Emergency Medicine Checklist Compendium: Problems

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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 (timesensitive 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):

0	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 \(\beta\theta\theta CG\), 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" (rather referred to as Clinical Risk Scores) 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

О	☐ 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	$1 (10-14.9), 2 (\geq 15)$
% Neutrofils	$1 (70-84\%), 2 (\geq 85\%)$
CRP	$1(10-49), 2 (\geq 50)$
Temp $\geq 38.5^{\circ}$	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

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	☐ Leg strength & gait
neuro	□ Romberg
	☐ Sensation leg & perineum
	☐ Patella & plantar reflexes

CONSIDER PERFORMING

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

TESTS

315
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:
 - o 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
 - o radiculopathy attributable to a single nerve root level AND (risk of metastatic cancer OR moderate to high risk of infection)
 - o 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:
 - o risk factors: current immunosuppression/hemodialysis, current or recent injection drug use/invasive epidural/spinal procedure/endocarditis or bacteremia
 - o 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:
 - o multiple risk factors (based on age, smoking history, family history, physical examination findings e.g. focal vertebral tenderness, recent weight loss)
 - o history of cancer
 - o strong clinical suspicion

VERTEBRAL COMPRESSION FRACTURE

- Plain radiography if at risk. Based on:
 - o advanced age
 - o prolonged systemic glucocorticoid use
 - o significant trauma
 - o 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 ($\Delta = difference$)

Rule-Out 30-day MACE	Rule-In 30-day MACE
0h hs-cTnT < 12 ng/L AND	$0h hs-cTnT \ge 52 ng/L OR$
$1h\Delta < 3 \text{ ng/L AND}$	$1h\Delta \ge 5 \text{ ng/L OR}$
History not high-risk AND	0h or 1h hs-cTnT $>$ 14 ng/L + either high-
EKG non-ischemic	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 \geq 1 high-risk feature/condition. High risk if score \geq 2.

AORTIC DISSECTION & d-dimer

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

AORTIC DISSECTION & EKG

An ECG that is diagnostic of acute coronary occlusion is highly unlikely to be secondary to aortic dissection, and performing a CT scan delays reperfusion.

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?	
	□ Allergies?	
P	☐ Past medical history? Prior cancer?	
L	☐ Life circumstances?	
E	☐ Alcohol: how often? How much?	
S	☐ Smoking: amount? Prior smoking?	

HISTORY

0	☐ 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	□ Orientation
Function	□ Dysphasia / dysarthria
	☐ Visual fields / neglect
Cranial	☐ Visual fields/neglect
Nerves	☐ 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
Coordi-	☐ Finger-nose
nation	□ Knee-shin
	□ Romberg

TESTS

CRP if > 50 y	/ears
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*

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	Neck stiffness
• Fever	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	Sudden onset of the headache
Abnormal findings on neurological examination	

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)	• Age > 50
Elevated CRP without alternative explanation	years

MIGRAINE: "POUNDing"

 \geq 4/5 of the following had LR 24 for migraine while \leq 2/5 had LR 0.41 for migraine:

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

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

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

\mathbf{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)
- \square Glucose (≥ 1 ml pink top)

OTHER TESTS FOR SEPTIC ARTHRITIS

- ☐ Blood cultures x 2: aerobic and anaerobic. Adult bottles (8-10 ml in each)
- □ WBC + Neutrophils
- ☐ Glucose
- \square 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%)	• Gonococcal arthritis
• Septic arthritis (8-27%)	• Viral arthritis
• Osteoarthritis (5-17%)	• Lyme disease
• Rheumatoid arthritis (11-16%)	 Drug-induced arthritis
• Reactive arthritis (2-19%)	• Reactive arthritis
• Systemic lupus erythematosus (7%)	• Rheumatic fever
• Pseudogout (3%)	 Seronegative spondyloarthropathies
• Spontaneous hemarthrosis (3%)	• Systemic lupus erythematosus
Charcot's joint	

SEPTIC ARTHRITIS

WBC COUNT

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

ACUTE PRIMARY GOUT

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

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

KNEE OSTEOARTHRITIS

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

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

Leg Pain/Swelling

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

BAC	CKGROUND	TEST
M	☐ Current medications?	□ CRP
	☐ Birth control pill? Hormones?	
A	□ Allergies?	CONSIDER
P	□ Past medical history?	1. Deep vein thrombosis
	□ Prior clots in the leg or lung?	2. Arterial insufficiency
L	☐ Life circumstances?	3. Infection
E	☐ Alcohol: how often? How much?	4. Compartment syndrome
S	☐ Smoking: amount? Prior smoking?	5. Ruptured Achilles tendon
HIS	TORY	
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?	
PHY	YSICAL	
Vit	als RR, SpO2%, HR, BP, Temp?	
Leg		
]	□ Palpation	

Leg Pain/Swelling: DDx & Clinical Diagnostic Rules

DIFFERENTIAL DIAGNOSIS

Arterial	• Chronic arterial	Muscle	Necrotizing soft-tissue
	insufficiency		infections
	• Acute arterial insufficiency,		 Muscle rupture, strain,
	e.g. secondary to embolism		hematoma
Venous	Deep venous thrombosis	Skin	• Cellulitis, erysipelas,
	Superficial		abscess
	thrombophlebitis		Erythema nodosum
	Venous insufficiency	Bursa	Rupture of a popliteal
	• Venous compression, e.g.		(Baker's) cyst
	tumor, advanced pregnancy	Tendon	Achilles tendon rupture
	Budd-Chiari		Tenosynovitis
	Right heart failure	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)	
Pitting edema confined to the symptomatic leg	1
Collateral superficial veins (nonvaricose)	1
Previously documented deep-vein thrombosis	
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
 - o negative ultrasound proximal veins + negative repeat ultrasound (+1 week)
 - o negative ultrasound proximal + distal veins

Scrotal/Testicular Pain

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

		•	
BAC	CKGRO	UND	TESTS
M	□ Curr	rent medications?	□ CRP
Α	□ Alle	rgies?	☐ Urine dipstick
P		medical history?	
L	_	circumstances? Sexual activity?	CONSIDER
E		ohol: how often? How much?	1. Testicular torsion
S	□ Smo	king: amount? Prior smoking?	2. Epididymitis
	TORY		٦
0		en did the pain start? What were you doing?	
	_	e till max intensity: sec? min? hr?	
P		location? Size of the painful area?	
	□ Radiation?		
Q	☐ Description of pain quality		
R			
S	□ VAS (1-10)?		
T			
	☐ Prior similar painful episodes?		
+	☐ Dysuria, urgency, discharge?		
	☐ Fever / chills? ☐ Nausea, vomiting?		
	□ Nau	sea, vointing:	
PHY	SICAL		_
Vit	tals	□ RR, SpO2%, HR, BP, Temp?	
Bu	k	□ Inspection	
		□ Palpation	
Ge	nitalia	□ 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?
	□ Allergies?
P	□ Past medical history?
L	☐ Life circumstances?
E	☐ Alcohol: how often? How much?
S	☐ Smoking: amount? Prior smoking?

