Emergency Medicine Checklist Compendium: Problems

Problem Checklists

Patients seek care at or are referred to the Emergency Department because of:
- subjective problems (e.g. chest pain)
- objective problems (e.g. rash, low sodium)
- potential problems (e.g. post-traumatic fracture or internal bleeding, suspected poisoning)

The diagnostic focus in Emergency Medicine lies in determining the probability of conditions whereby measures delivered within minutes to days reduces morbidity and mortality (time-sensitive diagnoses). There are three sources of diagnostic mistakes:
- inadequate information
- inadequate knowledge
- failure to consciously consider the actual diagnosis

Checklists have been used in the flight industry to prevent errors, and checklists are increasingly used in health care to promote patient safety. This section of the Emergency Medicine Checklist Compendium provides checklists for routine bedside information acquisition based on the patient's presenting problem. Use of these checklists may decrease the likelihood of diagnostic mistakes due to incomplete information acquisition. Associated with each symptom checklist features a list of pertinent time-sensitive conditions that ought to be consciously considered in all patients presenting with a given problem, to reduce the likelihood of diagnostic errors due to failure to consider the actual diagnosis.

Note the importance of using the appropriate checklist! The conditions for using a particular checklist feature in black font against a green background. For example, the conditions for using the Chest/Thoracic Pain checklist are as follows:

Pain or discomfort localized to or under the chest wall (including the back)
If pain in the midline of the back: use instead Back Pain

Bayes' theorem provides a theoretical framework for assessing the likelihood that the patient has a time-sensitive condition. According to Bayes' theorem, the posttest probability of a condition is the product of the pretest probability and a factor that depends on the test's characteristics. The pretest probability can be estimated by taking into account epidemiological factors such as age, gender, prior medical conditions, medications, smoking and alcohol consumption. Single test results usually modify the pretest probability only modestly, but test results combined into Clinical Decision Rules may lower the diagnostic likelihood below the test-threshold or raise the diagnostic likelihood above the treat-threshold.

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MAPLES

Background information is required to estimate the pretest probability of time-sensitive diagnoses and inform further management. MAPLES is a mnemonic for this information.

M	**Medications**, including over-the-counter medications, birth control pill
A	**Allergies**
P	**Past medical history**
L	**Life circumstances**, e.g. occupation, social support, home care
E	**Ethanol**: How often? How much?
S	**Smoking**: Amount? Prior smoking?

OPQRST+

Specific time-sensitive conditions ought to be consciously considered given the patient's problem. For example, the likelihoods of acute coronary syndrome, pulmonary embolism and aortic dissection ought to be estimated for patients presenting with chest pain. It follows that information necessary to determine these likelihoods should be routinely sought from patients seeking care in the ED with chest pain. OPQRST+ is a mnemonic for information from the history that should be acquired from patients presenting with pain and a number of other problems (e.g. vertigo, dyspnea):

O	**Onset**: When did the problem begin? Time to max intensity? Activity at onset?
P	**Position**: Pain location (or body position)? Radiation?
Q	**Quality**, e.g. type of pain (burning, pressure), nature of the deficit (motor, sensory)
R	**Relieving / aggravating factors**, e.g. worse with inspiration, movement
S	**Severity**, e.g. using a Visual Analogue Scale (VAS) 1-10
T	**Trend**: Constant or intermittent? Increasing? Prior similar episodes?
+	Additional pertinent questions

Bedside Tests

"Bedside tests" refers to information that can be acquired rapidly in the ED, such as information from history-taking, the physical examination, the electrocardiogram, bedside blood tests, ultrasound, urinanalysis and urine ß-hCG, capillary CRP and ketones. Given the patient's presenting problem, certain bedside tests should routinely be acquired to estimate the likelihoods of potential time-sensitive conditions. For example, and EKG should routinely be obtained from patients presenting with chest pain. The problem checklists provided in this section focus on bedside tests.

Clinical Decisions Rules

Clinical decision "rules" or "tools" help estimate the likelihoods of time-sensitive conditions using bedside tests.

Abdominal/Flank Pain

Pain between the lower border of the rib cage and the pelvis
If pain localized to the middle of the back: use instead Back Pain

BACKGROUND

M	☐ Current medications? ☐ NSAIDs?
A	☐ Allergies?
P	☐ Past medical history? ☐ Prior abdominal operations / procedures?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

O	☐ Time of onset? What were you doing? ☐ Time till max intensity: sec? min? hr?
P	☐ Pain location? Size of the painful area? ☐ Radiation?
Q	☐ Burning, aching, sharp?
R	☐ Worse with deep inspiration? ☐ Worse with movement?
S	☐ VAS (1-10)?
T	☐ Constant or intermittent? Increasing? ☐ Prior similar painful episodes?
+	☐ PO: nausea, vomiting? ☐ PR: diarrhea, constipation? ☐ PU: dysuria? ☐ PV (for fertile women): last period? Discharge?

PHYSICAL

Vitals	☐ RR, SpO2%, HR, BP, Temp?
Abdo	Including the groin: ☐ Inspection ☐ Auscultation ☐ Palpation
Testis	If male < 25 years: ☐ Inspection ☐ Palpation

TESTS

- ☐ WBC & CRP
- ☐ Urine dipstick
- ☐ Pregnancy test (for fertile women)
- ☐ EKG if > 50 years
- ☐ Ultrasound abdominal aorta if > 60 years

CONSIDER IF UNCLEAR

The cause of abdominal - flank pain may remain unclear after routine bedside information is obtained. Admission for observation and/or abdominal CT should be considered for these patients in the following situations:

1. Abdominal pain & shock
2. Severe & sudden abdominal pain
3. Decreased functional ability
4. Generalised peritonitis
5. Suspected bowel obstruction
6. Inflammed right lower quadrant

Abdominal/Flank Pain: Syndromes and Diagnostic Rules

1. ABDOMINAL PAIN & CHOCK

Abdominal pain with the following:

- Tachycardia and/or hypotension
- Elevated lactate, base deficit

Potential diagnoses:

- Ruptured abdominal aortic aneurysm
- Ruptured ectopic pregnancy
- Perforation (e.g. ulcer, diverticulus) and sepsis
- Severe pancreatitis, cholangitis

2. SEVERE & SUDDEN ABDOMINAL PAIN

- Sudden onset of diffuse abdominal pain
- Severe pain that does not respond to analgesics
- Peritoneal findings are absent

Potential diagnoses:

- Mesenteric ischemia
- Aortic dissection
- Perforated ulcer
- Ovarian torsion, testicular torsion

3. DECREASED FUNCTIONAL ABILITY

Patients (often elderly patients) who are sufficiently affected by their abdominal pain that they cannot function at home.

4. GENERALIZED PERITONITIS

- Pain worsens with movement
- Diffuse tenderness
- Rigidity or rebound tenderness

Potential diagnoses:

- Perforated ulcer
- Perforated diverticulitis
- Perforated appendicitis
- Cholecystitis, pancreatitis

5. BOWEL OBSTRUCTION

Pain with several of the following:

- Prior abdominal surgery
- Diffuse, crampy pain, intermittent spikes
- Vomiting, decreased bowel movements, absent flatus
- Swollen abdomen
- The abdomen is diffusely tender in the absence of peritoneal findings

6. RIGHT LOWER QUADRANT

- Right lower quadrant (RLQ) pain
- RLQ peritonitis OR elevated WBC/CRP

Potential diagnoses:

- Acute appendicitis
- Salpingitis
- Ovarial pathology
- Mesenteric adenitis
- Sigmoiditis

APPENDICITIS INFLAMMATORY RESPONSE SCORE

Criteria	Points
RLQ pain	1
Vomiting	1
Peritonitis	1, 2 or 3
WBC count	1 (10-14.9), 2 (≥ 15)
% Neutrofils	1 (70-84%), 2 (≥ 85%)
CRP	1 (10-49), 2 (≥ 50)
Temp ≥ 38.5°	1

Probability: 0-4 low, 5-8 indet., 9-12 high

APPENDICITIS vs SALPINGITIS

In fertile women:

Criteria	Salpingitis
Absent pain migration	OR 4.2
Bilateral tenderness	OR 16.7
No nausea or vomiting	OR 8.4
All of the above	99%

Back Pain

Pain localized to the middle of the back
If lateral pain: use instead Chest/Thoracic Pain or Abdominal/Flank Pain

BACKGROUND

M	☐ Current medications (corticosteroids, immunosuppressives, anticoagulants)? ☐ Analgesics: amount, frequency?
A	☐ Allergies?
P	☐ Past medical history? ☐ Prior cancer? ☐ Recent invasive procedures? ☐ Recent infections?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

O	☐ When did the pain start? What were you doing? ☐ Time till max intensity: sec? min? hr?
P	☐ Pain location? Size of the painful area? ☐ Radiation?
Q	☐ Type of pain: aching, sharp/riping?
R	☐ Decreased pain with analgesia? ☐ Decreased pain when lying down? ☐ Increased pain upon flexion, extension, walking?
S	☐ VAS (1-10)? Impact on daily function?
T	☐ Constant or intermittent? Increasing? ☐ Prior similar painful episodes?
+	☐ Leg weakness? ☐ Decreased perineal/leg sensation? ☐ Loss of bowel/bladder control? ☐ Fever/chills?

PHYSICAL

Vitals	☐ RR, SpO2%, HR, BP, Temp?
Back	☐ Inspection & palpation ☐ Range of motion
Leg neuro	☐ Leg strength & gait ☐ Romberg ☐ Sensation leg & perineum ☐ Patella & plantar reflexes

CONSIDER PERFORMING

- ☐ Straight leg raise (Lasègue)
- ☐ Per rectum (sensation-tonus?)
- ☐ Bladder scan (retention?)

TESTS

- ☐ CRP
- ☐ Ultrasound abdominal aorta if > 60 years

CONSIDER

1. Ruptured abdominal aortic aneurysm
2. Aortic dissection
3. Spinal cord compression (e.g. from spinal epidural metastasis, spinal epidural abscess, spinal epidural hematoma, central disc herniation)
4. Spinal infection (e.g. osteomyelitis, discitis, spinal epidural abscess)
5. Cancer
6. Fracture

Back Pain: Clinical Diagnostic Rules

SPINAL CORD COMPRESSION

- Immediate MRI in the presence of:
  - signs/symptoms of cauda equina: new urinary retention, urinary incontinence from bladder overflow, fecal incontinence, saddle anesthesia
  - radiculopathy attributable to a single nerve root with severe or progressive motor deficits
  - radiculopathy attributable to a single nerve root level AND (risk of metastatic cancer OR moderate to high risk of infection)
  - significant motor deficits not localized to a single nerve root

SPINAL INFECTION

- Immediate MRI (or CT) if moderate/high risk; ESR and/or CRP if low risk. Based on:
  - risk factors: current immunosuppression/hemodialysis, current or recent injection drug use/invasive epidural/spinal procedure/endocarditis or bacteremia
  - symptoms: fever, focal vertebral tenderness, neurological deficits
- Infection unlikely if ≤ 1 risk factor + ESR < 20.

CANCER

- If current or recent cancer: discuss choice of imaging with patient's oncologist
- Plain radiograph + ESR/CRP if moderate/high risk of cancer. Based on:
  - multiple risk factors (based on age, smoking history, family history, physical examination findings e.g. focal vertebral tenderness, recent weight loss)
  - history of cancer
  - strong clinical suspicion

VERTEBRAL COMPRESSION FRACTURE

- Plain radiography if at risk. Based on:
  - advanced age
  - prolonged systemic glucocorticoid use
  - significant trauma
  - mild trauma + history or risk factors for osteoporosis (previous fracture, low body weight, current smoking, excessive alcohol consumption, rheumatoid arthritis)

Adapted from
1-Wheeler SG et al. Evaluation of low back pain in adults. UpToDate 2020
2-Hsu PS et al. Acute lumbosacral radiculopathy: Pathophysiology, clinical features, and diagnosis. UpToDate 2020

Chest/Thoracic Pain

Pain or discomfort localized to or under the chest wall (including the back) If pain localized to the midline of the back: use instead Back Pain

BACKGROUND

M	☐ Current medications? ☐ Birth control pill, other hormonal treatments?
A	☐ Allergies?
P	☐ Past medical history? ☐ Prior heart or thromboembolic disease?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

O	☐ When did the pain start? What were you doing? ☐ Time till max intensity: sec? min? hr?
P	☐ Pain location? Size of the painful area? ☐ Radiation?
Q	☐ Cramping, aching, sharp, ripping, burning?
R	☐ Worse with deep inspiration? ☐ Worse with movement?
S	☐ VAS (1-10)?
T	☐ Constant or intermittent? Increasing? ☐ Prior similar painful episodes?
+	☐ Wind: shortness of breath? ☐ Walk: leg pain/swelling? ☐ Warm: fever/chills?

PHYSICAL

Vitals	☐ RR, SpO2%, HR, BP, Temp?
Heart	☐ S3/S4, murmurs? ☐ Elevated JVP?
Lungs	☐ Rales? ☐ Decreased breath sounds?
Chest	☐ Redness? Rash? ☐ Tenderness on palpation?
Abdo	☐ Upper abdominal tenderness?
Legs	☐ Swelling? Edema?

TESTS

- ☐ Troponin if > 40 years
- ☐ EKG

CONSIDER

1. Acute coronary syndrome
2. Pulmonary embolism
3. Aortic dissection

Other causes:

1. Pneumothorax
2. Pericarditis
3. Esophageal perforation

Chest/Thoracic Pain: Clinical Diagnostic Rules

ACUTE CORONARY SYNDROME

Age	< 40 years	40 - 65 years	> 65 years
ACS Prevalence	0-2%	8-10%	12-19%
0 Risk Factors*	LR 0.17	LR 0.53	LR 0.96
≥ 4 Risk Factors*	LR 7.4	LR 2.1	LR 1.09

* Diabetes, smoking, hypercholesterolemia, hypertension, heredity

History: high-risk features include pressure-type pain, radiation to one or both arms, worsening with exertion (but not with inspiration, position), similarity to prior ischemia.

EKG	ST Elevation	ST depression	T wave inversion
LR	22	5.3	1.8

0h-Troponin (see also www.compass-mi.com)
hs-cTnT < 5 ng/L + History not high-risk + EKG non-ischemic rules-out 30-day MACE (acute myocardial infarction, unstable angina, cardiac arrest, cardiogenic shock, death, high-risk arrhythmias) with 99.2% sensitivity and a negative predictive value of 99.7%.

0h/1h-Troponin (Δ = difference)

Rule-Out 30-day MACE	Rule-In 30-day MACE
0h hs-cTnT < 12 ng/L AND 1hΔ < 3 ng/L AND History not high-risk AND EKG non-ischemic	0h hs-cTnT ≥ 52 ng/L OR 1hΔ ≥ 5 ng/L OR 0h or 1h hs-cTnT > 14 ng/L + either high-risk history or ischemic EKG

Patients for whom 30-day MACE neither ruled-in nor ruled-out: consider additional troponin testing or stress testing / myocardial imaging (as out-patient?).

AORTIC DISSECTION DETECTION (ADD) RISK SCORE

High risk conditions: 1-Marfan syndrome 2-Family history of aortic disease 3-Known aortic valve disease 4-Recent aortic manipulation 5-Known thoracic aortic aneurysm
High risk pain features: 1-Abrupt in onset 2-Severe in intensity 3-Ripping or tearing
High risk examination features: 1-Evidence of perfusion deficit (pulse deficit, systolic BP differential, focal neurologic deficit in conjunction with pain) 2-Murmur of aortic insufficiency (new or not known to be old and in conjunction with pain) 3-Hypotension or shock state
ADD risk score: #categories featuring ≥ 1 high-risk feature/condition. High risk if score ≥ 2.

AORTIC DISSECTION & d-dimer

A negative serum D-dimer (< 500 ng/dL) rules out AD if the ADD risk score is ≤ 1.

WELLS SCORE FOR PULMONARY EMBOLISM

See **Dyspnea**.

Headache/Facial Pain

Pain localized to the head including the face
If throat or neck pain: use instead Throat/Neck Pain
If headache days from trauma: use instead Trauma to the Head & Neck

BACKGROUND

M	☐ Current medications? Birth control pill? Pain medications: how much / often?
A	☐ Allergies?
P	☐ Past medical history? Prior cancer?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

O	☐ When did the pain start? What were you doing? ☐ Time till max intensity: sec? min? hr?
P	☐ Pain location? Size of the painful area? ☐ Radiation?
Q	☐ Pulsating?
R	☐ Worse lying vs standing? ☐ Worse with valsalva / effort?
S	☐ VAS (1-10)? Impact on daily function?
T	☐ Constant or intermittent? Increasing? ☐ Worse in the morning or in the evening? ☐ Prior similar headaches?
+	☐ Neck pain / stiffness? ☐ Head trauma? ☐ Fever? ☐ Vision disturbance (e.g. aura, double vision)?

PHYSICAL

Vitals	☐ RR, SpO2%, HR, BP, Temp?
Head	☐ Focal tenderness to palpation? ☐ Meningismus?
Eye	☐ Conjunctivitis? ☐ Fundoscopy: papilledema? bleed?

NEUROLOGICAL EXAMINATION

Cortical Function	☐ Orientation ☐ Dysphasia / dysarthria ☐ Visual fields / neglect
Cranial Nerves	☐ Visual fields/neglect ☐ Pupil size, reactivity ☐ Eye movements ☐ Facial sensation ☐ Facial movement ☐ Soft palate and uvula ☐ Tongue movement
Motor	☐ Proximal and distal arm strength ☐ Proximal and distal leg strength
Sensory	☐ Sensation touch and pinch in the distal arm ☐ Sensation touch and pinch in the distal leg
Reflex	☐ Arm ☐ Patella
Coordination	☐ Finger-nose ☐ Knee-shin ☐ Romberg

TESTS

☐ CRP if > 50 years
☐ EKG if > 50 years

CONSIDER

1. Subarachnoid hemorrhage
2. Bacterial meningitis
3. Serious intracranial pathology
4. Giant cell arteritis
5. Carotid or vertebral artery dissection

Rare serious causes:
1. CO poisoning
2. Cerebral sinus thrombosis
3. Idiopathic intracranial hypertension
4. Acute closed-angle glaucoma

Headache/Facial Pain: Clinical Diagnostic Rules

OTTAWA SUBARACHNOID HEMORRHAGE RULE

Purpose: ruling-out SAH clinically
Inclusion: adults (≥ 16 years); nontraumatic headache reaching max intensity within 1 hour; alert and oriented (GCS 15); no fall or direct head trauma within previous 7 days; presenting to the ED within 14 days of headache onset
Exclusion: new neurologic deficits (e.g. isolated cranial nerve palsies, limb weakness); papilledema on fundoscopic examination; previous diagnosis of cerebral aneurysm, SAH, brain neoplasm, or hydrocephalus; history of recurrent headaches (≥ 3 episodes of the same character and intensity over the course of ≥ 6 months); returned for reassessment of the same headache if already investigated with both CT and lumbar puncture

The rule recommends investigating for SAH if ≥ 1 high-risk variable is present:

• Age ≥ 40 y • Onset during exertion • Thunderclap headache (instantly peaking)	• Witnessed loss of consciousness • Neck pain or stiffness (subjective) • Limited neck flexion on examination*

* defined as inability to touch chin to chest or raise the head 8 cm off the bed if supine

SUBARACHNOID HEMORRHAGE & CT HEAD

- CT head (modern, correctly interpreted) within 6 hours of onset of isolated headache (no primary neck pain, no loss of consciousness, normal neuro exam): SN 100%, LR- 0.01
- CT head beyond 6 hours from headache onset: SN 89%, LR- 0.07

BACTERIAL MENINGITIS

95% of adults with community-acquired bacterial meningitis had ≥ 2 of the following:

• Headache • Fever	• Neck stiffness • Change in mental status

SERIOUS INTRACRANIAL PATHOLOGY

Among alert (GCS 15) patients > 15 years presenting to the ED with nontraumatic headache, ≥ 1 of following had SN 98.6%, SP 34.4%, LR+ 1.50, LR- 0.04 for serious IC pathology:

• Age > 50 years • Abnormal findings on neurological examination	• Sudden onset of the headache

GIANT CELL ARTERITIS

The presence of the following combination motivates empiric treatment with corticosteroids and temporal artery biopsy:

• New onset headache without alternative explanation (e.g. normal CT) • Elevated CRP without alternative explanation	• Age > 50 years

MIGRAINE: "POUNDing"

≥ 4/5 of the following had LR 24 for migraine while ≤ 2/5 had LR 0.41 for migraine:

• Pulsatile quality • Duration 4-72 hOurs • Unilateral location	• Nausea and vomiting • Disabling intensity

Joint Pain/Swelling

Pain or swelling localized to a joint
If pain localized to the leg: use also Leg Pain/Swelling

BACKGROUND

M	☐ Current medications?
A	☐ Allergies?
P	☐ Past medical history?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

O	☐ When did the pain start? What were you doing? ☐ Time till max intensity: sec? min? hr?
P	☐ Location of the pain? One or several joints? ☐ Radiation?
Q	☐ Pain? Stiffness?
R	☐ Worse with movement? In such case, which?
S	☐ VAS (1-10)? Impact on daily function?
T	☐ Constant or intermittent? Increasing? ☐ Prior similar painful episodes?
+	☐ Fever / chills? ☐ Pain somewhere else?

PHYSICAL

Vitals	☐ RR, SpO2%, HR, BP, Temp?
Joint	☐ Inspection: red, swollen? ☐ Palpation: warm, tender, joint effusion? ☐ Range of motion?

