Pulmonary/ Critical Care Review 2015

Brenda Shinar, MD

Question 1.

 C; FEV1/FVC 0.82 FEV1 75% FVC 68% TLC 68% RV 125%

- 63 year-old man
- DOE x months
- Minimal tobacco use
- Tachypneic
- Reduced breath sounds/insp crackles
- Normal cardiac exam
- CXR atelectasis, low volume
- FEV1/FVC ratio 82% (no obstruction)
- Low TLC and high RV (weak inspiration and expiration)

Diagnose Respiratory Muscle Weakness by Pulmonary Function Tests

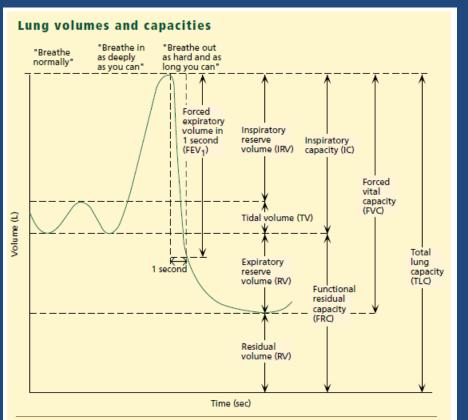
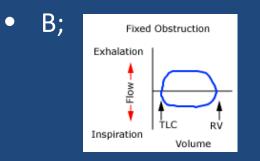


FIGURE 1. Lung volumes and capacities depicted on a volume-time spirogram. The most important values are the forced vital capacity (FVC), the forced expiratory volume in 1 second (FEV₁), and the FEV₁/FVC ratio. Spirometry cannot measure the residual volume or the total lung capacity.

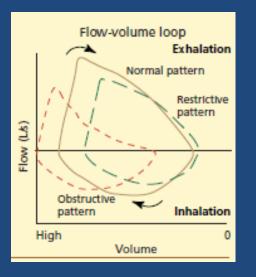
	FEV1 / FVC	TLC (= FVC + RV)	RV	DLCO
Inter- stitial diseases	≥70% predict ed	Low	Low	Low
Respira- tory muscle Weak- ness	≥ 70% predict ed	Low	High	Normal

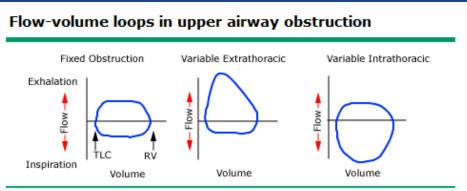
Question 2.



- 30 year-old woman
- Dyspnea x 2 weeks
- Intubated x 1 week 3 months ago
- Minimal tobacco use
- Mild intermittent asthma
- Tachypneic
- Inspiratory and expiratory wheezing
- FEV1/FVC ratio 65% (obstruction)
- FEV1 40% (severe)

Diagnose fixed airway obstruction using flow-volume loops





Left panel shows fixed upper airway obstruction with flow limitation and flattening of both the inspiratory and expiratory limbs of the flow-volume loop. Middle panel shows dynamic (or variable, nonfixed) extrathoracic obstruction with flow limitation and flattening of the inspiratory limb of the loop. Right panel shows dynamic (or variable, nonfixed) intrathoracic obstruction with flow limitation and flattening of the expiratory limb of the loop.

TLC: total lung capacity; RV: residual volume. Adapted from Stoller JK, Cleve Clin J Med 1992; 59:75.

KEY POINTS:

- Flow-volume loops plot inspiratory and expiratory flow (on the Y-axis) against volume (on the X-axis) during maximal forced inspiratory and expiratory maneuvers
- The contour of the loop helps to determine whether the obstruction is intrathoracic or extrathoracic and whether it is fixed or dynamic

Question 3.