HISTORY

О	☐ 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	
\square 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

0	☐ 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?
В	□ 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
\square EKG if > 50 years

CONSIDER IF UNCLEAR

- 1. Metabolic: Wernicke's, liver encaphalopathy, uremia
- 2. Electrolyltes (Na, Ca) and Endocrine (hypothyroid, addison's)
- 3. Diabetes: hypoglycemia, DKA, HHS
- 4. Vital encephalitis, bacterial meningitis
- 5. Non-convulsive and partial complex seizure
- 6. Poisoning, intoxication
- 7. Sepsis
- 8. Structural: stroke including basilar thrombosis, cerebral vein thrombosis

Altered Consciousness: DDx & Clinical Syndromes

DIFFERENTIAL DIAGNOSIS

Pathophysiology	Examples	
Vascular	• Arterial: basilar artery thrombosis, large stroke (including SAH),	
Cardiac	primary CNS vasculitis, giant cell arteritis	
	• Venous: cerebral sinus thrombosis (delirium, amnesia, mutism)	
	• Systemic: cardiogenic chock, thrombotic thrombocytopenic	
	purpura, hypertensive encephalopathy, PRES	
Infectious	• Intracranial infections: meningitis, encephalitis, cerebral abscess	
Infiltrative	• Extracranial infections: pneumonia, urosepsis	
Neurological	• Seizures: status epilepticus (including non-convulsive), post-ictal	
Neoplastic • Increased ICP: brain tumor, hydrocephalus, hematom		
Psychiatric • Psychiatric: depression, psychosis		
Degenerative	Degenerative: Alzheimer's dementia	
Deficiency	• Deficiency : Wernicke's encephalopathy, B12 deficiency	
Intoxication	• Traditional medications: overdose, withdrawal (e.g. opioids)	
Withdrawal	• 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	• Intracranial: concussion, shunt dysfunction	
Mechanical	• Extracranial: urinary retention, fat embolism syndrome	
Electrolytes	• Electrolytes: hypo- hypernatremia, hypo- hypercalcemia	
Endocrine	• Endocrine/Metabolic: hypoglycemia, HHS, hypothyroidism,	
Metabolic	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

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

TESTS

\square 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-	Deep brow ache	• Tearing, red, extremely painful
Angle Glaucoma	Nausea, vomiting	• Fixed mid-size pupil; hard eyeball
Vitreous	• Vision reduction propor-	• +/- decreased red reflex
Hemorrhage	tional to amount of blood	No relative afferent pupillary defect
Central Retinal	• Acute onset, painless	Relative afferent pupillary defect
Artery Occlusion	• Severe vision loss	• Milky-white retina + cherry-red spot
Central Retinal	• Subacute onset, painless	• +/- relative afferent pupillary defect
Vein Occlusion	• Vision loss up to severe	• "Blood-and Thunder" retina
Retinal	• Floaters, black dots,	• +/- relative afferent pupillary defect
Detachment	photopsias; painless	Decreased red reflex
	Visual field deficit	• Elevated retina with folds
Optic Neuritis	• Pain on eye movement	Relative afferent pupillary defect
	Washed-out colour	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)	Pituitary pathology
		hemianopsia	
		Binasal hemianopsia	Bilateral internal carotid artery
			aneurysms, hydrocephalus
		Left homonymous	Lesion affecting the right optic tract
		hemianopsia	Lesion affecting the right occipital lobe
		Left homonymous superior	Lesion affecting the lower right optic
		quadrantanopsia	radiations in the temporal lobe
		Left homonymous inferior	Lesion affecting the upper right optic
		quadrantanopsia	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

BAC	CKGROUND	VITALS	
M	☐ Current medications?	□ RR, S ₁	pO2%, HR, BP, Temp?
A	☐ Allergies to meds or contrast?		-
P	☐ Past medical history?	NEUROS	CREEN
L	☐ Life circumstances?	Cranial	□ Visual fields?
E	☐ Alcohol: how often? How much?	nerves	☐ Pupil size, reactivity?
S	☐ Smoking: amount? Prior smoking?		☐ Eye movements?
			☐ Facial movement?
HIS	TORY		☐ Soft palate and tongue?
O	☐ When did the double vision start?	Motor	☐ Proximal arm strength?
	What were you doing?		☐ Distal arm strength?
	☐ Time till max intensity: sec? min? hr?		☐ Proximal leg strength?
P	☐ Does the double vision persist when		☐ Distal leg strength?
	one eye is closed?	Coordi-	□ Romberg?
	☐ Is the separation of images horizontal	nation	☐ Finger-nose?
	or vertical/diagnonal?		□ Knee-shin?
	☐ Where are you looking when the		
	double vision is worst? Least?	EYE	
R	☐ Is there any corrective head position	□ Inspec	tion of the eyelids,
	that makes the double vision		nctiva, cornea
	tolerable?	□ Visual	acuity
	☐ Is the double vision worse when	□ Swing	ing flashlight test
	looking near or worse at distance?	☐ Fundo	scopy
	☐ Does the double vision worsen during		
	the day?	TESTS	
T	☐ Constant or intermittent? Increasing?	□ CRP i	f > 50 years
	☐ Prior similar episodes?		
+	☐ Eye or periorbital pain?	CONSIDI	ER
	☐ Pain upon eye movement?	1. Intracra	nial process (e.g. aneurysm)
	☐ Headache?	2. Orbital	
	☐ Fever?	3. Giant ce	
	☐ Other neurological deficits?	4 Wernick	ce's encenhalonathy

Double Vision: Differential Diagnoses & Clinical Clues

DIFFERENTIAL DIAGNOSIS BINOCULAR DIPLOPIA

Anatomy	Examples	
Extraocular	• Infections (orbital cellulitis)	
Muscle &	• Neoplasms of the orbit	
Orbit	Myositis	
	Trauma (e.g. muscle entrapment secondary to orbital fracture)	
	Graves' disease with exophthalmos and diplopia	
Neuromuscular	Myasthenia gravis (antibodies to the post-synaptic ACh receptors)	
Junction	Botulism (decreased presynaptic acetylcholine release)	
Cavernous	• Cavernous sinus thrombosis (e.g. from a facial infection or aseptic)	
Sinus	• 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	• Stroke, e.g. basilar artery thrombosis	
(affecting	• Infections, e.g. viral encephalitis	
cranial nerve	Neoplasms	
nuclei and	Wernicke's encephalopathy	
connections)	• 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 (V₁), maxillary sensory loss (V₂), 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?	