ARTHROCENTESIS

- ☐ Cultures: aerobic and anaerobic. Adult bottles (8-10 ml in each) or use culture bottles for children (2-3 ml in each)
- ☐ WBC + Neutrophil percentage (≥ 1 ml EDTA-purple top)
- ☐ Crystals (EDTA-purple top)
- ☐ Glucose (≥ 1 ml grey top)
- ☐ Lactate (grey top)

OTHER TESTS FOR SEPTIC ARTHRITIS

- ☐ Blood cultures x 2: aerobic and anaerobic. Adult bottles (8-10 ml in each)
- ☐ WBC + Neutrophils
- ☐ Glucose
- ☐ CRP + ESR
- ☐ Joint X-ray
- ☐ Ultrasound for hip or shoulder

CONSIDER

1. Septic arthritis
2. If shoulder pain: acute coronary syndrome

Joint Pain: DDx & Clinical Diagnostic Rules

DIFFERENTIAL DIAGNOSIS

Monarticular	Polyartricular
• Gout (15-27%) • Septic arthritis (8-27%) • Osteoarthritis (5-17%) • Rheumatoid arthritis (11-16%) • Reactive arthritis (2-19%) • Systemic lupus erythematosus (7%) • Pseudogout (3%) • Spontaneous hemarthrosis (3%) • Charcot's joint	• Gonococcal arthritis • Viral arthritis • Lyme disease • Drug-induced arthritis • Reactive arthritis • Rheumatic fever • Seronegative spondyloarthropathies • Systemic lupus erythematosus

SEPTIC ARTHRITIS

WBC COUNT

WBC < 25 x 10^9/L: LR 0.32 (0.23-0.43) WBC ≥ 25 x 10^9/L: LR 2.9 (2.5-3.4) WBC > 50 x 10^9/L: LR 7.7 (5.7-11.0) WBC > 100 x 10^9/L: LR 28.0 (12-66)	A low WBC count can occur in early infection, and WBC > 50 x 10^9/L can occur with rheumatoid arthritis, gout and pseudogout

PMN PERCENTAGE: Polymorphonuclear cells count > 90% in the synovial fluid suggests septic arthritis LR+ 3.4; LR- 0.34

GLUCOSE: Low synovial fluid glucose (defined as serum/synovial fluid glucose ratio < 0.75 and/or synovial fluid glucose < 1.5 mmmol/ml) is weakly associated with septic arthritis SN 51%, SP 85%, LR+ 3.4, LR- 0.58

LDH: LDH > 250 U/L in the synovial fluid is sensitive but not specific for septic arthritis SN 100%, SP 51%, LR+ 1.9, LR- 0.10

LACTATE: cut-off of > 2.8 mmol/L according to expert opinion

ACUTE PRIMARY GOUT

The presence of ≥ 7 suggests acute gout (SN 74%, SP 99%, +LR 74, -LR 0.26):

• More than 1 attack of acute arthritis • Maximum inflammation developed within 1 day • Attack of monoarthritis • Redness observed over joints • First metatarsophalangeal joint painful and swollen • Unilateral attack of first metatarsophalangeal joint • Unilateral attack of tarsal joint	• Tophus (proven or suspected) • Hyperuricemia • Asymmetric swelling within a joint on radiograph • Subcortical cysts without erosions on radiograph • Monosodium urate monohydrate microcrystals in joint fluid during attack • Culture of joint fluid negative for organisms during attack

KNEE OSTEOARTHRITIS

Knee pain + ≥ 3 of the following suggests OA (SN 95%, SP 69%, LR+ 3.1, LR- 0.07):

• Age > 50 years • Morning stiffness lasting < 30 min • Crepitus on active range of motion	• Bony tenderness • Bony enlargement • No palpable warmth

Leg Pain/Swelling

Pain and/or swelling localized to the leg
If pain localized to the knee: use also Joint Pain

BACKGROUND

M	☐ Current medications? ☐ Birth control pill? Hormones?
A	☐ Allergies?
P	☐ Past medical history? ☐ Prior clots in the leg or lung?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

O	☐ When did the pain/swelling start? What were you doing? ☐ Time till max intensity: sec? min? hr?
P	☐ Location of the pain/swelling? Size? ☐ Radiation (if pain is present)?
Q	☐ Pain? Swelling? Other symptoms (e.g. redness, itch)?
R	☐ Is the pain exacerbated by leg/foot movements? ☐ Is the pain/swelling affected by position (supine, sitting)?
S	☐ VAS (1-10)? Impact on daily function?
T	☐ Constant or intermittent? Increasing? ☐ Prior similar painful episodes?
+	☐ Chest pain? ☐ Shortness of breath? ☐ Fever?

PHYSICAL

Vitals	☐ RR, SpO2%, HR, BP, Temp?
Leg	☐ Inspection ☐ Palpation

TEST

☐ CRP

CONSIDER

1. Deep vein thrombosis
2. Arterial insufficiency
3. Infection
4. Compartment syndrome
5. Ruptured Achilles tendon

Leg Pain/Swelling: DDx & Clinical Diagnostic Rules

DIFFERENTIAL DIAGNOSIS

Arterial	• Chronic arterial insufficiency • Acute arterial insufficiency, e.g. secondary to embolism	**Muscle**	• Necrotizing soft-tissue infections • Muscle rupture, strain, hematoma
Venous	• Deep venous thrombosis • Superficial thrombophlebitis • Venous insufficiency • Venous compression, e.g. tumor, advanced pregnancy • Budd-Chiari • Right heart failure	**Skin**	• Cellulitis, erysipelas, abscess • Erythema nodosum
		Bursa	• Rupture of a popliteal (Baker's) cyst
		Tendon	• Achilles tendon rupture • Tenosynovitis
		Nerve	• Radiculopathy
Capillary	• Compartment syndrome	**Other**	• Hypoalbuminemia
Lymphatic	• Lymphedema		• Pretibial myxedema

SIMPLIFIED CLINICAL MODEL FOR ASSESSMENT OF DEEP VEIN THROMBOSIS

RISK FACTORS	POINTS
• Active cancer (treated within the previous 6 months or currently receiving palliative treatment)	1
• Paralysis, paresis, or recent plaster immobilization of the lower extremities	1
• Recently bedridden for ≥ 3 days or major surgery within the previous 12 weeks requiring general or regional anesthesia	1
• Localized tenderness along the distribution of the deep venous system	1
• Entire leg swollen	1
• Calf swelling at least 3 cm larger than on the asymptomatic side (measured 10 cm below the tibial tuberosity)	1
• Pitting edema confined to the symptomatic leg	1
• Collateral superficial veins (nonvaricose)	1
• Previously documented deep-vein thrombosis	1
• Alternative diagnosis at least as likely as deep-vein thrombosis	-2

In patients with symptoms in both legs, the more symptomatic leg is used.

PRETEST PROBABILITY + D-DIMER + ULTRASOUND

Deep-vein thrombosis can be ruled out in the following situations:

- Score < 2 + negative d-dimer
- Score < 2 + negative ultrasound proximal veins (despite positive d-dimer)
- Score ≥ 2 + negative d-dimer + negative ultrasound proximal veins
- Score ≥ 2 + one of the following
  - negative ultrasound proximal veins + negative repeat ultrasound (+1 week)
  - negative ultrasound proximal + distal veins

Scrotal/Testicular Pain

Pain localized to the scrotum or testicle
If concurrent abdominal pain: use also Abdominal/Flank Pain

BACKGROUND

M	☐ Current medications?
A	☐ Allergies?
P	☐ Past medical history?
L	☐ Life circumstances? Sexual activity?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

TESTS

☐ CRP
☐ Urine dipstick

CONSIDER

1. Testicular torsion
2. Epididymitis

HISTORY

O	☐ When did the pain start? What were you doing? ☐ Time till max intensity: sec? min? hr?
P	☐ Pain location? Size of the painful area? ☐ Radiation?
Q	☐ Description of pain quality
R	☐ Worse with movement?
S	☐ VAS (1-10)?
T	☐ Constant or intermittent? Increasing? ☐ Prior similar painful episodes?
+	☐ Dysuria, urgency, discharge? ☐ Fever / chills? ☐ Nausea, vomiting?

PHYSICAL

Vitals	☐ RR, SpO2%, HR, BP, Temp?
Buk	☐ Inspection ☐ Palpation
Genitalia	☐ Inspection ☐ Palpation ☐ Cremaster reflex

Scrotal/Testicular Pain: Clinical Diagnostic Rule

DIFFERENTIAL DIAGNOSIS

Anatomy	Examples
Spermatic cord	• Testicular torsion • Varicocele
Epididymis	• Epididymitis
Appendix testis	• Torsion of appendix testis
Testicle	• Orchitis • Post-traumatic hematocele, testicular rupture • Testicular cancer (pain from hemorrhage or infarction) • Mumps
Perineum	• Fournier's gangrene
Ureter	• Distal ureterolithiasis
Bowel	• Incarcerated hernia
Aorta	• Aortic dissection • Ruptured abdominal aortic aneurysm

TESTICULAR WORKUP FOR ISCHEMIA AND SUSPECTED TORSION (TWIST)

Purpose: stratify patients with suspected testicular torsion into risk groups
Inclusion: studies of the TWIST score have included patients with acute scrotum ranging in age from 1 month to 28 years

FEATURES	POINTS
• Testicular swelling	2
• Hard testicle	2
• Absent cremasteric reflex	1
• High-riding testis	1
• Nausea/vomiting	1

- The initial study included 338 patients. The study reported 100% negative predictive value associated with score 0-2.
- A prospective study including 128 patients (44 with torsion) reported a 100% negative predictive value associated with score 0. 3/44 patients with torsion has a score 1-2.
- A prospective study including 258 patients (19 with torsion) reported that 2/111 patients with score 0-1 had torsion.

Throat/Neck Pain

Pain localized to the throat or neck
If post-traumatic: use instead Trauma to the Head/Neck
If concurrent headache: use instead Headache/Facial Pain

BACKGROUND

M	☐ Current medications?
A	☐ Allergies?
P	☐ Past medical history?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

O	☐ When did the pain start? ☐ Time till max intensity: sec? min? hr?
P	☐ Pain location? ☐ Radiation?
Q	☐ Pain quality?
R	☐ Worse with swallowing?
S	☐ VAS (1-10)?
T	☐ Constant or intermittent? Increasing? ☐ Prior similar painful episodes?
+	☐ Fever / chills? ☐ Cough? ☐ Trauma to the head / throat / neck?

PHYSICAL

Vitals	☐ RR, SpO2%, HR, BP, Temp?
Throat	☐ Redness? Swelling? Exudate?
Neck	☐ Swelling (e.g. lymph nodes)? ☐ Tenderness?

TESTS

- ☐ CRP
- ☐ EKG if > 50 years

CONSIDER

1. Epiglottitis
2. Deep neck space infection (e.g. retropharyngeal abscess, Ludwig's angina
3. Dissection (carotid, vertebro-basilar)
4. Acute coronary syndrome
5. Lemierre's syndrome

Throat/Neck Pain: Clinical Syndromes and Decision Rule

EPIGLOTTITIS

Fever + the 4 D's:

- Dypnea
- Dysphagia (odynophagia)
- Dysphonia
- Drooling

DEEP NECK SPACE INFECTIONS

- Peritonsillar abscess (quinsy), parotitis
- Infection in the submandibular space (Ludwig's angina)
- Infection in the parapharyngeal space
- Infection in the retropharyngeal space

Symptoms that may occur:

- Sore throat
- Trismus (the inability to open the jaw)
- Purulent oral discharge, pooling of saliva in the mouth, asymmetry of the oropharynx
- Lymphadenopathy is usually present.
- Dysphagia and odynophagia are secondary to inflammation of the cricoarytenoid joints.
- Dysphonia and hoarseness are late findings in neck infections and may indicate involvement of the tenth cranial nerve
- Unilateral tongue paresis indicates involvement of the twelfth cranial nerve.
- Stridor and dyspnea signify airway obstruction and may be manifestations of local pressure or spread of infection to the mediastinum.

MODIFIED CENTOR CRITERIA

Criteria	Points
• Temperature > 38.0	1
• Tonsillar swelling or exudate	1
• Swollen tender anterior cervical nodes	1
• Absence of cough	1
• 3-14 years	1
• ≥ 45 years	-1

Points	Likelihood of positive throat culture for Group A Streptococcal Pharyngitis
≤ 0	1-2.5%
1	5-10%
2	11-17%
3	28-35%
≥ 4	51-53%

Some recommend performing a throat culture or rapid antingen-detection test if ≥ 2 points.

Altered Consciousness

Decreased level of consciousness or confusion
If trauma to the head: use instead Trauma to the Head or Neck
If suspected poisoning/overdose: use also Poisoning

BACKGROUND

M	☐ Current medications? ☐ Recent changes?
A	☐ Allergies?
P	☐ Past medical history? ☐ Prior episodes with altered consciousness?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

O	☐ When did the problem start? ☐ Activity at the time? ☐ How rapid onset?
P	☐ Place where patient found? ☐ Any evidence of poisoning/trauma?
Q	☐ Altered or decreased LOC?
R	☐
S	☐
T	☐ Constant or fluctutating? Increasing? ☐ Prior similar episodes?
+	☐ Fever/chills/infectious symptoms? ☐ Pain (e.g. headache)?

PHYSICAL

A	☐ Trauma to the head? ☐ Tongue bite?
B	☐ SpO2? ☐ Respiratory rate? ☐ Lung auscultation? ☐ Chest wall examination
C	☐ Pulse/blood pressure? ☐ Heart rate? ☐ QRS width, regularity?
D	☐ Level of consciousness? ☐ Eye / pupil examination ☐ Focal neurological deficits arm/leg? ☐ Glucose level?
E	☐ Front side of the body ☐ Back side of the body ☐ Temperature?

TESTS

- ☐ Acid-base: pH, pCO_2, HCO_3/BE
- ☐ Electrolytes: Na, K, Ca
- ☐ Hb, WBC, CRP
- ☐ Trombocytes, INR
- ☐ Creatinine
- ☐ Liver function tests
- ☐ EKG if > 50 years

CONSIDER IF UNCLEAR

1. Stroke including basilar thrombosis
2. Sepsis
3. Meningitis
4. Herpes encephalitis
5. Non-convulsive status
6. Wernicke's encephalopathy

Altered Consciousness: DDx & Clinical Syndromes

DIFFERENTIAL DIAGNOSIS

Pathophysiology	Examples
Vascular Cardiac	• **Arterial**: basilar artery thrombosis, large stroke (including SAH), primary CNS vasculitis, giant cell arteritis • **Venous**: cerebral sinus thrombosis (delirium, amnesia, mutism) • **Systemic**: cardiogenic chock, thrombotic thrombocytopenic purpura, hypertensive encephalopathy, PRES
Infectious Infiltrative	• **Intracranial infections**: meningitis, encephalitis, cerebral abscess • **Extracranial infections**: pneumonia, urosepsis
Neurological Neoplastic Psychiatric	• **Seizures**: status epilepticus (including non-convulsive), post-ictal • **Increased ICP**: brain tumor, hydrocephalus, hematoma • **Psychiatric**: depression, psychosis
Degenerative Deficiency	• **Degenerative**: Alzheimer's dementia • **Deficiency**: Wernicke's encephalopathy, B12 deficiency
Intoxication Withdrawal	• **Traditional medications:** overdose, withdrawal (e.g. opioids) • **Intake of other substances** (e.g. alcohol, illicit drugs) • **Toxic drug levels**, e.g. of Digoxin in renal failure, antiepileptics
Collagen	• **Lupus cerebritis**
Autoimmune	• **Anti-NMDAR encephalitis**
Trauma Mechanical	• **Intracranial**: concussion, shunt dysfunction • **Extracranial**: urinary retention, fat embolism syndrome
Electrolytes Endocrine Metabolic	• **Electrolytes**: hypo- hypernatremia, hypo- hypercalcemia • **Endocrine/Metabolic**: hypoglycemia, HHS, hypothyroidism, thyrotoxicosis, uremia, hepatic encephalopathy, hypoxia, hypercapnia, hypothermia, heatstroke, porphyria

METABOLIC CAUSE

The presence of the following three findings suggests a metabolic cause of coma (SN 96%):

• Age ≤ 50 years	• SBT ≤ 150 mm Hg	• Lack of focal neurological findings

BACTERIAL MENINGITIS

95% of adults with community-acquired bacterial meningitis had ≥ 2 of the following:

• Headache	• Fever	• Neck stiffness	• Change in mental status

WERNICKE'S ENCEPHALOPATHY

Classic triad of encephalopathy, ocular abnormalities and gait ataxia present in only 17% of cases. Operational criteria to identify patients with Wernicke's encephalopathy: ≥ 2 of

- **Dietary deficiencies** (e.g. chronic alcohol abuse, anorexia nervosa, GI surgery including bariatric surgery, hyperemesis of pregnancy, prolonged IV feeding without proper suppl.)
- **Altered mental status** (e.g. confusion, apathy, inattentiveness, inability to concentrate, disorientation) **or mild memory impairment**
- **Oculomotor abnormalities** (e.g. nystagmus, symmetrical or asymmetrical palsy of both lateral recti or the other ocular msucles, conjugated-gaze palsies)
- **Cerebellar dysfunction** (incoordination of gait or truncal ataxia)

Altered Vision

Decreased visual acuity and/or visual symptoms excluding diplopia
If double vision: use Double Vision
If headache: use also Headache/Facial Pain
If weakness/paresthesia: use also Weakness/Paresthesia

BACKGROUND

M	☐ Current medications?
A	☐ Allergies?
P	☐ Past medical history?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

O	☐ When did the vision disturbance start? What were you doing? ☐ Time till max intensity: sec? min? hr?
P	☐ Does the disturbance affect vision from one or both eyes? ☐ Which part of the visual field is affected?
Q	☐ Description of the distrubance: decreased visual acuity, shadows, flashes, floaters, halo?
S	☐ Degree of deficit (e.g. ability to read, count fingers)?
T	☐ Constant or intermittent? Increasing? ☐ Prior similar episodes?
+	☐ Eye pain? Headache? ☐ Fever?

PHYSICAL

Vitals	☐ RR, SpO2%, HR, BP, Temp?
Eye	☐ Inspection of the eyelids, conjunctiva, cornea ☐ Visual acuity ☐ Visual fields ☐ Pupil size, reaction to light ☐ Swinging flashlight test ☐ Red reflex ☐ Fundoscopy

NEUROSCREEN

Cranial nerves	☐ Eye movements? ☐ Facial movement? ☐ Soft palate and tongue?
Motor	☐ Proximal arm strength? ☐ Distal arm strength? ☐ Proximal leg strength? ☐ Distal leg strength?
Coordi-nation	☐ Romberg? ☐ Finger-nose? ☐ Knee-shin?

TESTS

☐ CRP if > 50 years

IMMEDIATE TREAMENT

1. Central retinal artery occlusion
2. Stroke
3. Giant cell arteritis + vision changes
4. Acute closed-angle glaucoma

EMERGENCY Tx (< 24 hours)

1. Infectious keratitis
2. Endophthalmitis or severe uveitis
3. Acute retinal necrosis
4. Hyphema
5. Retinal detachment

URGENT REFERRAL (24-48 hours)

1. Non-infectious uveitis
2. Vitreous hemorrhage
3. Acute maculopathy
4. Central retinal vein occlusion
5. Optic neuritis

Altered Vision: Clinical Diagnostic Clues

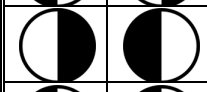
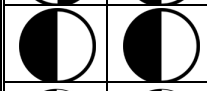
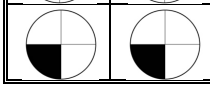
MONOCULAR VISION DISTURBANCE

	History	Physical
Keratitis	• Sharp superficial pain	• Tearing, red, irritated eye
Acute Closed-Angle Glaucoma	• Deep brow ache • Nausea, vomiting	• Tearing, red, extremely painful • Fixed mid-size pupil; hard eyeball
Vitreous Hemorrhage	• Vision reduction proportional to amount of blood	• +/- decreased red reflex • No relative afferent pupillary defect
Central Retinal Artery Occlusion	• Acute onset, painless • Severe vision loss	• Relative afferent pupillary defect • Milky-white retina + cherry-red spot
Central Retinal Vein Occlusion	• Subacute onset, painless • Vision loss up to severe	• +/- relative afferent pupillary defect • "Blood-and Thunder" retina
Retinal Detachment	• Floaters, black dots, photopsias; painless • Visual field deficit	• +/- relative afferent pupillary defect • Decreased red reflex • Elevated retina with folds
Optic Neuritis	• Pain on eye movement • Washed-out colour	• Relative afferent pupillary defect • Optic disc normal or swollen

BINOCULAR VISION DISTURBANCE

Acute binocular vision disturbance may be caused by either:

- a systemic process affecting both sides, e.g. giant cell arteritis resulting in bilateral ischemic optic neuropathy
- a chiasmal or post-chiasmal process

Field Loss*	Terminology	Pathology
	Bitemporal (bipolar) hemianopsia	Pituitary pathology
	Binasal hemianopsia	Bilateral internal carotid artery aneurysms, hydrocephalus
	Left homonymous hemianopsia	Lesion affecting the right optic tract Lesion affecting the right occipital lobe
	Left homonymous superior quadrantanopsia	Lesion affecting the lower right optic radiations in the temporal lobe
	Left homonymous inferior quadrantanopsia	Lesion affecting the upper right optic radiations in the parietal lobe

* from the patient's perspective

Double Vision

Double vision (minimal diplopia may lead to "blurry vision")
If decreased visual acuity/visual phenomena: see Altered Vision
If weakness/paresthesia: use also Weakness/Paresthesia
If headache: use also Headache/Facial Pain

BACKGROUND

M	☐ Current medications?
A	☐ Allergies to meds or contrast?
P	☐ Past medical history?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

O	☐ When did the double vision start? What were you doing? ☐ Time till max intensity: sec? min? hr?
P	☐ Does the double vision persist when one eye is closed? ☐ Is the separation of images horizontal or vertical/diagnonal? ☐ Where are you looking when the double vision is worst? Least?
R	☐ Is there any corrective head position that makes the double vision tolerable? ☐ Is the double vision worse when looking near or worse at distance? ☐ Does the double vision worsen during the day?
T	☐ Constant or intermittent? Increasing? ☐ Prior similar episodes?
+	☐ Eye or periorbital pain? ☐ Pain upon eye movement? ☐ Headache? ☐ Fever? ☐ Other neurological deficits?

VITALS

☐ RR, SpO2%, HR, BP, Temp?

NEUROSCREEN

Cranial nerves	☐ Visual fields? ☐ Pupil size, reactivity? ☐ Eye movements? ☐ Facial movement? ☐ Soft palate and tongue?
Motor	☐ Proximal arm strength? ☐ Distal arm strength? ☐ Proximal leg strength? ☐ Distal leg strength?
Coordi -nation	☐ Romberg? ☐ Finger-nose? ☐ Knee-shin?

