Cardiology

Clinical features of acute decompensated heart failure	
Clinical presentation	• Acute dyspnea, orthopnea, paroxysmal nocturnal dyspnea • Hypertension common; hypotension suggests severe disease • Accessory muscle use, tachycardia, tachypnea • Diffuse crackles with possible wheezes (cardiac asthma) • Possible S3, jugular venous distention, peripheral edema
Treatment	**Normal or elevated blood pressure with adequate end-organ perfusion** • Supplemental oxygen • Intravenous loop diuretic (eg, furosemide) • Consider intravenous vasodilator (eg, nitroglycerin) **Hypotension or signs of shock** • Supplemental oxygen • Intravenous loop diuretic (eg, furosemide) as appropriate • Intravenous vasopressor (eg, norepinephrine)

Poor prognostic factors in systolic heart failure	
Clinical	• Higher NYHA functional class • Resting tachycardia • Presence of S3 gallop • Elevated jugular venous pressure • Low blood pressure (< 100/60 mm Hg) • Moderate to severe mitral regurgitation • Low maximal oxygen consumption (peak VO_2)
Laboratory	• Hyponatremia • Elevated pro-BNP levels • Renal insufficiency
Electrocardiography	• QRS duration >120 msec • Left bundle branch block pattern
Echocardiography	• Severe LV dysfunction • Concomitant diastolic dysfunction • Reduced right ventricular function • Pulmonary hypertension
Associated conditions	• Anemia • Atrial fibrillation • Diabetes mellitus

CHA_2-DS_2-VASc score		
Risk criteria		Score
C	Congestive heart failure	1
H	Hypertension	1
A_2	Age ≥75*	2
D	Diabetes mellitus	1
S_2	Stroke/TIA/thromboembolism	2
V	Vascular disease (prior myocardial infarction, peripheral artery disease, or aortic plaque)	1
A	Age 65-74*	1
Sc	Sex category (ie, female)	1
	Maximum score	**9**

Anticoagulation	
CHA_2-DS_2-VASc score	Stroke
0	Lo
1	Interme
≥2	Hig

Indications for urgent dialysis (AEIOU)	
Acidosis	• Metabolic acidosis ◦ pH <7.1 refractory to medical therapy

Electrolyte abnormalities	• Symptomatic hyperkalemia
	  ◦ ECG changes or ventricular arrhythmias
	• Severe hyperkalemia
	  ◦ K >6.5 mEq/L refractory to medical therapy

Ingestion	• Toxic alcohols (methanol, ethylene glycol) • Salicylate • Lithium • Sodium valproate, carbamazepine
Overload	Volume overload refractory to diuretics

Uremia	• Symptomatic:
	  ◦ Encephalopathy
	  ◦ Pericarditis
	  ◦ Bleeding

*Normal changes to the aging heart include decreased resting and maximal cardiac output, decreased maximum heart rate, increased contraction and relaxation time of heart muscle, increased myocardial stiffness during diastole, decreased myocyte number, and pigment accumulation in myocardial cells.

Clinical manifestations of amyloidosis (primary & secondary)	
Renal	• Heavy **proteinuria** with nephrotic syndrome • Peripheral edema
Cardiac	• **Restrictive cardiomyopathy** • Conduction defects, low voltage
CNS	• Peripheral &/or autonomic neuropathy • Stroke
GI	• Hepatomegaly • Dysmotility, malabsorption • GI bleeding due to vascular fragility
Pulmonary	• Pulmonary nodules, tracheobronchial infiltration, pleural effusions
Musculoskeletal	• Enlarged tongue, shoulder pad enlargement
Skin	• Thickening of skin, subcutaneous nodules/plaques • Ecchymoses, periorbital purpura
Heme	• Anemia, thrombocytopenia

Amyloidosis	

Etiology	• Extracellular deposition of insoluble polymeric protein fibrils in tissues & organs
	• Can be primary (AL type) or secondary (AA) to chronic inflammatory conditions such as:
	  ○ **Inflammatory arthritis** (eg, rheumatoid arthritis)
	  ○ **Chronic infections** (eg, bronchiectasis, tuberculosis, osteomyelitis)
	  ○ **Inflammatory bowel disease** (eg, Crohn's disease)
	  ○ **Malignancy** (eg, lymphoma)
	  ○ **Vasculitis**

Clinical presentation	• Asymptomatic proteinuria or nephrotic syndrome • Restrictive cardiomyopathy • Hepatomegaly • Peripheral neuropathy &/or autonomic neuropathy • Visible organ enlargement (eg, macroglossia) • Bleeding diathesis • Waxy thickening, easy bruising of skin
Diagnosis	• Abdominal fat pad aspiration biopsy

Drugs/supplements that affect warfarin metabolism (selected)	
CYP450 Inhibitors **↑ Warfarin effect** (↑ bleeding risk)	• Acetaminophen, NSAIDs • Antibiotics/antifungals (eg, metronidazole) • Amiodarone • Cimetidine • Cranberry juice, *Ginkgo biloba*, vitamin E • Omeprazole • Thyroid hormone • SSRIs (eg, fluoxetine)
CYP450 Inducers **↓ Warfarin effect** (↓ in efficacy)	• Carbamazepine, phenytoin • Ginseng, St. John's wort • Oral contraceptives • Phenobarbital • Rifampin

AAA: The imaging modality of choice for diagnosis and follow-up is abdominal ultrasound, as it has nearly 100% sensitivity and specificity, facilitates measurement of aneurysm size, and can show the presence of any associated thrombus.

Ascending aortic aneurysms (60% of cases) typically start anywhere from the aortic valve to the innominate artery. These are most often due to cystic medial necrosis (which usually occurs with aging) or connective tissue disorders (eg, Marfan syndrome, Ehlers-Danlos syndrome). Descending aortic aneurysms (40% of cases) arise distal to the left subclavian artery. Descending aortic aneurysms, as in this patient, are usually due to atherosclerosis; risk factors include hypertension, hypercholesterolemia, andsmoking.

men aged 65-75 who have smoked cigarettes as having the greatest benefit from screening,

The main risk factors associated with aneurysm expansion and rupture include large diameter, rate of expansion, and current cigarette smoking.

Diabetes is a strong risk factor for atherosclerosis and cardiovascular disease. However, for unclear reasons, the risk of AAA. in patients with diabetes is lower than in those without diabetes. In observational studies, AAA. growth rate was also noted to be lower in diabetics.
The current indications for operative or endovascular repair include
aneurysm size >5.5 cm, rapid rate of aneurysm expansion (>0.5 cm in 6 months or >1 cm per year), and presence of symptoms (abdominal, back, or flank pain; limb ischemia) regardless of aneurysm size.

Coarctation of the aorta	
Etiology	• Congenital • Acquired (rare) (eg, Takayasu arteritis)

Clinical features	• Upper body
	  ○ Well developed
	  ○ Hypertension (headaches, epistaxis)
	• Lower extremities
	  ○ Underdeveloped
	  ○ Claudication
	• Brachial-femoral pulse delay
	• Upper & lower extremity blood pressure differential
	• Left interscapular systolic or continuous murmur

Diagnostic studies	• ECG: Left ventricular hypertrophy
	• Chest x-ray
	  ○ Inferior notching of the 3rd to 8th ribs
	  ○ "3" sign due to aortic indentation
	• Echocardiography: Diagnostic confirmation

Treatment	• Balloon angioplasty ± stent placement • Surgery

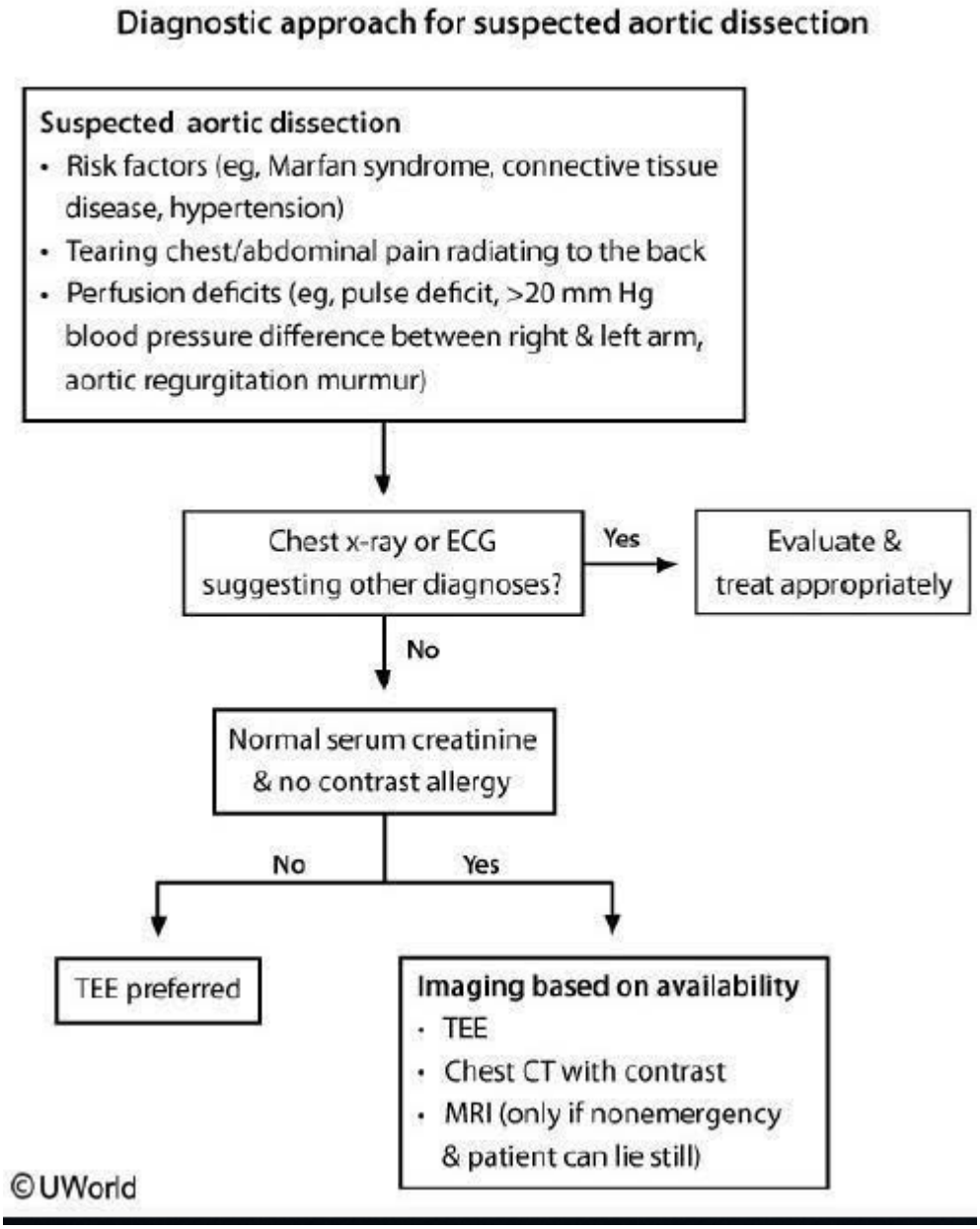
ECG findings consistent with left ventricular hypertrophy (LVH) (eg, high-voltage QRS complexes, lateral ST segment depression, lateral T wave inversion) are suggestive of long-standing systemic hypertension. Essential hypertension rarely leads to end-organ damage (eg, LVH) in young patients (eg, age <40), and therefore a cause of secondary hypertension should be sought.