TESTS

□ Acid-base: pH, pCO ₂ , HCO ₃ /BE
☐ Electrolytes: Na, K, Ca
□ Hb, WBC, CRP
□ Creatinine
☐ Liver function tests
\square EKG if > 50 years

HISTORY (from patient and/or other)

0	☐ 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?	

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
В	☐ 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)
С	☐ 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 PSYCHIATRIČ

Suggests Organic	Suggests Psychiatric
• Age < 12 years or > 40 years without	• Age 12 - 40 years
previous psychiatric diagnosis	Previous psychiatric diagnosis
Sudden onset of symptoms	Gradual onset of symptoms
 Visual or tactile hallucinations 	Auditory hallucinations
 History of substance abuse 	• No recent ingestions of mind-altering stuff
New medications including herbal meds	No new medications
Seizure	No seizures
No family history of psychiatric	Significant family history of psychiatric
disorders	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	• Intracranial infections: herpes encephalitis
Infiltrative	• Extracranial infections: sepsis, botulism
Neurological	Space occupying lesion
Neoplastic	• Paraneoplastic syndrome: NMDAR-antibody encephalitis
Degenerative	Wernicke's encephalopathy, B12 deficiency
Deficiency	Wilson's disease
Intoxication	• Traditional medications: overdose, withdrawal, toxic levels
Withdrawal	• Other substances: alcohol (delirium tremens), cocaine, synthetic
	drugs, carbon monoxide, heavy metals
Autoimmune	• SLE, myasthenia gravis, MS
Trauma	• Intracranial: concussion, shunt dysfunction
Mechanical	• Extracranial: urinary retention, fat embolism syndrome
Electrolyte	• Electrolytes: hypercalcemia
Endocrine	• Endocrine/Metabolic: hyperthyroidism/toxicosis, Addison's,
Metabolic	hypothyroidism/myxedema coma, hepatic encephalopathy, uremia

SUICIDE RISK

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

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? meningoencephalitis?
- medications/non-compliance (order blood levels of antiepileptic meds)?
- 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 is defined by the European Society of Cardiology as "a state of real or apparent LOC with loss of awareness, characterized by amnesia for the period of unconsciousness, abnormal motor control, loss of responsiveness, and a short duration."

Conditions that Can be Misinterpreted as transient loss of Consciousness

Complex partial seizure	No loss of motor control (no fall)
Psychogenic pseudosyncope	Duration many minutes to hours
Fall without TLOC	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 or impairment of consciousness
Intoxication	Prolonged loss or impairment of consciousness

2-NON-TRAUMATIC TRANSIENT LOSS OF CONSCIOUNESS: CAUSE?

Syncope	Due to transient cerebral hypoperfusion:	
	• Reflex syncope: vasovagal, situational (micturition, swallow,	
	defecation, cough, sneeze, post-exercise), carotic sinus syndrome	
	• Orthostatic hypotension: drug-induced, volume depletion, Iary (e.g.	
	Parkinson's disease) vs IIay autonomic failure (e.g. diabetes)	
	• Cardiovascular: arrhythmia (tachy, brady), structural (cardiac, PE, AD,	
	pulm HTN) structural cardiac (aortic stenosis, myocardial ischemia,	
	hypertrophic cardiomyopathy, pericardial disease/tamponade),	
	structural great vessels (PE, AD, pulmonary hypertension)	
Seizure	Due to abnormal excessive brain activity. Generalized seizure:	
	Tonic, clonic, tonic-clonic	
	• Atonic	
Psychogenic	Due to the psychological process of conversion	
	Psychogenic pseudosyncope	
	Psychogenic non-epileptic seizures	
Rare Causes	Subclavian steal syndrome	
	Vertebrobasilar transient ischemic attack	
	Subarachnoid hemorrhage	
	Cyanotic breath holding spell	

MAPLES

This	suggests
Certain medications	Bradycardia, orthostatic, prolonged QTc
Heart disease	Cardiac
Brain disease	Seizure
Hypertension	Orthostatic
Parkinsonism	Orthostatic
Diabetes mellitus	Cardiac, orthostatic

Situation

This	suggests
During exercise	Arrhythmia
After exercise	Reflex, orthostatic
After standing up	Orthostatic
During micturition, defecation, coughing attack	Reflex
Head trauma	Commotio
During laugh, emotion, surprise	Cataplexia
Head rotation, pressure on neck	Carotid-sinus
Pain, fear, procedure	Reflex
Lack of sleep	Seizure, reflex
Warm environment	Reflex, orthostatic
Crowded room	Reflex

Symptoms Preceding LOC

This	suggests
"Epigastric rising"	Seizure (vasovagal)
Palpitations	Tachyarrhythmia (vasovagal)
Weird smell/taste, déjà vu	Seizure
Nausea, sweating, pale	Reflex
Pain in the shoulders and neck	Orthostatic
Sudden onset of severe headache	Subarachnoidal hemorrhage
Crying out upon LOC	Seizure

During LOC

This	suggests
Jerks: onset before/upon LOC	Seizure
Jerks: symmetrical, synchronous, 20-100	Seizure
Jerks: asymmetrical, asynchronous, < 10	Syncope
Urinary incompetence	No diagnostic value
LOC < 30 sec	Syncope >> seizure
LOC > 1 min	Seizure > syncope*
Pelvic thrusting	Psychogenic, frontal seizure
Cyanosis	Seizure, cardiac

Post LOC

This	suggests
Direct cognitive recovery, disoriented < 10 sec	Syncope
Confused for minutes	Seizure, arrhythmia with hypoxia
Muscular pain	Seizure
Chest pain	Cardiac ischemia