EYE

☐ Inspection of the eyelids, conjunctiva, cornea ☐ Visual acuity ☐ Swinging flashlight test ☐ Fundoscopy

TESTS

☐ CRP if > 50 years

CONSIDER

1. Intracranial process (e.g. aneurysm)
2. Orbital process
3. Giant cell arteritis
4. Wernicke's encephalopathy

Double Vision: Differential Diagnoses & Clinical Clues

DIFFERENTIAL DIAGNOSIS BINOCULAR DIPLOPIA

Anatomy	Examples
Extraocular Muscle & Orbit	• Infections (orbital cellulitis) • Neoplasms of the orbit • Myositis • Trauma (e.g. muscle entrapment secondary to orbital fracture) • Graves' disease with exophthalmos and diplopia
Neuromuscular Junction	• Myasthenia gravis (antibodies to the post-synaptic ACh receptors) • Botulism (decreased presynaptic acetylcholine release)
Cavernous Sinus	• Cavernous sinus thrombosis (e.g. from a facial infection or aseptic) • Intracavernous carotid artery aneurysm or carotid-cavernous fistula • Pituitary tumors or pituitary apoplexy, metastatic tumors, direct extension of nasopharyngeal tumor • Tolosa-Hunt syndrome (idiopathic granulamatous disease)
Cranial nerves	• Infarction, e.g. from diabetes or giant-cell arteritis • Skull-based tumors • Aneurysms • Meningitis • Miller-Fisher variant of Guillain-Barré syndrome
Brainstem (affecting cranial nerve nuclei and connections)	• Stroke, e.g. basilar artery thrombosis • Infections, e.g. viral encephalitis • Neoplasms • Wernicke's encephalopathy • Auto-immune, e.g. multiple sclerosis, systemic lupus erythematosus • Trauma, e.g. subdural hematoma, basilar skull fracture

DIAGNOSTIC CLUES

- **CN III palsy** (eye in "down and out" position, ptosis) + **fixed dilated pupil** suggests nerve compression by aneurysm of posterior communicating artery or skull based tumor
- **CN III palsy** (eye in "down and out" position, ptosis) + **pupillary sparing** suggests microvascular infarction as seen in diabetes, or Wernicke's encephalopathy
- **Horizontal diplopia** worse when looking at a distance + **papilledema** suggests CN VI pathology due to increased intracranial pressure or pseudotumor cerebri
- **Involvement of CN III, IV and VI without involvement of CN II** suggests sinus cavernosus syndrome; other potential findings include decreased corneal reflex (V_1), maxillary sensory loss (V_2), chemosis (obstruction of venous flow)
- **Involvement of CN III, IV, VI and CN II** suggests orbital pathology
- **Pain localized directly to the eye** or **upon eye movement** suggests intraorbital pathology
- **Headache preceding palsy** suggests ischemic etiologies (e.g. diabetes mellitus or giant cell arteritis)

DIFFERENTIAL DIAGNOSIS MONOCULAR DIPLOPIA

• Corneal irregularity	• Lens dislocation	• Cataract	• Psychiatric

Psychiatric Disturbance

Suspected psychosis, mania, depression
Use also Decreased level of consciousness or confusion
If suspected poisoning/overdose: use also Poisoning

BACKGROUND

M	☐ Current / alternative medications? ☐ Recent changes?
A	☐ Allergies?
P	☐ Past medical / psychiatric history?
L	☐ Life circumstances? Children?
E	☐ Alcohol: how often? How much?
S	☐ Substance abuse: illicit drugs?

HISTORY (from patient and/or other)

O	☐ When did the problem start? ☐ Gradual versus sudden onset?
Q	☐ Decreased or altered consciousness?
S	☐ Impact on daily function?
T	☐ Time course? Diurnal fluctuation? ☐ Prior similar episodes?
+	☐ Recent trauma? ☐ Other symptoms?

TESTS

- ☐ Acid-base: pH, pCO_2, HCO_3/BE
- ☐ Electrolytes: Na, K, Ca
- ☐ Hb, WBC, CRP
- ☐ Creatinine
- ☐ Liver function tests
- ☐ EKG if > 50 years

CONSIDER

1. Organic cause
2. Risk for suicide / self-harm
3. Risk for violence; access to firearms?
4. Children-contact social services?

MENTAL STATUS EXAMINATION

A	☐ Appearance: *posture* (stooped, relaxed, stiff, shaky, slouched ...), *clothes* (appropriate to age, season, setting; colours ...), *grooming* (clean, dirty, unbathed ...)
	☐ Attitude: friendly, cooperative, hostile, secretive, evasive, suspicious, apathetic, easily distracted, seductive, defensive, oppositional, resistant, irritable, shy ...
B	☐ Behavior: mannerisms, psychomotor activity, expression, compulsions, gait, agitation, grimaces, tics, twitches, ritualistic behaviour, chewing movements...
	☐ Babbling: *quantity* (expansive, paucity...), *rate* (fast, slow, pressured ...), *volume*, *flow* (hesitant, rambling ...), *clarity* (slurred, mumbled), *content* (neologims ...)
C	☐ Cognition-Process: logical, relevance, circumstantial, tangential, loose associations, incoherent, evasive, racing, blocking, perseveration, flight of ideas, vague ...
	☐ Cognition-Content: ruminations, delusions, grandiosity, preoccupations, ideas of reference, suicidal /paranoid ideation, obsessions, phobias, magical thinking ...
D	☐ Distorsion: hallucinations (false sensory perceptions without external stimuli), illusions, depersonalisation, derealisation, déjà vu, jamais vu ...
	☐ Dissociation: *insight* (complete denial; recognizes there is a problem but projects blame; both intellectual and emotional awareness), *perception of illness*
E	☐ Emotion-Affect i.e. observed expression of inner feeling: sad, hostile, indifferent, euthymic, dysphoric, detached, elated, labile, anxious, irritable, inappropriate ...
	☐ Emotion-Mood i.e. sustained state of inner feeling: happy, sad, despondent, fearful, discouraged/depressed, energized/elated/out of control, angry/irritable ...

Psychiatric Disturbance

ORGANIC VS PSYCHIATRIC

Suggests Organic	Suggests Psychiatric
• Age < 12 years or > 40 years without previous psychiatric diagnosis • Sudden onset of symptoms • Visual or tactile hallucinations • History of substance abuse • New medications including herbal meds • Seizure • No family history of psychiatric disorders	• Age 12 - 40 years • Previous psychiatric diagnosis • Gradual onset of symptoms • Auditory hallucinations • No recent ingestions of mind-altering stuff • No new medications • No seizures • Significant family history of psychiatric disorders (first-degree relatives)

Adapted from Tucci et al Emerg Med Clin N Am 2015;33:721

ORGANIC CAUSES OF PSYCHIATRIC DISTURBANCES

Pathophysiology	Examples
Vascular	• Thrombotic thrombocytopenic purpura
Infectious Infiltrative	• **Intracranial infections**: herpes encephalitis • **Extracranial infections**: sepsis, botulism
Neurological Neoplastic	• **Space occupying lesion** • **Paraneoplastic syndrome:** NMDAR-antibody encephalitis
Degenerative Deficiency	• Wernicke's encephalopathy, B12 deficiency • Wilson's disease
Intoxication Withdrawal	• **Traditional medications:** overdose, withdrawal, toxic levels • **Other substances:** alcohol (delirium tremens), cocaine, synthetic drugs, carbon monoxide, heavy metals
Autoimmune	• **SLE, myasthenia gravis, MS**
Trauma Mechanical	• **Intracranial**: concussion, shunt dysfunction • **Extracranial**: urinary retention, fat embolism syndrome
Electrolyte Endocrine Metabolic	• **Electrolytes**: hypercalcemia • **Endocrine/Metabolic**: hyperthyroidism/toxicosis, Addison's, hypothyroidism/myxedema coma, hepatic encephalopathy, uremia

SUICIDE RISK

Precipitating Factors	Predisposing Factors	Protective Factors
• Drug and alcohol misuse • Access to lethal means • Life events (e.g. recent loss) • New diagnosis of terminal or chronic physical illness • Media effects (e.g. local epidemic of suicide)	• Neuropsychiatric disorders • Family history of suicidal behavior • Previous suicide attempt • Adverse childhood experiences • Socioeconomic deprivation	• Family and community support • Ongoing medical and mental health care relationships • Cultural and religious beliefs that discourage suicide • Skills in problem solving

Adapted from Fazel et al. Suicide. NEJM 2020;382:266-74 + cdc.gov

Syncope/Seizure

Transient loss of consciousness with rapid onset & complete recovery
If residual decreased consciousness: use also Altered Consciousness

BACKGROUND

M	☐ Current medications? ☐ Recent additions, dosage changes?
A	☐ Allergies?
P	☐ Past medical history? ☐ Prior episodes with transient loss of consciousness?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

Prior
- ☐ Circumstances (Activity, standing, sitting, supine?)
- ☐ Prodrome? Pain? Palpitations?
- ☐ Trauma upon loss of consciousness?

During (if witnessed)
- ☐ Shaking?
- ☐ Skin colour?
- ☐ Duration of loss of consciousness?

After
- ☐ Confusion? If so, duration?
- ☐ Pain (muscle, head, chest, back, abdomen, leg)?

PHYSICAL

Vitals	☐ RR, SpO_2, HR, BP, Temp?
Mouth	☐ Tongue bite?
Head	☐ Head trauma?
Heart	☐ S3/S4, murmurs? ☐ Elevated JVP?
Legs	☐ Swelling?
Neuro	☐ See Weakness/Paresthesia for screening neurological exam

EKG*

Rate	☐ Tachy- bradycardia?
Rhythm	☐ AV block? ☐ Atrial fibrillation?
P	☐ Left atrial hypertrophy?
PR	☐ Short PR segment?
Q	☐ Deep, narrow in lateral leads? ☐ Signs of prior infarction?
QRS	☐ Bundle branch block? ☐ Delta wave? ☐ Epsilon wave?
R/S	☐ Tall precordial R waves?
ST	☐ Ischemia? ☐ Brugada pattern?
T	☐ Ischemia? RV strain?
QTc	☐ Prolonged? Short?

* EKG taken in the ED and prior available EKGs (e.g. taken by paramedics, GP)

CONSIDER

1. Seizure? Consider triggers:
- infection
- medications/non-compliance
- intoxication/withdrawal (esp alcohol)
- hypo-gluc, Na, Ca, Mg; hyper-Na, Ca
- sleep deprivation
- Head CT following first seizure and in the setting of posttraumatic seizure.

2. Syncope? Consider vascular causes:
- pulmonary embolism
- subarachnoid hemorrhage
- aortic dissection
- ruptured abdominal aortic aneurysm
- ruptured ectopic pregnancy

3. Syncope? Consider cardiogenic causes:
- arrhythmia
- valvulopathy

4. Driving restriction

Transient LOC: Syndromes & Clinical Decision Rules

1-TRANSIENT LOSS OF CONSCIOUSNESS?

Transient loss of consciousness: rapid onset of real or apparent loss of consciousness with loss of responsiveness, loss of motor control, amnesia for the period of unconsciousness, short duration, complete spontaneous recovery.

Conditions that do not fullfill these criteria:

• Complex partial seizure	No loss of motor control (no fall)
• Psychogenic pseudosyncope	Duration many minutes to hours
• Fall	Not unresponsive, no amnesia
• Cataplexy	No amnesia
• Vertebrobasilar TIA	Loss of consciousness (if present) is prolonged
• Carotid TIA	No loss of consciousness
• Metabolic disoders	Prolonged loss of consciousness
• Intoxication	Prolonged loss of/impaired consciousness

2-SEIZURE?

INFORMATION	SUGGESTS SEIZURE
MAPLES	• Known brain pathology
Circumstances	• Sleep deprivation
Prodrome	• Epigastric rising sensation • Strange smell/taste • Déjà vu, jamais vu • Shout upon loss of consciousness
During	• Convulsion onset prior to/upon LOC • Convulsion: symmetrical, synchronous, several (20-100) • Cyanosis
After	• Confused for several minutes • Myalgia

3-SYNCOPE CATEGORY?

Reflex	• Vasovagal • Situational (micturition, swallow, defecation, cough, sneeze, post-exercise) • Carotid sinus syndrome
Orthostatic Hypotension	• Drug-induced • Volume depletion • Primary autonomic failure (e.g. Parkinson's disease) • Secondary autonomic failure (e.g. diabetes, spinal cord injuries)
Cardiac	• Arrhythmia (bradycardia or tachycardia) • Structural: aortic stenosis, myocardial ischemia, hypertrophic cardiomyopathy, pericardial disease/tamponade . . . . • Great vessels: aortic disection, pulmonary embolism, pulmonary hypertension

Transient LOC: Clinical Decision Rule

CANADIAN SYNCOPE ARRHYTHMIA RISK SCORE

Purpose: predict death, arrhythmia or procedural interventions to treat arrhythmias within 30 days of ED evaluation among patients for whom arrythmia and non-arrhythmic serious conditions were not identified during the ED evaluation
Inclusion: adults (≥ 16 yr) with syncope presentin within 24 hours after the event
Exclusion: prolonged loss of consciousness (> 5 min), change in mental status from baseline after the syncope, obvious witnessed seizure or head trauma causing loss of consciousness, major trauma requiring hospital admission, intoxication with alcohol or illicit drugs, language barrier

CATEGORY	POINTS
Clinical Evaluation	
• Vasovagal predisposition*	-1
• History of heart disease÷	+1
• Any ED SBP < 90 or > 180 mm Hg‡	+1
Investigations	
• Troponin > 99%ile	+1
• QRS duration > 130 ms	+2
• QTc interval > 480 ms	+1
Diagnosis in Emergency Department	
• ED diagnosis of vasovagal syncope	-1
• ED diagnosis of cardiac syncope	+2

SCORE	RISK
-2	0.2%
-1	0.5%
0	0.9%
1	1.9%
2	3.8%
3	7.5%
4	14.3%
5	25.4%
6	41.1%
7	58.8%
8	74.5%

Score of ≥ 0 had SN 97% and SP 53% for death/arrhythmia/intervention within 30 days.

*Warm-crowded place, prolonged standing, fear, emotion or pain
÷ Includes history of coronary or valvular heart disease, cardiomyopathy, congestive heart failure or non-sinus rhythm (ECG evidence during the index visit or documented history of ventricular or atrial arrhythmias, or device implantation)
‡ Includes blood pressure values from triage until ED disposition

Syncope: Risk Stratification

BACKGROUND

Low	• Long history (years) of recurrent syncope with low-risk features with the same characteristics of the current episode • Absence of structural heart disease
High	• Severe structural or coronary artery disease (heart failure, low left ventricular ejection fraction or previous myocardial infarction)

HISTORY

Low	• Associated prodrome typical of reflex syncope (e.g. lightheadedness, feeling of warmth, sweating, nausea, vomiting) • After sudden unexpected unpleasant sight, sound, smell or pain • After prolonged standing or crowded, hot places • During a meal or postprandial • Triggered by cough, defaecation or micturition • With head rotation or pressure on carotid sinus (e.g. tumour, shaving, tight collars) • Standing from supine/sitting position
High Major	• New onset of chest discomfort, breathlessness, abdominal pain or headache • Syncope during exertion or when supine • Sudden-onset palpitation immediately followed by syncope
High Minor*	• No warning symptoms or short (<10 s) prodrome • Family history of sudden cardiac death at young age • Syncope in the sitting position

*High risk only if associated with structural heart disease or abnormal ECG

PHYSICAL

Low	• Normal examination
High	• Unexplained systolic BP in the ED <90 mm Hg • Suggestion of gastrointestinal bleed on rectal examination • Persistent bradycardia (<40 bpm) in awake state and in absence of physical training • Undiagnosed systolic murmur

EKG

Low	• Normal EKG
High	• ECG changes consistent with acute ischaemia • Mobitz II second-degree and third-degree atrioventricular (AV) block • Slow atrial fibrillation (AF) (<40 bpm) • Persistent sinus bradycardia (<40 bpm), or repetitive sinoatrial block or sinus pauses >3 s in awake state and in absence of physical training. • Bundle branch block, intraventricular conduction disturbance, ventricular hypertrophy or Q waves consistent with ischaemic heart disease or cardiomyopathy • Sustained and non-sustained ventricular tachycardia • Dysfunction of an implantable cardiac device (pacemaker or implantable cardioverter defibrillator) • ST-segment elevation with type 1 morphology in leads V1−V3 (Brugada pattern) • QTc >460 ms in repeated 12-lead ECGs indicating long QT syndrome

Vertigo/Dizziness

Transient or permanent illusion of motion or unsteadiness
If feeling of impending faint: use instead Syncope/Seizure

BACKGROUND

M	☐ Current medications?
A	☐ Allergies?
P	☐ Past medical history?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

O	☐ When did vertigo start? Activity at the time? ☐ Time till max intensity: sec? min? hr?
P	☐ Is the vertigo brought on by changing position?
Q	☐ Illusion of motion? Faintness?
R	☐ Worse with movement of the head?
S	☐ Impact on daily function (e.g. able to walk?)
T	☐ Duration: sec, min, hr, days? ☐ Prior similar episodes?
+	☐ Diplopia? ☐ Dysarthria? ☐ Dysphagia? ☐ Decreased hearing / tinnitus? ☐ Decreased strength or sensation? ☐ Dysmetria? ☐ Headache / neck pain? ☐ Trauma to the head / neck recently?

PHYSICAL

☐ RR, SpO2%, HR, BP, Temp?

NEUROSCREEN

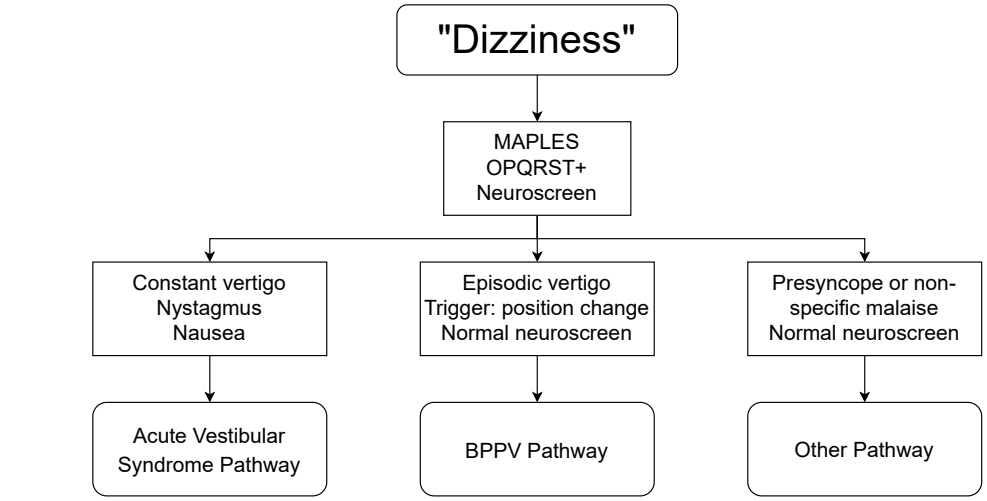
Cranial nerves	☐ Visual fields? ☐ Pupil size, reactivity? ☐ Eye movements?* ☐ Facial movement? ☐ Soft palate and tongue?
Motor	☐ Proximal arm strength? ☐ Distal arm strength? ☐ Proximal leg strength? ☐ Distal leg strength?
Coordi-nation	☐ Romberg? ☐ Finger-nose? ☐ Knee-shin?

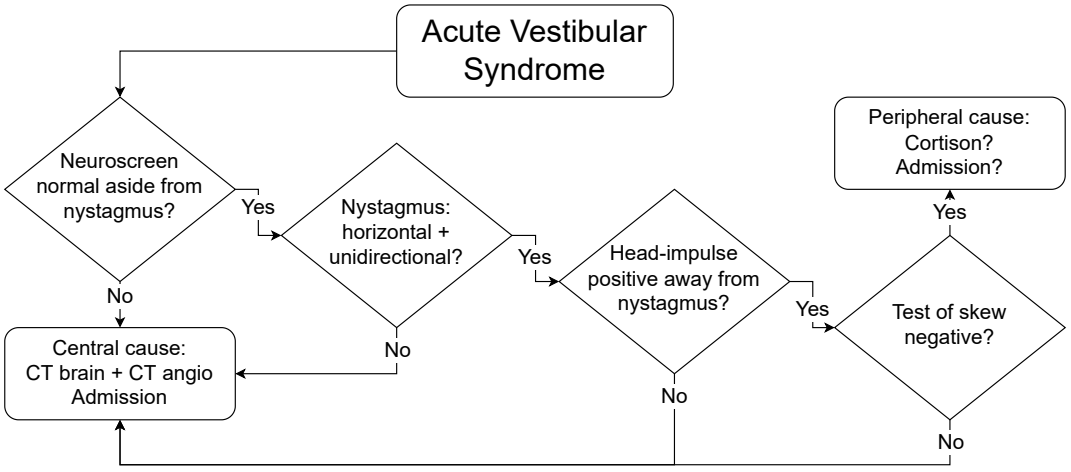
* Use Frenzel's glasses to detect subtle nystagmus

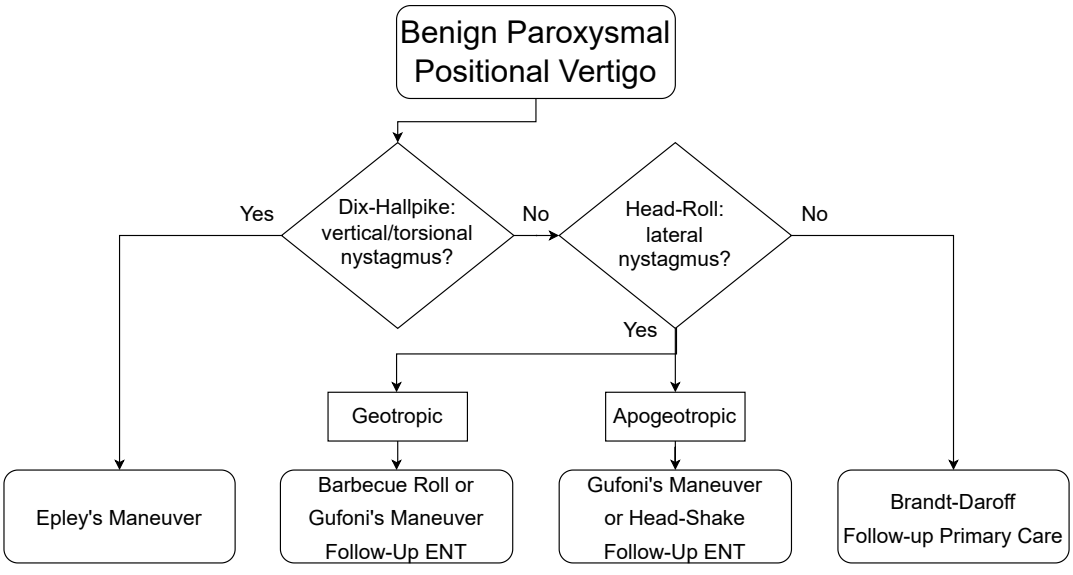
CONSIDER

1. Stroke, vertebrobasilar dissection
2. Bacterial labyrinthitis
3. Metabolic-Cardiovascular-Toxic conditions

Vertigo: Algorithms







Weakness/Paresthesia

Weakness and/or paresthesia
If head trauma: use also Trauma to the Head/Neck

BACKGROUND

M	☐ Current medications?
A	☐ Allergies?
P	☐ Past medical history?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

O	☐ When did the deficit start? ☐ What were you doing? ☐ Time till max intensity: sec? min? hr?
P	☐ Location of the deficit?
Q	☐ Weakness? Paresthesia? Both?
S	☐ Degree of deficit? Impact on daily function?
T	☐ Constant or intermittent? Increasing? Progressing (ascending vs descending)? ☐ Prior similar episodes?
+	☐ Trouble finding/understanding words? ☐ Vision problems? ☐ Urinary incontinence/retention? ☐ Pain (head, neck, chest, back)? ☐ Fever?

