (usually non-Hodgkin’s) lymphoma
Other features

- Constitutional features: fatigue, malaise, myalgia
- Raynaud phenomenon
- Cutaneous vasculitis
- CNS abnormalities
  - Cerebritis, CNS vasculitis
  - Stroke
  - Multiple sclerosis-like illness
- Peripheral neuropathy
  - Sensory
  - Autonomic
  - Heart aortic regurgitation


Osteoarthritis

15. Perform a focused physical examination for the causes of spondyloarthritis.

- Seropositive conditions (rheumatoid factor [RF] positive)
  - Musculoskeletal
    - Rheumatoid arthritis
    - Sjögren syndrome
    - Systemic lupus erythematosus (SLE)
    - Scleroderma
  - Heart
    - Subacute bacterial endocarditis (SBE)
  - Lung
    - Idiopathic pulmonary fibrosis
  - Blood
    - Mixed cryoglobulinemia
  - Infections
    - Infectious mononucleosis
    - Influenza
    - Chronic active hepatitis
    - Vaccinations
    - Tuberculosis
    - Syphilis
  - Malignancy
    - Malignancies
• Seronegative (rheumatoid factor [RF] negative; commonly an asymmetrical arthritis)

➤ Ankylosing spondylitis
  o Sacroiliac joints and spine
  o Hips, knees and shoulders
  o Psoriatic arthritis
  o Terminal interphalangeal joints
  o Sacroiliac joints
  o Rheumatoid pattern

➤ Reiter’s syndrome
  o Sacroiliac joints and spine
  o Hips
  o Knees
  o Ankles, and most of the joints of the feet

➤ Infections
  o Sarcoidosis
  o Infectious mononucleosis
  o Influenza
  o Chronic active hepatitis
  o Vaccinations
  o Tuberculosis
  o Syphilis
  o Brucellosis
  o Leprosy
  o Salmonellosis
  o Malaria
  o Kala-azar
  o Schistosomiasis
  o Filarisis
  o Trypanosomiasis

➤ Miscellaneous
  o Hypergammaglobulinemic purpura
  o Asbestosis

**Ankylosing spondylitis (AS)**

Useful background: Remember:

- The four ‘A’s of ankylosing spondylitis: anterior uveitis, pulmonary apical fibrosis, aortic regurgitation, Achilles tendinitis
- Psoriasis and Reiter’s syndrome can also cause sacroiliitis


16. Take a directed history for ankylosing spondylitis.

- Back stiffness and back pain-worse in the morning, improves on exercise and worsens on rest
- Symptoms in the peripheral joints (in 40%), particularly shoulders and knees.
- Onset of symptoms is typically insidious, and in the third to fourth decade

- Extra-articular manifestations:
  - Red eye (uveitis)
  - Diarrhea (GI involvement)
  - Aortic regurgitation
  - Pulmonary apical fibrosis (worse in smokers)

- Causes of sacro-iliitis
  - Musculoskeletal
    - Ankylosing spondylitis
    - Reiter’s disease
    - Psoriatic arthritis
    - Juvenile rheumatoid arthritis
  - IBD
    - Ulcerative colitis and Crohn’s disease
  - Infection
    - TB
    - Brucellosis
    - Whipple’s disease

17. Perform a focused physical examination for ankylosing spondylitis.

- Postural change in advanced AS, 'question mark posture'

A focused physical examination for ankylosing spondylitis.

- Postural change in advanced AS, ‘question mark posture’

- Loss of lumbar lordosis and thoracic kyphosis
- Severe flexion deformity of the lumbar spine (rare)
- Tenderness of the lumbar vertebrae
- Reduction of movement of the lumbar spine in all directions (whole body turns)
- Tenderness of the sacroiliac joints
- Late involvement of cervical spine, with grating sensations on movement of neck
- Occiput-to-wall distance (inability to make contact when heel and back are against the wall indicates upper thoracic and cervical limitation)
- ‘Question mark’ posture (loss of lumbar lordosis, fixed kyphoscoliosis of the thoracic spine with compensatory extension of the cervical spine)
- Protuberant abdomen
- Perform Schober’s test - this involves marking points 10 cm above and 5 cm below a line joining the ‘dimple of Venus’ on the sacral promontory. An increase in the separation of less than 5 cm during full forward flexion indicates limited spinal mobility
Finger-floor distance (a simple indicator but is less reliable because good hip movement may compensate for back limitation)
- Chest expansion at nipple line < 5 cm (costovertebral involvement)
- Sacroiliac tenderness

**Distal arthritis**
- Distal arthritis occurs in up to 30% of patients and may precede the onset of the back symptoms. Small joints of the hand and feet are rarely affected.