Physical

This	suggests	
Tongue bite: lateral	Seizure	
Tongue bite: tip	Syncope	

Syncope: Risk Stratification

BACKGROUND

Low	•	Years of recurrent syncope with low-risk features + same characteristics of current episode
	•	Absence of structural heart disease
High	•	Severe structural or coronary artery disease (heart failure, low LVEF, previous MI

HISTORY

Low	Prodrome typical of reflex syncope (e.g. lightheaded, sweating, nausea, vomiting) After syndom uncorrected symplescent sight, count, are all on rain.
	After sudden unexpected unpleasant sight, sound, smell or pain
	After prolonged standing or in crowded, hot place
	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	New onset of chest discomfort, breathlessness, abdominal pain or headache
Major	Syncope during exertion or when supine
	Sudden-onset palpitation immediately followed by syncope
High	No warning symptoms or short (<10 s) prodrome
Minor*	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

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
Major	Mobitz II second-degree and third-degree atrioventricular (AV) block
	• Slow atrial fibrillation (AF) (<40 bpm)
	• Persistent sinus brady (<40 bpm), repetitive sinoatrial block or sinus pauses >3 s
	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 ICD)
	• 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
High	Mobitz I 2° AV block and 1° AV block with markedly prolonged PR interval
Minor*	Asymptomatic inappropriate mild sinus bradycardia or slow AF (40-50 bpm)
	Paroxysmal SVT or atrial fibrillation
	Pre-excited QRS complex
	• Short QTc interval (≤ 340 ms)
	Atypical Brugada patterns
	Negative T waves in right precordial leads, epsilon waves suggestive of ARVC

^{*} High-risk only if history consistent with arrhythmic syncope

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?

NEUROSCREEN

Cranial	□ Visual fields?	
nerves	☐ 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-	□ Romberg?	
nation	☐ Finger-nose?	
	☐ Knee-shin?	

^{*} Use Frenzel's glasses to detect subtle nystagmus

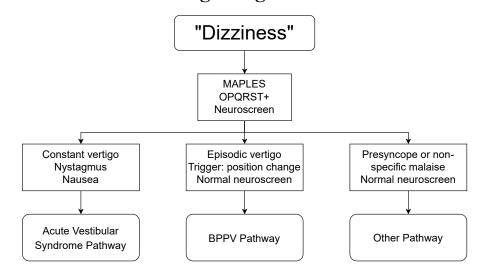
CONSIDER

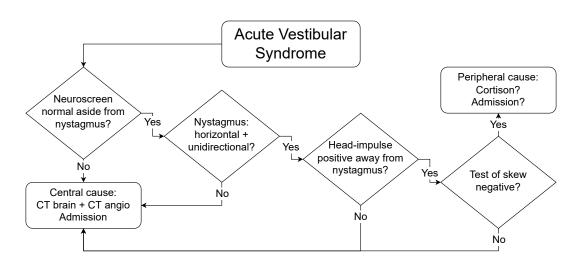
- 1. Stroke, vertebrobasilar dissection
- 2. Bacterial labyrinthitis
- 3. Metabolic-Cardiovascular-Toxic conditions

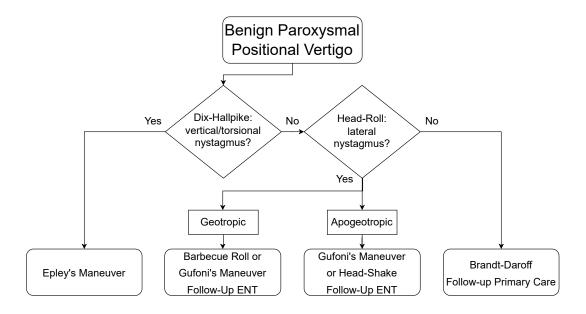
PHYSICAL

□ RR, SpO2%, HR, BP, Temp?	
----------------------------	--

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	□ Visual fields?		
nerves	☐ 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-	□ Romberg?		
nation	☐ Finger-nose?		
	□ Knee-shin?		

TESTS

\square EKG if > 50 years	
\square 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

BAC	CKGROUND
M	□ Current medications?
	□ Platelet inhibitors? Anticoagulant?
A	□ Allergies?
P	□ Past medical history?
HIS	ΓORY
	Mechanism of injury?
ll .	Visual acuity?
	Double vision?
	Pain?
PHY	SICAL 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
Ger	ntly place a Tegaderm over the eyelid, apply LOTS of gel and "float" the linear probe
ove	r 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
1	ight in other eye and/or a light through the eyelid
TES	TS
ll .	EKG if > 50 years
	NR and thrombocytes if the patient is taking an anticoagulant
CON	NSIDER

- 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)	• decreased visual acuity
• ophthalmoplegia	 afferent pupillary defect
• ocular pressure greater than 40 mm Hg	• 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.

Head/Neck Trauma

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

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

^{*} with oral and written instructions

CANADIAN C-SPINE RULE	NEXUS LOW-RISK CRITERIA
No cervical spine x-ray is required if all 4	No cervical spine x-ray is required if all
are present:	5 are present:
• Inclusion Criteria: all	Normal level of alertness
High Risk Factors: 0	 No evidence of intoxication
• Low Risk Factors: ≥ 1	 No painful distracting injuries
• Can actively rotate neck > 45° left and	No focal neurologic deficit
right	No posterior cervical-spine tenderness
Inclusion Criteria	High Risk Factors
• > 15 years	• Age ≥ 65 years
No history of back or vertebral disease	• Paresthesias in the extremities
Normal level of consciousness	• Dangerous mechanism of injury:
• Trauma < 48 hrs old	○ Fall from \geq 1 m or 5 stairs
Low Risk Factors	 Axial load on the head
Simple rear-end motor vehicle collision	 Motor vehicle collision at high
• Sitting position in the ED	speed (> 100 km/h) or with rollover
• Ambulatory at any time	or ejection
Delayed (not immediate) onset of neck	A collision involving a motorized
pain	recreational vehicle
Absence of midline C-spine tenderness	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	Focal neurologic findings
• Acute skull fracture, including depressed or basilar	Skull fracture, especially
fracture	findings of basilar skull fracture
Seizure following injury	Seizure
• Altered mental status (eg, lethargy or irritability)	• Persistent altered mental status
• Definite loss of consciousness if longer than a few	(eg, agitation, lethargy,
seconds	repetitive questioning, or slow
Bulging fontanelle	response to verbal questioning)
Persistent vomiting	• Prolonged loss of consciousness
• Suspicion of child abuse	