Clinical features of aortic dissection	
Risk factors/ associations	• Hypertension (most common) • Marfan syndrome • Cocaine use
Clinical features	• Severe, sharp, tearing chest or back pain • >20 mm Hg variation in systolic blood pressure between arms
Complications (involved structure)	• Stroke (carotid arteries) • Acute aortic regurgitation (aortic valves) • Horner syndrome (superior cervical sympathetic ganglion) • Acute myocardial ischemia/infarction (coronary artery) • Pericardial effusion/cardiac tamponade (pericardial cavity) • Hemothorax (pleural cavity) • Lower-extremity weakness or ischemia (spinal or common iliac arteries) • Abdominal pain (mesenteric artery)

Systemic hypertension is the most important predisposing risk factor associated with the development of aortic dissection. It is twice as common in patients with type B (descending aorta) dissection compared with type A (ascending aorta) aortic dissection. A sudden and transient increase in blood pressure is frequently noted in patients with acute aortic dissection. Patients are usually older (age >60) and may have a prior aortic aneurysm. Atherosclerosis alone does not increase the predisposition to aortic dissection, although it is frequently seen, particularly in association with thoracic aortic and abdominal aortic aneurysms**.**

Marfan syndrome is responsible for almost 50% of the aortic dissections seen in patients age <40, it is an uncommon cause in older patients (age >60).

Management of patients with acute aortic dissection
• Pain relief with morphine • Intravenous beta blockers for a target systolic blood pressure of 100-120 mm Hg (labetalol, propranolol, esmolol) • Transfer to intensive care unit • Initiate additional vasodilator (eg, sodium nitroprusside) if systolic blood pressure remains elevated • Surgery for acute ascending aortic dissections & complicated descending aortic dissections



In Aortic Dissection CT angiography is the initial study of choice in hemodynamically stable patients with no evidence of renal dysfunction. It can reveal an intimal flap separating the true and false lumens in the aorta.

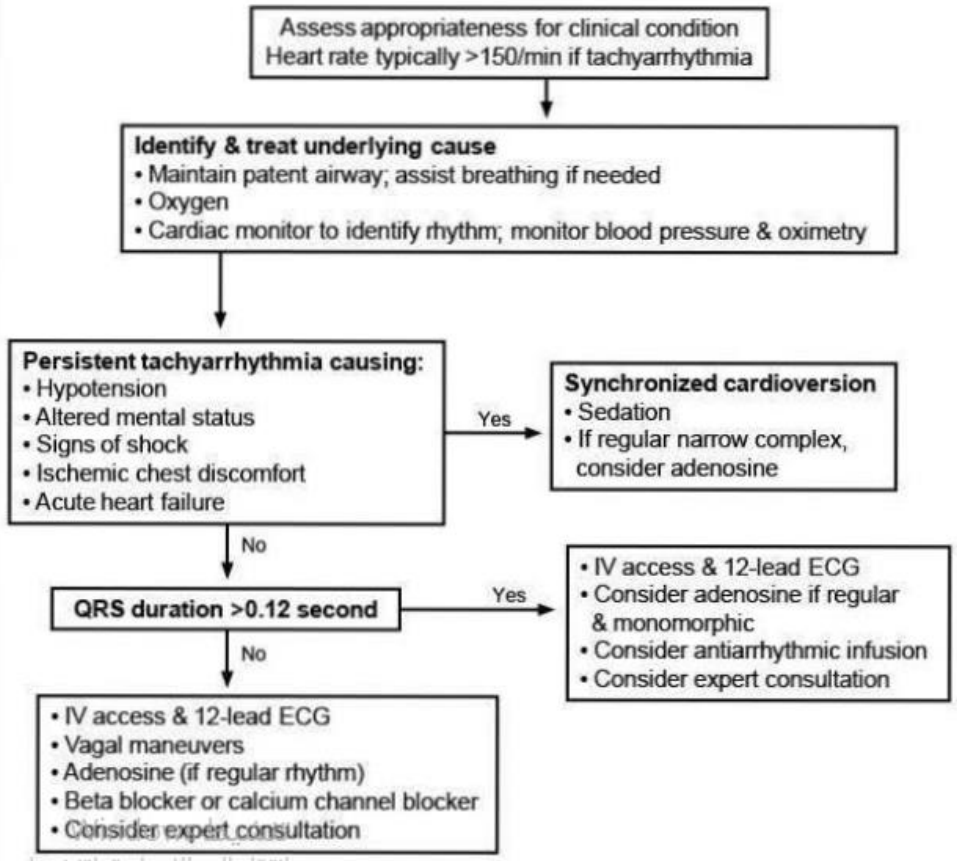
Supravalvular aortic stenosis (AS), the second most common type of AS, usually refers to congenital left ventricular outflow tract obstruction due to discrete or diffuse narrowing of the ascending aorta. This causes a systolic murmur similar to that seen with valvular AS; however, the murmur is usually best heard at the first right intercostal space, higher than where the valvular AS murmur is best heard. Patients may also have unequal carotid pulses, differential blood pressure in the upper extremities (high-pressure jet in ascending aorta), and a palpable thrill in the suprasternal notch. Patients with significant supravalvular AS develop left ventricular hypertrophy over time and can also have coronary artery stenosis as an associated anomaly. These changes, along with the increase in myocardial oxygen demand during exercise, can lead to subendocardial or myocardial ischemia, which is likely responsible for this patient's anginal symptoms during exercise.

Cholesterol crystal embolism (atheroembolism)	
Risk factors	• Comorbid conditions (hypercholesterolemia, hypertension, type 2 diabetes mellitus) • **Cardiac catheterization** or vascular procedure
Clinical features	• Dermatologic (**livedo reticularis**, ulcers, gangrene, **blue toe syndrome**) • Renal (**acute or subacute kidney injury**) • Central nervous system (stroke, amaurosis fugax) • Ocular involvement (**Hollenhorst plaques**) • Gastrointestinal (intestinal ischemia, pancreatitis)
Diagnosis	• **Laboratory findings** ◦ Elevated serum creatinine, **eosinophilia**, hypocomplementemia ◦ Urinalysis – typically benign with few cells or casts, may have **eosinophiluria** • **Skin or renal biopsy** ◦ Biconvex, needle-shaped clefts within occluded vessels ◦ Perivascular inflammation with eosinophils

Amiodarone is a class Ill antiarrhythmic drug often used for management of ventricular arrhythmias in patients with coronary artery disease and ischemic cardiomyopathy.
Amiodarone can cause several potential adverse effects, and long-term monitoring of several parameters (eg, pulmonary function and thyroid tests) is recommended for early detection and recognition of potential side effects from its use.
Pulmonary toxicity is a serious adverse effect of long-term amiodarone use that usually presents with chronic Interstitial pneumonitis (most common) but can cause acute respiratory distress syndrome. Pulmonary toxicity correlates with the total cumulative dose and usually occurs months to several years after the initiation of the drug therapy.
A baseline chest rad iograph and pulmonary function testing (PFT) are usually obtained prior to initiating therapy, and long-term surveillance is guided by development of signs or symptoms (cough, fever. dyspnea) suggestive of pulmonary toxicity.

Major side effects of amiodarone	
Cardiac	• Sinus bradycardia, heart block • Risk of proarrhythmias – QT prolongation & risk of torsades de pointes
Pulmonary	• **Chronic** interstitial pneumonitis (cough, fever, dyspnea, pulmonary infiltrates) most common
Endocrine	• Hypothyroidism • Hyperthyroidism
Gastrointestinal/ Hepatic	• Elevated transaminases, hepatitis
Ocular	• Corneal microdeposits • Optic neuropathy
Dermatologic	• Blue-gray skin discoloration
Neurologic	• Peripheral neuropathy

Adult tachycardia algorithm (with pulse) – Advanced Cardiac Life Support guidelines



Based on the Advanced Cardiac Life Support guidelines, all patients with a pulse who have persistent tachyarrhythmia (narrow or wide complex) causing clinical or hemodynamic instability (hypotension, cardiogenic shock, signs of ischemia, acute heart failure) should be managed with immediate direct current cardioversion. With cardioversion, energy delivery is synchronized to the QRS complex to minimize the likelihood of the shock occurring during repolarization, which can precipitate ventricular fibrillation.
By contrast, immediate defibrillation (as opposed to cardioversion) provides a highenergy shock at a random point in the cardiac cycle (unsynchronized shock) and is indicated in patients with ventricular fibrillation or pulseless ventricular tachycardia.