• B; Bedside vital capacity

- 52 year-old woman
- Progressive dyspnea and weakness x 48 hrs.
- Hx of myasthenia gravis
- Medication noncompliance
- Tachypneic
- Signs of increased work of breathing/impending respiratory failure

Diagnose and monitor neuromuscular respiratory failure



MIP = Maximal Inspiratory Pressure VC = Vital Capacity (maximal amount of gas exhaled from a maximal inspiration) Normal = -70 cm H20 MIP and 70 cc/kg VC

< 20 cc/kg VC or < -30 MIP = impending respiratory failure



		MIP*	
	Children (ages 7 to 13) ^[1]	Male: 77 to 114	
		Female: 71 to 108	
	Adolescents (ages 13 to 35) ^[2]	Male: 114 to 121	
		Female: 65 to 85	
	Adults (ages 18 to 65) ^[3]	Male: 92 to 121	
		Female: 68 to 79	
	Older adults (ages 65 to 85) ^[4]	Male: 65 to 90	
		Female: 45 to 60	
* Mean values in cm H2O.			

TABLE 51.4. Respiratory Consequences of Neuromuscular Weakness			
Vital Capacity (mL/kg)	Consequences	Management	
70	Normal respiratory muscle strength	Observe	
30	Impaired cough, with difficulty clearing secretions	Chest physiotherapy	
25	Accumulation of secretions, with risk of infection and airways obstruction	Tracheal intubation	
20	Atelectasis and progressive hypoxemia	Supplemental oxygen	
10	Alveolar hypoventilation and hypercapnia	Mechanical ventilation	

Question 4.

• D; Sleep diary

- 24 year old man
- Excessive daytime sleepiness
- Erratic sleep schedule
- Normal BMI
- No upper airway signs of obstruction

Manage excessive daytime sleepiness

Distinguish between:

- Excessive Daytime Sleepiness
- Hypersomnolence
- Fatigue

4 categories:

- Insufficient sleep
- Sleep disorders
- Neurologic, psychiatric, or medical chronic conditions
- Medications

Insufficient sleep

Sleep deprivation

Environmental intrusions

Sleep disorders

Obstructive sleep apnea

Central sleep apnea

Sleep related hypoventilation or hypoxemia

Central disorders of hypersomnolence

Narcolepsy type 1 or 2

Kleine-Levin syndrome

Idiopathic hypersomnia

Circadian rhythm sleep-wake disorders

Delayed sleep phase disorder

Advanced sleep phase disorder

Jet lag

Shift work

Restless legs syndrome

Periodic limb movement disorder

Key questions in evaluating the tired patient

Que	estions about sleepiness
Do	o you feel sleepy during the day?
Is	daytime sleepiness a problem for you?
Is	it difficult to keep your eyes open at times during the day?
Do	you struggle to stay awake during the day?
Do	o you take naps?
Но	w often and how long do you nap during the day?
Do	you fall asleep at times you do not want to (ie, watching a movie, reading a book, or on long drives)?
Que	estions about tiredness, fatigue, and low energy
Do	you lack the energy to go about your daily activities?
Do	you tire easily, or sooner than others, when you are active?
Do	you feel physically or mentally exhausted?
Que	estions to differentiate sleepiness from related complaints
Do	es your problem bother you more if you sit to read for an hour, or if you go out shopping for an hour?
W	hich of the following is the single most important problem for you: sleepiness, tiredness, fatigue, or lack of energy?
W	hich of the following most interferes with your ability to accomplish what you would like to: sleepiness, tiredness, fatigue, or lack of energy
w	hich of the following is the one problem you would most like to address effectively: sleepiness, tiredness, fatigue, or lack of energy?

Adapted from:

- 1. Bodkin CL, Manchanda S. Office evaluation of the "tired" or "sleepy" patient. Semin Neurol 2011; 31:42.
- 2. Chervin RD. Sleepiness, fatigue, tiredness, and lack of energy in obstructive sleep apnea. Chest 2000; 118:372.

Multiple sleep latency test: Narcolepsy or central somnolence
 Polysomnography: Obstructive sleep apnea, limb movement disorders, narcolepsy, insomnia

3) Sleep diary: Sleep deprivation (8 hours per night recommended)

Question 5.

B; Perform a hypoxia altitude stimulation test



- 72 year-old man
- Severe COPD and systolic heart failure
- 91% saturation on RA
- Pa02 68 mm Hg on RA
- Anticipate commercial flight

Manage air travel in a patient with chronic obstructive lung disease

 The FAA requires commercial airlines cabins to be pressurized to 8,000 ft. with transient decreases in pressurization to 10,000 ft in circumstances to avoid weather.