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

< 2 years	≥2 years
Vomiting that is self-limited	Vomiting
• Loss of consciousness that is uncertain, or isolated and very	Headache
brief (less than a few seconds)	 Questionable or brief
History of lethargy or irritability, now resolved	loss of consciousness
Behavioral change reported by caregiver	(LOC)
• 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)	• Injury caused by highrisk mechanism of 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	

No neuroimaging if all of the following criteria are met

< 2 years	≥2 years
No severe mechanism of injury*	No severe mechanism of
Normal mental status	injury*
No palpable skull fracture	• Normal mental status
• No history of LOC ≥ 5 sec	 No signs of basilar skull
No occipital or parietal or temporal scalp haematoma	fracture
Acting normally per parent	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 \geq 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	Head-on collision, rollover, ejection from the vehicle, death
crash	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, complex crush
- Deep or contaminated lacerations on the bottom of the foot
- Human bite wounds

Methods of primary closure:

- Non-suture alternatives e.g. tissue adhesives +/- steristrips
- Absorbable sutures

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 CONSIDER ☐ Recently taken/terminated medications/substances? 1. Anaphylaxis 2. Angioedema ☐ Recent NSAID use? ☐ Known allergies to medications, food, other? □ Past medical history? ☐ Recent medical test (e.g. with contrast agent)? ☐ Life circumstances? ☐ Alcohol: how often? How much? ☐ Smoking: amount? Prior smoking? **HISTORY** ☐ When did the symptoms start? What were you doing? ☐ Time till max intensity: sec? min? hr? ☐ Which body parts are affected? ☐ Rash? Swelling? Itch? Pain? 0 ☐ Effect of measures if taken (e.g. corticosteroids, R antihistamine)? ☐ Impact on daily function? S □ Constant, intermittent, increasing symptoms? ☐ Prior similar episodes? □ Food intake? ☐ Insect bite? □ New soap / washing detergent? **PHYSICAL** ☐ Hoarse? Stridor? □ Lip- tongue swelling? □ SpO2% ☐ Respiratory rate? □ Lung auscultation? ☐ Chest wall examination □ Pulse/blood pressure ☐ Heart rate ☐ Level of consciousness? ☐ Front side of the body

□ Back side of the body

☐ Temperature?

Allergic Reaction: Clinical Diagnostic Clues

ANAPHYLAXIS

Anaphylaxis is highly likely when any one of the two criteria are fulfilled:

1-Acute onset of **hypotension** or **bronchospasm** or **laryngeal involvement** after exposure to a **known or highly probable allergen** for that patient (minutes to several hours), even in the absence of typical skin involvement.

2-Acute onset of an illness (minutes to several hours) with simultaneous involvement of the **skin, mucosal tissue, or both** (eg, generalized hives, pruritus or flushing, swollen lipstongue-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)
- Severe gastrointestinal symptoms (eg, severe crampy abdominal pain, repetitive vomiting), especially after exposure to non-food allergens

See Anaphylaxis

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)

See Angioedema

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 (e.g.			
	ate hand-picked mushrooms?)			
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		
	□ Auscultation	
	□ Palpation	
PR	☐ Stool colour?	

TESTS

CRP		

CONSIDER

- 1. Sepsis
- 2. Gastrointestinal bleeding3. Colonic and mesenteric ischemia
- 4. Invasive bacterial syndrome
- 5. 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	TravelHospitalized > 3	 80% probability of bacterial etiology Persistent diarrhea suggests a protozoa Clostridium difficile 	 Presumptive antibiotic therapy combined with clinical observation Stools for Clostridium
	daysAntibiotic useContact with health care personnel		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	t □ S3/S4, murmurs?		
	□ Elevated JVP?		
Lungs	Lungs ☐ Chest wall movements?		
	☐ Auscultation: rales? ronchi? decreased		
	breath sounds?		
Legs	☐ Swelling? Edema?		

TESTS

- □ pH, pCO2, HCO3/BE
- \square CRP
- \square 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 of 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 \ge 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	SCORE	RISK
Initial assessment		0	3%
History of stroke or TIA	1	1	5%
 History of intubation for respiratory distress 	2	2	9%
 Heart rate on ED arrival ≥ 110 	2	3	16%
• Room air SaO2 < 90% on EMS or ED arrive	al 1	4	26%
Investigations		5	40%
EKG has acute ischemic changes	2	6	55%
• Urea ≥ 12 mmol/L	1	7	70%
• Serum CO2 ≥ 35 mmol/L	2	8	81%
Troponin I or T elevated to MI level	2	9	89%
• NT-ProBNP \geq 5,000 ng/L	1	*Patient is as	ked to walk at
Walk Test* after ED treatment		-	e for 3 minutes
• One of the following:	1		gardless of the
○ SaO2 < 90% on room air or usual O2		distance cove	red
○ HR \geq 110 during 3-minute walk test			
Too ill to walk			

PNEUMONIA SEVERITY INDEX: SCORE

Risk Factors		Points	Risk Factors		Points
Demo-	Men	Yrs	Coexisting	Neoplastic disease	+30
graphics	Women	Yrs - 10	illnesses	Liver disease	+20
	Nursing home	+10		Congest. heart failure	+10
Labs &	pH < 7.35	+30		Stroke	+10
CXR	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 $\geq 30/\min$	+20
	Hematocrit < 30%	+10		SBP < 90 mm Hg	+20
	PaO2 < 60 mm Hg	+10		Temp $< 35^{\circ}$ C or $\ge 40^{\circ}$ C	+15
	Pleural effusion	+10		$HR \ge 125 \text{ bpm}$	+10

[&]quot;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 $\geq 2/3$ 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 POI	NTS	SCORE	RISK
History		0	2%
 Coronary bypass graft 	1	1	4%
Peripheral vascular disease intervention	1	2	7%
Intubation for respiratory distress	2	3	13%
Examination		4	21%
• Heart rate on arrival in ED ≥ 110 /min	2	5	33%
• Too ill to do the Walk Test* after treatn	nent in ED	6	48%
$(SaO2 < 90\% \text{ or heart rate} \ge 120/\text{min})$	2	7	63%
Investigations		8	76%
 Acute ischemic changes on ECG 	2	9	NA
• Pulmonary congestion evident on chest	X-ray 1	10	91%
• Hemoglobin < 100 g/L	3	*Patient is asked	d to walk at their
• Urea ≥ 12 mmol/L	1	own pace for 3 r	
• Serum CO2 ≥ 35 mmol/L	1	ED, regardless of	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