The term "lone AF" is occasionally used for patients with paroxysmal, persistent, or permanent AF with no evidence of cardiopulmonary or structural heart disease. Patients with lone AF are generally age <60 and, by definition, have a CHA,DS,-VASc score of 0. The risk of systemic embolization in such patients is extremely low, and anticoagulant therapy is not indicated.

Conditions associated with atrial fibrillation	
Cardiac	• Hypertensive heart disease (most common) • Coronary artery disease • Rheumatic/Valvular heart disease (eg, mitral stenosis, mitral regurgitation) • Congestive heart failure • Hypertrophic cardiomyopathy • Congenital heart disease (eg, atrial septal defect) • Post cardiac surgery
Pulmonary	• Obstructive sleep apnea • Pulmonary embolism • Chronic obstructive pulmonary disease • Acute hypoxia (eg, pneumonia)
Miscellaneous	• Obesity • Endocrine (eg, hyperthyroidism, diabetes) • Alcohol abuse • Drugs (eg, amphetamines, cocaine, theophylline)

Class I antiarrhythmic drugs act by blocking sodium channels and inhibiting the initial depolarization phase (phase 0) of the action potential. Flecainide and propafenone are class IC antiarrhythmic drugs that are occasionally used in treatment for atrial fibrillation (for maintenance of sinus rhythm) in patients with structurally normal hearts. They have the slowest rate of drug binding and dissociation from the sodium channel receptor. Under normal circumstances, flecainide does not cause any significantprolongation of the QRS or QT interval.
In patients with faster heart rates, the drug has less time to dissociate from the sodium channels, leading to a higher number of blocked channels, which in turn leads to a progressive decrease in impulse conduction and a widening of the **QRS** complex. This phenomenon is termed "use

dependence," and is seen most frequently with class IC antiarrhythmic agents (less with class IA drugs and rarely with class IB drugs), and is the mechanism behind their efficacy against supraventricular arrhythmias.

Cardiac myxoma	
Tumor characteristics	• 80% located in the left atrium

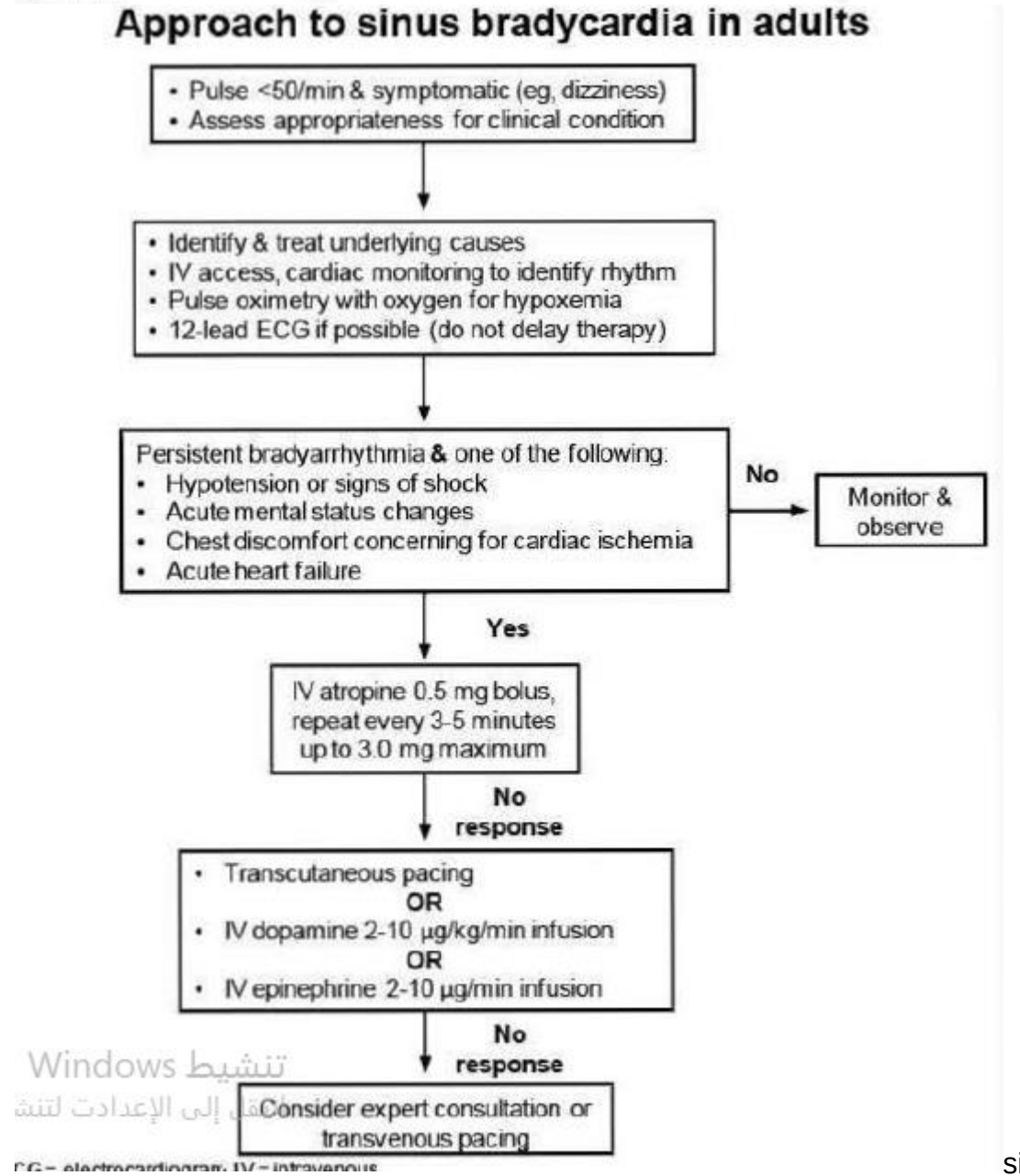
Clinical features	• **Constitutional** symptoms (eg, fever, weight loss, Raynaud phenomenon)
	• **Cardiovascular** complications
	  ○ Valvular abnormalities (eg, mitral disease)
	  ○ Heart failure due to anatomic obstruction
	  ○ Myocardial invasion causing arrhythmias, heart block, or pericardial effusion
	• **Embolization**
	• Lung invasion causing respiratory symptoms mimicking bronchogenic carcinoma

Diagnosis & management	• Echocardiogram • Prompt surgical resection

About 50% of patients report constitutional symptoms (due to overproduction of interleukin-6) such as fever, weight loss, or Raynaud phenomenon.
Although transesophageal echocardiography is the most sensitive test for diagnosis, transthoracic echocardiography is usually adequate.

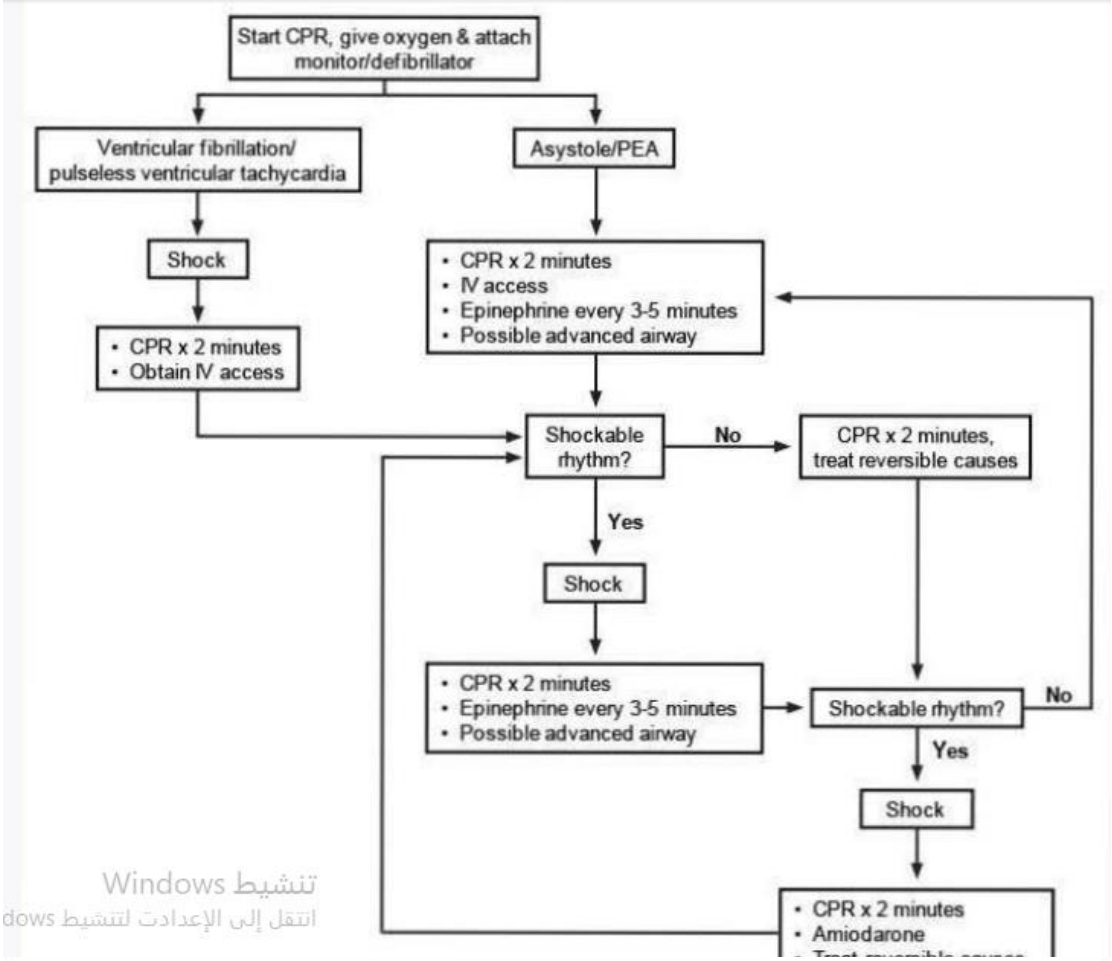
This patient's presentation- bradycardia, atrioventricular block, hypotension, and diffuse wheezing- is suggestive of beta blocker overdose. Intoxication with calcium channel blockers, digoxin, and cholinergic agents could also cause similar symptoms, but wheezing is more specific for beta blocker toxicity. The most common presentation is bradycardia and hypotension leading to cardiogenic shock, as evidenced by this patient's cold and clammy extremities. Other effects include hypoglycemia, bronchospasm (beta 2 blockade), and neurological dysfunction (eg, delirium, seizures).
The first steps in management are to secure the airway and give isotonic fluid boluses and intravenous (IV) atropine for initial treatment of hypotension and bradycardia. In patients with refractory or profound hypotension, the next step is to administer IV glucagon. Glucagon increases the intracellular levels of cyclic AMP and has been effective in treating both beta blocker and calcium channel blocker toxicity. Other therapies that can be used simultaneously or in succession include IV calcium, vasopressors (epinephrine or norepinephrine), high-dose insulin and glucose, and IV lipid emulsion therapy.