Hypoxia Altitude Stimulation Test:

- Artificially reduces inspired oxygen to levels at 8000 feet (FI02 to 15%) and has patient breath this for 20 minutes
- Decreases barometric pressure to 565 Torr in a hypobaric chamber
 - Pa02 < 50 or < 55 requires 02 prescription for flight
 - Who to screen?

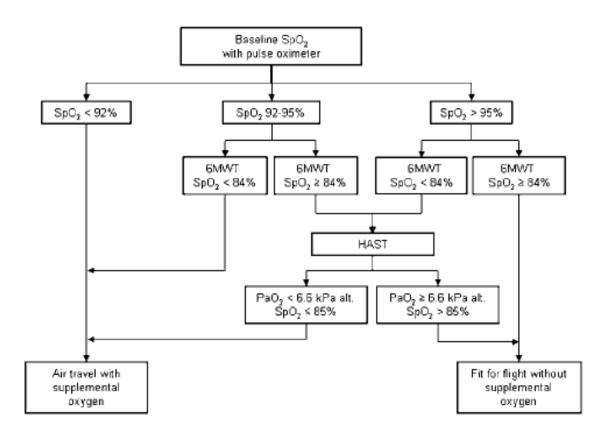


Figure 4 Pre-flight evaluation algorithm. 6MWT, 6 min walk test; alt, alternatively; HAST, hypoxia-altitude simulation test; PaO₂, arterial oxygen pressure; SpO₂, arterial oxygen saturation measured with pulse oximetry.

ORIGINAL ARTICLE

Air travel and chronic obstructive pulmonary disease: a new algorithm for pre-flight evaluation

Anne Edvardsen,^{1,3} Aina Akerø,² Carl C Christensen,¹ Morten Ryg,¹ Ole H Skjønsberg^{2,3}

Question 6.

• D; Tiotropium inhaler

- 56 year-old man
- New dx COPD, moderate FEV1 58%
- Stopped smoking 1 week ago
- Started short-acting bronchodilator and vaccinated
- Morning productive cough, dyspnea with mod exertion (MMRC 3), prolonged expiration

Spirometry

GOLD Stage	FEV1/FVC	FEV1
Stage 1	< 0.70	> 80% predicted
Stage 2	< 0.70	>50% <80% of predicted
Stage 3	< 0.70	>30% and <50% of predicted
Stage 4	< 0.70	< 30% of predicted

MODIFIED MEDICAL RESEARCH COUNCIL DYSPNEA SCALE

Please choose the one best response to describe your shortness of breath.

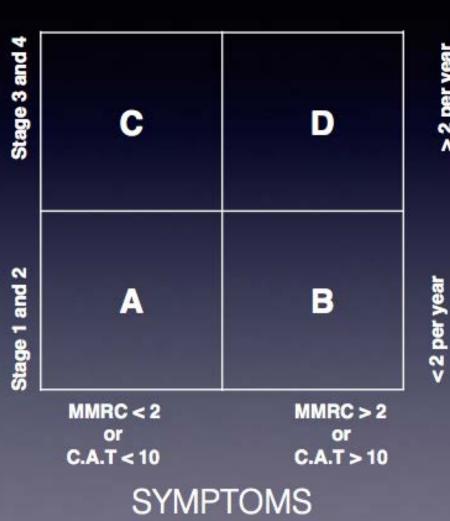
Grade

- 0 "I only get breathless with strenuous exercise"
- 1 "I get short of breath when hurrying on the level or walking up a slight hill"
- 2 "I walk slower than people of the same age on the level because of breathlessness or have to stop for breath when walking at my own pace on the level"
- 3 "I stop for breath after walking about 100 yards or after a few minutes on the level"
- 4 "I am too breathless to leave the house" or "I am breathless when dressing"

1. Grade

Exacerbations

> 2 per year or >1 requiring hospitalization



SPIROMETRY

GOLD Stage C

GOLD Stage D

Inhaled corticosteroid + long-acting beta2-agonist or Long-acting anticholinergic Supplemental Oxygen	Inhaled corticosteroid + long-acting beta2-agonist and/or Long-acting anticholinergic Supplemental Oxygen
GOLD Stage A	GOLD Stage B
Short-acting anticholinergic or	Long-acting anticholinergic or

Question 7.