0	☐ 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 \geq 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

Pathor	ohysiology	Examples
Blood	Coagulo-	Thrombocytopenia
	pathy	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	Immune-complex small vessel vasculitides
(ANCA-associated)	(non-ANCA-associated)
 Granulomatosis with polyangiitis 	Henoch Schönlein Purpura
• Churg-Straus	Cryoglobulinemia
Microscopic Polyangiitis	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?
шіс	ΓORY
ш5	IUNI

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

PHYSICAL

TESTS			
□ Urinalysis	☐ Creatinine, Na, K	□ Cap CRP	☐ Bladder Scan: pre- + post-void

□ Leg strength, sensation

DIFFERENTIAL DIAGNOSIS OF URINARY RETENTION

☐ Per rectum: obstruction, sensation, prostate hypertrophy?

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 postobstructive diuresis. Observe for 4 hours; if urine output > 200 ml/hr for 2 hours, admit.

Acid-Base

Acid-Base Interpretation (Mnemonic: ACID)

11014 2450 111001	or ctation (which onic. ACID)				
Replace	• If the blood gas comes from peripheral venous blood, add 0.03 to the				
Values?	venous pH to estimate the arterial pH and remove 0.6 kPa (5 mm Hg)				
	from the venous pCO ₂ to estin	nate the arterial pCo	O_2 .		
	• If the pCO ₂ is between 3.3 and	17.3 kPa (25 and 5	5 mm Hg),	, the	
	standard bicarbonate HCO ₃ (st)) is a reasonable ap	proximatio	on of the	
	actual HCO ₃ . Otherwise, use t	he actual HCO3 (se	e table bel	ow)	
1. Acidosis /	• pH < 7.38 and HCO ₃ < 22 mm	nol/L: M	etabolic A	cidosis	
alkalosis?	• pH < 7.38 and pCO ₂ > 5.7 kPa	a (42 mm Hg): Re	spiratory	Acidosis	
	• pH > 7.42 and HCO ₃ > 26 mm		etabolic A		
	• pH > 7.42 and pCO ₂ < 5 kPa (38 mm Hg): Re	spiratory	Alkalosis	
2. Compen-	Disorder	Expected Comp	kPa	mm Hg	
sation?	Metabolic Acidosis	$\Delta pCO_2 = SBE x$	0.1	1	
	Metabolic Alkalosis	$\Delta pCO_2 = SBE x$	0.1	1/2	
	Respiratory Disorder < 2 days	SB	E = 0		
	Respiratory Acidosis > 5 days	$SBE = \Delta pCO_2 x$	3	1/3	
	Respiratory Alkalosis > 5 days				
3. Ions?	1. Calculate the Anion Gap (AG): Na - Cl - HCO ₃				
	2. Calculate the Delta AG (\(\Delta \) AG): Actual AG - Expected AG				
	• Expected AG is around 8 mm	ol/L when modern	nachines a	ire used	
	3. Calculate \triangle AG + HCO ₃				
	• A sum > 26 mmol/L suggests	the presence of eith	er a Meta	bolic	
	Alkalosis or a metabolic comp	pensation for a Res	piratory A	Acidosis	
	• A sum < 22 mmol/L suggests	the presence of eith	er a Norm	nal Anion	
	Gap Metabolic Acidosis or co	ompensation for a I	Respirator	\mathbf{y}	
	Alkalosis				
4. Diagnoses?	☐ Speculate on the most plausib	le causes of the aci	d-base dis	orders	
	using all available clinical inf	ormation			

Actual HCO₃ based on the pH and pCO₂

							pН				
	kPa	mm Hg	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
2	9.0	68	13	16	20	25	32	40	51	64	81
	9.5	71	13	17	21	27	34	43	54	68	85
bC	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/Condition	Anion(s)
M	Methanol	Formate and L-lactate
	Metformin	L-lactate
U	Uremia	Phosphates, sulphates, urate and hippurate
D	Diabetic ketoacidosis*	ß-hydroxybutyrate (main ketone) and acetoacetate
P	Propylene glycol	Pyruvate, L-lactate and D-lactate
	Pyroglutamic acid	Pyroglutamic acid (5-oxoproline)
I	Iron	L-lactate
	Isoniazid	L-lactate
L	L-Lactate	L-lactate
	D-Lactate	D-lactate
E	Ethylene glycol	Glycolate, glyoxylate, oxalate, lactate (may be falsely high)
	Ethanol ketoacidosis	ß-hydroxybutyrate
S	Salicylates	Pyruvate, L-lactate and ketones
	Starvation ketoacidosis	ß-hydroxybutyrate, acetoacetate, acetone

^{*} Euglycemic DKA (with P-glucose < 12 mmol/L) can occur during treatment with SGLT2 inhibitors such as Dapagliflozin (Forxiga) and Empagliflozin (Jardiance).

Normal Anion Gap Metabolic Acidosis (Hyperchloremic Metabolic Acidosis)

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

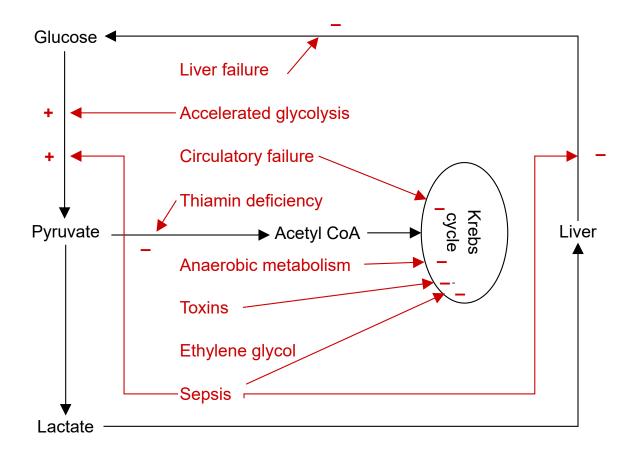
Decreased Anion Gap

	Causes	Pathophysiology
L	Lithium	Lithium is a cation
	Low albumin	Albumin is negatively charged
I	Iodide	Falsely elevated chloride value
M	M Myeloma Positively charged monoclonal IgG	
В	Bromide	Falsely elevated chloride value
S	Salicylates	Falsely elevated chloride value