PHYSICAL

Vitals	☐ RR, SpO2%, HR, BP, Temp?
Heart	☐ S3/S4, murmurs? ☐ Irregular rhythm?

NEUROSCREEN

Cranial nerves	☐ Visual fields? ☐ Pupil size, reactivity? ☐ Eye movements? ☐ Facial movement? ☐ Soft palate and tongue?
Motor	☐ Proximal arm strength? ☐ Distal arm strength? ☐ Proximal leg strength? ☐ Distal leg strength?
Coordi-nation	☐ Romberg? ☐ Finger-nose? ☐ Knee-shin?

TESTS

☐ EKG if > 50 years
☐ CRP if > 50 years

CONSIDER

1. Stroke / TIA within 5 hours
2. Dissection (aorta, carotid, vertebrobasilar arteries)
3. Myelopathy
4. Giant cell arteritis
5. Guillain-Barré syndrome
6. Hyper- and hypokalemia

Stroke Mimics ("5M-3P")

- Migraine
- Mass (tumor, bleed)
- Metabolic
- Meningitis + systemic infections
- Multiple sclerosis
- Peripheral (e.g. mononeuropathy)
- Post-ictal + non-convulsive status
- Psychiatric

Weakness/Paresthesia: Clinical Syndromes

FOCAL FOREBRAIN LESION

- Unilateral weakness in the face (forehead sparing), arm and/or leg
- Dysphasia, neglect, conjugated eye deviation, homonymous hemianopsia: cortical involvement

FOCAL BRAINSTEM and/or CEREBELLAR LESION

- Unilateral cranial nerve dysfunction (no forehead sparing)
- Contralateral weakness and/or decreased sensation with long tract involvement

MYELOPATHY

- Absence of cortical and cranial nerve involvement; a sensory or motor level is present
- **Total cord syndrome**: bilateral weakness, loss of sensation for all modalities and sphincter dysfunction
- **Anterior cord syndrome**: bilateral weakness and loss of sensation for pain; preserved touch
- **Posterior cord syndrome**: bilateral loss of touch; preserved strength and pain sensation
- **Central cord syndrome**: bilateralt loss of strength and pain sensation in the arms
- **Brown-Séquard**: ipsilateral weakness and loss of sensation for touch; preserved pain sensation
- **Conus medullaris/cauda equina syndromes**: leg weakness in specific myotomes; saddle anesthesia; incontinence

RADICULOPATHY

	Paresthesia	Weakness	Hyporeflexia
C5	Lateral upper arm	Arm abduction	Supinator
C6	Lateral forearm, dig 1-2	Elbow flexion	Biceps
C7	Dorsal forearm, dig 3	Elbow extension	Triceps
C8	Medial forearm, dig 4-5	Finger flexion	
T1	Medial elbow	Finger abduction	
L3	Anteromedial thigh	Hip flexion/adduction	
L4	Anterior thigh + medial shin	Knee extension	Patella
L5	Anterolateral shin, foot dorsum	Ankle dorsiflexion	
S1	Posterolateral leg, sole of foot	Ankle plantar flexion, hip extension	Achilles

PERIPHERAL MONONEUROPATHY

Nerve	Paresthesia*	Weakness*
Axillary	Lateral upper arm	Arm abduction
Musculocutaneus	Lateral forearm	Elbow flexion supinated forearm
Radial	Radial aspect of hand dorsum	Elbow/wrist/finger extension
Median	Radial aspect of hand palm	Thumb opposition
Ulnar	Ulnar hand, dig 5 + medial dig 4	Finger abduction/adduction
Lateral cutaneous	Lateral thigh	
Obturator	Medial thigh	Hip adduction
Femoral	Anterior thigh + medial shin	Knee extension
Sciatic	Foot	Knee flexion, ankle (dorsi/plantar)
Tibial	Foot sole, lateral dig 5	Ankle plantarflexion + inversion
Peroneal, deep	Web space between dig 1-2	Ankle & toe dorsiflexion
Peroneal, superficial	Lateral calf, foot dorsum	Ankle eversion

* The distribution of the deficit depends on the level of injury

Eye Trauma

Blunt or penetrating trauma to the eye
See also Trauma to the head/neck

BACKGROUND

M	☐ Current medications? ☐ Platelet inhibitors? Anticoagulant?
A	☐ Allergies?
P	☐ Past medical history?

HISTORY

- ☐ Mechanism of injury?
- ☐ Visual acuity?
- ☐ Double vision?
- ☐ Pain?

PHYSICAL EXAMINATION OF THE EYES

- ☐ Inspection (including symmetry)
- ☐ Visual acuity
- ☐ Pupillary size
- ☐ Pupillary reactivity to light direct/indirect
- ☐ Swinging flashlight test (dimmed lighting)
- ☐ Visual fields
- ☐ Extraocular movements

EYE ULTRASOUND

Gently place a Tegaderm over the eyelid, apply LOTS of gel and "float" the linear probe over the eye without applying pressure

- ☐ Is the anterior chamber present? Absence suggests globe rupture/perforation
- ☐ Is the posterior chamber black, round and smooth?
- ☐ Is there retinal detachment (a linear bright white anechoic segment flapping off the posterior globe)?
- ☐ Is the overall shape of the globe round? A triangular shape ("guitar pick sign") suggests retrobulbar hematoma
- ☐ Pupillary response can be assessed in the transverse and coronal plane by shining a light in other eye and/or a light through the eyelid

TESTS

- ☐ EKG if > 50 years
- ☐ INR and thrombocytes if the patient is taking an anticoagulant

CONSIDER

1. Retrobulbar hematoma with orbital compartment syndrome
2. Hyphema
3. Retinal detachment
4. Globe rupture

Eye Trauma

FINDINGS ON EYE EXAMINATION

Inspection	Enophthalmos suggests globe rupture Exophthalmos suggests retrobulbar process
Visual acuity	Impaired with orbital compartment syndrome, large retinal detachment
Pupils	RAPD suggests optic nerve dysfunction, large retinal detachment, orbital compartment syndrome
Visual field defect	Suggests retinal detachment
Extraocular movements	Extraocular pathology
Increased intraocular pressure	Suggests traumatic acute glaucoma associated with hyphema or orbital compartment syndrome

LATERAL CANTHOTOMY

INDICATIONS

Lateral canthotomy and cantholysis is for patients with orbital compartment syndrome within 60-120 min of the onset of ischemic features, as a temporizing measure prior to surgical evacuation of the retrobulbar hematoma. Suggestive symptoms:

Mechanical Consequences	Ischemic Consequences
• proptosis (best seen from head of bed) • ophthalmoplegia • ocular pressure greater than 40 mm Hg	• decreased visual acuity • afferent pupillary defect • blown pupil • optic nerve pallor • severe eye pain • cherry-red macula

CONTRAINDICATION: ruptured globe

TECHNIQUE

1-Anesthetize the lateral cantus with carbocain and adrenalin
2-Crush the lateral cantus with a clamp to devascularize the area for 1 minute
3-Cut the lateral cantus (lateral incision)
4-Pull the lower eyelid away from the globe with toothed forceps
5-“Strum” the tissue under the canthotomy with the scissors to identify the inferior crus of the lateral canthal ligament (feels like a guitar string). Cut the inferior crus. The scissors are directed inferiorly during this step, perpendicular to the canthotomy incision.
6-Recheck the intraocular pressure. If it remains over 40 mm Hg, cut the superior crus in a similar manner.

Trauma to the head or neck
If altered consciousness: use instead Altered Consciousness
If loss of consciousness prior to trauma: use also Syncope/Seizure

Head/Neck Trauma

BACKGROUND

M	☐ Current medications? ☐ Platelet inhibitors? Anticoagulant?
A	☐ Allergies?
P	☐ Past medical history?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

Prior ☐ Circumstances? ☐ Prior symptoms (e.g. palpitations?)
Trauma ☐ Mechanism of injury? ☐ Loss of consciousness?
After ☐ Amnesia (retrograde, anterograde)? ☐ Vomiting? ☐ Headache? Neck pain? ☐ Seizure? ☐ Paresthesia? ☐ Vision disturbance? ☐ Altered bite?

PHYSICAL

Vitals	☐ RR, SpO2%, HR, BP, Temp?
Head	☐ Inspection ☐ Palpation
C-spine	☐ Palpation
Face	☐ Visual acuity ☐ Swinging flashlight test ☐ Eye movements ☐ Palpation of the orbital rims ☐ Palpation of the nasal bridge ☐ Examination of the nasal septum ☐ Inspection of the oral cavity ☐ Examintion of jaw movement ☐ Otoscopy
Neuro	☐ Level of consciousness ☐ Gross extremity sensation and strength

TESTS

- ☐ EKG if > 50 years
- ☐ INR and thrombocytes if the patient is taking an anticoagulant

CONSIDER

1. Cause (e.g. assault, seizure poisoning, arrhythmia, etc.). Contact social services?
2. Intracranial bleed
3. C-spine fracture
4. Facial fracture
5. Admission for observation

Head/Neck Trauma: Clinical Diagnostic Rules Adults

SCANDINAVIAN NEUROTRAUMA COMMITTEE GUIDELINES

For adults with minimal-moderate head injury (GCS 9-15 / RLS 1-3) ≤ 24 hrs of injury:

GCS 9-13 / RLS 3	CT head **and** admission for observation > 24 hrs
GCS 14-15 / RLS 1-2 + **any of**: • posttraumatic seizures • focal neurological deficits • clinical signs of depressed or basal skull fracture • shunt-treated hydrocephalus • therapeutic anticoagulation or coagulation disorders	CT head **and** admission for observation > 24 hrs
GCS 14-15 / RLS 1-2 + **both of**: • age ≥ 65 years • anti-platelet medication	CT head **or** admission for observation ≥ 12 hours; discharge* if CT normal
GCS 14 / RLS 2 **or** GCS 15 / RLS 1 **and any of:** • suspected/confirmed loss of consciousness • repeated vomiting (≥ 2 episodes)	S100B if < 6 hrs since injury; discharge* if < 0.1 ug/L CT head **or** admission for observation ≥ 12 hrs if > 6 hrs or S100B not available or S100B > 0.1 ug/L; discharge* if CT normal
GCS 15 / RLS 1 and none of the risk factors listed above	Discharge*

* with oral and written instructions

CANADIAN C-SPINE RULE

No cervical spine x-ray is required if **all 4** are present:

- **Inclusion Criteria: all**
- **High Risk Factors: 0**
- **Low Risk Factors: ≥ 1**
- Can actively rotate neck > 45° left and right

Inclusion Criteria

- > 15 years
- No history of back or vertebral disease
- Normal level of consciousness
- Trauma < 48 hrs old

Low Risk Factors

- Simple rear-end motor vehicle collision
- Sitting position in the ED
- Ambulatory at any time
- Delayed (not immediate) onset of neck pain
- Absence of midline C-spine tenderness

NEXUS LOW-RISK CRITERIA

No cervical spine x-ray is required if **all 5** are present:

- Normal level of alertness
- No evidence of intoxication
- No painful distracting injuries
- No focal neurologic deficit
- No posterior cervical-spine tenderness

High Risk Factors

- Age ≥ 65 years
- Paresthesias in the extremities
- Dangerous mechanism of injury:
  - Fall from ≥ 1 m or 5 stairs
  - Axial load on the head
  - Motor vehicle collision at high speed (> 100 km/h) or with rollover or ejection
  - A collision involving a motorized recreational vehicle
  - A bicycle collision

Head/Neck Trauma: Clinical Diagnostic Rules Children

NEUROIMAGING HEAD IN CHILDREN

Neuroimaging in the presence of ≥ 1 of the following criteria

< 2 years	≥ 2 years
• Focal neurologic findings • Acute skull fracture, including depressed or basilar fracture • Seizure following injury • Altered mental status (eg, lethargy or irritability) • Definite loss of consciousness if longer than a few seconds • Bulging fontanelle • Persistent vomiting • Suspicion of child abuse	• Focal neurologic findings • Skull fracture, especially findings of basilar skull fracture • Seizure • Persistent altered mental status (eg, agitation, lethargy, repetitive questioning, or slow response to verbal questioning) • Prolonged loss of consciousness

Observation or neuroimaging in the presence of ≥ 1 of the following criteria

< 2 years	≥ 2 years
• Vomiting that is self-limited • Loss of consciousness that is uncertain, or isolated and very brief (less than a few seconds) • History of lethargy or irritability, now resolved • Behavioral change reported by caregiver • Injury caused by high-risk mechanism of injury (eg, fall more than three feet, patient ejection, death of a passenger, rollover, high-impact head injury) • Scalp hematoma (particularly nonfrontal) • Skull fracture more than 24 hours old (nonacute) • Unwitnessed trauma of concern (eg, fall heard in adjacent room with possible loss of consciousness) • Age younger than three months with nontrivial trauma	• Vomiting • Headache • Questionable or brief loss of consciousness (LOC) • Injury caused by high-risk mechanism of injury

No neuroimaging if all of the following criteria are met

< 2 years	≥ 2 years
• No severe mechanism of injury* • Normal mental status • No palpable skull fracture • No history of LOC ≥ 5 sec • No occipital or parietal or temporal scalp haematoma • Acting normally per parent	• No severe mechanism of injury* • Normal mental status • No signs of basilar skull fracture • No history of LOC • No history of vomiting • No severe headache

* Fall > 1.5 m for child > 2 years; fall > 0.9 m if child < 2 years; head struck by high impact object; motor vehicle collision with patient ejection, death of another passenger, or rollover; pedestrian or bicyclist without a helmet struck by a motorized vehicle

C-SPINE INJURY IN CHILDREN

One case-control study including 540 children < 16 years with cervical spine injury sustained after blunt trauma identified 8 risk factors. Having ≥ 1 risk factor had SN 98% (96-99) and SP 26% (23-29) for cervical spine injury.

Risk Factors	Details
1-Predisposing condition	Condition predisposing to cervical spine injury*
2-High-risk motor vehicle crash	Head-on collision, rollover, ejection from the vehicle, death in the same crash, or a speed of more than 55 mph (90 km/h)
3-Diving	
4-Substantial torso injury	Injuries warranting surgical intervention or inpatient observation affecting the thorax, including the clavicles, abdomen, flanks, back including the spine and the pelvis (e.g. rib fractures, visceral or solid organ injury, pelvic fracture)
5-Altered mental status	GCS < 15, < A on the AVPU scale
6-Focal neurologic findings	Paresthesias, sensory loss, motor weakness
7-Neck pain	Any documented tenderness on examination of the neck in the history or physical examination
8-Torticollis	Limited range of motion or difficulty moving the neck

* Down syndrome, Klippel-Feil syndrome, achondrodysplasia, mucopolysaccharidosis, Ehlers-Danlos syndrome, Marfan syndrome, osteogenesis imperfecta, Larsen syndrome, juvenile rheumatoid arthritis, juvenile ankylosing spondylitis, renal osteodystrophy, rickets, history of cervical spine injury or surgery

Wound

If trauma to the head or neck: use also Trauma to the Head/Neck

BACKGROUND

M	☐ Current medications?
A	☐ Allergies (e.g. to anesthetics used during dental procedures)?
P	☐ Past medical history?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

When	☐ When did the wound occur?
What	☐ What were you doing at the time? ☐ Mechanism of injury? ☐ Might foreign material still be present in the wound?
Why	☐ Accident? Poisoning? Loss of consciousness? Assault? Self-harm?

PHYSICAL

1. Protective gear	☐ Put on gloves, consider eye guard, mouth guard
2. Distal function	☐ Assess touch (two point discrimination?) ☐ Assess motor function (specific tendon function?) ☐ Assess perfusion
3. Anesthesia	1. Apply antiseptic solution to intact skin around wound 2. Anesthetize the wound with lidocain +/- adrenalin
4. Inspection	1. Apply hemostatic measures if needed 2. Irrigate with NaCl or tap water under pressure (use splash guard) 3. Inspect for injured structures (e.g. tendons) 4. Inspect for foreign material +/- scrub, debride as needed

CONSIDER

1. Imaging to rule-out foreign material (e.g. ultrasound, X-ray)
2. Primary closure
3. Tetanus prophylaxis
4. Antibiotics
5. Child/elder abuse

Wound Tips

PRIMARY CLOSURE

Primary closure is contraindicated in the following settings:

- Wounds that are already infected
- Contamination with soil, organic matter, faeces
- Extensive tissue damage, e.g. explosion injuries, high-velocity missile injuries, complex crush injuries
- Deep or contaminated lacerations on the bottom of the foot
- Human bite wounds

Alternatives to primary closure include:

- Secondary closure (excision of the wound followed by primary closure)
- Delayed primary closure on day 4-5
- Primary healing i.e. healing by secondary intention

TETANUS PROPHYLAXIS

Minimally contamined minor wound:

- Fully immunized ≤ 10 years since last dose: no prophylaxis
- Not fully immunized or > 10 years since last dose: tetanus toxoid

Tetanus-prone wound (contaminated or complex wound, e.g. deep puncture wound):

- Fully immunized ≤ 5 years since last dose: no prophylaxis
- Fully immunized 5-10 years since last dose: tetanus toxoid
- Fully immunized > 10 years since last dose OR non-fully immunized: tetanus toxoid + human tetanus immune globulin

ANTIBIOTICS

Consider 72 hours of antibiotic treatment in the following settings:

- extremity bite wounds
- puncture-type bite wounds in any location
- intraoral lacerations that are sutured
- orocutaneous lip wounds
- wounds that cannot be cleaned or débrided satisfactorily
- highly contaminated wounds (e.g. with soil, organic matter, purulence, faeces, saliva)
- wounds involving tendons, bones, or joints
- wounds requiring extensive débridement in the operating room
- wounds in lymphedematous tissue
- distal extremity wounds when treatment is delayed for 12 to 24 hours
- patients with orthopedic prostheses
- patients at risk for the development of infective endocarditis

The choice of antibiotics depends on the cause of the wound (e.g. the species responsible for the bite) and evolving bacterial resistance.

BRUISES SUGGESTING CHILD ABUSE

- Bruises on torso, ear, neck, or buttocks
- Any bruising in infants < 6 months of age
- > 1 bruise in a pre-mobile infant
- > 2 bruises in a crawling child

Allergic Reaction

Suspected allergic reaction (rash, pruritus, swelling etc)

BACKGROUND

M	☐ Recently taken/terminated medications/substances? ☐ Recent NSAID use?
A	☐ Known allergies to medications, food, other?
P	☐ Past medical history? ☐ Recent medical test (e.g. with contrast agent)?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

CONSIDER

1. Anaphylaxis
2. Angioedema

HISTORY

O	☐ When did the symptoms start? What were you doing? ☐ Time till max intensity: sec? min? hr?
P	☐ Which body parts are affected?
Q	☐ Rash? Swelling? Itch? Pain?
R	☐ Effect of measures if taken (e.g. corticosteroids, antihistamine)?
S	☐ Impact on daily function?
T	☐ Constant, intermittent, increasing symptoms? ☐ Prior similar episodes?
+	☐ Food intake? ☐ Insect bite? ☐ New soap / washing detergent?

PHYSICAL

A	☐ Hoarse? Stridor? ☐ Lip- tongue swelling?
B	☐ SpO2% ☐ Respiratory rate? ☐ Lung auscultation? ☐ Chest wall examination
C	☐ Pulse/blood pressure ☐ Heart rate
D	☐ Level of consciousness?
E	☐ Front side of the body ☐ Back side of the body ☐ Temperature?

Allergic Reaction: Clinical Diagnostic Clues

ANAPHYLAXIS

Anaphylaxis is a severe, systemic hypersensitivity reaction that affects airway, breathing and/or circulation and is usually associated with skin (e.g. urticarial) and/or mucosal symptoms. Anaphylaxis is highly likely in any one of the following three contexts:

1. Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissue, or both (eg, generalized hives, pruritus or flushing, swollen lips-tongue-uvula) and at least one of the following:

- Respiratory compromise (eg, dyspnea, wheeze-bronchospasm, stridor, reduced PEF, hypoxemia)
- Reduced BP or associated symptoms of end-organ dysfunction (eg, hypotonia [collapse], syncope, incontinence)

2. Two or more of the following that occur rapidly after exposure to a *likely* allergen for that patient (minutes to several hours):

- Involvement of the skin-mucosal tissue (eg, generalized hives, itch-flush, swollen lips-tongue-uvula)
- Respiratory compromise (eg, dyspnea, wheeze-bronchospasm, stridor, reduced PEF, hypoxemia)
- Reduced BP or associated symptoms (eg, hypotonia [collapse], syncope, incontinence)
- Persistent gastrointestinal symptoms (eg, crampy abdominal pain, vomiting)

3. Reduced BP after exposure to *known* allergen for that patient (minutes to several hours):

- Infants and children: low SBP (age specific) or > 30% decrease in SBP
- Adults: SBP of less than 90 mm Hg or > 30% decrease from that person's baseline.