• D; Noninvasive positive pressure ventilation

- 66 year-old man
- Severe COPD exacerbation s/p intubation, ready for extubation
- Baseline CO2 retainer (pC02 55, pH 7.36)

Manage weaning a patient from invasive to non-invasive ventilation

Evidence for post-extubation NPPV:

- 164 patients at risk for post-extubation respiratory failure
 - Age ≥ 65
 - APACHE II Score > 12
 - Intubated for CHF
 - Hypercarbia on spontaneous breathing trial
- 106 Randomized to conventional medical therapy with or without NPPV for 24 hours immediately following extubation.
- NPPV was effective in reducing the reintubation rate from 48% (25) to 15% (8) and the **90 day mortality** was significantly lower in the NPPV group: 11% (6) vs. 31% (16).
- Length of stay in the ICU and mortality during the hospital stay did not differ between the groups.

 NPPV should be started immediately after extubation and should not be delayed until patient fails in patients with PaC02 > 45 mm Hg during a spontaneous breathing trial (most of whom will have chronic lung disease)

Lancet. 2009;374(9695): 1082-1088

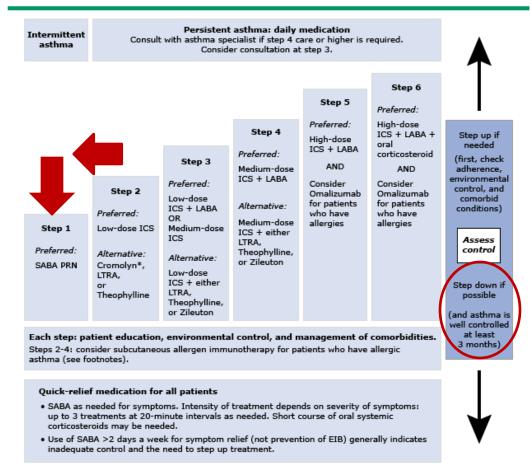
Question 8.

• B; Discontinue inhaled corticosteroids

- 28 year-old woman
- Hoarseness
- Asthma, well controlled on daily low dose inhaled corticosteroid and prn B2 agonist
- Last exacerbation > 1 year ago
- Thrush
- Normal spirometry

Manage asthma with step-down therapy

Stepwise approach for managing asthma in youths greater than or equal to 12 years of age and adults



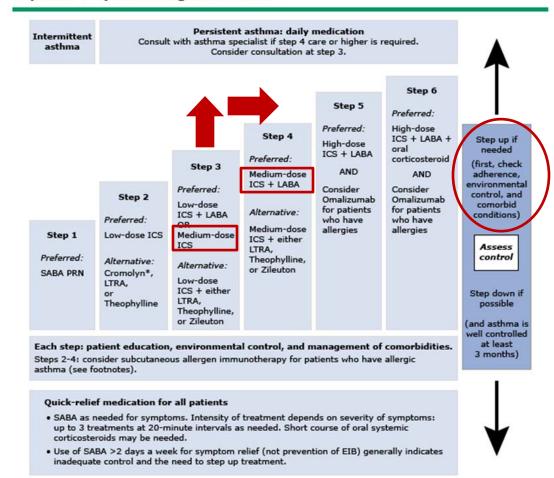
Question 9.