**Legs/Feet**
- Achilles tendinitis
- Plantar fasciitis
- Cauda equine compression (rare lower limb weakness)
- Loss of sphincter control
- Saddle sensory loss

**Lungs**
- Decreased chest expansion (<5 cm, suggesting costo-vertebral involvement)
- Pulmonary apical fibrosis, cavities
- Mild restrictive lung disease

**Heart**
- Aortic regurgitation

**The eyes**
- Acute iritis (recurrent) – painful red eye in 10-15%

**CNS**
- Tetraplegia

**GI**
- Ulcerative colitis or Crohn’s disease
- Hepatosplenomegaly, amyloidosis

**GU**
- Renal enlargement (amyloidosis)
- Proteinuria

18. Perform a focused physical examination for primary vs secondary osteoarthritis.

<table>
<thead>
<tr>
<th></th>
<th>Primary</th>
<th>Secondary</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Symmetrical</td>
<td>o +</td>
<td>o -</td>
</tr>
<tr>
<td>➢ Many joints</td>
<td>o Many</td>
<td>o Few</td>
</tr>
<tr>
<td>➢ Previously damaged joints</td>
<td>o No</td>
<td>o Yes</td>
</tr>
<tr>
<td>➢ Fingers affected</td>
<td>o Yes</td>
<td>o No</td>
</tr>
<tr>
<td>➢ Sites affected</td>
<td>o Distal (Heberden’s nodes) and proximal (Bouchard’s nodes) interphalangeal joints, and metacarpophalangeal (MCP) joints of the thumbs</td>
<td>o Hip o Knees o Intervertebral disc</td>
</tr>
<tr>
<td></td>
<td>o Acromioclavicular joints</td>
<td></td>
</tr>
<tr>
<td></td>
<td>o Small joints of the spine (lower cervical and lumbar)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>o Knees,</td>
<td></td>
</tr>
<tr>
<td></td>
<td>o Metatarsophalangeal (MTP) joints of the great toes</td>
<td></td>
</tr>
</tbody>
</table>


**Psoriatic arthritis**

19. Perform a focused physical examination for psoriatic arthritis.

➢ Arthritis (in only 5% of persons with psoriasis)
  o Asymmetrical terminal joint involvement
    - Monoarticular and oligoarticular arthritis of the hands and feet.
    - May occur with psoriatic nail changes
  
  o Symmetrical joint involvement (most common like rheumatoid arthritis, but seronegative)
    - Arthritis mutilans (destructive polyarthritis, with telescoping of digits)
Nail changes (nails involved in 80% of persons with associated arthritis)
  - Pitting, onycholysis, ridging, hyperkeratosis, discoloration
  - Psoriatic, reddish plaques, with silvery scales and well-defined edges (most prominent on elbows [extensor surfaces], scalp, submamillary and umbilical regions)


Useful background: The radiological features of psoriatic arthritis

- ‘Fluffy’ periostitis
- Destruction of small joints
- ‘Pencil and cup’ appearance
- Osteolysis, ankylosis and telescoping in arthritis mutilans


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

Q: How does sacroilitis of psoriatic arthritis differ from ankylosing spondylitis?

A: In psoriatic arthritis, the syndesmophytes are usually from the internal and anterior surfaces of the vertebral bodies, and not from the margins of the bodies as is usually the case in ankylosing spondylitis

Systemic lupus erythematosus

20. Perform a focused physical examination for systemic lupus erythematosus (SLE) and its complications.

- General appearance
  - Cushingoid
  - Weight
  - Altered mental state
  - Fever

- Head
  - Hair: Alopecia, lupus hairs
  - Eyes – scleritis, cytoid lesions
  - Mouth – ulcers, infection
  - Butterfly rash
  - Cranial nerve lesions
  - Cervical lymph adenopathy

- Arms
  - Livedo reticularis
  - Purpura
  - Proximal myopathy (active disease or result steroid treatment)

Useful background: Diagnostic criteria for SLE. Four out of eleven criteria present at any time is required for diagnosis of SLE