ANGIOEDEMA

Angioedema results from the fast onset of increased vascular permeability in subcutaneous or submucosal tissue. Symptoms and signs include:

- Swelling of the face (eyelids, lips, tongue), extremities and genitalia
- Swelling of the larynx, resulting in throat tightness, dyspnea, dysphonia, dysphagia
- Swelling of the intestine, resulting in abdominal pain, nausea and vomiting
- Urticaria, flushing, generalized pruritus, bronchospasm and/or hypotension are present in the setting of histamine-induced angioedema but absent in the setting of bradykinin-induced angioedema (e.g. ACE-inhibitor induced, hereditary or acquired C1-inhibitor deficiency)

Diarrhea

Loose or watery bowel movements

BACKGROUND

M	☐ Current medications? ☐ Recent antibiotic use?
A	☐ Allergies?
P	☐ Past medical history?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

O	☐ When did the diarrhea start? ☐ Travel history? Food prior to diarrhea onset?
Q	☐ Watery? Bloody? Tarry?
R	☐ Worsened with food / fluid intake?
S	☐ Volume? Frequency?
T	☐ Duration? ☐ Prior similar episodes?
+	☐ Fever? ☐ Abdominal pain?

PHYSICAL

Vitals	☐ RR, SpO2%, HR, BP, Temp?
Abdo	☐ Inspection ☐ Auscultation ☐ Palpation
PR	☐ Stool colour?

TESTS

☐ CRP

CONSIDER

1. Sepsis
2. Gastrointestinal bleeding
3. Invasive bacterial syndrome
4. Epidemiological features justifying presumptive antimicrobial therapy

Diarrhea: Clinical Diagnostic Clues

BAYESIAN APPROACH TO ACUTE INFECTIOUS DIARRHEA IN ADULTS

Goodgame 2006 recommends categorizing adults with acute infectious diarrhea (≥ 3 loose stools per day for < 14 days) into three categories for the sake of further management:

Category	Features	Infectious agent	Management
Viral or "norovirus-like" diarrhea	• No specific epidemiologic risk factor • No clinical feature suggestive of severe bacterial infection	• Norovirus • Bacteria (e.g. Salmonella) and protozoa producing an uncomplicated gastroenteritis syndrome	• No specialized diagnostic testing or antimicrobial management • Avoid milk products • Loperamid 4 mg once and 2 mg with each liquid stool
Severe bacterial infection	• Fever > 38.5°C • Bloody diarrhea • Voluminous diarrhea • Severe abdominal pain • > 6 stools per 24 hours • Diarrhea persisting > 7 days	• Salmonella, Campylobacter, Shigella • Shiga-toxin producing E coli • Yersinia • Vibrio • Clostridium difficile	• Stool testing for bacterial (or amoebic) infection, shiga toxin • If the signs and symptoms are severe, presumptive antibiotic therapy is recommended (unless E coli O157:H7 is suspected)
Epidemiologic risk factors	• Travel	• 80% probability of bacterial etiology • Persistent diarrhea suggests a protozoa	• Presumptive antibiotic therapy combined with clinical observation
	• Hospitalized > 3 days • Antibiotic use • Contact with health care personnel	• Clostridium difficile	• Stools for Clostridium difficile toxin • Presumptive treatment while awaiting test results is appropriate in severely ill patients
	• Immuno-compromised host	• Virus, bacteria, mycobacteria, protozoa	• Consult infectious disease specialist

HEMOLYTIC-UREMIC SYNDROME

Diarrhea occurring in the setting of hemolysis, thrombocytopenia and uremia suggests hemolytic-uremic syndrome. Most cases are caused by E coli O157:H7.

Dyspnea

Shortness of breath

BACKGROUND

M	☐ Current medications? ☐ Birth control pill, other hormonal treatments?
A	☐ Allergies?
P	☐ Past medical history? ☐ Prior heart or thromboembolic disease?
L	☐ Life circumstances (e.g. occupation, pets)?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

O	☐ When did the dyspnea start? What were you doing? ☐ Time till max intensity: sec? min? hr?
P	☐ Worse when lying down?
Q	☐ Air hunger? Chest tightness?
R	☐ Worse with exertion?
S	☐ Impact on daily function?
T	☐ Constant or intermittent? Increasing? ☐ Prior similar episodes?
+	☐ Chest pain or discomfort? ☐ Leg pain or swelling? ☐ Fever / chills? ☐ Cough (dry or productive-sputum colour)?

PHYSICAL

Vitals	☐ RR, SpO2%, HR, BP, Temp?
Heart	☐ S3/S4, murmurs? ☐ Elevated JVP?
Lungs	☐ Chest wall movements? ☐ Auscultation: rales? ronchi? decreased breath sounds?
Legs	☐ Swelling? Edema?

TESTS

- ☐ pH, pCO2, HCO3/BE
- ☐ CRP
- ☐ EKG if > 40 years
- ☐ Ultrasound:
  - Heart: Pericardial fluid? Dilated RV? Decreased contractility?
  - IVC: Dilated IVC? Decrease upon inspiration?
  - Juice: Pleural fluid?
  - Lung: Lung-sliding? A-lines vs B-lines?

CONSIDER

1. Upper respiratory tract problem (e.g. anaphylaxis, epiglottitis, retropharyngeal abscess)
2. Acute coronary syndrome
3. Pulmonary embolism
4. Pneumonia

Dyspnea: Clinical Diagnostic Rules & Clues

PULMONARY EMBOLISM: THE SIMPLIFIED WELLS SCORING SYSTEM

Purpose: ruling-out PE with a negative d-dimer
Inclusion: clinically suspected PE: sudden onset of dyspnea, sudden deterioration of existing dyspnea, or sudden onset of pleuritic chest pain without another apparent cause
Exclusion: therapeutic doses of unfractionated or low-molecular-weight heparin for > 24 hrs, life expectancy < 3 mo, pregnancy, < 18 years, allergy to IV contrast, renal insufficiency (Crea clearance < 30 ml/min), too ill to undergo CT scanning, hemodynamic instability

RISK FACTORS	POINTS
• Clinical signs and symptoms of deep venous thrombosis*	3
• Alternative diagnosis less likely than pulmonary embolism	3
• Heart rate > 100/min	1.5
• Immobilization (> 3 days) or surgery in the previous 4 weeks	1.5
• Previous pulmonary embolism or deep ven thrombosis	1.5
• Hemoptysis	1
• Malignancy (receiving treatment, treated in the last 6 mo or palliative)	1

* minimum of leg swelling and pain with palpation of the deep veins
PE unlikely (score ≤ 4) + negative d-dimer: 0.5% nonfatal PE/DVT at 3 month follow-up

HEART FAILURE

Background	• Heart failure	LR+ 5.8	LR- 0.45
	• Myocardial infarction	LR+ 3.1	LR- 0.69
Symptoms	• Paroxysmal nocturnal dyspnea	LR+ 2.6	LR- 0.70
	• Orthopnea	LR+ 2.2	LR- 0.65
	• Dyspnea on exertion	LR+ 1.3	LR- 0.48
Physical	• S3	LR+ 11	LR- 0.88
	• Jugular venous distension	LR+ 5.1	LR- 0.66
	• Rales	LR+ 2.8	LR- 0.51
	• Wheezing	LR+ 0.5	LR- 1.3
EKG	• Atrial fibrillation	LR+ 3.8	LR- 0.79
	• Any abnormal finding	LR+ 2.2	LR- 0.64
Ultrasound	• Reduced EF*	LR+ 4.1	LR- 0.24
	• IVC ≥ 20.5 mm	SN 90%	SP 73%
	• Pleural effusion(s)	LR+ 2.0	LR- 0.49
	• Positive B-line scan	LR+ 7.4	LR- 0.16
Chest X-ray	• Venous congestion	LR+ 12.0	LR- 0.48
	• Cardiomegaly	LR+ 3.3	LR- 0.33
BNP	• > 100 pg/ml	LR+ 2.2	LR- 0.11
NT-proBNP	• > 300 pg/ml	LR+ 1.8	LR- 0.09

* EPSS (E-Point Septal Separation): normal <7 mm. EF = 75.5 - (EPSS x 2.5)

•

OTTAWA HEART FAILURE RISK SCALE

Purpose: predict death from any cause within 30 days or ED visit or serious adverse event within 14 days of ED visit (regardless of whether admitted): admission to critical care or acute monitoring unit where the patient is too ill to ambulate, endotracheal intubation or NIV, myocardial infarction, unplanned CABG/PCI/cardiac surgery, return to ED for any related medical problem (e.g. for respiratory distress, fever, sepsis) and admission
Inclusion: ≥ 50 yr, presenting to ED with shortness of breath < 7 days duration due to exacerbation of chronic HF or new-onset HF (pulmonary or peripheral fluid retention + abnormal cardiac structure or function)
Exclusion: too ill to be discharged after 2-15 hrs of ED management: SpO2 < 85% or after being on home oxygen levels > 20 min, heart rate ≥ 120/min on arrival, SBP < 85 mm Hg on arrival, confusion / disorientation / dementia, ischemic chest pain or acute ST-T changes, STEMI, terminal status, nursing home or chronic care facility, chronic hemodialysis

CATEGORY	POINTS
Initial assessment	
• History of stroke or TIA	1
• History of intubation for respiratory distress	2
• Heart rate on ED arrival ≥ 110	2
• Room air SaO2 < 90% on EMS or ED arrival	1
Investigations	
• EKG has acute ischemic changes	2
• Urea ≥ 12 mmol/L	1
• Serum CO2 ≥ 35 mmol/L	2
• Troponin I or T elevated to MI level	2
• NT-ProBNP ≥ 5,000 ng/L	1
Walk Test* after ED treatment	
• One of the following: ○ SaO2 < 90% on room air or usual O2 ○ HR ≥ 110 during 3-minute walk test ○ Too ill to walk	1

SCORE	RISK
0	3%
1	5%
2	9%
3	16%
4	26%
5	40%
6	55%
7	70%
8	81%
9	89%

*Patient is asked to walk at their own pace for 3 minutes in the ED, regardless of the distance covered

PNEUMONIA SEVERITY INDEX: SCORE

Risk Factors		Points	Risk Factors		Points
Demo-graphics	Men	Yrs	Coexisting illnesses	Neoplastic disease	+30
	Women	Yrs - 10		Liver disease	+20
	Nursing home	+10		Congest. heart failure	+10
Labs & CXR	pH < 7.35	+30		Stroke	+10
	BUN ≥ 11 mmol/L	+20		Renal failure	+10
	Na < 130 mmol/L	+20	Physical	Altered mental status	+20
	Gluc ≥ 14 mmol/L	+10		Resp rate ≥ 30/min	+20
	Hematocrit < 30%	+10		SBP < 90 mm Hg	+20
	PaO2 < 60 mm Hg	+10		Temp < 35°C or ≥ 40°C	+15
	Pleural effusion	+10		HR ≥ 125 bpm	+10

"MDCALC PSI": https://www.mdcalc.com/psi-port-score-pneumonia-severity-index-cap

OTTAWA COPD RISK SCALE

Purpose: predict death from any cause within 30 days or ED visit or serious adverse event within 14 days of ED visit (regardless of whether admitted): admission to critical care or acute monitoring unit where the patient is too ill to ambulate, endotracheal intubation or NIV, myocardial infarction, unplanned CABG/PCI/cardiac surgery/new hemodialysis, return to ED for any related medical problem (e.g. for respiratory distress, fever, sepsis) and admission

Inclusion: ≥ 50 years, COPD previously diagnosed or diagnosed in ED on the basis of 1 year of chronic dyspnea or cough with sputum production, ≥ 15 pack year smoking history, prior or current evidence of moderate airflow obstruction, COPD exacerbation (increase in ≥2/3 of breathlessness, sputum volume, sputum purulence)

Exclusion: too ill to be discharged: resting SpO2 < 85%; heart rate ≥ 130/min; SBP < 85 mm Hg; confusion, disorientation or severe dementia, ischemic chest pain requiring treatment on arrival; STEMI on arrival; death from chronic illness expected within weeks; arrival from a nursing home or chronic care facility

CATEGORY	POINTS
History	
• Coronary bypass graft	1
• Peripheral vascular disease intervention	1
• Intubation for respiratory distress	2
Examination	
• Heart rate on arrival in ED ≥ 110 /min	2
• Too ill to do the Walk Test* after treatment in ED (SaO2 < 90% or heart rate ≥ 120/min)	2
Investigations	
• Acute ischemic changes on ECG	2
• Pulmonary congestion evident on chest X-ray	1
• Hemoglobin < 100 g/L	3
• Urea ≥ 12 mmol/L	1
• Serum CO2 ≥ 35 mmol/L	1

SCORE	RISK
0	2%
1	4%
2	7%
3	13%
4	21%
5	33%
6	48%
7	63%
8	76%
9	NA
10	91%

*Patient is asked to walk at their own pace for 3 minutes in the ED, regardless of the distance covered

PNEUMONIA SEVERITY INDEX: INTERPRETATION

Risk Class (Points)	Mortality	Recommendation
I (< 50)	0.1%	Outpatient
II (51-70)	0.6%	Outpatient
III (71-90)	0.9%	Out- or inpatient
IV (91-130)	9.3%	Inpatient
V (> 130)	27.0%	Inpatient

Fever

Elevated body temperature not caused by exogenous factors
If other symptoms present (e.g. headache): use other checklists

BACKGROUND

M	☐ Current medications? New medications? ☐ Acetaminophen usage?
A	☐ Allergies?
P	☐ Past medical history?
L	☐ Life circumstances? (e.g. travel history?)
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

O	☐ When did the fever begin?
S	☐ Degree of fever?
T	☐ Constant or intermittent? Increasing? ☐ Prior similar episodes?
+	☐ Headache? Neck stiffness? ☐ Shortness of breath? Cough? Chest pain? ☐ Abdominal pain? Diarrhea? ☐ Back pain? Dysuria? ☐ Leg pain or swelling? ☐ Rash?

PHYSICAL

Vitals	☐ RR, SpO2%, HR, BP, Temp?
Head	☐ Meningismus?
Heart	☐ S3/S4, insufficiency murmurs?
Lung	☐ Rales?
Abdo	☐ Inspection ☐ Auscultation ☐ Palpation
Back	☐ Inspection ☐ Percussion tenderness over the kidneys?
Leg	☐ Unilateral swelling?
Skin	☐ Rash on the trunk / extremities? ☐ Inflammation around IV's, port-a-cath, PICC-line? ☐ Splinter hemorrhages, Janeway lesions?

TESTS

☐ WBC (+ Neutrophils if available)
☐ CRP

CONSIDER FOR ALL

1. Sepsis
2. Risk for contagion (e.g. COVID, influensa, gastroenteritis)

CONSIDER IF UNCLEAR

The list of causes of fever is long. If the history and physical examination do not suggest a specific cause, consider the following diagnoses:

1. Pulmonary embolism
2. Cholecystitis-cholangitis
3. Pyelonephritis
4. Obstructive nephrolithiasis
5. Appendicitis
6. Diverticulitis
7. Necrotizing fasciitis
8. Infective endocarditis
9. Drug fever
10. Malignancy

Fever: Clinical Syndromes & Prediction Rule

SEPSIS

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection. The clinical criteria for sepsis are the presence of both:

- Suspected or documented infection
- Acute increase in the Sequential Organ Failure Assessment (SOFA) score ≥ 2 points consequent to infection. The SOFA score assigns 0-4 points depending on the degree of dysfunction in each of six organ systems (respiration, cardiovascular, central nervous system, renal, coagulation, liver). Bilirubin, platelet count, PaO2 and creatinine are necessary to calculate the SOFA score.

QUICK SEQUENTIAL ORGAN FAILURE ASSESSMENT (qSOFA)

The qSOFA score uses bedside clinical criteria to identify patients with suspected infection who have an increased risk of mortality or prolonged ICU admission, i.e. those with ≥ 2 of the following criteria:

- Respiratory rate ≥ 22/min
- Systolic blood pressure ≤ 100 mm Hg
- Altered mentation

The qSOFA score had similar predictive validity to the full SOFA score outside the ICU. Its purposes are to:

- help identify adults with infections who are likely to have a poor outcome
- prompt consideration of possible infection if infection is not yet suspected
- prompt testing for biochemical organ dysfunction
- prompt the physician to initiate or escalate therapy
- increase the frequency of monitoring or refer to critical care

SEPTIC CHOCK

Septic shock is a subset of sepsis associated with substantially increased mortality due to profound circulatory and cellular/metabolic abnormalities. The clinical criteria for severe sepsis (associated with a hospital mortality > 40%) are the presence of both:

- Persisting hypotension requiring vasopressors to maintain MAP ≥ 65 mm Hg
- Serum lactate level > 2 mmol/L despite adequate volume resuscitation (30 ml/kg cystalloid during the first 3 hours, or 1000 ml over the first 30 min)

TOXIC CHOCK SYNDROME

Toxic shock syndrome (TSS) is cause by exotoxins synthesize by Staphylococcus aureus or Group A Streptococcus (GAS). These exotoxins act as ‘superantingens’ and activate large numbers of T cells resulting in massive cytokine production. Staphylococcal toxic shock syndrome is associated with a variety of clinical settings, e.g. menstruation, postpartum and postsurgical states, barrier contraceptive use, staphylococcal pneumonia. The cytokines cause capillary leak and tissue damage, leading to

- Shock
- Diffuse, sunburn-like erythematous rash
- Multiorgan failure

Petechiae / Purpura

DEFINITIONS

Pink or purple, non-blanching, appearing on the skin, mucous membranes, conjunctiva, retina.
Caused by hemorrhage of capillaries.
Petechiae: ≤ 4 mm. Purpura: 5-10 mm. Ecchymosis > 10 mm

SPECIFIC INVESTIGATIONS

- Physical examination: location of the petechiae? "Palpable purpura"?
- Hb, WBC, Thrombocytes, INR, CRP, urinalysis (proteinuria?)

DIFFERENTIAL DIAGNOSIS OF PETECHIAE / PURPURA

Pathophysiology		Examples
Blood	Coagulo-pathy	• Thrombocytopenia • Deficiency of coagulation factors
	Emboli	• Fibrin (DIC, upon starting Warfarin) • Thrombocytes (TTP, HUS) • Thrombi (non-bacterial thrombotic endocarditis), fat, cholesterol
Vessel	Fragility	• Trauma, senile purpura • Steroid purpura, solar purpura • Amyloidosis, collagen problem (e.g. Ehlers Danlos, scurvy)
	Vasculitis	• Primary vasculitides (small vessel) • Secondary vasculitides (SLE, reumatoid arthritis, Sjögrens, Behcet) • Septic vasculopathy (meningococcemia, disseminated gonococcemia, bacterial endocarditis, Rickettsia)

DIC: Disseminated intravascular coagulation
TTP: Thrombotic thrombocytopenic purpura
HUS: Hemolytic uremic syndrome
SLE: Systemic lupus erythematosus

DIFFERENTIAL DIAGNOSIS OF PRIMARY SMALL VESSEL VASCULITIS

Pauci-immune small vessel vasculitides (ANCA-associated)	Immune-complex small vessel vasculitides (non-ANCA-associated)
• Granulomatosis with polyangiitis • Churg-Straus • Microscopic Polyangiitis	• Henoch Schönlein Purpura • Cryoglobulinemia • Drug-induced

OTHER DERMATOLOGIC TERMINOLOGY

Macule	Flat	< 1 cm
Patch	Flat	> 1 cm
Papule	Raised	< 1 cm
Plaque	Raised	> 1 cm
Vesicule	Fluid filled	< 1 cm
Bulla	Fluid filled	> 1 cm
Pustule	Pus filled	

Urinary Retention

BACKGROUND

M	☐ What medications are you taking? Any new medications?
A	☐ Allergies?
P	☐ Current or previous cancer? Recent surgery/radiation therapy?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking? Substance abuse?

HISTORY

☐ Hematuria, dysuria, fever?
☐ Back pain?
☐ Leg weakness/paresthesia
☐ Perineal paresthesia?

PHYSICAL

☐ Per rectum: obstruction, sensation, prostate hypertrophy?
☐ Leg strength, sensation

TESTS

☐ Urinalysis ☐ Creatinine, Na, K ☐ Bladder Scan: pre-void + post-void

DIFFERENTIAL DIAGNOSIS OF URINARY RETENTION

Pathophysiology	Examples
Infectious	• Urinary tract infection • Acute prostatitis, prostatic abscess • Acute vulvovaginitis • Genital herpes, varicella zoster infection
Neurological	• Spinal cord: transverse myelitis, infarction, multiple sclerosis • Spinal cord/cauda equina compression: epidural abscess, metastases • Guillain-Barré, diabetic neuropathy
Medications	• Antidepressants (e.g. tricyclics), Antipsychotics (e.g. halperidol) • Antihistamines (e.g. diphenhydramine), Anticholinergics (e.g. atropine) • Antiparkinsonian agents (e.g. levodopa, bromocriptine) • Sympathomimetics: ephedrine, pseudoephedrine, amphetamine • Miscellaneous: opioids, carbamazepine, dopamine
Mechanical	• Bladder cancer, bladder stones • Urethral stricture, phimosis, paraphimosis • Benign prostatic hyperplasic, prostate cancer • Vaginal prolapse, gynecological mass • Fecal impaction

URETHRAL CATHETER

- Indication: post-void residual > 300 ml
- Size: 16 F, 10-12 F if urethral stricture, 20-22 F coudé if enlarged prostate
- Tips: slowly preinject large amounts of lubricant with lidocaine
- Duration: the catheter should usually remain in place for 7-10 days
- Antibiotics are not recommended unless underlying infection present
- Patients with abnormal electrolytes or newly elevated Creatinine are at risk for post-obstructive diuresis. Observe for 4 hours; if urine output > 200 ml/hr for 2 hours, admit.