• A; Add a long-acting B2 agonist inhaler

- 55 year-old woman
- Asthma exacerbation
- No triggers to modify (post-nasal drip, heartburn, NSAIDS)
- No fever or pneumonia on exam

Manage asthma with step-up therapy

Stepwise approach for managing asthma in youths greater than or equal to 12 years of age and adults



Inhaled Steroids Approximate Dose Conversions for Adults

Inhaled Steroid	Low dose	Medium Dose	High dose
Qvar 40, Qvar 80 (Beclomethasone HFA) 40 mcg or 80 mcg/puff	80-240 mcg Qvar 40 1-2 puff BID or Qvar 80 1 puff BID	240-480 mcg Qvar 80 2 puffs BID	>480 mcg
Vanceril , Vanceril DS (Beclomethasone) 42 or 84 mcg per puff	168-504 mcg Vanceril DS 1-2 puffs BID to TID	504-840 mcg	>840 mcg
Pulmicort (Budesonide) 200 mcg/inhalation	200-400 mcg Pulmicort 1 inhalation once daily	400-600 mcg Pulmicort 1 inhalation BID	>600 mcg Pulmicort 2 inhalations BID
Aerobid (Flunisolide) 250 mcg/puff	500 -1000 mcg Aerobid 1 to 2 puffs BID	1000 -2000 mcg	>2000 mcg Aerobid 4-5 puffs BID
Flovent HFA (Fluticasone) 44, 110 or 220 mcg	88-264 mcg Flovent 44 mcg 1- 2 puff BID or Flovent 110 one puff BID	264-660 mcg Flovent 110 mcg 2 puff BID to 3 puffs BID	>660 mcg Flovent 220 mcg 2 puff BID
Advair (Fluticasone/ Salmeterol) 100/50, 250/50 or 500/50 mcg/inhalation	100-300 mcg Advair 100/50 one inhalation daily to BID	300-600 mcg Flovent 250/50 one inhalation BID	>600 mcg Flovent 500/50 mcg one inhalation BID
Asmanex (Mometasone Furoate) 220 mcg/inhalation	220 - 440 mcg Asmanex 1-2 inhalations daily in evening	440-660 mcg Asmanex 2-3 inhalations daily	>660 mcg Asmanex 2 inhalations twice daily
Azmacort (Triamcinolone) 100 mcg/puff	400-1000 mcg Azmacort 2 puff BID to QID	1000-2000 mcg	>2000 mcg

Question 10.

• A; Alpha-1 antitrypsin level measurement

Problem List:

- 38 year old man (young!)
- Productive cough x 1 yr
- DOE x 6 months, progressive
- Minimal history of smoking
- Decreased breath sounds bilaterally
- CT bibasilar lucency
- FEV1/FVC 64% (obstruction)
- FEV1 53% (GOLD 2); no bronchodilator response
- DLCO low

Diagnose Alpha-1 Antitrypsin Deficiency

Characteristics of alpha-1 antitrypsin deficiency phenotypes

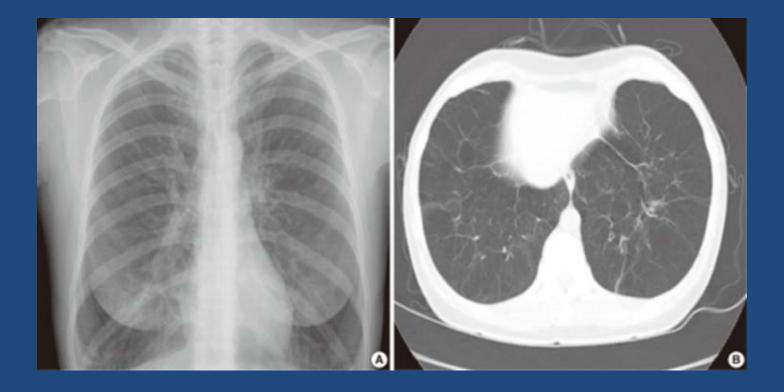
Phenotype	Risk for emphysema	True plasma level, micromol/L (SI units)	Commercial standard plasma level, mg/dL
MM (normal)	No increase	20 to 48	80 to 220
MZ	Possible mild increase	17 to 33	90 to 210
SS	No increase	15 to 33	100 to 200
SZ*	Mild increase (20 to 50 percent)	8 to 16	75 to 120 •
ZZ	High risk (80 to 100 percent)	2.5 to 7	20 to 45
Null	High risk (100 percent by age 30)	0	0

Pulmonary and plasma features of the different phenotypes of alpha-1 antitrypsin deficiency. Standard commercial measurements of AAT serum levels are obtained by nephelometry.