<table>
<thead>
<tr>
<th>Clinical</th>
<th>Laboratory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin</td>
<td>Hematological abnormalities</td>
</tr>
<tr>
<td>- Malar rash</td>
<td>- Immunological abnormalities</td>
</tr>
<tr>
<td>- Photosensitive rash</td>
<td>- ANA positive (95%)</td>
</tr>
<tr>
<td>- Discoid lupus rash</td>
<td>- Hemolytic anemia</td>
</tr>
<tr>
<td>- Mucosal aphthous ulceration</td>
<td>- Leukopenia</td>
</tr>
<tr>
<td>- Photo sensitivity</td>
<td>- Thrombocytopenia</td>
</tr>
<tr>
<td>CNS</td>
<td>- Positive LE cell</td>
</tr>
<tr>
<td>- Neurological involvement</td>
<td>- Anti-DNA antibody</td>
</tr>
<tr>
<td>- Seizures or psychosis</td>
<td>- False-positive</td>
</tr>
<tr>
<td>Kidney</td>
<td>- Proteinuria or casts</td>
</tr>
<tr>
<td>Serositis: Pleuritis or pericarditis</td>
<td>- Immunological abnormalities</td>
</tr>
<tr>
<td></td>
<td>- ANA positive (95%)</td>
</tr>
<tr>
<td></td>
<td>- Hemolytic anemia</td>
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<td>- Leukopenia</td>
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<td>- Positive LE cell</td>
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<tr>
<td></td>
<td>- Anti-DNA antibody</td>
</tr>
<tr>
<td></td>
<td>- False-positive</td>
</tr>
</tbody>
</table>
- Arthritis
  - Nonerosive

- Mouth
  - Oral ulcers


Useful background: Physical examination for systemic lupus erythematosus

Scleroderma


- General appearance
  - ‘Bird-like’ facies
  - Weight-loss (malabsorption)
  - Fever

- Head
  - Hair - alopecia – hair loss
  - Eyes – loss of eyebrows, anemia, dryness (Sjögren’s), difficulty opening eyes
  - Mouth – difficulty smiling, raising forehead skin
  - Ischemia, end of nose
  - Pigmentation
  - Telangiectasia
  - Neck muscles – wasting and weakness

- Chest
  - Tight skin
  - Heart – cor pulmonale, pericarditis, congestive heart failure, hypertension (renal involvement)
  - Lungs – fibrosis, reflux aspiration pneumonitis, chest infections, alveolar cell carcinoma, vasculitis

- GI/GU
  - Dysphagia
  - Delayed gastric emptying (succussion splash)
  - Malnutrition (small bowel bacterial overgrowth [diarrhea, malabsorption])
  - Wide-mouthed colonic diverticulae
  - Proteinuria

- Arms
  - Edema (early) or skin thickening and tightening
  - Pigmentation
  - Vitiligo
  - Proximal myopathy

- Hands
  - CRST syndrome – calcinosis, Raynaud’s (atrophy distal tissue pulp), sclerodactyly and telangiectasia
  - Dilated capillary loops in nailfolds
  - Small joint arthropathy and tendon crepitus
  - Fixed flexion deformity
  - Hand function

- Legs
  - Skin lesions
Raynaud’s phenomenon

Useful background: Causes of Raynaud’s phenomenon

- Definition: Paroxysmal digital ischemia, usually accompanied by pallor and cyanosis and followed by erythema (white-blue-red).

- General
  - Malnutrition and cachexia

- Collagen vascular disease
  - Systemic sclerosis
  - Polyarteritis
  - SLE
  - RA
  - Sjogren’s
  - Dermatomyositis
  - Raynaud’s disease

- MSK
  - Cervical spondylosis
  - Paralysis or disuse of a limb
  - Cervical rib, hyperabduction syndrome

- Blood
  - Embolus, thrombosis or stenosis
  - Arteriosclerosis
  - Increased blood agglutination
    - Cold agglutinins
    - Dysproteinaemias
      - Cryoglobulinemia
      - Macroglobulinemia
      - Hyperglobulinemia
  - Polycythaemia, leukemia
  - Reflex vasoconstriction

- Infection

- Trauma
Achieving Excellence in the OSCE Part 2 © A.B.R Thomson

- Cold injury
- Frost bite
- Trench foot
- Vibrating machinery
- Injury (Volkmann’s ischemia)

➢ Toxin
  - Toxins: ergot, heavy metals, tobacco


22. Perform a directed physical examination for Raynaud’s phenomenon (white->blue->red fingers/toes in response to cold temperature).