Acid-Base

Acid-Base Interpretation (Mnemonic: ACID)

Replace Values?

- If the blood gas comes from peripheral venous blood, add 0.03 to the venous pH to estimate the arterial pH and remove 0.6 kPa (5 mm Hg) from the venous pCO_2 to estimate the arterial pCO_2.
- If the pCO_2 is between 3.3 and 7.3 kPa (25 and 55 mm Hg), the standard bicarbonate $HCO_3(st)$ is a reasonable approximation of the actual HCO_3. Otherwise, use the actual HCO_3 (see table below)

1. Acidosis / alkalosis?

- pH < 7.38 and HCO_3 < 22 mmol/L: **Metabolic Acidosis**
- pH < 7.38 and pCO_2 > 5.7 kPa (42 mm Hg): **Respiratory Acidosis**
- pH > 7.42 and HCO_3 > 26 mmol/L: **Metabolic Alkalosis**
- pH > 7.42 and pCO_2 < 5 kPa (38 mm Hg): **Respiratory Alkalosis**

2. Compensation?

Disorder	Expected Comp	kPa	mm Hg
Metabolic Disorder	ΔpCO_2 = SBE x	0.1	0.75
Metabolic Acidosis	$\Delta pCO_2 = \Delta HCO_3$ x	0.16	1.2
Metabolic Alkalosis	$\Delta pCO_2 = \Delta HCO_3$ x	0.09	0.7
Respiratory Disorder < 2 days	SBE = 0		
Respiratory Acidosis < 2 days	$\Delta HCO_3 = \Delta pCO_2$ x	0.75	0.1
Respiratory Alkalosis < 2 days	$\Delta HCO_3 = \Delta pCO_2$ x	1.5	0.2
Respiratory Disorder > 5 days	SBE = ΔpCO_2 x	3	0.4
Respiratory Acidosis > 5 days	$\Delta HCO_3 = \Delta pCO_2$ x	2.62	0.35
Respiratory Alkalosis > 5 days	$\Delta HCO_3 = \Delta pCO_2$ x	3	0.4

3. Ions?

1. Calculate the Anion Gap (AG): Na - Cl - HCO_3
2. Calculate the Delta AG (Δ AG): Actual AG - Expected AG
- Expected AG is around 8 mmol/L when modern machines are used

3. Calculate Δ AG + HCO_3
- A sum > 26 mmol/L suggests the presence of either a **Metabolic Alkalosis** or a metabolic compensation for a **Respiratory Acidosis**
- A sum < 22 mmol/L suggests the presence of either a **Normal Anion Gap Metabolic Acidosis** or compensation for a **Respiratory Alkalosis**

4. Diagnoses?

☐ Speculate on the most plausible causes of the acid-base disorders using all available clinical information

Actual HCO_3 based on the pH and pCO_2

pCO_2 kPa	pCO_2 mm Hg	pH 6.9	7.0	7.1	7.2	7.3	7.4	7.5	7.6	7.7
1.0	8	1	2	2	3	4	4	6	7	9
1.5	11	2	3	3	4	5	7	8	11	13
2.0	15	3	4	5	6	7	9	11	14	18
2.5	19	4	4	6	7	9	11	14	18	22
3.0	22	4	5	7	8	11	13	17	21	27
7.5	56	11	13	17	21	27	34	42	53	67
8.0	60	11	14	18	23	29	36	45	57	72
8.5	64	12	15	19	24	30	38	48	60	76
9.0	68	13	16	20	25	32	40	51	64	81
9.5	71	13	17	21	27	34	43	54	68	85
10.0	75	14	18	23	28	36	45	57	71	90
10.5	79	15	19	24	30	37	47	59	75	94
11.0	82	16	20	25	31	39	49	62	78	99
11.5	86	16	21	26	33	41	52	65	82	103
12.0	90	17	21	27	34	43	54	68	85	107

Metabolic Acidosis with Increased Anion Gap

Substance		Anion(s)
M	**M**ethanol	Formate and L-lactate
	Metformin	L-lactate
U	**U**remia	Phosphates, sulphates, urate and hippurate
D	**D**iabetic ketoacidosis	ß-hydroxybutyrate (main ketone) and acetoacetate
P	**P**ropylene glycol	Pyruvate, L-lactate and D-lactate
	Pyroglutamic acid	Pyroglutamic acid (5-oxoproline)
I	**I**ron	L-lactate
	Isoniazid	L-lactate
L	L-**L**actate	L-lactate
	D-**L**actate	D-lactate
E	**E**thylene glycol	Glycolate, glyoxylate, oxalate, lactate (may be falsely high)
	Ethanol ketoacidosis	ß-hydroxybutyrate
S	**S**alicylates	Pyruvate, L-lactate and ketones
	Starvation ketoacidosis	ß-hydroxybutyrate, acetoacetate, acetone

Normal Anion Gap Metabolic Acidosis (Hyperchloremic Metabolic Acidosis)

Pathophysiology		Examples
Chloride administration		• Aggressive fluid resuscitation with NaCl • Hyperalimentation (lysine, histidine, or arginine hydrochloride)
HCO3 loss	Bowel loss	• Diarrhea • Urinary intestinal diversions • Biliary, pancreatic or small bowel fistulas
	Renal loss	• Renal tubular acidosis • Carbonic anhydrase inhibitor • Early renal failure (impaired acid excretion)

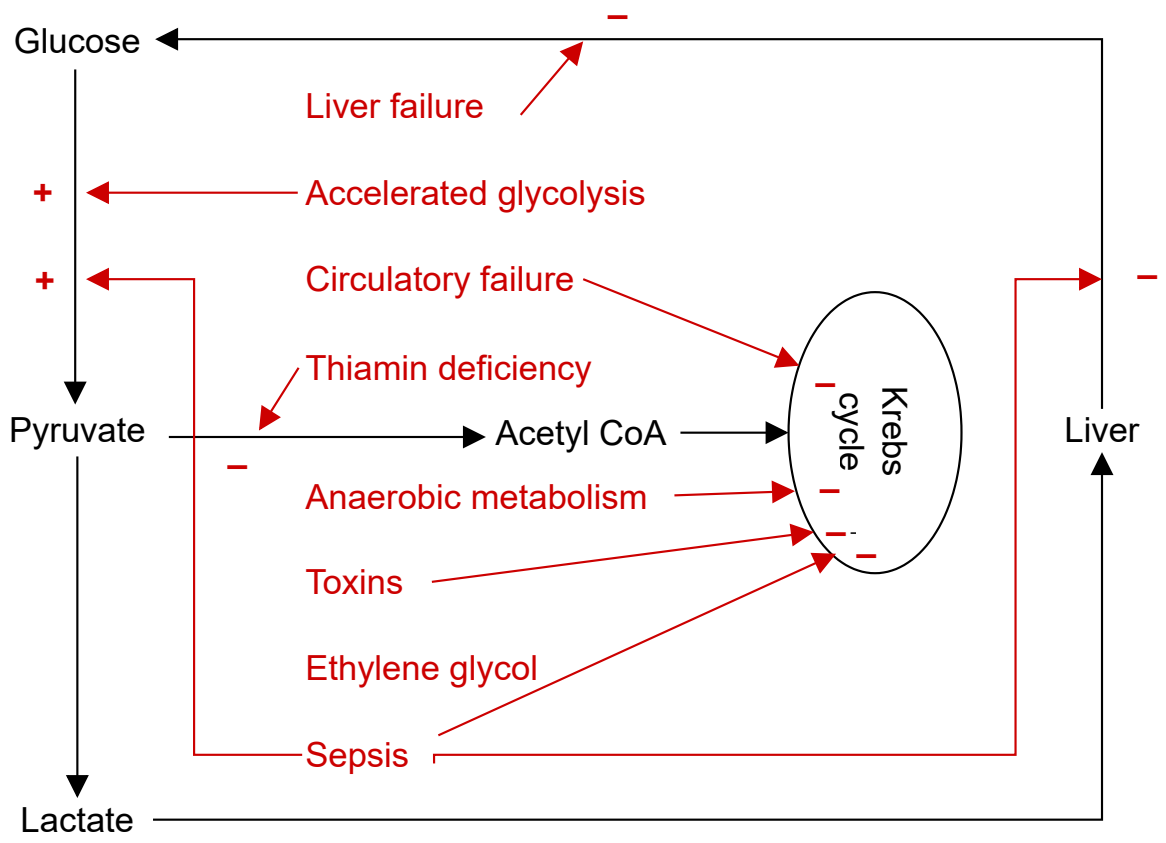
Decreased Anion Gap

Causes		Pathophysiology
L	**L**ithium	Lithium is a cation
	Low albumin	Albumin is negatively charged
I	**I**odide	Falsely elevated chloride value
M	**M**yeloma	Positively charged monoclonal IgG
B	**B**romide	Falsely elevated chloride value
S	**S**alicylates	Falsely elevated chloride value

L-Lactic Acidosis

Pathophysiology		Examples
Increased lactate production	Decreased O2 delivery to tissues / anaerobic metabolism	• Shock (hypovolemic, cardiogenic, septic) • Severe hypoxemia, anemia (Hb < 50 g/L) • Severe methemoglobinemia • Carbon monoxide poisoning • Pheochromocytoma, severe poisoning with sympathomimetics • Cancers (due to tumor tissue hypoxia) • Seizure, intensive exercise
	Increased glycolysis / pyruvate production	• Stimulation of ß2-receptors via endogenous adrenalin: sepsis, stress, seizure, shivering, intensive exercise, pheochromocytoma • Stimulation of ß2-receptors via exogenous agents: adrenalin, caffein-/theophyllin-/ ß2-agonist poisoning, severe poisoning with sympathomimetics • Stimulation of glycolysis via other mechanisms-respiratory alkalosis, cancer (lymphoma, leukemia, solid tumors)
	Decreased pyruvate metabolism to acetyl CoA	• Thiamin deficiency
	Interference with oxidative phosphorylation	• Methanol, ethylene glycol (usually moderate lactate elevation) • Salicylates (elevated lactate in severe poisoning) • Massive early-stage paracetamol poisoning • Carbon monoxide, iron, cyanid poisoning • Metformin • Nucleoside reverse-transcriptase inhibitors • Propofol (during prolonged high-dose infusion) • Linezolid
Decreased lactate metabolism		• Acute ethanol intoxication • Metformin • Liver failure (cirrhosis, hepatitis, severe paracetamol poisoning in late phase) • Sepsis
Falsely elevated lactate		• Ethylene glycol (glycolate and glyoxylate falsely interpreted as lactate by certain point-of-care machines)

L-Lactic Acidosis



Ethylene glycol poisoning may lead to falsely elevated lactate measurement by certain point-of-care machines

Metabolic Alkalosis

Pathophysiology		Examples
HCO3 administration		• Overzealous correction of a metabolic acidosis
H+ shifts intracellular		• Hypokalemia
H+ loss	Gastrointestinal loss	• Vomiting • Chloride wasting enteropathy • Cystic fibrosis • Laxative abuse
	Renal loss	• Extracellular volume depletion • Diuretic therapy • Renal artery stenosis • Conn's syndrome, Cushing's syndrome • Exogenous mineralocorticoids (e.g. licorice, fludrocortisone)

Respiratory Acidosis

Anatomy	Examples
Central Nervous System	• Vascular problems, e.g. stroke, hemorrhage • Infectious conditions, e.g. encephalitis, transverse myelitis • Primary tumors or metastases • Degenerative conditions, e.g. amyotrophic lateral sclerosis • Drugs, e.g. opioids, alcohol, benzodiazepines, barbiturates • Trauma to the brain or spinal cord • Metabolic encephalopathies, e.g. hepatic encephalopathy
Peripheral Nervous System	• Nerve dysfunction, e.g. phrenic nerve paralysis, Guillain Barré syndrome • Neuromuscular junction conditions, e.g. myasthenia gravis, botulism
Musculoskeletal	• Muscular conditions, e.g. myopathies, muscular dystrophy • Skeletal: kyphoscoliosis, ankylosing spondylitis
Pulmonary	• Upper airway obstruction, e.g. angioedema • Lower airway obstruction, e.g. COPD, life-threatening asthma • Alveoli: pneumonia, pulmonary edema • Blood vessels: massive pulmonary embolism • Pleura: pneumothorax, hemothorax

Respiratory Alkalosis

Pathophysiology	Examples
Hypoxia-driven	• Intrinsic lung disease and/or ventilation-perfusion mismatch, e.g. pulmonary edema, pneumonia, pulmonary embolism, aspiration • Severe anemia
Non hypoxia-driven	• Anxiety, pain • Salicylates, methylxanthines (theophyllamine, koffein), nicotine • Pregnancy (progesterone effect on the central nervous system) • Liver cirrhosis (progesterone effect on the central nervous system) • Gram-negative sepsis • Hepatic encephalopathy • Brainstem pathology

A-a Gradient

A-a Gradient

The **alveolar-arterial oxygen gradient** (A-a gradient) is the difference between the partial pressure of oxygen in the alveoli (PAO_2) and the partial pressure of oxygen in the arterial blood (PaO_2). The A-a gradient helps narrow the differential diagnosis of hypoxemia. Hypoxemia with normal A-a gradient suggests hypoventilation (e.g. CNS depression, musculoskeletal disoders). Hypoxemia with an elevated A-a gradient suggests ventilation-perfusion mismatch (e.g. pulmonary edema, pneumonia, pulmonary embolism), intrinsic lung disease or right-to-left shunt (intracardiac or intrapulmonary). Assuming sea level:

When the pressures are measured in **mm Hg**, the following formulas apply:

- $PAO_2 = FiO_2 \times 713 - PaCO_2 \times 1.25$
- PAO_2 when patient breathing room air = $21\% \times 713 - PaCO_2 \times 1.25 =$ **$150 - PaCO_2 \times 1.25$**
- A-a gradient = $PAO_2 - PaO_2$.
- A normal A-a gradient in young persons is < 10, whereas a normal A-a gradient in the elderly is < 20. Alternatively, a normal A-a gradient is (age + 4)/4.

When the pressures are measured in **kPa**, the following formulas apply:

- $PAO_2 = FiO_2 \times 95 - PaCO_2 \times 1.25$
- PAO_2 when patient breathing room air = $21\% \times 95 - PaCO_2 \times 1.25 =$ **$20 - PaCO_2 \times 1.25$**
- A-a gradient = $PAO_2 - PaO_2$.
- A normal A-a gradient in young persons is < 1.3, whereas a normal A-a gradient in the elderly is < 2.7. Alternatively, a normal A-a gradient is (age + 4)/30.

The FiO_2 can be estimated from the delivered supplemental oxygen using the table below on the left. The PaO_2 can be estimated from the SpO_2 using the table below on the right.

Method	O_2 flow (L/min)	Estimated FiO_2%
Nasal cannula	1	24
	2	28
	3	32
	4	36
	5	40
	6	44
Face mask	5	40
	6-7	50
	7-8	60
Face mask with reservoir	6	60
	7	70
	8	80
	9	90
	10	95

SpO_2	PaO_2 (mm Hg)	PaO_2 (kPa)
80	44	5.9
81	45	6.0
82	46	6.1
83	47	6.3
84	49	6.5
85	50	6.7
86	52	6.9
87	53	7.1
88	55	7.3
89	57	7.6
90	60	8.0
91	62	8.3
92	65	8.7
93	69	9.2
94	73	9.7
95	79	10.5
96	86	11.5
97	96	12.8
98	112	14.9
99	145	19.3

Anemia

INVESTIGATIONS

1. History

- Why was the Hb measured? Prior hematological blood tests?
- Background: medications, allergies, past med Hx, life circumstances, smoking, ethanol
- Bleeding: hematemesis? hemoptysis? melena/hematochezia? hematuria? menorrhagia?
- Symptoms: shortness of breath, presyncope, chest pain?

2. Tests

- To identify bone marrow failure: leukocytes, thrombocytes, reticulocytes
- To identify hemolysis: bilirubin, INR
- To identify common deficiencies: Fe, TIBC, Ferritin, B12, Folate, MCV
- Potential ruptured ectopic pregnancy: urine ß-hCG and POCUS
- Suspected hemolytic anemia: haptoglobin, LDH, Coombs test
- Suspected thrombotic microangiopathy syndrome: blood smear (schistocytes?)
- Probable tranfusion: blood type

MANAGEMENT

1. Acute Hemorrhage?

- See Hemorrhagic Shock

2. Auto-Immune Hemolytic Anemia?

- Contact hematologist: Prednisolon 1 mg/kg PO? High-dose Methylprednisolon IV?

3. Thrombotic Microangiopathy?

- Consider TTP, HUS and other thrombotic microangiopathies in the setting of hemolytic anemia + thrombocytopenia.
- Contact hematologist regarding treatment with FFP as a bridge to plasmaphoresis

4. Bone Marrow Failure?

- Contact hematologist.

5. Blood Transfusion?

- Numerous studies report no benefit of transfusion threshold higher than 70 g/L
- Transfusion at Hb > 70 g/L is indicated in the setting of
  - significant on-going bleeding
  - acute coronary syndrome
  - severe symptoms likely anemia-related
- Withholding transfusion despite Hb < 70 g/L is justifiable in certain situations (e.g. young relatively asymptomatic patient with iron-deficiency anemia)
- For patients with heart failure receiving transfusion, consider Lasix IV to reduce the risk of TACO (Transfusion-Associated Circulatory Overload).

6. Iron Transfusion?

- Consider IV iron infusion (e.g. MonoFer® 100 mg/ml 10 ml in 100 ml NaCl IV over 30 min) for iron-deficiency anemia; observe for 1 hour afterwards for allergic reactions.

7. Follow-Up

- Arrange for follow-up of Hb +/- investigations (e.g. gastroscopy, colonoscopy)

DIFFERENTIAL DIAGNOSIS OF ANEMIA

Pathophysiology		Examples
Decreased production	Hemoglobin	• Iron, B12, folate deficiencies • Anemia of chronic disease, lead poisoning • Thalassemia
	Hematopoiesis	• Aplastic anemia, pure red cell aplasia (immune-mediat.) • Lymphoma, carcinoma (bone-marrow infiltration) • Leukemia (hematopoietic stem cell lesion) • Renal failure (decrease EPO)
Increased loss	Hemorrhage	• Trauma • Gastrointestinal bleeding, ruptured AAA • Ruptured ectopic pregnancy, post-partum hemorrhage
	Hemolysis	• Auto-immune hemolytic anemia (CLL, Mycoplasma) • Thrombotic microangiopathy (TTP, HUS, drug-induced...) • Disseminated intravascular coagulation • Intracellular parasites: malaria, babesiosis • Congenital membranopathies, enzymopathies (e.g. G6PD deficiency), hemoglobinopathies (e.g. sickle cell) • Paroxysmal nocturnal hemoglobinuria • Pregnancy-associated: severe preeclampsia, HELLP
Other		• In vitro hemolysis • Acute splenic sequestration in sickle cell disease

Elevated Creatinine

DEFINITIONS

Creatinine is a breakdown product of creatinine phosphate (energy reserve found in muscle) and protein metabolism. Normal values are roughly 80 µmol/L in women and 100 µmol/L in men. Elevated creatinine values suggest acute kidney injury or chronic renal failure.

Glomerular filtration rate (GFR) corresponds to the summative filtration rate of all the nephrons. Creatinine is used (together with age, gender +/- weight +/- length) to estimated glomerular filtration rate (GFR*) assuming steady state in creatinine production and renal filtration*. (use absolute eGFR as opposed to relative eGFR Nyman 2017)

Acute kidney injury is defined as a sudden reduction in GFR as indicated by:

- Increase in serum creatinine by ≥ 26.5 µmol/L within 48 hours
- Increase in serum creatinine to ≥ 1.5 times baseline (known or presumed to have occurred within prior 7 days)
- Urine volume <0.5 mL/kg/h for 6 hours

INVESTIGATIONS

1. History

- Why was the Creatinine measured? Prior hematological blood tests?
- Background: medications, allergies, past med Hx, life circumstances, smoking, ethanol
- Fluid intake and loss (urinproduction, vomiting, diarrhea), pain, fever?

2. Tests

- Potassium; EKG if hyperkalemia is present
- pH, HCO3/BE, Na, Cl, Hb
- POCUS: bladder (post-void)? Hydronephrosis? IVC/IJV? B-lines (pulmonary edema)?
- Urinalysis: proteinuria suggests intrinsic renal disease
- Total CK and myoglobin if potential rhabdomyolysis (e.g. found lying, crush injury)

MANAGEMENT

1. Initial Management

- Hyperkalemia? See **Hyperkalemia.**
- Foley-catheter if urinary retention
- Ringer's acetate 1 Liter IV over 1-2 hours if suspected prerenal acute kidney injury
- Contact nephrology if suspected renal cause of renal failure
- Discontinue/avoid nephrotoxic medications, e.g. NSAIDs, ACE-inhibitors, ARBs
- Discontinue/dose-adjust medications cleared by the kidney e.g. Metformin, Digoxin, antibiotics

Webpage Estimate GFR / dose-adjust medications: _____ SE: janusinfo.se
Note: In the setting of acute kidney injury, estimates of GFR using Creatinine are unreliable.