* Heterozygotes with the SZ phenotype rarely have evidence of clinical pulmonary disease.

• Protective threshold of 11 micromol/L is approximately equal to a commercial standard level of 80 mg/dL by immunodiffusion (older assay) or ~57 mg/dL by nephelometry.

Adapted from: American Thoracic Society/European Respiratory Society statement: standards for the diagnosis and management of individuals with alpha-1 antitrypsin deficiency. Am J Respir Crit Care Med 2003; 168:818.



Indications of severe alpha-1 antitrypsin deficiency

- Emphysema in a young individual (ie, less than or equal to 45 years)
- Emphysema in a nonsmoker or minimal smoker

Emphysema characterized by predominant basilar changes on the chest x-ray

A family history of emphysema and/or liver disease, especially unexplained cirrhosis or hepatoma

Clinical findings or history of panniculitis

Clinical findings or history of unexplained chronic liver disease

- Three subtypes of emphysema are described:
- A. centrilobular emphysema (CLE)
- B. paraseptalemphysema (PSE), and
- C. panlobular emphysema (PLE) = AAT deficiency= LOWER LOBE

The pulmonary lobule is more or less uniformly destroyed from the respiratory bronchiole to the terminal distal alveoli.

Question 11.

• D; Restart anticoagulation

PROBLEM LIST:

- 45 year-old man
- Unprovoked PE
- S/P 3 months anticoagulation, doing well off AC x 1 month
- Elevated D-dimer

Risk of VTE recurrence after discontinuation of anticoagulation:

- 1) First VTE provoked by surgery
 - 1% for the first year
 - 0.5 percent/year thereafter
- 2) First VTE provoked by non-surgical risk factor
 - 5% for the first year
 - 2.5% /year thereafter
- 3) First episode of unprovoked VTE
 - 10% for the first year
 - 5%/ year thereafter
- 4) Second episode of unprovoked VTE
 - 15 % for the first year
 - 7.5% /year thereafter

Assess and manage risk for recurrent pulmonary embolism

HAS-BLED Score for Major Bleedin Estimates risk of major bleeding for patients on antico	
Hypertension History	+1 NO
(Uncontrolled, >160 mmHg systolic)	
Renal Disease	+1 NO
Dialysis, transplant, Cr>2.6 mg/dLor>200 µmol/L	
Liver Disease	+1 💷 NO
Cirrhosis or Bilirubin >2x Normal or AST/ALT/AP >3x Normal	
Stroke History	+1 III NO
Prior Major Bleeding or Predisposition to Bleeding	+1 NO
Labile INR	+1 III NO
(Unstable/high INRs), Time in Therapeutic Range < 60%	
Age>65	+1 III NO
Medication Usage Predisposing to Bleeding	+1 NO
(Antiplatelet agents, NSAIDs)	
Alcohol or Drug Usage History	+1 III NO
≥8 drinks/week	



- Weighing risk of bleeding vs. benefit of anticoagulation to determine how long to treat is important.
- HASBLED score ≥ 3 is considered too high risk for anticoagulation
- Aspirin 100 mg/day decreases risk by 30%

Question 12.

• D; Unfractionated heparin

- 62 year-old woman
- Dyspnea and chest pain following prolonged travel
- Hypotension responding to fluids
- Hypoxemia responding to oxygen therapy
- CT angiography with multiple PEs
- Echocardiogram with right ventricle dilation

Management of Massive versus Submassive PE

Which patients with acute pulmonary embolism should I treat with systemic thrombolytics?

- The ACCP suggests using systemic thrombolytics to treat patients with acute PE who are hypotensive (they propose a cutoff of systolic blood pressure less than 90 mm Hg). (Grade 2C).
- ACCP recommends **against** treating most patients with acute PE without hypotension with systemic thrombolytics (Grade 1C).
- However, patients deemed to be at high risk for becoming hypotensive according to clinical course are suggested to receive systemic thrombolytics, if they have a low bleeding risk (Grade 2C). "Looking sick," dyspneic and hypoxic, right ventricular dysfunction on echocardiogram, elevated troponins, elevated neck veins, severe tachycardia have all been proposed as risk factors.