L-Lactic Acidosis

L-Lactic Ac	Pathophysiology	Examples
Increased	Decreased O2 delivery to	Shock (hypovolemic, cardiogenic, septic)
lactate	tissues / anaerobic	• Severe hypoxemia, anemia (Hb < 50 g/L)
production	metabolism	Severe methemoglobinemia
		Carbon monoxide poisoning
		Pheochromocytoma, severe poisoning with
		sympathomimetics
		Cancers (due to tumor tissue hypoxia)
		Seizure, intensive exercise
	Increased glycolysis /	• Stimulation of \(\mathbb{B}2\)-receptors via endogenous
	pyruvate production	adrenalin: sepsis, stress, seizure, shivering,
		intensive exercise, pheochromocytoma
		• Stimulation of \(\text{B2-receptors via exogenous} \)
		agents: adrenalin, caffein-/theophyllin-/ß2-
		agonist poisoning, severe poisoning with
		sympathomimetics
		Stimulation of glycolysis via other machanisms require to a leader of the conservation of the conser
		mechanisms-respiratory alkalosis, cancer (lymphoma, leukemia, solid tumors)
	Decreased pyruvate	Thiamin deficiency
	metabolism to acetyl CoA	Thianini deficiency
	Interference with oxidative	Methanol, ethylene glycol (usually moderate
	phosphorylation	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 1	actate metabolism	Acute ethanol intoxication
		Metformin
		Liver failure (cirrhosis, hepatitis, severe
		paracetamol poisoning in late phase)
		• Sepsis
Falsely elev	ated 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

Pat	hophysiology	Examples
HCO3 ad	lministration	• Overzealous correction of a metabolic acidosis
H+ shifts	intracellular	Hypokalemia
H+ loss	Gastrointestinal	• Vomiting
	loss	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	Vascular problems, e.g. stroke, hemorrhage
System	• 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	• Nerve dysfunction, e.g. phrenic nerve paralysis, Guillain Barré
System	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 (PAO₂) and the partial pressure of oxygen in the arterial blood (PaO₂). 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:

- $PAO_2 = FiO_2 \times 713 PaCO_2 \times 1.25$
- PAO₂ when patient breathing room air = $21\% \times 713$ PaCO₂ x 1.25 = 150 PaCO₂ x 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:

- $PAO_2 = FiO_2 \times 95 PaCO_2 \times 1.25$
- PAO₂ when patient breathing room air = $21\% \times 95$ PaCO₂ x 1.25 = 20 PaCO₂ x 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 FiO₂ can be estimated from the delivered supplemental oxygen using the table below on the left. The PaO₂ can be estimated from the SpO₂ using the table below on the right.

Method	O ₂ flow	Estimated
	(L/min)	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	6	60
reservoir	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
- To identify common deficiencies: Fe, TIBC, Ferritin, B12, Folate, MCV
- Potential ruptured ectopic pregnancy: urine β-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/Pancytopenia?

• 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
 - o significant on-going bleeding
 - o acute coronary syndrome
 - o 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

Patho	physiology	Examples
Decreased	Hemoglobin	• Iron, B12, folate deficiencies
production		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	Hemorrhage	• Trauma
loss		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 (eGFR) assuming steady state in creatinine production and renal filtration. Use absolute eGFR as opposed to relative eGFR

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

- Increase in serum creatinine by $\geq 26.5 \,\mu\text{mol/L}$ 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 Creatin	ine are unreliable

2. Urgent Hemodialysis or Hemofiltration

- Acidemia (pH < 7.1) not responding to hemodynamic optimisation
- Electrolytes: K > 6.1 mmol/L refractory to medical management or rapidly rising K levels
- Ingestion of nephrotoxic drugs amenable to dialysis (salicylates, lithium, methanol . . .)
- Overload: respiratory distress resulting from pulmonary edema
- Uremic 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	• Renal artery stenosis (atherosclerotic or fibrodysplastic)
	occlusion	Renal artery emboli
	Renal arteriolar	Severe hypercalcemia
	vasoconstriction	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, fibrosisKidney 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)
 - o Ethanol, Paracetamol, PETh
 - Viral serologi
 - o Autoimmune serology (ANA, AMA, SMA)

MANAGEMENT

- Consider
 - o 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:
 - o Highly elevated tests
 - o Signs of liver failure (e.g. spontaneously elevated INR)
 - o Hepatic encephalopathy
 - o Acute cholecystitis, cholangitis, pancreatitis
- Out-of-hospital follow-up:
 - o Discontinue potential culprit medications
 - Advice to abstain from alcohol

DIFFERENTIAL DIAGNOSIS OF ELEVATED AST - ALT - AP - GT

Pathophysiology	Examples
Vascular,	• Acute ischemia (AST > ALT): shock, cocaine, metamphetamine etc.
Cardiac	Acute Budd-Chiari syndrome
	Congestive heart failure
Infectious,	• Viral hepatitis A, B, C, D, E
Infiltrative	• 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,	Non-alcoholic steatohepatitis
Metabolic	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	Hemolytic anemia (unconjugated)
Acute ischemic liver injury	Hematoma resorption (unconjugated)
Acute viral hepatitis	• Gilbert syndrome (unconjugated)
Severe auto-immune hepatitis	• Rotor syndrome (conjugated)
Wilson's disease	Dubin-Johnson syndrome (conjugated)

Hyponatremia

INVESTIGATIONS

- Urine sodium, Urine osmolarity, Serum osmolarity, TSH, T4, Cortisol
- For each 10 mmol/L increase in glucose, sodium drops by 3 mmol/L through dilution

MANAGEMENT

1. Hyponatremic Encephalopathy?

• Severe symptoms (confusion, 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 500 ml IV over 4 hours
- Suspected SIADH or heart failure/edema: fluid restriction (< 800 ml/day)
- Monitor urine output. If > 100 ml/hr: measure U-osm. If U-osm < 100 mOsm: give Desmopressin 1 ug IV and fluid restrict
- Repeat [Na] measurement initially every 2-6 hours; target rise Na 6-8 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	Primary polydypsia
• U < 100 mOsm/L	Secondary polydypsia (e.g. hypothalamic pathology)
Too little salt in	Anorexia nervosa
• U < 100 mOsm/L	• "Tea-and-toast" hyponatremia
	Beer potomania
Too little water out	"Appropriately" elevated ADH (U-Na < 30 mmol/L):
• $U > 100 \text{ mOsm/L}$	• 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	• Diuretic use (renal solute loss), especially thiazides
• $U > 100 \text{ mOsm/L}$	Primary adrenal insufficiency (hypoaldosteronism)
• U-Na > 30 mmol/L	• 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)