Symptoms of digoxin toxicity	
Cardiac	• **Life-threatening arrhythmias**
Gastrointestinal	• Anorexia • Nausea & vomiting • Abdominal pain
Neurologic	• Fatigue • Confusion • Weakness • **Color vision alterations**



sinus bradycardia (heart rate <60/min with regular rhythm and a constant PR interval). The normal resting heart rate is usually 60-100/min. Sinus bradycardia can occur normally in adolescents and younger adults, in well-conditioned athletes, and in some elderly patients, especially during sleep. Pathologic causes

include sick sinus syndrome, myocardial ischemia or infarction, obstructive sleep apnea, hypothyroidism, increased intracranial pressure, and medications.



Reversible causes of asystole/pulseless electrical activity	
5 Hs	**5 Ts**
Hypovolemia	Tension pneumothorax
Hypoxia	Tamponade, cardiac
Hydrogen ions (acidosis)	Toxins (narcotics, benzodiazepines)
Hypo- or hyperkalemia	Thrombosis (pulmonary or coronary)
Hypothermia	Trauma

Patients with RVMI are initially treated similarly to others with acute MI: dual antiplatelet therapy, statins, anticoagulation, and urgent revascularization (thrombolytics or primary percutaneous intervention). Patients with RVMI require a high preload to maintain adequate right heart output. Therefore, patients with hypotension and low/normal jugular venous pressure (JVP) should be given high-flow intravenous (IV) fluids to increase RV preload (if JVP is elevated, IV fluids are less

likely to be helpful). Drugs that decrease preload, such as nitrates, diuretics and opioids, can cause profound hypotension and should be avoided. Drugs that slow the heart rate (beta blockers) or decrease contractility (calcium channel blockers) should be used with caution.

Management of ST-segment elevation myocardial infarction (STEMI)

- Oxygen for arterial saturation <90%
- Nitrates
  - Caution with hypotension, right ventricular infarction, or severe aortic stenosis
- Antiplatelet therapy
  - Aspirin + $P2Y_{12}$ receptor blocker
- Anticoagulation
  - Unfractionated heparin, low-molecular weight heparin, or bivalirudin
- Beta blockers
  - Contraindicated in overt heart failure
  - High risk for cardiogenic shock
  - Bradycardia
- Prompt reperfusion with PCI
  - Ideal first medical contact to PCI ≤90 minutes
- Statin therapy as soon as possible

Hemodynamic measurements in shock

Parameter	Normal	Hypovolemic shock	Cardiogenic shock	Septic shock
Right atrial pressure (preload)	Mean of 4 mm Hg	↓	↑	Normal to slight ↓
Pulmonary capillary wedge pressure (preload)	Mean of 9 mm Hg	↓	↑	Normal to slight ↓
Cardiac index (pump function)	2.8-4.2 L/min/m^2	↓	↓↓	↑
Systemic vascular resistance (afterload)	Mean of 1,150 dynes•sec/cm^5	↑	↑	↓
Mixed venous oxygen saturation	60%-80%	↓	↓	↑

Indications for carotid endarterectomy	
Men	**Asymptomatic:** • 60-99% stenosis **Symptomatic:** • 50-69% stenosis (Grade IIA) • 70-99% stenosis (Grade IA)
Women	**Both symptomatic & asymptomatic:** • 70-99% stenosis

Symptomatic carotid stenosis is defined by the occurrence (within the past 6 months) of sudden-onset focal neurological symptoms corresponding to a carotid artery lesion.

*Atrial tachycardia with AV block is the arrhythmia most specific for digitalis toxicity.

- High Output HF

Congenital	Acquired
Patent ductus arteriosus	Trauma
Angiomas	Iatrogenic (e.g., femoral catheterization)
Pulmonary AVF	Atherosclerosis (e.g., aortocaval fistula)
CNS AVF	Cancer

- Symptomatic AVF can be congenital or acquired (as shown in the table above) and creates an abnormal connection between the arterial and venous systems that bypasses the capillary beds. Shunting of a large amount of blood through the fistula decreases systemic vascular resistance, increases cardiac preload, and increases cardiac output. Clinical signs include widened pulse pressure, strong peripheral arterial pulsation (e.g., brisk carotid upstroke), systolic flow murmur (as in this patient), tachycardia, and usually flushed extremities. The left ventricle hypertrophies, and the point of maximal impulse is displaced to the left. An ECGusually shows left ventricular hypertrophy.

Causes of heart failure with preserved left ventricular function	
Diastolic heart failure	• Hypertension with left ventricular hypertrophy • Restrictive cardiomyopathy • Infiltrative cardiomyopathy (eg, sarcoidosis) • Hypertrophic cardiomyopathy • Occult coronary artery disease
Valvular heart disease	• Aortic stenosis or regurgitation • Mitral stenosis or regurgitation
Pericardial disease	• Constrictive pericarditis • Cardiac tamponade
Systemic disorders (high-output cardiac conditions)	• Thyrotoxicosis • Severe anemia

Etiologies of pericarditis	
Infection	Viral (most common), bacterial
Iatrogenic	Surgery, trauma, radiation & drug-related
Connective tissue disease	Rheumatoid arthritis, systemic lupus erythematosus
Cardiac	Dressler syndrome (post-myocardial pericarditis), usually 1-6 weeks after myocardial infarction
Uremic	Serum BUN usually >60 mg/dL, but degree of pericarditis does not always correlate with degree of elevation
Malignancy	Can be due to cancer (eg, lung & breast, Hodgkin lymphoma) or treatment (eg, radiation, chemotherapy)

Clinical features of cocaine use	
Clinical features	• Sympathetic hyperactivity - tachycardia, hypertension, dilated pupils • Chest pain due to coronary vasoconstriction • Psychomotor agitation, seizures
Complications	• Acute myocardial ischemia • Aortic dissection • Intracranial hemorrhage
Management of chest pain	• **Benzodiazepines** for blood pressure & anxiety • Aspirin • Nitroglycerin & calcium channel blockers for pain • Beta blockers **contraindicated** • Fibrinolytics not preferred due to increased risk of intracranial hemorrhage • Immediate cardiac catheterization with reperfusion when indicated

Atrophic Nasal mucosa

- Cocaine acts as a sympathomimetic agent that induces coronary vasoconstriction, promotes thrombus formation, and increases the risk of myocardial ischemia and infarction by causing increased myocardial oxygen demand. Patients with acute cocaine intoxication should initially be treated with intravenous benzodiazepines to alleviate psychomotor agitation and sympathomimetic effects (eg, tachycardia, hypertension).Those with acute coronary syndrome (ACS) are managed with antiplatelet therapy,nitrates, and percutaneous coronary intervention if indicated. However, the use of beta blockers should be avoided due to the risk of unopposed cocaine-induced alpha agonist activity and resultant worsening vasoconstriction.

Features of constrictive pericarditis	
Etiology	• Idiopathic or viral pericarditis • Cardiac surgery or radiation therapy • Tuberculous pericarditis (in endemic areas)
Clinical presentation	• Fatigue & dyspnea on exertion • Peripheral edema & ascites • ↑ Jugular venous pressure • Pericardial knock may be heard • Pulsus paradoxus • Kussmaul's sign
Diagnostic findings	• ECG may be nonspecific or show atrial fibrillation or low-voltage QRS complex • Imaging shows pericardial thickening & calcification • Jugular venous pulse tracing shows prominent x & y descents

Characteristic findings of cor pulmonale	
Common etiologies	• COPD (most common) • Interstitial lung disease • Pulmonary vascular disease (eg, thromboembolic) • Obstructive sleep apnea
Symptoms	• Dyspnea on exertion, fatigue, lethargy • Exertional syncope (due to ↓ cardiac output) • Exertional angina (due to ↑ myocardial demand)
Examination	• Peripheral edema • ↑ Jugular venous pressure with prominent *a* wave • Loud S2 • Right-sided heave • Pulsatile liver from congestion • Tricuspid regurgitation murmur
Imaging	• **Electrocardiogram (ECG)**: Partial or complete right bundle branch block, right axis deviation, right ventricular hypertrophy, right atrial enlargement • **Echocardiogram**: Pulmonary hypertension, dilated right ventricle, tricuspid regurgitation • **Right heart catheterization**: Gold standard for diagnosis showing right ventricular dysfunction, pulmonary hypertension & no left heart disease

- Prinzmetal's angina. It is caused by temporary spasm of the coronary arteries, as opposed to atherosclerotic narrowing which is seen in myocardial infarction. Young women are classically affected, and the greatest risk factor for variant angina is smoking. Aside from smoking, there is often an absence of cardiovascular risk factors. Variant angina is associated with other vasospastic disorders, such as Raynaud's phenomenon and migraine headaches. The episodes often occur in the middle of the night (midnight to 8 am) and are precipitated by exercise, hyperventilation, emotional stress, cold exposure or cocaine use. The angina episodes are accompanied by transient ST elevations with return of ST segments to baseline upon resolution of symptoms. This is in contrast to the ST depressions seen in unstable angina, and the longer duration of ST elevations seen in myocardial infarction. Medical therapy for variant angina typically involves calcium channel blockers or nitrates.
-)Choice B) Nonselective 13-Biockers such as propranolol should be avoided in variant angina because 132 receptor inhibition can lead to worsened coronary vasospasm
-)Choice C) Aspirin should also be avoided in variant angina because it causes prostacyclin inhibition, which may promote coronary vasospasm.