How should I treat acute pulmonary embolism with systemic thrombolytics?

- A short infusion time of 2 hours for systemic thrombolytics is suggested, rather than a longer infusion (Grade 2C). Tissue plasminogen activator (tPA) has a short infusion time and has been recommended as the best agent for this reason.
- Infuse systemic thrombolytics through a peripheral vein, rather than a pulmonary artery catheter (Grade 2C).

Massive PE:

- sustained hypotension (systolic blood pressure <90 mm Hg for at least 15 minutes) OR
- requiring inotropic support AND
- not due to a cause other than PE.

Submassive PE:

- without systemic hypotension (systolic blood pressure ≥90 mm Hg) but
- *with* either RV dysfunction OR
- myocardial necrosis (trop I >0.4 ng/mL)

RV dysfunction:

- RV dilation or RV systolic dysfunction on echocardiography or CT
- Elevation of N-terminal pro-BNP >500 pg/mL or BNP > 90 pg/mL
- EKG changes
 - New complete or incomplete RBBB
 - Anteroseptal ST elevation or depression
 - Anteroseptal T wave inversion

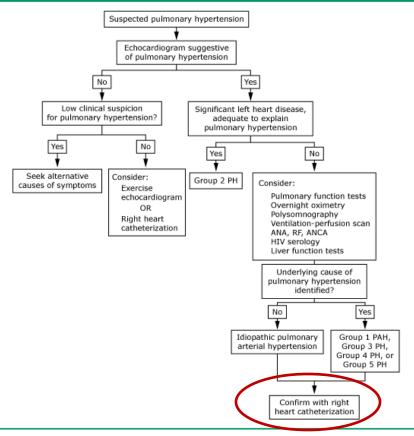
Question 13.

• C; Right heart catheterization

- 33 year-old woman
- DOE x 2 years
- Distended neck veins
- Prominent pulmonic component of S2
- Clear lungs
- Edema bilateral lower extremities
- CXR: prominent central pulmonary arteries
- Normal PFTs x low DLCO
- EKG: Right axis deviation
- Echo: dilated RV
- VQ scan: normal

Diagnose pulmonary arterial hypertension

Algorithm for investigation of suspected pulmonary hypertension



PH: pulmonary hypertension; PAH: pulmonary arterial hypertension; ANA: anit-nuclear antibody; RF: rheumatoid factor; ANCA: anti-neutrophil cytoplasmic antibody.

WHO Classification of PAH:

- 1) Idipathic
 - Hereditary
 - Toxin/drug associated
 - Connective-tissue dz related
 - HIV
 - Portal hypertension related
 - Congenital heart related
 - Schistosomiasis
 - Chronic hemolytic anemia associated
- 2) Left heart-related
- 3) Lung dz/hypoxemia related
- 4) Chronic thromboembolic
- 5) Miscellaneous

Question 14.

C; Perform high-resolution CT imaging

- 78 year-old man
- Dyspnea and dry cough x 3 months
- Afib with RVR, newly started amiodarone 4 months ago
- No JVD, normal cardiac exam
- Crackles bilaterally
- FEV1/FVC 78% (no obstruction)
- TLC 65% (low) and DLCO 50% (low) (Restrictive)

Diagnose amiodarone pulmonary toxicity

Amiodarone side effects :

- Photosensitivity
- Blue-gray skin discoloration
- Thyroid dysfunction
- Corneal deposits
- Abnormal LFTs
- Bone Marrow Suppression
- Pulmonary toxicity (5%)*

Pulmonary toxicity due to amiodarone (foamy macrophages):

- Interstitial pneumonitis
 - Most common, usually a couple months at > 400 mg/day
- Organizing pneumonia
- Acute respiratory distress syndrome (ARDS)
- Diffuse alveolar hemorrhage
- Pulmonary mass







Bronchoalveolar lavage (BAL) cytopreparation smear with normal differential and marked foamy appearance of alveolar macrophages. *Courtesy of Talmadge E King Jr, MD.*

Question 15.