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

О	• 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 20 mmol (Addex®-Magnesium 1 mmol/ml 20 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 $K \ge 3.0$ mmol/L can usually be follow-up in the primary care setting

DIFFERENTIAL DIAGNOSIS OF HYPOKALEMIA

Pathophysiology	Examples		
Decreased potassium	Poor dietary intake		
intake	Geophagia		
Shift (extracellular	• Alkalosis		
to intracellular)	• 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	Renal	Diuretics, osmotic diuresis, diabetes insipidus	
loss		• High aldosterone:	
		 Primary hyperaldosteronism e.g. Conn' 	
		syndrome	
		 Secondary to intravascular volume depletion 	
		Renal tubular acidosis	
		Licorice ingestion, chewing tobacco	
		Hypomagnesemia	
	Gastro-	Vomiting, nasogastric suction	
	intestinal	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

О	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, give Glucose 300 mg/ml 100 ml IV instead of Glucose 50 mg/ml 500 ml (avoid a very peripheral PVC).

3. Beta-2 Agonist?

- Indication: potassium \geq 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
 - o Sodium zirconium cyclosilicate (Lokelma) 10 g x3/day PO
 - o Patiromer 8.4 g/day PO
 - o Calcium Resonium 15 g x3/day PO

5. Hemodialysis?

- **Indicated** in the following settings:
 - o severe life-threatening hyperkalemia
 - o hyperkalemia resistent to medical therapy
 - o end-stage renal disease
 - o oliguric acute kidney injury (< 400 mL/day urine output)
 - o 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-rich foods, potassium-containing drugs	
potassium intake	Intravenous administration	
Shift (intracellular	r • Acidosis (with a lipophobic anion)	
to extracellular)	Rhabdomyolysis, heavy exercise, hemolysis, tumor lysis	
	syndrome	
	Decreased N-K ATPase activity: insulin deficiency, digitalis intoxication	
Decreased renal	• Renal failure (acute kidney injury or chronic kidney disease)	
potassium loss	• Potassium-sparing diuretics (e.g. spironolactone), ACE-inhibitors,	
	ARBs, NSAIDs, beta-blockers, trimethoprim	
	Aldosterone deficiency (e.g. Addisons)	
Pseudo-	Tourniquet use	
hyperkalemia	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% 20 ml IV over 10 min
- Magnesium Sulphate 2 g = 8 mmol IV (Addex®-Magnesium 1 mmol/ml 8 ml) IV over 10 min

2. Asymptomatic Hypocalcemia

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

DIFFERENTIAL DIAGNOSIS OF HYPOCALCEMIA

DIFFERENTIAL DIAGNOSIS OF HYPOCALCEMIA			
Pathophysiology		Examples	
Decreased	Нуро-	Surgical	
oral intake,	parathyroidism	Autoimmune	
intestinal absorption,		Hypomagnesemia (PTH resistance / suppressed secretion)	
bone resorption		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:	
omanig	phosphatenna	 Increased phosphorus intake 	
		Tissue breakdown (rhabdomyolysis, tumor lysis SD)	
		Decreased renal excretion (e.g. acute renal failure)	
	Citrate	Transfusion of blood products	
	Bone	• Osteoblastic bone metastases (breast, prostate cancer)	
	mineralization	Hungry bone syndrome	
	Fat	Pancreatitis	
	Respiratory	Both acute and chronic respiratory alkalosis decrease	
	alkalosis	ionized Ca	
Increased	Hypoparathyroidism leads to increased calcium renal excretion		
renal loss			

Hypercalcemia

INVESTIGATIONS

• PTH, EKG

ELECTROCARDIOGRAM

О	• 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-	• Primary hyperparathyroidism (leading cause, 50% of cases)
parathyroidism	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	Sarcoidosis
diseases	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	Glucagon 1 mg IM	Glucagon 1 mg IM to children > 6 years
access		Glucagon 0.5 mg IM to children < 6 years
IV access	Glucose 300 mg/ml	Glucose 100 mg/ml (10%) 2 ml/kg IV
	(30%) 30 ml 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 insufficienc	
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)	

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	• $\text{CVP} > 10 \text{ cm H}_2\text{O}$
Caval index* < 50%	• 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 H₂O 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	• Focal posterolateral B-line may be found
• Interstitial pneumonia / pneumonitis	in a normal lung, due to gravity alone
• Pulmonary fibrosis	Pneumonia and pneumonitis
Acute respiratory distress syndrome	Atelectasis
(ARDS)	Pulmonary infarction or contusion
	Pleural disease
	Malignancy

Electrocardiogram

EKG Interpretation

	<u> </u>
0	□ 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)	• P wave > 2.5 mm, occasionally negative, < 0.12 sec
	in depth	=

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

Right Bundle Branch Block

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

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 hypertrophyLeft posterior hemiblockLateral 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 variantIntoxication with sodium channel blockersHyperkalemia

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		
	eciprocal 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	• ST-segment elevation associated with a notch at the J point in V4.		
Repolarization	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		
	rongly 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	Takostubo cardiomyopathy is also referred to as apical ballooning		
cardiomyopathy	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	Hyperacute T waves refer to tall, symmetrical T waves seen in the	
ischemia	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 occurring 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

Definition

• Acute kidney injury occurring shortly after administration of iodinated contrast and that may or may not be directly caused by the contrast material.

Risk factors: patient

- Chronic renal disease (especially diabetic nephropathy) with GFR < 45 ml/min
- Diabetes mellitus
- Chronic heart failure (NYHA III/IV)
- Dehydration (vomiting, diarrhea, ileus)
- Nephrotoxic medications (e.g. NSAID, certain antibiotics or chemotherapy agents)

Risk factors: procedure

- Dose and type of contrast agent
- Specific procedure: intra-arterial vs intravenous, interventional vs diagnostic angiography

Strategies to mitigate the risk of CAN:

- Hydration, e.g. Ringer-Acetat 3 ml/kg/hr during 1 hour pre och 1 ml/kg/h \geq 6 hrs post
- Discontinue Metformin for at least 48 hours if GFR < 45 ml/min or if angiography/intervention
- Avoid nephrotoxic agents