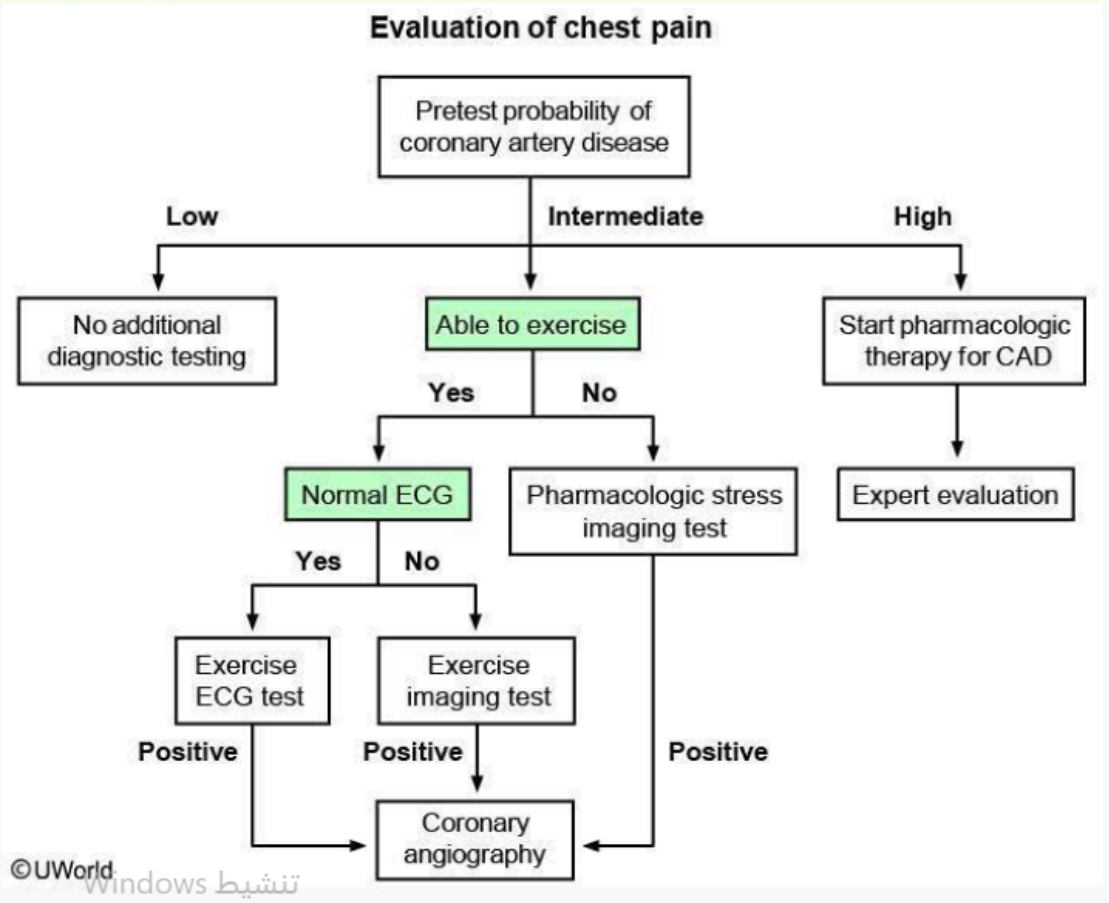
Classification of angina	
Classic	• Typical location (eg, substernal), quality (eg, pressure) & duration (eg, >20 min) • Provoked by exercise/emotional upheaval • Relieved with nitroglycerin or rest
Atypical	• 2 of 3 classic angina characteristics
Non-anginal	• 0 or 1 of 3 classic angina characteristics

Pretest probability of coronary artery disease	
Low (<10%)	• Asymptomatic people of all ages • Atypical chest pain in women age <50
Intermediate (20%-80%)	• Atypical angina in men of all ages • Atypical angina in women age ≥50 • Typical angina in women age 30-50
High (>90%)	• Typical angina in men age ≥40 • Typical angina in women age ≥60

- Nitrates exert their effect by direct vascular smooth muscle relaxation causing systemic venodilation and an increase in peripheral venous capacitance. Their primary anti-ischemic effect is mediated by systemic vasodilation and decrease in cardiac preload resulting in a decrease in left ventricular end-diastolic and end-systolic volume. This, in turn, leads to a reduction in left ventricular systolic wall stress - which reflects afterload and is proportional to (pressure • radius / thickness) - and a decrease in myocardial oxygen demand, resulting in relief of anginal symptoms.

Treatment for stable chronic angina	
Antianginal	**Beta blocker** • **1st-line therapy** for anginal symptoms, improves exercise tolerance • Relieves angina by decreasing myocardial contractility & heart rate • Improves survival in those with myocardial infarction **Calcium channel blocker** • Can combine with beta blocker if angina persists or as alternate therapy • Improves angina by causing peripheral & coronary vasodilation **Nitrates** • Short-acting form is used in the **acute** setting • Long-acting form is an add-on therapy for persistent angina
Preventive	• Aspirin • Statin • Smoking cessation • Regular exercise & weight loss • Control of blood pressure & diabetes

Medications to withhold prior to cardiac stress testing	
Hold for 48 hours	Beta blockers, calcium channel blockers, nitrates
Hold for 48 hours prior to vasodilator stress test	Dipyridamole
Hold for 12 hours prior to vasodilator stress test	Caffeine-containing food or drinks
Continue	Angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, digoxin, statins, diuretics



- Erythropoietin stimulates erythropoiesis for CKD patient's anemia but can cause serious cardiovascular events, thromboembolic events, stroke, and increased mortality in patients with cardiovascular disease.
- Cyanide toxicity can occur in patients receiving prolonged infusions or higher doses of nitroprusside, and is most common in patients with renal insufficiency. It is characterized by altered mental status, lactic acidosis, seizures, and coma.

Alcohol withdrawal syndrome		
Manifestations	**Symptoms/signs**	**Onset since last drink (hours)**
Mild withdrawal	Anxiety, insomnia, tremors, diaphoresis, palpitations, gastrointestinal upset, intact orientation	6-24
Seizures	Single or multiple generalized tonic-clonic	12-48
Alcoholic hallucinosis	Visual, auditory, or tactile; intact orientation; stable vital signs	12-48
Delirium tremens	Confusion, agitation, fever, tachycardia, hypertension, diaphoresis, hallucinations	48-96

- In this patient with no evidence of coronary artery disease on angiography, the most likely etiology is dilated cardiomyopathy due to heavy alcohol consumption (macrocytic anemia

(mean corpuscular volume >100)Jm~). thrombocytopenia, >2:1 ratio of aspartate aminotransferase to alanine aminotransferase).

- Alcoholic cardiomyopathy is a diagnosis of exclusion in patients with dilated cardiomyopathy and history of alcohol abuse in whom no other potential causes of cardiomyopathy (eg, coronary artery disease, valvular heart disease) are suspected or identified. The degree of LV dysfunction in alcoholic cardiomyopathy is directly related to the daily amount and overall duration of alcohol intake. The primary therapy for such patients is complete abstinence from alcohol use; this intervention is associated with improvement or normalization of LV function over time.

Viral myocarditis	
Clinical presentation	• Relatively young adults (eg, age <60) • Viral prodrome (eg, fever, malaise, myalgias) • Heart failure (eg, dyspnea, orthopnea, edema) • Chest pain • Sudden cardiac death
Diagnosis	• ECG: Nonspecific • Echocardiogram: 4-chamber dilation • Cardiac MRI: Enhancement of the epicardium • Biopsy: Lymphocytic infiltration, viral DNA or RNA
Treatment	• Medication (eg, diuretics, ACE inhibitor, beta blocker) • Temporary ventricular assist device, if needed • Heart transplant if no recovery

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Guidelines for lipid-lowering therapy	
Indication	**Recommended therapy**
Clinically significant atherosclerotic disease • ACS, MI • Stable or unstable angina • Coronary or other arterial revascularization • Stroke, TIA, PAD	• **Age ≤75:** High-intensity statin • **Age >75:** Moderate-intensity statin
LDL ≥190 mg/dL	**High-intensity statin**
Age 40-75 with diabetes	• **10-year ASCVD risk ≥7.5%:** High-intensity statin • **10-year ASCVD risk <7.5%:** Moderate-intensity statin
Estimated 10-year ASCVD risk ≥7.5% (Pooled Cohort Equations)	Moderate- to high-intensity statin*

ACS = Acute coronary syndrome; **ASCVD** = atherosclerotic cardiovascular disease; **MI** = myocardial infarction; **PAD** = peripheral arterial disease; **TIA** = transient ischemic attack.