2. Urgent Hemodialysis or Hemofiltration

- **A**cidemia (pH < 7.1) not responding to hemodynamic optimisation
- **E**lectrolytes: K > 6.1 mmol/L refractory to medical management or rapidly rising K levels
- **I**ngestion of nephrotoxic drugs amenable to dialysis (salicylates, lithium, methanol . . .)
- **O**verload: respiratory distress resulting from pulmonary edema
- **U**remic complications (pericarditis/pericardial effusion, encephalopathy, coagulopathy)

DIFFERENTIAL DIAGNOSIS OF ACUTE RENAL INJURY

Anatomy		Examples
Prerenal	Decreased renal perfusion	• Hypovolemia (e.g. decreased intake, bleed, GI loss, burns) • Congestive heart disease, cirrhosis, sepsis
	Renal arterial occlusion	• Renal artery stenosis (atherosclerotic or fibrodysplastic) • Renal artery emboli
	Renal arteriolar vasoconstriction	• Severe hypercalcemia • Radiocontrast agents • NSAIDs • ACE-inhibitors and ARBs • Amphotericin, vasopressors, hepatorenal syndrome
Renal	Glomeruli	• Post-infectious glomerulonephritis after Group A Strep infection • Anti-glomerular basement membrane disease, immune-complex disorders, ANCA-vasculitis
	Interstitium	• Infections • Infiltrative causes, e.g. lymphoma, sarcoidosis • Hypersensitivity reactions, e.g. secondary to NSAIDs, sulfa medication, penicillins • Autoimmune, e.g. SLE, Goodpasture syndrome • Pigment-induced conditions e.g. hemolysis, rhabdomyolysis
	Tubules	• Medications, toxins • ATN from prolonged acute prerenal kidney injury
	Vascular	• Hemolytic-uremic syndrome, TTP, renal vein thrombosis
Postrenal	Pre-bladder	• Abdominal and pelvic tumors, adhesions, fibrosis • Kidney or bladder stones
	Bladder	• Neurogenic bladder
	Post-bladder	• Prostate hypertrophy • Clogged in-dwelling urinary catheter

Elevated Liver Tests

INVESTIGATIONS

1. History

- Why were the liver tests measures? Prior values?
- Background: medications, allergies, past med Hx, life circumstances, ethanol, smoking
- New medications? Over-the-counter/alternative meds? Supplements? Herbs? Mushrooms?
- Recent travel? Sexual contacts? Tattoos? Transfusion of blood products? IV drug use?

2. Physical

- ABCDE: used in this context as a generic physical exam. Fever? Ascites? RUQ pain?

3. Tests

- ASAT, ALAT, ALP, GT, Bilirubin, Amylas, Albumin, INR
- Consider one or several of the following:
  - Ultrasound liver + gallbladder (+/- doppler to detect hepatic/portal vein thrombosis)
  - Ethanol, Paracetamol, PETh
  - Viral serologi
  - Autoimmune serology (ANA, AMA, SMA)

MANAGEMENT

- Consider
  - Piperacillin-Tazobactam in the setting of suspected cholangitis
  - Acetylcystein infusion if suspected liver failure secondary to Paracetamol overdose; may even be of benefit in cases of liver failure secondary to causes other than Paracetamol overdose
- Admission if:
  - Highly elevated tests
  - Signs of liver failure (e.g. spontaneously elevated INR)
  - Hepatic encephalopathy
  - Acute cholecystitis, cholangitis, pancreatitis
- Out-of-hospital follow-up:
  - Discontinue potential culprit medications
  - Advice to abstain from alcohol

DIFFERENTIAL DIAGNOSIS OF ELEVATED AST - ALT - AP - GT

Pathophysiology	Examples
Vascular, Cardiac	• Acute ischemia (AST > ALT): shock, cocaine, metamphetamine etc. • Acute Budd-Chiari syndrome • Congestive heart failure
Infectious, Infiltrative	• Viral hepatitis A, B, C, D, E • EBV, CMV, HSV, VZV, Parvovirus B19 • Sepsis (can cause intrahepatic cholestasis) • Tropical infections (e.g. malaria, leptospirosis, scrub typhus) • Sarcoidosis, amyloidosis, tuberculosis
Neoplastic	• Malignant infiltration e.g. lymphoma, leukemia, breast and colon cancer • Obstruction e.g. pancreas cancer, cholangiocarcinoma
Deficiency	• Wilson's disease • Hereditary hemochromatosis • Alpha 1-antitrypsin deficiency (early-onset emphysema?)
Drugs, Toxins	• Alcohol (AST:ALT > 2) • Paracetamol • Medications* e.g. anti-tuberculosis, anti-fungal, antiepileptic drugs • Herbal and nutritional supplements • Amanita phalloides (AST > ALT)
Autoimmune	• Autoimmune hepatitis (ALT:AP > 5) • Primary biliary cirrhosis (ALT:AP < 2) • Primary sclerosing cholangitis (ALT:AP < 2)
Mechanical	• Acute biliary obstruction (AST and ALT may be up to x 25 upper limit)
Endocrine, Metabolic	• Non-alcoholic steatohepatitis • Acute fatty liver of pregnancy • HELLP (Hemolysis, Elevated Liver enzymes, Low Platelets)

*Isoniazid, rifampicin, pyrazinamide, sulfonamides, trimethoprim-sulfamethoxazole, amoxicillin-clavulanate, dapsone, ketoconazole, ofloxacin, didanosine, efavirenz, allopurinol, diclofenac, halothane, isoflurane, phenytoin, valproic acid, nicotinic acid, statins, imipramine, propylthiouracil, disulfiram, lisinopril, labetalol, methyldopa, amiodarone, flutamide, metformin, etoposide, gemtuzumab, anabolic steroids

AST - ALT > 25 x upper limit	Isolated hyperbilirubinemia
• Toxin/drug-induced liver damage • Acute ischemic liver injury • Acute viral hepatitis • Severe auto-immune hepatitis • Wilson's disease	• Hemolytic anemia (unconjugated) • Hematoma resorption (unconjugated) • Gilbert syndrome (unconjugated) • Rotor syndrome (conjugated) • Dubin-Johnson syndrome (conjugated)

Hyponatremia

INVESTIGATIONS

- Urine sodium, Urine osmolarity, Serum osmolarity, TSH, T4, Cortisol

MANAGEMENT

1. Hyponatremic Encephalopathy?

- Severe symptoms (vomiting, confusion, somnolence, coma, seizures): see **Hyponatremic Encephalopathy.**

2. Initial Treatment & Follow-Up

- Admission if [Na] < 120 mmol/L, symptomatic, risk for rapid [Na] change, no available short-term follow-up outside of the hospital.
- Suspected intravascular volume depletion: Ringer's acetate 1000 ml IV over 4 hours
- Suspected SIADH or heart failure/edema: fluid restriction (< 800 ml/day)
- Monitor urine output; repeat [Na] measurement initially every 2-6 hours
- Target rise in serum Na ≤ 10 mmol/L/24 hours

3. Overcorrection

- Glucose 50 mg/ml 500 ml IV over 4 hours +/- water PO/NG +/- Desmopressin 1 µg IV

DIFFERENTIAL DIAGNOSIS OF HYPONATREMIA

Pathophysiology	Examples
Too much water in • U < 100 mOsm/L	• Primary polydypsia • Secondary polydypsia (e.g. hypothalamic pathology)
Too little salt in • U < 100 mOsm/L	• Anorexia nervosa • "Tea-and-toast" hyponatremia • Beer potomania
Too little water out • U > 100 mOsm/L	"Appropriately" elevated ADH (U-Na < 30 mmol/L): • Intravascular volume depletion, e.g. diarrhea, vomiting, pancreatitis, burns, bowel obstruction • Heart failure (including heart failure from severe hypothyroidism) • Nephrotic syndrome (hypoalbuminemia) • Cirrhosis (hypoalbuminemia)
	"Inappropriately" elevated ADH-SIADH (U-Na > 30 mmol/L): • Lung pathology e.g. pneumonia, abscess, tuberculosis • Brain pathology e.g. meningoencephalitis, abscess • Cancer e.g. lung, pancreatic, ovarian, lymphoma • Medications e.g. morphine, carbamazepine, vincristine • Pain, nausea, delivery, psychosis • Secondary adrenal insufficiency
Too much salt out • U > 100 mOsm/L • U-Na > 30 mmol/L	• Diuretic use (renal solute loss), especially thiazides • Primary adrenal insufficiency (hypoaldosteronism) • Salt-losing nephropathy e.g. renal tubular acidosis, polycystic kidney disease, obstructive uropathy • Cerebral salt wasting (mainly due to subarachnoid hemorrhage) • Osmotic diuresis (mannitol, glucose, urea)

If S-osm > 275 mOsm/L: hyperglycemia, mannitol, ethanol, high urea, pseudohyponatremia

Hypernatremia

INVESTIGATIONS
- Urine osmolarity, Urine sodium

MANAGEMENT
1. Marked Hypovolemia?
- NaCl 0.9% 500 ml IV bolus. Reassess volume status, remeasure [Na].

2. Acute Hypernatremia (i.e. Salt Poisoning)
- Glucose 5% 500 ml IV bolus + Water PO/NG +/- hemodialysis. [Na] + [Glu] every 2h.

3. Initial Treatment & Follow-Up
- NaCl 0.9% (154 mmol/L), Ringer's (134 mmol/L Na + 4 mmol/L K) or Glucose 5% + NaCl 80 mmol/L + KCl 40 mmol/L depending on [Na] and desired rate of fluid repletion.
- Measure [Na] initially every 4 hours, target [Na] drop ≤ 10 mmol/L/24 hours

DIFFERENTIAL DIAGNOSIS OF HYPERNATREMIA

Pathophysiology	Examples
Decreased water intake	• Primary hypodipsia (impaired thirst) from hypothalamic pathology • Secondary hypodipsia i.e. inability to obtain or swallow free water
Increased salt intake	• Iatrogenic e.g. hypertonic saline, sodium bicarbonate • Oral e.g. salt tablets, salt water ingestion

Increased water loss	• Gastrointestinal tract
	  ○ Vomiting, nasogastric drainage
	  ○ Diarrhea, osmotic cathartic agents (e.g. lactulose)

	• Renal
	  ○ Osmotic diuresis (e.g. from hyperglycemia)
	  ○ Diuretics (loop or thiazide) in critically ill patients
	  ○ Relief of complete postrenal urinary obstruction
	  ○ Central diabetes insipidus (low ADH)
	  ○ Nephrogenic diabetes insipidus (high ADH)

	• Increased insensible losses
	  ○ Skin: fever, diaphoresis
	  ○ Respiratory tract: tachypnea

	• Drugs e.g. alcohol, lithium (most common cause of drug-induced nephrogenic diabetes insipidus), phenytoin, sulfonylureas
Decreased salt loss	• Primary aldosteronism • Cushing syndrome • Ectopic adrenocorticotrophic hormone production

Urine Osmolarity	Pathophysiology
> 600 mOsm/L	• Decreased water intake • GI (vomiting, diarrhea) or insensible losses (U-Na < 25 mOsm/L) • Salt poisoning (U-Na > 100 mOsm/L)
300-600 mOsm/L	• Osmotic diuresis • Diabetes insipidus
< 300 mOsm/L	• Diabetes insipidus (central or nephrogenic)

Hypokalemia

DEFINITIONS

- Hypokalemia is defined as a serum potassium < 3.5 mmol/L.
- Severe hypokalemia is defined as a serum potassium < 2.5 mmol/L.

SYMPTOMS

- Patients may present with **fatigue, constipation, leg cramps, weakness, ascending paralysis, cardiac arrhythmias.**

INVESTIGATIONS

- EKG: Arrhythmias? Prolonged QTc? U waves? Increased risk in patients on antiarrhythmics, e.g. digoxin and sotalol
- Serum Magnesium: Hypomagnesemia?

ELECTROCARDIOGRAM

O	• **Arrhythmias**, especially if the patient is taking digoxin: premature supraventricular and ventricular beats, sinus bradycardia, paroxysmal atrial or junctional tachycardia, atrioventricular block • **Mild PR prolongation** • **Cardiac arrest**: PEA, asystole, pVT, VF
S	• **ST-segment depression**
T	• **T-wave flattening or inversion** • **QTc prolongation**
+	• **U waves**, often seen in V4-V6

MANAGEMENT

1. Fast Potassium Chloride Infusion?

- **Indication:** unstable arrhythmias where cardiac arrest is considered imminent.
- **Potassium 20 mmol** (Addex®-Kaliumklorid 2 mmol/ml 10 ml in 250 ml NaCl) **IV over 10 min** (ideally via a central vein catheter).
- The dose can be followed up by an infusion of Potassium 10 mmol over 10 minutes.

2. Fast Magnesium Sulphate Infusion?

- **Indication:** ventricular tachycardia and severe hypokalemia.
- **Magnesium Sulphate 2.5 g (10 mmol)** (Addex®-Magnesium 1 mmol/ml 10 ml mixed with 10 ml NaCl 0.9%) **IV over 10 min**.
- The dose may be repeated after 10-15 min.
- Hypomagnesemia is very common in patients with hypokalemia, magnesium administration may be the fastest way to decrease the risk of malignant arrhythmias.

3. Slow Potassium Chloride and Magnesium Sulphate Infusion < 3.0 mmol/L

- **Indication:** [K] < 3.0 mmol/L
- **Potassium Chloride 40 mmol** (Addex®-Kaliumklorid 2 mmol/ml 20 ml) + **Magnesium Sulfate 10 mmol** (Addex®-Magnesium 1 mmol/ml 10 ml) in 1 L NaCl 0.9% **IV over 4 hours**, ideally with an infusion pump. KCl infusion rate is thus 10 mmol/hr.
- The maximum recommended KCl infusion rate is 20 mmol/hr. Two peripheral infusions at the rate of 10 mmol/hr may be given.
- Patients receiving KCl infusion require in addition 10-20 mmol Magnesium daily.

4. Oral Potassium Chloride

- **Indication:** [K] < 3.5 mmol/L
- **Potassium Chloride PO.** Oral and IV supplementation may be carried out simultaneously. The recommended dose is Potassium Chloride 40 mEq PO three to four times per day. Kaleorid depottablett à 750 mg contains 10 mEq Kalium, can give 2-4 tabletts PO initially.

5. Heart Monitoring

- **Continuous cardiac monitoring** is recommended as long as the K is < 2.5 mmol/L

6. Monitor Serum Potassium

- **Monitor serum potassium** initially every 2-4 hours. Risk of hyperkalemia (i.e. over-correction) especially in patients with GFR < 30 ml/min and in patients for whom the hypokalemias was caused by shift (se table below).

7. Further Care

- Kaleorid depottablett à 750 mg (10 mEq KCl) 2-3 x 3-4 daily PO
- Emgesan 250 mg 1x2 PO daily for several days if severe hypokalemia
- Patients with K ≥ 3.0 mmol/L can usually be follow-up in the primary care setting

DIFFERENTIAL DIAGNOSIS OF HYPOKALEMIA

Pathophysiology	Examples	
Decreased potassium intake	• Poor dietary intake • Geophagia	
Shift (extracellular to intracellular)	• Alkalosis • Insulin e.g. treatment of diabetic ketoacidosis • Adrenalin (exogenous and endogenous) • Beta-adrenergics (e.g. when treating asthma exacerbations) • Hypokalemic or thyrotoxic periodic paralysis	

Increased potassium loss	Renal	• Diuretics, osmotic diuresis, diabetes insipidus
		• High aldosterone:
		  ○ Primary hyperaldosteronism e.g. Conn' syndrome
		  ○ Secondary to intravascular volume depletion
		• Renal tubular acidosis
		• Licorice ingestion, chewing tobacco
		• Hypomagnesemia

	Gastro-intestinal	• Vomiting, nasogastric suction • Diarrhea, malabsorption, laxative abuse, enema abuse
Pseudohypokalemia	• Acute leukemia	

Hyperkalemia

INVESTIGATIONS

- EKG: Widened QRS complex? Arrhythmias? Peaked T-waves?
- Repeat S-potassium (hemolysis?), Creatinine (renal failure?), PoCUS (urinary retention?)

ELECTROCARDIOGRAM

O	• **Bradycardia** may occur • **First degree AV-block** may occur • **Sinoventricular rhythm** (K 8-9 mmol/L). Given absence of P waves and wide QRS complexes, can be mistaken for ventricular tachycardia • **Junctional escape rhythm** (K around 10 mmol/L) • **Sine-wave pattern** (K > 10 mmol/L) as QRS complex merges with the T wave • **Asystole**, **ventricular tachycardia** and **ventricular fibrillation** may occur
P	• The P waves **flatten** until they disappear • **PR shortening** is an early manifestation of hyperkalemia • **PR interval becomes prolonged** as hyperkalemia progresses
Q	• **Right and left bundle branch blocks** occur with hyperkalemia • **Widening of the QRS complexes** • **Sine-wave pattern** from merging of a wide QRS complex with the T wave
S	• **ST-segment depression** may occur • **ST-segment elevation** may mimic a STEMI ("pseudoinfarct" pattern) • **Brugada pattern** (down-sloping ST elevation in V1) may be present
T	• **Peaked ('tented') T waves**, i.e. tall, narrow, symmetric T waves that are larger than the R wave in more than 1 lead; earliest manifestation of hyperkalemia • **Shortened QTc interval** may be present in the early stages of hyperkalemia

MANAGEMENT

1. Calcium?

- **Indication**: potassium ≥ 6.0 mmol/L AND (wide QRS OR bradycardia OR arrhythmia). Its effects occur within 1-3 minutes and last for 30-60 minutes.
- **Calcium Gluconate** 10% 30 ml (0.3 ml/kg) IV over 5 min or **Calcium Chloride** 10% 10 ml (0.1 ml/kg) IV over 5 min through a central venous catheter or a secure large-bore antecubital peripheral line (tissue necrosis if it extravasates)
- Repeat dose after 5-10 minutes if EKG indications persist.

2. Insulin?

- **Indication**: potassium ≥ 6.0 mmol/L. Insulin is the most reliable method for shifting potassium intracellularly and is indicated in all cases of hyperkalemia requiring emergency treatment. Insulin drives potassium intracellularly by stimulating the Na-K-ATPase pump in skeletal muscle. Onset of action is 15-30 min.
- **Short acting insulin** (e.g. Novorapid) **10 U in Glucose 50 mg/ml 500 ml** IV over 15-30 minutes (0.1 U/kg with D25W infusion 2 ml/kg in children). Glucose may be omitted if the patient is significantly hyperglycemic on presentation.
- **Glucose 50 mg/ml 500 ml** should then be administered IV over 5 hours to prevent hypoglycemia in patients with an initial [Glucose] < 7 mmol/L.
- If desirable to reduce infused volume, **Glucose 300 mg/ml 100 ml IV** can be administered (avoid a very peripheral PVC).

3. Beta-2 Agonist?

- **Indication**: potassium ≥ 6.5 mmol/L. Shifts K intracellularly by stimulating Na-K-ATPase pump. Combination of insulin and albuterol is synergistic. Onset of action 15-30 min.
- **Salbutamol (Albuterol) 10-20 mg nebulized** (2.5 mg if < 25 kg or 5 mg if > 25 kg)
- **Terbutalin (Bricanyl) 0.5 mg/ml 1 ml SC or IV** is an alternative to inhaled therapy

4. Potassium exchange resin?

- **Indication**: potassium ≥ 6.0 mmol/L. Remove potassium via the gastrointesintal tract.
- Options consist of
  - Sodium zirconium cyclosilicate (Lokelma) 10 g x3/day PO
  - Patiromer 8.4 g/day PO
  - Calcium Resonium 15 g x3/day PO

5. Hemodialysis?

- **Indicated** in the following settings:
  - severe life-threatening hyperkalemia
  - hyperkalemia resistent to medical therapy
  - end-stage renal disease
  - oliguric acute kidney injury (< 400 mL/day urine output)
  - marked tissue breakdown (e.g. rhabdomyolysis)

6. Loop diuretics?

- **Indication**: sufficient renal function
- Furosemide 40-80 mg IV in conjunction with hydration with 0.9% NaCl

7. Further Management

- Measure serum potassium and glucose levels 1-2 hours after initiation of therapy.
- Address the cause of the hyperkalemia (see **Differential Diagnosis** below).

Therapies of Unproven Benefit

- **Sodium bicarbonate** NaHCO3 50 mg/ml 100 ml is recommended in the setting of severe hyperkalemia combined with severe acidosis or renal failure
- **Hypertonic NaCl 3%** reverses EKG changes of hyperK in patients with hyponatremia

DIFFERENTIAL DIAGNOSIS OF HYPERKALEMIA

Pathophysiology	Examples
Increased potassium intake	• Potassium-rich foods, potassium-containing drugs • Intravenous administration
Shift (intracellular to extracellular)	• Acidosis (with a lipophobic anion) • Rhabdomyolysis, heavy exercise, hemolysis, tumor lysis syndrome • Decreased N-K ATPase activity: insulin deficiency, digitalis intoxication
Decreased renal potassium loss	• Renal failure (acute kidney injury or chronic kidney disease) • Potassium-sparing diuretics (e.g. spironolactone), ACE-inhibitors, ARBs, NSAIDs, beta-blockers, trimethoprim • Aldosterone deficiency (e.g. Addisons)
Pseudo-hyperkalemia	• Tourniquet use • Hemolysis (in vitro) • Leukocytosis, thrombocytosis

Hypocalcemia

INVESTIGATIONS

- Ionized calcium, PTH, 25-OH-Vitamin D, Magnesium, Phosphate
- EKG: heart block, prolonged QTc, T wave inversions

MANAGEMENT

1. Symptomatic Acute Hypocalcemia

- **Indication**: muscle cramps (including carpopedal spasm, laryngospasm), seizures
- **Contraindication**: IV calcium contraindicated in the presence of hyperphosphatemia because of the risk of precipitation
- **Calcium Gluconate 10%** 30 ml IV
- **Magnesium Sulphate** 1-2 g = 4-8 mmol IV (Addex®-Magnesium 1 mmol/ml 4-8 ml)

2. Asymptomatic Hypocalcemia

- Oral calcium
- Consider vitamin D supplementation
- Consider changing from loop diuretics to thiazide diuretics

DIFFERENTIAL DIAGNOSIS OF HYPOCALCEMIA

Pathophysiology		Examples
Decreased oral intake, intestinal absorption, bone resorption	Hypo-parathyroidism	• Surgical • Autoimmune • Hypomagnesemia (PTH resistance / suppressed secretion) • Hypermagnesemia (when acute & severe: suppressed PTH secretion)
	Vitamin D deficiency	• Decreased intake/absorption of foods containing vitamin D • Decreased exposure to ultraviolet light • Decreased 25-hydroxylation in the liver • Decreased 1-hydroxylation in the kidney • Increased metabolism of Vitamin D to inactive metabolites
Shift-binding	Hyper-phosphatemia	Hyperphosphatemia leads to increased calcium deposition in bone (mostly) and in extraskeletal tissues. Causes: • Increased phosphorus intake • Tissue breakdown (rhabdomyolysis, tumor lysis SD) • Decreased renal excretion (e.g. acute renal failure)
	Citrate	• Transfusion of blood products
	Bone mineralization	• Osteoblastic bone metastases (breast, prostate cancer) • Hungry bone syndrome
	Fat	• Pancreatitis
	Respiratory alkalosis	• Both acute and chronic respiratory alkalosis decrease ionized Ca
Increased renal loss	Hypoparathyroidism leads to increased calcium renal excretion	

Hypercalcemia

INVESTIGATIONS
- PTH, EKG

ELECTROCARDIOGRAM

O	• **Arrhythmias** (VF, conduction defects, bradycardia) rarely occur • **Atrioventricular block** progressing to complete heart block
P	• **PR prolongation**
Q	• **QRS widening**
S	• **ST-segment elevation** can occur with severe hypercalcemia
T	• **Short QTc** interval due to shortening of the heart's action potential

MANAGEMENT
1. Normal Saline
- **Indication**: normal saline infusion to start lowering the calcium is the first-line therapy. The goal is to replenish intravascular volume and lead to urine output of 100-150 ml/hr.
- **Normal Saline**: the volume and rate depend on comorbidities (e.g. heart failure) and symptoms. In general, 1000 ml over 4 hours.