➢ Reflex
  - Raynaud’s disease (idiopathic)
  - Vibrating machinery injury (jackhammer use)
  - Cervical spondylosis
  - Shoulder hand syndrome
  - Causalgia

➢ Connective tissue disease
  - Scleroderma (90-100%), CRST syndrome, mixed connective tissue disease (90-100%)
  - Systemic lupus erythematosus (SLE;15%)
  - Polyarteritis nodosa (PAN)
  - Rheumatoid arthritis (10%)
  - Polymyositis
  - Sjogren’s
  - Dermatomyositis
  - Aortic arch syndrome (Takayashu’s)

➢ Arterial disease
  - Thoracic outlet obstruction
  - Embolism or thrombosis
  - Buerger’s disease (thromboangiitis obliterans)
  - Trauma – vibration-induced

➢ Hematological disease
  - Polycythemia (increased blood viscosity)
  - Leukemia
  - Dysproteinemia (cryo-, macro-, hyperglobulinemia)

➢ Neurological
  - Paralysis
  - Disuse of limb

➢ Drugs/Poisons
  - Beta-blockers, ergotamine, bleomycin, vinyl
chloride

- Cold Injury

- Malnutrition, cachexia


**Vasculitis/arteritis**

23. Take a directed history and perform a focused physical examination for systemic vasculitis.

- **General**
  - Fever
  - Fatigue
  - Weight loss

- **CNS**
  - Seizures
  - Cerebrovascular accident
  - Mononeuritis multiplex
  - Peripheral neuropathy (mononeuritis multiplex)

- **Eye/sinuses**
  - Retinal hemorrhage
  - Necrotizing (hemorrhagic) sinusitis

- **CVS**
  - Coronary artery disease

- **Lung**
  - Interstitial pneumonitis
  - Hemoptysis
  - Pulmonary infiltrates or nodules

- **MSK**
  - Myalgia
  - Arthralgia
  - Arthritis

- **GI**
  - Ischemic bowel

- **GU**
  - Focal necrotizing glomerulonephritis
  - Abnormal renal sediment
  - Hypertension
  - Testicular pain

- **Skin**
  - Palpable purpura
  - Livedo reticularis
  - Cutaneous infarctions
Nodules
Ulcerations


- Chapel Hill consensus on the nomenclature of systemic vasculitis
  - Large-vessel vasculitis
    - Giant cell (temporal) arteritis
    - Takayasu arteritis
  - Medium-size vessel vasculitis
    - Classic polyarteritis nodosa
    - Kawasaki disease
  - Small-vessel vasculitis
    - Microscopic polyangiitis*
    - Wegener granulomatosis*
    - Churg-Strauss syndrome*
    - Henoch-Schönlein purpura
    - Essential cryoglobulinemic vasculitis
    - Cutaneous leukocytoclastic vasculitis
    - Anti-glomerular basement membrane disease

- Other causes of small – vessel vasculitis
  - Systemic vasculitis
    - Polyarteritis (primary and secondary)
    - Takayasu arteritis
    - Serum sickness
    - Goodpasture syndrome
  - Nonsystemic
    - Hypocomplementemic urticarial vasculitis
    - Leukocytoclastic vasculitis related to:
      - Rheumatoid arthritis
      - Sjögren syndrome
      - Systemic lupus erythematosus
      - Other connective tissue diseases
    - Drug-induced and postinfectious angiitis
    - Malignancy-associated vasculitis
    - Inflammatory bowel disease
    - Organ transplant-associated vasculitis
    - Hypergammaglobulinemic purpura of Waldenström

* Strongly associated with antineutrophil cytoplasmic autoantibody (ANCA).

Polymyalgia rheumatica-like syndromes

24. Take a directed history and perform a focused physical examination for causes of polymyalgia rheumatica-like syndromes.