* High-intensity statins include atorvastatin 40-80 mg, rosuvastatin 20-40 mg; moderate-intensity statins include atorvastatin 10-20 mg, rosuvastatin 5-10 mg, simvastatin 20-40 mg, pravastatin 40-80 mg, lovastatin 40 mg.

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Infective endocarditis in IV drug users

- HIV infection increases IE risk in intravenous drug users
- *Staphylococcus aureus* the most common organism
- Tricuspid valve involvement (right-sided) more common than aortic valve
- Holosystolic murmur increases with inspiration indicating tricuspid involvement
- Septic pulmonary emboli common
- Fewer peripheral IE manifestations (eg, splinter hemorrhages, Janeway lesions)
- Heart failure more common in aortic valve involvement but rare with tricuspid valve disease

- Augmentation of intensity with inspiration was shown to have 100% sensitivity and 88% specificity in differentiating right-sided systolic murmurs from all others.
- When aortic regurgitation) AR) is due to valvular disease, the early diastolic murmur is best heard along the left sternal border (3rd and 4th intercostal spaces). However, when the AR murmur is due to aortic root disease, it is best heard along the right sternal border.

Treatment of hypertension

Modification	Recommended plan	Approximate ↓ systolic BP (mm Hg)
Weight loss	Reduce BMI <25 kg/m^2	5-20 per 10 kg loss
DASH diet	Diet high in fruits and vegetables and low in saturated fat and total fat	8-14
Exercise	30 minutes/day for 5-6 days/week	4-9
Dietary sodium	<3 g/day	2-8
Alcohol intake	2 drinks/day in men and 1 drink/day in women	2-4

Fibromuscular dysplasia	

Clinical presentation	• 90% women (in adults)
	• Internal carotid artery stenosis
	  ○ Recurrent headache
	  ○ Pulsatile tinnitus
	  ○ Transient ischemic attack
	  ○ Stroke
	• Renal artery stenosis
	  ○ Secondary hypertension
	  ○ Flank pain

Physical examination	• Subauricular systolic bruit • Abdominal bruit
Diagnosis	• Imaging preferred (eg, duplex US, CTA, MRA) • Catheter-based arteriography
Treatment	• Antihypertensives (ACE inhibitors or ARBs 1st line) • PTA • Surgery (if PTA unsuccessful)

- Although it is considered a benign finding, the presence of first-degree AV block has been associated with a higher risk of heart failure, atrial fibrillation, and overall mortality.
- Some patients with first-degree AV block and marked prolongation of the PR interval can develop "pacemaker syndrome." This refers to an uncomfortable sensation with the awareness of heartbeat due to atrial contraction against a closed mitral valve during ventricular systole. Such symptomatic patients are potential candidates for pacemaker explantation.

Second-degree AV block: Distinguishing features of Mobitz type I & type II AV block		
	Mobitz type I	**Mobitz type II**
Level of block	Usually AV node	Below the level of AV node (eg, bundle of His)
ECG findings	Progressive prolonged PR interval leads to a nonconducted P wave **("group beating")**	PR interval remains constant with intermittent nonconducted P waves
QRS complex	Narrow	Narrow or wide
Exercise or atropine	Improves type I AV block	Worsens type II AV block
Vagal maneuvers (carotid sinus massage)	Worsens type I AV block	Paradoxically improves type II AV block
Risk of complete heart block	Low risk	Higher risk, indication for pacemaker

Exertional heat stroke	
Risk factors	• Strenuous activity during **hot & humid weather** • Dehydration • Poor acclimatization • Lack of physical fitness • Obesity • Medications: Anticholinergics, antihistamines, phenothiazines, tricyclics
Clinical manifestations	• Core temperature >40 C (104 F) immediately after **collapse AND** • Central nervous system dysfunction: Altered mental status, confusion, irritability, seizure • Additional organ or tissue damage: Renal/hepatic failure, disseminated intravascular coagulation, acute respiratory distress syndrome
Management	• Rapid cooling: Ice water immersion preferred; can consider: high-flow cool water dousing, ice/wet towel rotation, evaporative cooling • Fluid resuscitation • Electrolyte correction • Management of end-organ complications • No role for antipyretic therapy

- Non exertional (or classic) heat stroke can have similar clinical features and potential complications. It occurs in the absence of strenuous activity and typically affects elderly patients with significant underlying comorbidities that limit their ability to escape or cope with excessive heat. Management for nonexertional heat stroke emphasizes evaporative cooling (eg, spraying lukewarm water while fans blow air on the patient's skin). rather than ice-water immersion, which is associated with increased mortality in nonexertional heat stroke.

Serotonin syndrome	
Causes	• Serotonergic medications (eg, SSRI/SNRI, TCA, tramadol, linezolid) • Drug interactions: Serotonergic medication & MAOI • Intentional overdose of serotonergic medications
Clinical features	• Mental status changes (eg, anxiety, agitation, delirium) • Autonomic dysregulation (eg, diaphoresis, hypertension, tachycardia, hyperthermia, vomiting, diarrhea) • Neuromuscular hyperactivity (eg, tremor, myoclonus, hyperreflexia)
Management	• Discontinue all serotonergic medications • Supportive care, sedation with benzodiazepines • Serotonin antagonist (cyproheptadine) if supportive measures fail

Hypertrophic cardiomyopathy	
Pathophysiology	• Mutations in **sarcomere** protein genes (most common) • **Autosomal dominant** • Variable expressivity/penetrance
Clinical features	• Asymptomatic or identified by family screening • Fatigue, chest pain, palpitations, syncope • **SEM** exacerbated by dehydration/impaired LV filling
Diagnostic evaluation	• ECG: LVH, repolarization abnormalities • **TTE**: LVH, ↑ **LVOT** gradient, SAM of mitral valve • Exercise testing • Family screening
Management	• Avoidance of volume depletion • BBs/CCBs • Surgery if persistent symptoms
Complications	• **Sudden cardiac death** • **Heart failure** • Stroke

- HCM is often characterized by left ventricular hypertrophy most prominent inthe basal anterior septum.

Hypertensive complications	
Hypertensive urgency	• Severe hypertension (usually ≥180/120 mm Hg) with no symptoms or acute end-organ damage
Hypertensive emergency	Severe hypertension with acute, life-threatening, end-organ complications • **Malignant hypertension**: Severe hypertension with retinal hemorrhages, exudates, or **papilledema** • **Hypertensive encephalopathy**: Severe hypertension with cerebral edema & non-localizing neurologic symptoms & signs

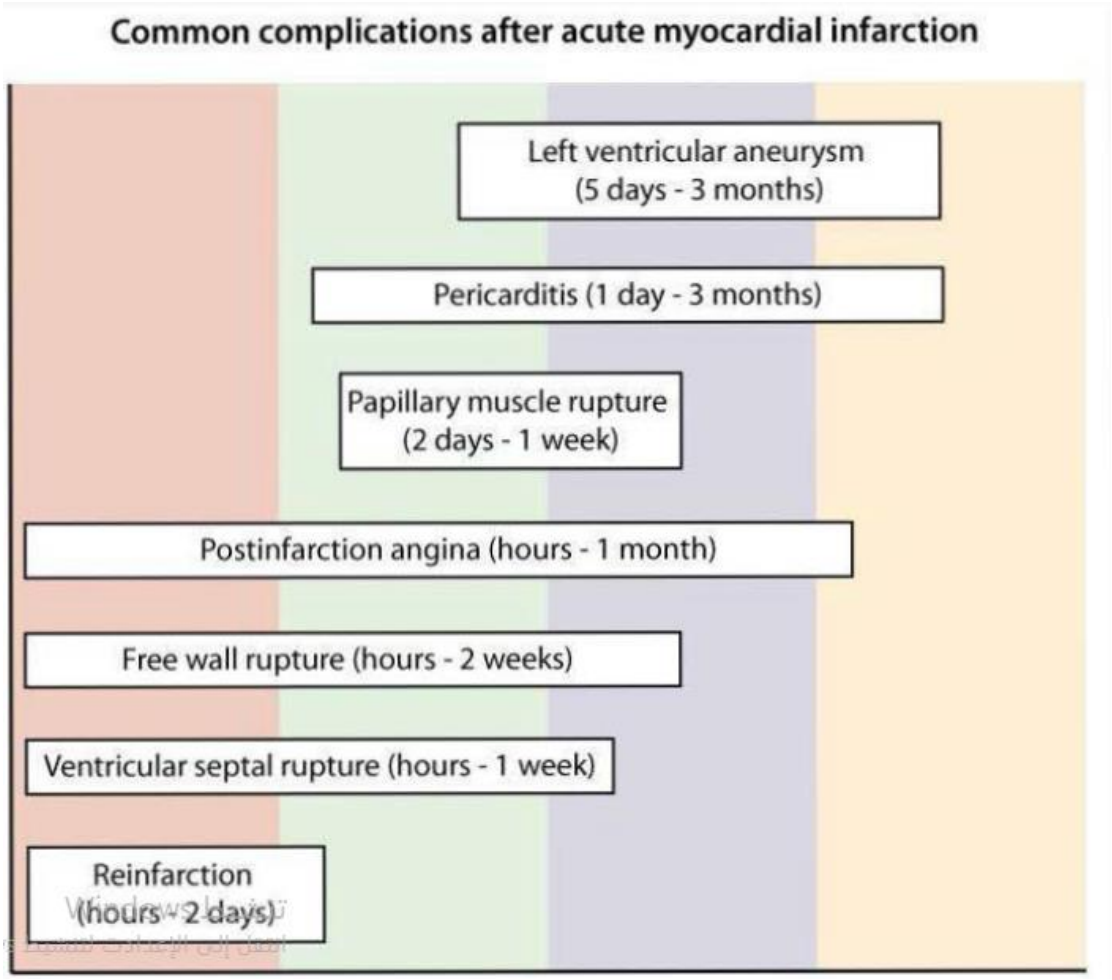
- sudden onset of chest pain, ST segment elevation, holosystolic murmur at apex, and bibasilar crackles-is consistent with acute inferior myocardial infarction (MI) with papillary muscle displacement, leading to acute mitral regurgitation (MR) and pulmonary edema. Patients with acute Ml can also develop acute MR 2 to 7 days after the infarct due to papillary muscle rupture.