2. Loop Diuretics
- **Indicated** once the patient is euvolemic to block calcium reabsorption in the kidney
- **Furosemide** 1 mg/kg IV

3. Biphosphonate
- **Indication**: Biphosphonates such as Zoledronic acid and Pamidronate are first-line treatments for malignancy-induced hypercalcemia and a mainstay of treatment of moderate to severe hypercalcemia in general
- **Zoledronic acid** 4 mg IV over 15 min or **Pamidronate** 60-90 mg IV over 2 hours. Dose adjustment in patients with renal disease.

4. Other Therapies?
- **Prednisolone 40 mg/day** in the setting of Hodgkin's disease, multiple myeloma, granulomatous disorders, excessive intake of Vitamin D
- **Hemodialysis** and **peritoneal dialysis** are effective therapies for lowering calcium levels e.g. in patients refractory to other therapies or patients with congestive heart failure or renal failure with fluid overload for whom hydration cannot be used.

5. Medication Changes
- Discontinue medications that promote hypercalcemia, e.g. thiazide diuretics, Vitamin D, Vitamin A, Calcium supplements.

6. Admission?
- Severe hypercalcemia (ionized Ca > 2.5 mmol/L)
- Moderate hypercalcemia (ionized Ca 2.0 - 2.5 mmol/L) AND mental status changes
- Cardiac monitoring is recommended for patients with severe hypercalcemia due to the risk of arrhythmias

DIAGNOSIS DIFFERENTIAL DIAGNOSIS OF HYPERCALCEMIA

Pathophysiology	Examples
Hyper-parathyroidism	• Primary hyperparathyroidism (leading cause, 50% of cases) • Tertiary hyperparathyroidism (hyperplasia of the parathyroid gland in response to chronic hypocalcemia, unresponsive to calcium levels)
Malignancy	• Direct bone destruction, e.g. breast cancer, multiple myeloma • PTH-rP (parathyroid related peptide), which mimics the biological effects of normal PTH, e.g. squamous-cell lung cancer • Increased production of calcitriol, e.g. Hodgkin's lymphoma • Other squamous cell tumors, , T-cell tumors, renal-cell carcinoma
Granulomatous diseases	• Sarcoidosis • Tuberculosis • Leprosy
Immobilization	
Drugs	• Thiazide diuretics (increase renal calcium reabsorption) • Antacids • Lithium • Vitamin A • Vitamin D

Hypoglycemia

MANAGEMENT

1. Severe Symptoms

	Adult	Child
No IV access	**Glucagon** 1 mg IM	**Glucagon** 1 mg IM to children > 6 years **Glucagon** 0.5 mg IM to children < 6 years
IV access	**Glucose 300 mg/ml** (30%) 30 ml IV	**Glucose 100 mg/ml** (10%) 2 ml/kg IV followed by 4 ml/kg/hour infusion.

- **Thiamine** 500 mg IV should be given in conjunction with glucose if thiamine deficiency is suspected (e.g. chronic alcohol abuse, malnutrition, possible Wernicke's encephalopathy)
- **Octreotide** 100 µg IM or SC (1 µg/kg) should be added to glucose therapy in the case of sulfonylurea overdose. Overdose with sulfonylureas leads to increased release of endogenous insulin; treatment with glucose alone results in transient hyperglycemia, which in turn increases insulin secretion and leads to recurring episodes of hypoglycemia.

2. Mild Symptoms

- **Oral glucose** is preferable if available, since extravasation of glucose administered intravenously results in tissue necrosis.

3. Monitor

- **Remeasure glucose** 15-30 min later.
- **Anticipate duration of risk** e.g. depending on the half-life of the culprit substance

DIAGNOSIS DIFFERENTIAL DIAGNOSIS OF HYPOGLYCEMIA

Pathophysiology	Examples
Too much insulin	• Exogenous insulin • Sulfonylurea or herbal products contaminated with a sulfonylurea • Meglitinides • Insulinoma • Gastric bypass or Nissen fundoplication • Insulin auto-immune hypoglycemia
Other	• Ethanol (inhibits gluconeogenesis but not glycogenolysis) • Drugs, e.g. beta-blockers • Critical illness, e.g. septic shock, renal insufficiency • Malnourishment (e.g. anorexia nervosa) • Cortisol deficiency (primary and secondary adrenal insufficiency)
Artefactual	• Absence of antiglycolytic agent in the blood collection tube, delayed processing, especially in the setting of leukemia and hemolytic anemia (in vitro glucose consumption)

Diabetes medication (Swedish tradenames)
SGTL-2 inhibitors: Empaglifozin (Jardiance),
Sulfonylureas:
Megltitindes
Metformin and company
Other medications (With Swedish tran

Ultrasound

Generic Point-of-Care Ultrasound Protocol (mnemonic HIJKL)

1. Heart	☐ Pericardial fluid? ☐ Right ventricular dilatation? ☐ Hypokinesia?
2. IVC	☐ Size < > 2 cm? ☐ Decrease upon inspiration < > 50%?
3. Juice	☐ Intraperitoneal fluid? ☐ Pleural fluid?
4. Koils	☐ Abdominal aortic aneurysm? ☐ Dilated loops of small bowel?
5. Lung	☐ Lung-sliding? ☐ A-lines vs B-lines? ☐ Hepatization?

Inferior Vena Cava

Findings	Suggests
IVC size > 2 cm and Caval index* < 50%	• CVP > 10 cm H_2O • In the setting of shock, these findings suggest obstructive (e.g. pericardial tamponade) or cardiogenic shock.
IVC size > 2 cm and Caval index* < 50%	• CVP < 10 cm H_2O • In the setting of shock, these findings suggest hypovolemic or distributive shock

* Caval index refers to the relative decrease in IVC diameter during one respiratory cycle.

Absent Lung Sliding

- Pneumothorax
- Absence of ventilation, e.g. apnea, phrenic nerve palsy, jet ventilation, esophageal intubation, intubation of the opposite main-stem bronchus, pneumectomy
- Dense lobar consolidation, e.g. pneumonia, lung contusion, atelectasis (B-lines may be present, ruling-out pneumothorax)
- Pleural adhesions, pleurodesis, bullae (A-lines or B-lines may be present)

Diffuse Bilateral B Lines	Focal B lines
• Pulmonary edema • Interstitial pneumonia / pneumonitis • Pulmonary fibrosis • Acute respiratory distress syndrome (ARDS)	• Focal posterolateral B-line may be found in a normal lung, due to gravity alone • Pneumonia and pneumonitis • Atelectasis • Pulmonary infarction or contusion • Pleural disease • Malignancy

Electrocardiogram

EKG Interpretation

O	☐ Overview: rate? ☐ Overview: rhythm?
P	☐ P wave: positive in lead II? signs of atrial hypertrophy? ☐ PR segment: duration? depression?
Q	☐ Pathological Q waves? ☐ QRS complexes: wide? bundle-branch block pattern?
R	☐ Axis deviation? ☐ R waves: ventricular hypertrophy?
S	☐ S waves: ventricular hypertrophy? ☐ ST segment: elevation or depression?
T	☐ T waves: peaked? inverted? ☐ QTc time: prolonged?
+	☐ Additional findings (e.g. U wave)?

Rate

- Paper speed of 25 mm/sec: rate is 300 /# of 5 mm squares between QRS complexes
- Paper speed of 50 mm/sec: rate is 600 /# of 5 mm squares between QRS complexes

Atrial Hypertrophy

	Left	Right
II	• Humped or notched P wave > 0.12 sec	• P wave > 2.5 mm, < 0.12 sec
V1	• Biphasic P wave, terminal negative deflection of > 0.04 sec or > 1 mm (0.1 mV) in depth	• P wave > 2.5 mm, occasionally negative, < 0.12 sec

PR Segment

- Short PR segment (< 120 msec): consider pre-excitation (delta wave)
- Prolonged PR segment (> 200 msec): consider 1st degree AV block
- Depressed PR segment: consider pericarditis

Pathological Q Waves

Q-waves can be physiological or caused by:

- myocardial ischemia or infiltration
- ventricular enlargement or hypertrophy
- conduction abnormalities

Q-wave duration, depth and location on the EKG determine whether the Q-wave is pathological or not, yet there is no current consensus regarding the exact criteria that distinguish pathological from physiological Q waves.

As an example, the Fourth Universal Definition of Myocardial Infarction states that the following Q-waves are associated with prior myocardial infarction:

- Q-wave > 0.02 s or QS complex in V2 or V3
- Q-wave ≥ 0.03 s and ≥ 1 mm deep or QS complex in any two leads belonging to a contiguous lead grouping: I + aVL; V1-V6; II, III, aVF

Differential Diagnosis of Wide QRS Complexes

- Depolarization originates in the ventricle (e.g. VT, premature ventricular beat)
- Bundle branch block
- Accessory pathway
- Hyperkalemia
- Intoxication with a sodium channel blocking agent

Left Bundle Branch Block

Suggestive Findings	Differential Diagnosis
• Wide, entirely negative QS complex in V1 (rarely, a wide rS complex) • Wide, tall R wave without a Q wave in V6	• Long-standing hypertensive disease • Valvular lesion (e.g. aortic stenosis, aortic regurgitation) • Cardiomyopathies • Coronary artery disease • Degenerative changes

Right Bundle Branch Block

Suggestive Findings	Differential Diagnosis
• rSR' appearance in V1 or V2. The R' deflection is usually wider than the initial r wave. • qRS pattern in V6 with a wide S wave of greater duration than the R wave or > 40 ms in leads I and V6 in adults. • When a pure dominant R wave with or without a notch is present in V1, the R peak time in leads V5 and V6 is normal while the R peak time in lead V1 is > 50 ms.	• Atrial septal defect with left-to-right shunt • Chronic pulmonary disease with pulmonary artery hypertension • Pulmonary stenosis • Cardiomyopathies • Coronary artery disease • Chronic degenerative changes

Ventricular Hypertrophy

Left	Right
• R in aVL > 11-13 mm • S in V1 + R in V5/V6 > 35 mm (i.e. > 3.5 mV) • S in V3 + R in aVL > 28 mm in men; > 20 mm in women • Slight ST-segment depression followed by an asymmetrically inverted T wave in V5-V6 • EKG findings of left atrial hypertrophy	• R wave exceeding the S wave in lead V1 • Right axis deviation • T wave inversions in V1-V3 • EKG findings of right atrial hypertrophy

Tall R waves in V1 (R/S ratio ≥ 1)

- Posterior myocardial infarction
- Acute right ventricular dilatation (strain)
- Hypertrophic cardiomyopathy
- Right ventricular hypertrophy
- Right bundle branch block
- Left ventricular ectopy
- Dextrocardia
- Progressive muscular dystrophy
- Misplaced leads
- Normal variant (1% of the population)

QRS Axis

Lead I	Lead II	QRS Axis	Differential Diagnosis
QRS+	QRS+	- 30° to 90°	• Normal
QRS -	QRS +	Rightward shift +90° to + 150°	• Right ventricular hypertrophy • Left posterior hemiblock • Lateral wall myocardial infarction • Chronic lung disease (e.g. emphysema) • Acute right ventricular overload (e.g. PE)
QRS +	QRS -	Leftward shift -30° to - 90°	• Left ventricular hypertrophy • Left anterior hemiblock • Left bundle branch block • Inferior wall myocardial infarction • Endocardial cushion defects (congenital)
QRS -	QRS -	"Northwest" axis +150° to - 90°	• Incorrect lead placement • Situs inversus • Heart transplant
Iso-electric QRS	Iso-electric QRS	Indeterminate axis	• Normal variant • Intoxication with sodium channel blockers • Hyperkalemia

Differential Diagnosis of ST-Segment Elevation

Pathophysiology	Characteristics
STEMI	• Horizontal or convex (dome-shaped) ST-segment elevation • ST-segment elevation in lead III > ST-segment elevation in lead II • Check-mark sign refers to a QR-T complex, i.e. a complex where the QR complex seems to merge directly with the T wave • Reciprocal ST-segment depressions may be present • The location of the ST elevation corresponds to the culprit lesion
Diffuse Ischemia	• Type 2 myocardial infarctions can lead to ischemic ST-segment elevations that are not limited to a specific coronary territory
Normal	• Normal ST-segment elevation occurs in 90% of healthy young men in the precordial leads (concave up, no reciprocal ST depressions).
Early Repolarization	• ST-segment elevation associated with a notch at the J point in V4. The ST-segment is concave up and the T waves are upright in V2 – V6.
Pericarditis	• ST-segment elevations are concave (saddle-shaped) and diffuse, i.e. not limited to a specific coronary territory • Reciprocal ST-segment depressions are absent • ST-segment elevation to T wave amplitude ratio ≥ 0.25 in lead V6 strongly suggests pericarditis
LVH	• ST-segment elevation in the precordial leads can occur in the context of left ventricular hypertrophy
LBBB	• A LBBB results in ST-segment elevation in the precordial leads. • A pacemaker that stimulates the right ventricle will also result in a LBBB pattern. • A STEMI equivalent in the setting of a LBBB can be detected using the Sgarbossa criteria
Hyperkalemia	• Hyperkalemia can cause ST-segment elevation ("pseudoinfarct pattern")
Brugada	• The Brugada pattern consists of : • downward sloping ST-segment elevation in leads V1 + V2 • complete or incomplete right bundle branch block
Flutter	• Flutter waves may lead to ST-segment elevation
Takotsubo cardiomyopathy	• Takostubo cardiomyopathy is also referred to as apical ballooning syndrome, stress cardiomoypathy and broken heart syndrome • ST-segment elevation on EKG which usually yields to T wave inversions within hours

Differential Diagnosis of ST-Segment Depression

Pathophysiology	Characteristics
Ischemia	• ST-segment depression from subendocardial ischemia • Reciprocal ST-segment depression from transmural infarction
LVH / RVH	• ST-segment depression resulting from hypertrophy: "strain pattern."
BBB	• Bundle branch blocks lead to ST-segment depression in certain leads.
Medications	• 'Scooping" or 'coving' ST-segment depression suggests a pharmacological effect, e.g. secondary to digoxin.
Metabolic	• Hypokalemia can result in ST-segment depression

Differential Diagnosis of Large Positive T waves

Pathophysiology	Characteristics
Myocardial ischemia	• Hyperacute T waves refer to tall, symmetrical T waves seen in the acute phase of a transmural infarction resulting from localized extracellular hyperkalemia
Hyperkalemia	• 'Tenting' and 'peaking' of the T wave refer to tall, symmetrical T waves generally considered to be the earliest EKG sign of hyperkalemia

Differential Diagnosis of Negative T waves

Pathophysiology	Characteristics
Normal	• Normal, negative T waves can be seen in leads with a negative QRS complex, e.g. in V1
Left ventricular hypertrophy	• The typical LV strain pattern consists of an initially convex, gradually downward sloping ST-segment leading to an inverted, asymmetric T wave with abrupt return to the baseline in lateral leads (I, aVL, V5, V6)
Pulmonary embolism	• Negative T waves in the precordial leads (V1-V4) are often seen in patients with acute coronary syndrome (ACS) and pulmonary embolism (PE). Negative T waves in both III and V1 suggested PE.
Myocardial infarction	• Negative T waves occur during the evolving phase of a Q wave and sometimes a non-Q wave myocardial infarction.
Myocardial ischemia	• Deep symmetrical T wave inversions (type 1) or biphasic T wave changes (type 2) in V2 and V3, in a patient with a history of angina pain who is pain free, suggest tight LAD stenosis. This pattern is refered to as 'Wellens' syndrome' or 'LAD coronary-T wave syndrome' and suggests left anterior descending artery stenosis.
Takotsubo	• Takotsubo (stress) cardiomyopathy is a cardiac syndrome characterized by ST-segment elevation, negative T waves, elevated cardiac enzymes and transient left ventricular apical ballooning without obstructive coronary disease.
CVA-T waves	• Very deep, widely splayed negative T waves may occur in the setting of cerebrovascular accidents such as subarachnoid hemorrhage, and are referred to as 'CVA-T waves'
Pericarditis	• Diffusely inverted T waves may be seen weeks following acute pericarditis

Abnormal T Wave Morphology

Pathophysiology	Characteristics
Pseudo-normalization	• Pseudonormalization of the T waves refers to a normal T wave replacing a negative T wave in a patient with acute chest pain or angina equivalent. Such a phenomenon suggests acute coronary syndrome.
Biphasic, notched T wave	• The T waves of patients with hereditary long QT syndromes are frequently abnormal with a biphasic contour or a prominent notched component.

QTc Interval

The **QT interval** is measured from the Q wave until the end of the T wave. The QT interval varies with the heart rate, and the Bazett formula is used to correct for the heart rate: QTc = QT / square root of the RR interval expressed in seconds. The lower limit of a normal QTc interval is around 330 msec but has not been well defined. The upper limit of a normal QTc time is 450 msec in adult men, 470 msec in adult women and 460 msec in 1-15 year-olds.

Differential Diagnosis of Prolonged QTc Interval

Pathophysiology	Examples
Electrolytes	• Hypokalemia, hypomagnesemia, hypocalcemia (less commonly)
Medications	• Antiarrhythmics, especially Class IA (Quinidine, pronainamide) and Class III (Ibutilide, Sotalol, Amiodarone) • Antidepressants, e.g. tricyclic antidepressants. • Antipsychotics, e.g. phenothiazines • Antihistamines, e.g. • Miscellaneous, see http://www.azcert.org for a complete list
Hereditary	• Congenital Long QT Syndrome is caused by 'channelopathies,' i.e. abnormal ion channel function in the heart that result in prolonged repolarization.
Ischemia	• Myocardial ischemia
Other	• Cerebrovascular accidents • Hypothermia prolongs the QT interval by slowing the repolarization of myocardial cells

Differential Diagnosis of Short QTc Interval

Pathophysiology	Examples
Electrolytes	• Hypercalcemia, hyperkalemia
Medications	• Digitalis

Differential Diagnosis of U Waves

Pathophysiology	Examples
Electrolytes	• Hypokalemia, hypercalcemia
Metabolic	• Thyrotoxicosis
Medications	• Sotalol, phenothiazines, digitalis and other medications
Other	• Cerebrovascular accidents can lead to prominent U waves in conjunction with CVA T waves

Episolon waves are low amplitude notches found right after the QRS in the right precordial leads (V1-V3). They suggest arrhythmogenic right ventricular dysplasia (ARVD), a genetic disorder leading to fibro-fatty changes that can cause sudden cardiac death in young people. Other EKG findings that may be present in patients with ARVD include:

- QRS-duration ≥ 110 msec in V1-V3
- S wave upstroke (from the nadir of the S wave to the isoelectric line) ≥ 55 msec in V1-V3 (95% of patients); the interval between the nadir of the S wave and the end of all depolarization deflections is referred to as the Terminal Activation Duration (TAD).
- T wave inversions in V1-V3 (85% of patients)
- QRS-duration > 110 msec in I

Ionizing Radiation in Pregnancy

A fetal dose **< 50 mGy** is considered safe. The radiation dose during pregnancy from naturally occuring background radiation is 0.1 - 0.5 mGy.

Examination	Fetal Dose (mGy)*
Extremities	< 0.001
Chest X-ray (2 views)	0.002
CT head or neck	0
CT chest (with/without angiography)	0.2
Low-dose perfusion scintigraphy	0.1 - 0.5
CT abdomen	4
CT abdomen and pelvis	25
CT angiography of the aorta	34

* Depends on type of equipment, mother's abdominal girth, fetal distance from maternal skin

In trauma, treat pregnant women like any other trauma patient. Contrast CT will allow to assess the placenta.

Contrast Associated Nephropathy

ADefinitino.

Risk factors
Diabetes
Chronic heart failure (NYHA Class III/IV)
Dehydration (vomiting, diarrhea, ileus)
Nephrotoxic medations (e.g. NSAID)
State these in the referral to radiology

De svenska rekommendationerna bygger
på europeiska (www.esur.org) och andra
internationella [14] rekommendationer.
Rekommendationerna påbjuder försiktighet
på grund av risk för kontrastmedelsnefropati
hos patienter med skattad GFR
<45 ml/min eller då multipla icke-renala
riskfaktorer föreligger. Detta gäller också
patienter i dialys
med betydande restfunktion,
medan man inte behöver spara på doserna
om restfunktion saknas.

(www.sfmr.se/sidor/jodkontrastmedel)
fortfarande

Relativt GFR (ml/min/1,73 m_2) används för att klassificera patientens njurfunktion oberoende av kroppsstorlek för att kunna bedöma om, och till vilken grad, njurarna är skadade.
Absolut GFR (ml/min) är den enskildes faktiska utsöndringskapacitet och avgör vilken kontrastmedelsdos i förhållande till denna kapacitet (gram jod/absolut GFR ratio) som är lämpligt att använda beroende av graden av njurfunktion/njurskada.

https://www.sfmr.se/sidor/kontrastmedel/