- MSK
  - Fibromyalgia (FMN)
  - Polymyalgia rheumatica (PMR)
  - Seronegative rheumatoid arthritis (SRA)
  - Polymyositis
  - Systemic vasculitis
  - Systemic lupus erythematosus (SLE)
  - Polyarticular osteoarthritis
  - Osteomalacia (OA)
  - Remitting seronegative, symmetric synovitis and peripheral edema

- Metabolic
  - Hypo-/Hyperthyroidism
  - Hyperparathyroidism

- Infection
  - Infectious endocarditis

- Infiltration
  - Paraneoplastic syndromes
  - Systemic amyloidosis

- Mental health
  - Depression


Aseptic necrosis of the bone

25. Take a directed history of the cause of aseptic necrosis of the bone.

(acronym: ASEPTIC)

- Alcohol, artherosclerotic vascular disease
- Steroids, sickle cell anemia (Gaucher storage disease)
- Emboli (fat, cholesterol)
- Postradiation necrosis
- Trauma
- Idiopathic
- Connective tissue disease (especially SLE), caisson disease

Abbreviations: SLE, systemic lupus erythematosus

Charcot's joint

26. Perform a directed physical examination for Charcot’s joint (neuroarthropathy).

- Chronic, progressive, degenerative arthropathy arising from loss of sensory innervations of joint
  - Early redness, heat, swelling, tenderness;
  - Late enlargement of affected joint with crepitus, deformity, swelling and instability (usually hypermobile joint)
  - Associated muscle atrophy (compare joint with the normal contralateral joint)
  - Feet and ankles affected commonly, from peripheral neuropathy and local injury
  - May be complicated by osteomyelitis from skin ulcers

- Decreased sensation (position and vibration, pain and temperature)

- Examine patient for conditions possibly associated with the development of Charcot’s joint
  - Diabetes mellitus
  - Tabes dorsalis
  - Syringomyalgia
  - Myelomeningocele
  - Leprosy


Arthropathy

Useful background: Patterns of polyarthopathy

- Primary osteoarthritis
  - Symmetrical, affecting many joints
    - Knees
    - Great toes and thumbs, MP joints
    - Fingers: terminal IP joints
    - Acromio-clavicular joints
    - Small joints of spine

- Secondary osteoarthritis
  - Asymmetrical, affecting weight bearing joints
    - Knee
    - Hip
    - Intervertebral discs
- Rheumatoid Arthritis
  - Hands: intercarpal joints, MP joints and proximal IP joints
  - Feet: tarsal and lateral MP joints
  - Knees
  - Small joints of cervical spine and subacromial bursae
- Psoriasis
  - Hands, terminal IP joints
  - Sacro-iliac joints
  - 'Rheumatoid' pattern
- Ankylosing spondylitis
  - Spine both sacro-iliac joints
  - Knees, shoulders, wrists
- Reiter’s
  - Ankles and all joints of feet
  - Knees
  - Hips, sacro-iliac joint and spine

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 112.

Clinical Gems: No!

- No osteoporosis or new bone formation with gout
- No ankylosis with osteoarthritis
- No sclerosis or new bone formation with rheumatoid arthritis
Useful background: Causes of hypermobile joints

- Marfans
- Ehlers-Danlos
- Osteogenesis imperfecta
- Inflammatory polyarthritis (e.g. RA)
- Charcot’s arthropathy
- Homocystinuria
- Hyperlysinemia
- Idiopathic


Useful background: Key features of inflammation

- **Symptoms**
  - Stiffness: worst in the early morning, or after prolonged inactivity, progressively easing as the day goes on
  - Pain: inflammatory pain is usually present at rest as well as on movement
  - Both are greatly relieved by non-steridal anti-inflammatory drugs (NSAIDs)

- **Examination**
  - Overlying skin is warm and may be red
  - Tenderness is elicited across the joint line
  - Swelling is fluid in nature, demonstrated by shifting the fluid within the joint cavity (the bulge or balloon sign)
  - Pain is elicited throughout the range of both active and passive movement

- **Laboratory tests**
  - Acute phase reactants raised (e.g. ESR and C-reactive protein)

27. Perform a focused physical examination for polymyositis/dermatomyositis.

28. Perform a focused physical examination for Marfan’s syndrome.

- **Eyes**
  - Iridodonesis or ectopia lentis (subluxation upwards)
  - Thick spectacles
  - Blue sclera

- **Head**
  - Long-headedness (dolichocephalic, with bossing of frontal eminences and prominent supraorbital ridges)

- **Palate**
  - High-arched palate

- **Skin**
  - Small papules in the neck (Miescher’s elastoma)