Mitral stenosis	
Clinical features	• **Dyspnea**, orthopnea, PND, **hemoptysis** • Atrial fibrillation, systemic thromboembolism • Voice hoarseness from recurrent laryngeal nerve compression due to LAE (Ortner syndrome)
Physical examination	• Mitral facies (pinkish-purple patches on cheeks) • **Loud S1**, loud P2 if pulmonary hypertension • Opening snap (high-frequency early diastolic sound) • **Mid-diastolic rumble** (best heard at cardiac apex)
Diagnosis	• CXR: Pulmonary blood flow redistribution to upper lobes, dilated pulmonary vessels, LAE, flattened left heart border • ECG: **"P mitrale"** (broad & notched P waves), atrial tachyarrhythmias, RVH (tall R waves in V1 & V2) • **TTE**: MV thickening/calcification/↓ mobility, coexisting MR

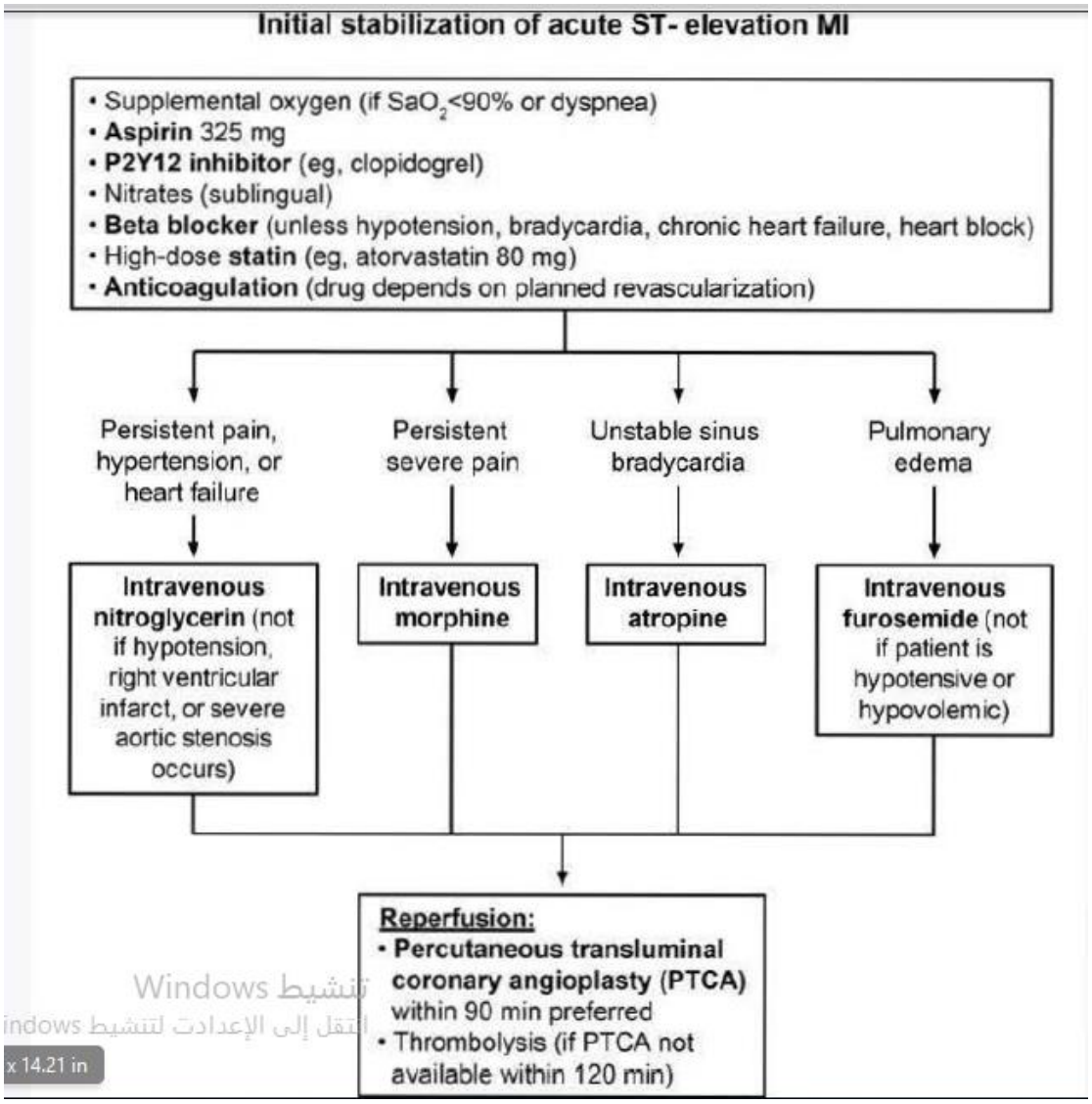
Auscultation of cardiac murmurs			
Maneuver	**What it does**	**Murmurs that get louder**	**Murmurs that get softer**
Valsalva	• ↓ Venous return (during strain; ↑ during relaxation)	• HCM • MVP	• All others
Standing	• ↓ Venous return	• HCM • MVP	• All others
Squatting	• ↑ Venous return • ↑ Afterload • ↑ Regurgitant fraction	• AR • MR • VSD	• HCM • MVP
Handgrip	• ↑ Afterload • ↑ Blood Pressure • ↑ Regurgitant fraction	• AR • MR • VSD	• HCM • AS

Mechanical complication of acute MI	Time course	Coronary artery typically involved	Clinical findings	Echocardiography
RV failure	Acute	RCA	• Hypotension & clear lungs • Kussmaul sign	• Hypokinetic RV
Papillary muscle rupture	Acute & within 3-5 days	RCA	• Acute, severe pulmonary edema • New holosystolic murmur	• Severe mitral regurgitation with flail leaflet
Interventricular septum rupture/defect	Acute & within 3-5 days	• LAD → apical septal rupture • RCA → basal septal rupture	• Shock & chest pain • New holosystolic murmur • Biventricular failure	• Left-to-right shunt at level of ventricle • Step-up oxygen level between right atrium & ventricle
Free wall rupture	Within first 5 days to 2 weeks	LAD	• Shock & chest pain • Jugular venous distension • Distant heart sounds	• Pericardial effusion with tamponade

- Acute MR in 50% is silent w/o murmur .



- Ventricular aneurysm occurs as a late complication of acuteST-segment elevation or transmural myocardial infarction. ECG often shows persistent ST-segment elevation along with deep Q waves. Progressive left ventricular enlargement can cause heart failure, refractory angina, ventricular arrhythmias, functional mitral regurgitation, or mural thrombus.
- Ventricular remodeling in the weeks to months following myocardial infarction can lead to dilatation of the ventricle. This process is lessened by ACE inhibitors .So , An ACE inhibitor should be initiated within 24 hours of myocardial infarction in all patients without a contraindication.



- Ventricular fibrillation is the most frequent underlying arrhythmia responsible for sudden cardiac arrest in the setting of acute MI;more than 50% occur within the first hour of symptom onset. Reentry is the predominant mechanism responsible for ventricular arrhythmias in the immediate post-infarction period.The underlying mechanism responsible for peri-infarction ventricular arrhythmias varies according to the time elapsed since the onset of MI. Arrhythmias occurring within 10minutes of coronary occlusion are known as "immediate" or phase 1a ventricular arrhythmias. Acute ischemia causes heterogeneity of conduction with areas of marked conduction slowing and delayed activation, which in turn predisposes to reentrant arrhythmias. On the contrary, "delayed" or phase 1b arrhythmias occur about 10-60minutes after acute infarction and are thought to result from abnormal automaticity..

- **Myocardial infarction location based on coronary vessel involvement**

Involved myocardium	Blocked vessel	ECG leads involved
Anterior MI	LAD	• Some or all of leads V1-V6
Inferior MI	RCA or LCX	• ST elevation in leads II, III & aVF
Posterior MI	LCX or RCA	• ST depression in leads V1-V3 • ST elevation in leads I & aVL (LCX) • ST depression in leads I & aVL (RCA)
Lateral MI	LCX, diagonal	• ST elevation in leads I, aVL, V5 & V6 • ST depression in leads II, III & aVF
Right ventricle MI (occurs in ½ of inferior MI)	RCA	• ST elevation in leads V4-V6R

- **Modified Wells criteria for pretest probability of PE**

Score +3 points

- Clinical signs of DVT
- Alternate diagnosis less likely than PE

Score +1.5 points

- Previous PE or DVT
- Heart rate >100
- Recent surgery or immobilization

Score +1 point

- Hemoptysis
- Cancer

Total score for clinical probability

≤ 4 = PE unlikely

> 4 = PE likely

Gallop heart sounds			
	Features	**Normal**	**Abnormal/associated conditions**
Third heart sound (S3)	• Ventricular gallop sound (after S2) • Heard during rapid filling of ventricles in diastole • Turbulent blood flow to the ventricles due to increased volume	• Children • **Young** adults • Pregnancy	• Age >40 • Heart failure • Restrictive cardiomyopathy • High-output states
Fourth heart sound (S4)	• Atrial gallop sound (before S1) • Heard immediately after atrial contraction phase as blood is forced into a stiff ventricle	• Healthy **older** adults	• Younger adults, children • Ventricular hypertrophy • Acute myocardial infarction

- Patients with myocardial ischemia/infarction canhave paradoxical splitting of 52 due to delayed myocardial relaxation and delayedclosure of the aortic valve.
- Electrical alternans is a pathognomonic ECG finding for pericardia! effusion.