- **Hands**
  - Hypermobile joints
  - Spider-like fingers (arachnodactyly)
  - Positive thumb sign – asking the patient to clench his thumb in his fist; the thumb should not exceed the ulnar side of the hand in normal subjects but because of hypermobility and laxity of the joint in Marfan’s disease it protrudes beyond his clenched fingers
  - Positive wrist sign – put the patient’s fingers around his other wrist; normal subjects cannot overlap the thumb and little finger around the wrist but in 80% of Marfan’s syndrome persons the little finger will overlap by at least 1 cm

- **Arms, legs**
  - Long arms and legs
  - Arm span (A) longer than the person’s height (H) (A:H>1.05)

- **Chest**
  - Pectus excavatum
  - Cystic lung disease

- **Heart**
  - Mitral valve prolapse (MVP)
  - Aortic aneurysm
  - Aortic regurgitation (AR)

- **Spine**
  - Scoliosis
  - Kyphosis

Miscellaneous

Useful background: Interesting trivia

➢ Where is the pain felt in the following conditions?
  o Osteoarthritis – radiation of pain to groin
  o Bursitis – pain over the superior margin of the greater trochanter
  o Sacroiliitis – pain localizing to sacroiliac joint

➢ Describe true and apparent leg lengths and what the discrepancies in these lengths suggest.
  o True leg length – distance between anterior superior iliac spine to medial malleolus. Differences in length suggest hip joint pathology.
  o Apparent leg length – distance between umbilicus and medial malleolus. Differences in length suggest a pelvic tilt, possibly resulting from adduction abnormality.


➢ Clinical Gems: No!
  o No!
    ▪ No osteoporosis or new bone formation with gout
    ▪ No ankylosis with osteoarthritis
    ▪ No sclerosis or new bone formation with rheumatoid arthritis
  o Reiter’s Disease
    ▪ “Calcaneal spur” (calcification of plantar fascia-highly suggestive of diagnosis)
    ▪ Plantar fasciitis
    ▪ Tendinitis
    ▪ Periostial calcification


➢ Variants of polyarteritis
  o Giant cell or temporal arteritis.
  o Wegener’s granulomatosis.
  o Senile arteritis (polymyalgia rheumatica).
  o Takayasu’s disease.

SO YOU WANT TO BE A RHEUMATOLOGIST!

Q: The skeletal phenotype of homocystinuria is similar to Marfan’s syndrome. How are the two distinguished on physical examination.
A:  
  o In homocystinuria the lens is dislocated downwards (and there is homocystine in the urine)


SO YOU WANT TO BE A RHEUMATOLOGIST!

Q: How is the diagnosis for Marfan’s syndrome made?
A:  
  o With family history: features from 2 systems
    - Skeletal features (including pectus carinatum or excavatum, reduced lower upper-lower segment ratio, arm-span-to-height ratio > 1.05, scoliosis and reduced elbow extension
    - Involvement of at least two other systems and one of the major criteria
    - Ectopia lentis
    - Dilation of the aortic root or aortic dissection
    - Lumbosacral dural ectasia by CT or MRI


➤ 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>o Monoarticular arthritis</td>
<td>o Podagra</td>
<td>- Gout, septic arthritis, hemorrhosis, pseudogout</td>
</tr>
<tr>
<td>o Knee post op</td>
<td></td>
<td>- Pseudogout</td>
</tr>
<tr>
<td>o With fever</td>
<td></td>
<td>- Septic</td>
</tr>
<tr>
<td>o Leg swelling</td>
<td>o Acute onset with pain</td>
<td>- Bakers Cyst</td>
</tr>
<tr>
<td>o Following travel</td>
<td></td>
<td>- DVT</td>
</tr>
<tr>
<td>o With fever</td>
<td></td>
<td>- Cellulitis</td>
</tr>
<tr>
<td>o Polyarticular arthritis</td>
<td>o Young woman with palmar rash</td>
<td>- Juvenile Rheumatoid arthritis</td>
</tr>
<tr>
<td>o 40 yr old female, symmetrical small joint</td>
<td>o Knees</td>
<td>- Rheumatoid arthritis</td>
</tr>
<tr>
<td>o With facial rash &amp; anemia</td>
<td></td>
<td>- Osteoarthritis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- SLE</td>
</tr>
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Source: Kindly provided by Dr. P Hamilton (U of Alberta)
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*Note: Page references to Parts One and Two of the text are preceded by I: and II: respectively.*

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