Clinical features of pulmonary hypertension	
Classification	• PAH (WHO group 1) • Due to left heart disease (WHO group 2) • Due to chronic lung disease (eg, COPD, ILD) (WHO group 3) • Due to chronic thromboembolic disease (WHO group 4) • Due to other causes* (WHO group 5)
Symptoms	• Dyspnea, fatigue/weakness • Exertional angina, syncope • Abdominal distension/pain
Signs	• Left parasternal lift, prominent **right ventricular heave** • **Loud P2**, right-sided S3 • Pansystolic murmur of tricuspid regurgitation • JVD, ascites, peripheral edema, hepatomegaly

- PE CXR : Westermark's sign (peripheral hyperlucency due to oligemia), Hampton's hump (peripheral wedge of lung opacity due to pulmonary infarction), and Fleischner sign (enlarged pulmonary artery).

Clinical clues to renovascular disease	
HTN-related symptoms	• Resistant HTN (uncontrolled despite 3-drug regimen) • Malignant HTN (with end-organ damage) • Onset of severe HTN (>180/120 mm Hg) after age 55 • Severe HTN with diffuse atherosclerosis • Recurrent flash pulmonary edema with severe HTN
Supportive evidence	**Physical examination** • Asymmetric renal size (>1.5 cm) • Abdominal bruit **Laboratory results** • Unexplained rise in serum creatinine (>30%) after starting ACE inhibitors or ARBs **Imaging results** • Unexplained atrophic kidney

Classification of multiple endocrine neoplasia (MEN)	
Type I	• Primary hyperparathyroidism (>90%) • Enteropancreatic tumors (60%-70%) • Pituitary tumors (10%-20%)
Type 2A	• MTC (>90%) • Pheochromocytoma (40%-50%) • Parathyroid hyperplasia (10%-20%)

Type 2B	• MTC
	• Pheochromocytoma
	• Other
	  ◦ Mucosal & intestinal neuromas
	  ◦ Marfanoid habitus

Likely etiology	Clinical clues to diagnosis
Vasovagal or neurally mediated syncope	• Triggers: Prolonged standing or emotional distress, painful stimuli • Prodromal symptoms: Nausea, warmth, diaphoresis
Situational syncope	• Triggers: Cough, micturition, defecation
Orthostatic hypotension	• Postural changes in heart rate/blood pressure after standing suddenly
Aortic stenosis, HCM, anomalous coronary arteries	• Syncope with exertion or during exercise
Ventricular arrhythmias	• Prior history of CAD, MI, cardiomyopathy, or ↓ EF
Sick sinus syndrome, bradyarrhythmias, atrioventricular block	• Sinus pauses, ↑ PR or ↑ QRS duration
Torsades de pointes (acquired long QT syndrome)	• Hypokalemia, hypomagnesemia, medications causing ↑ QT interval
Congenital long QT syndrome	• FHx of sudden death, ↑ QT interval, syncope with triggers (exercise, startle, sleeping)

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Vasovagal syncope	
Clinical presentation	• Inciting event (eg, stress, prolonged standing) • Prodrome (eg, pallor, nausea, diaphoresis) • Consciousness regained rapidly (eg, <1 minute)
Diagnosis	• Mainly clinical diagnosis • Upright tilt-table testing in uncertain cases
Treatment	• Reassurance • Avoidance of triggers • Counterpressure techniques for recurrent episodes

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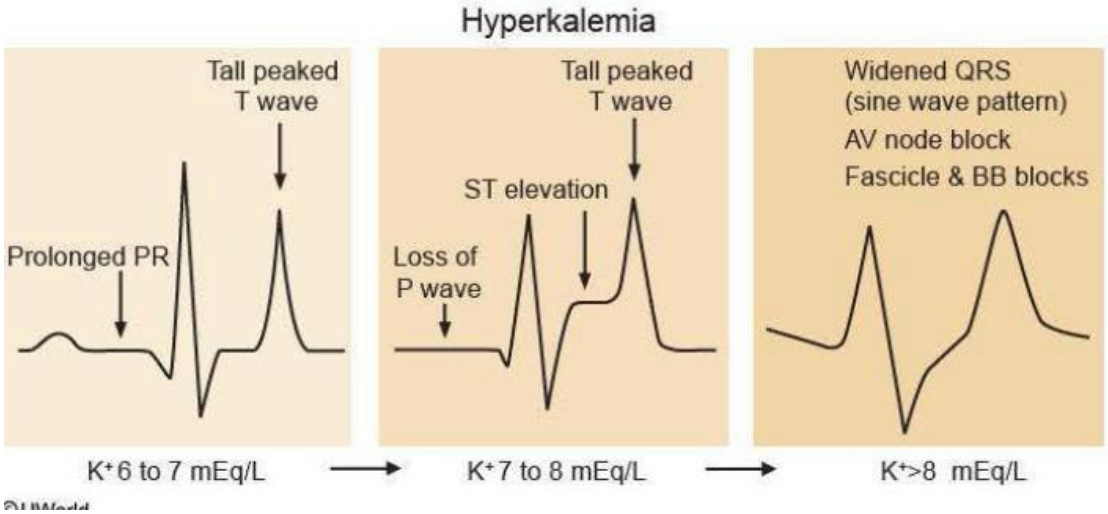
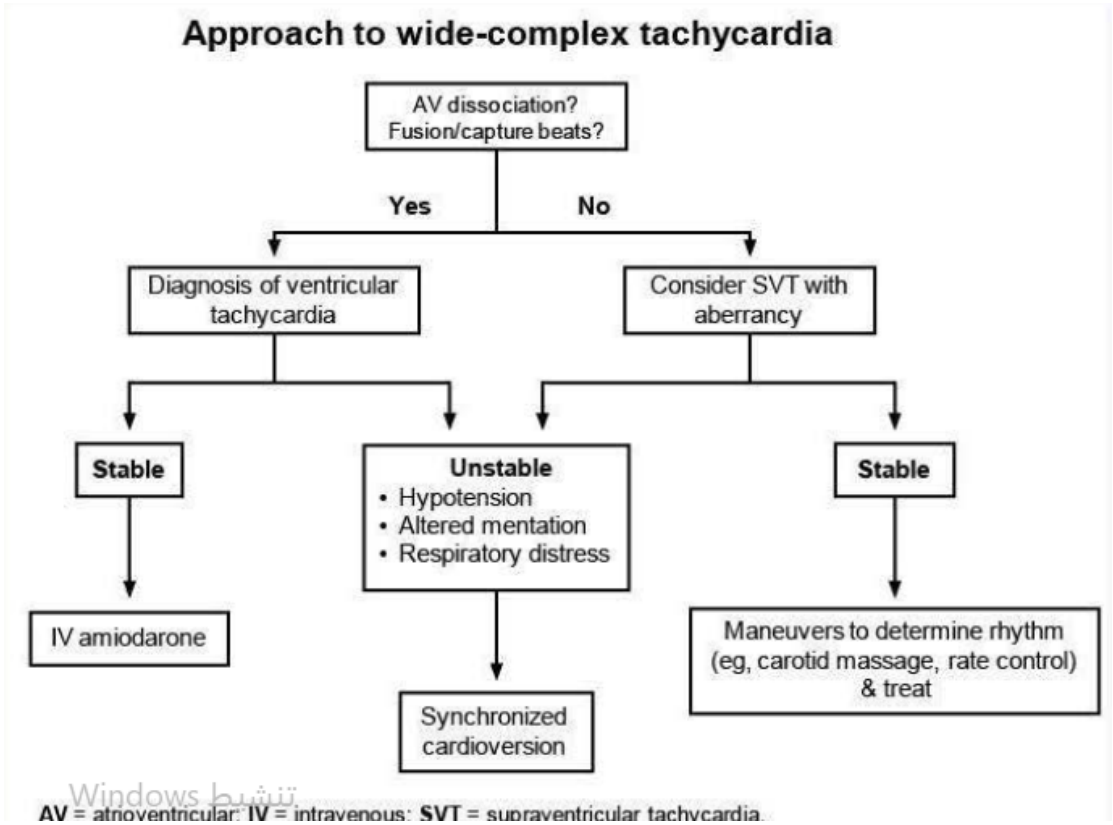
- Physical counter pressure maneuvers (eg, leg crossing with tensing of muscles, handgrip and tensing of arm muscles with clenched fists) during the prodromal phase can improve venous return and cardiac output, sometimes aborting syncopal episodes.

Factors associated with poor outcome after witnessed out-of-hospital sudden cardiac arrest
• Time elapsed prior to effective resuscitation (delayed bystander CPR, delayed defibrillation) • Initial rhythm of pulseless electrical activity or asystole • Prolonged CPR (>5 minutes) • Absence of vital signs • Advanced age • Prior history of cardiac disease • ≥2 Chronic illnesses • Persistent coma after CPR • Need for intubation or vasopressors • Pneumonia or renal failure after CPR • Sepsis, cerebrovascular accident, or class III or IV heart failure

Causes of acquired long QT syndrome	
Medications	• Diuretics (due to electrolyte imbalances) • Antiemetics (eg, ondansetron) • Antipsychotics (eg, haloperidol, quetiapine, risperidone) • Tricyclic antidepressants • Selective serotonin reuptake inhibitors (eg, citalopram) • Antiarrhythmics (eg, amiodarone, sotalol, flecainide) • Antianginal drugs (eg, ranolazine) • Anti-infective drugs (eg, macrolides, fluoroquinolones, antifungals)
Metabolic disorders	• Electrolyte imbalances (↓K, ↓Mg, ↓Ca) • Starvation • Hypothyroidism
Bradyarrhythmias	• Sinus node dysfunction • Atrioventricular block (2nd or 3rd degree)
Others	• Hypothermia • Myocardial ischemia/infarction • Intracranial disease • HIV infection

- 

- 

- AV = atrioventricular; IV = intravenous; SVT = supraventricular tachycardia.
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