• A; Obtain detailed history of current work exposures

- 28 year old woman
- Cough, SOB, low grade fevers x 12 weeks
- Failed outpatient respiratory fluoroquinolone therapy
- No travel or animal exposures
- No tobacco use
- Sheet metal worker
- Normal exam
- Diffuse bilateral opacities on CXR
- CT diffuse centrilobular ground glass opacity

Diagnose occupational lung disease

Occupational Lung Dz:

- 1. Occupational asthma
- 2. Diffuse Parenchymal
 - Lung Disease
 - Pneumoconiosis (inorganic)
 - Coal, asbestos, silica
 - Hypersensitivity Pneumonitis (organic)
 - Fungus, plant, animal proteins
- 3. Acute Toxic Inhalant syndrome

Box 2 Industries that Use Beryllium Industries that Use Beryllium Heavily Beryllium and beryllium alloy machine shops Beryllium extraction Electronics Nuclear weapons manufacturing Industries that Use Beryllium Less Heavily Aerospace Automotive Ceramics Dental appliances

2-minute Occupational History:

- 1. What kind of work do you do? Please be as specific as possible and tell me exactly what you do at work.
- 2. Do you think your medical problems are related to your work?
- 3. Do your symptoms get better when you are away from work, such as on weekends or vacation?
- 4. Are you now, or have you ever been, exposed to fumes, dusts, or gases?

Question 16.

• D; Tuberculosis testing

- 70 year-old man
- Cough, night sweats, weight loss x 3 months
- Pulmonary silicosis x 15 years
- Lifelong nonsmoker
- PFTs no change
- CXR with small upperlobe predominant lung nodules, no change

Evaluate for tuberculosis in a patient with pulmonary silicosis

Silica dust and MTB:

- Exposure to silica dust increases the risk of development of pulmonary TB by 2.9x-39x in the absence of silicosis, even after the exposure to dust ends
- The risk of TB increases with the radiologic presence of silicosis, with increasing amounts of dust exposure, and with tobacco pack-years.
- TB was diagnosed an average of 7.6 years after the end of exposure to dust (age 60)
- Silicosis preceded dx of TB in 90% of cases

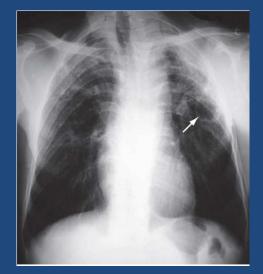




Figure 2 – Tomography scan of the chest of the same patient. Note the thick-walled cavity with an irregular surface in the left lower lobe (black arrow), as well as multiple, diffusely distributed nodules, predominantly in the left lung (white arrowheads).

Question 17.

• C; Diurese

- 67 year-old man
- Pauses in breathing during sleep
- Minimal snore
- No insomnia or daytime sleepiness
- Heart failure history
- BMI 24
- Widely patent airway
- Cheyne-Stokes breathing

Treat central sleep apnea in a patient with heart failure

The incidence, pathophysiology, treatment and prognosis of Cheyne-Stokes breathing disorder in patients with congestive heart failure.

Ingbir M, Freimark D, Motro M, Adler Y.

Cardiac Rehabilitation Institute and Heart Failure Unit, Sheba Medical Center, Tel Hashomer, Israel.

Abstract

DEFINITION: Cheyne-Stokes respiration is a breathing disorder characterized by recurrent central sleep apneas, mainly during sleep, alternating with a crescendo-decrescendo pattern of tidal volume.

PATHOPHYSIOLOGY AND PROGNOSIS: The pathophysiology of Cheyne-Stokes respiration, involving the cardiovascular, pulmonary and sympathetic nervous systems, is still not well understood. Although 50% of moderate to severe congestive heart failure patients suffer from significant Cheyne-Stokes respiration, studies been undertaken to determine the prevalence of this phenomenon and its implications regarding patients' life expectancy and quality of life were conducted only in recent years. Other studies suggest that Cheyne-Stokes respiration has a negative prognostic value upon congestive heart failure patients.

TREATMENT: Novel therapeutic approaches have been attempted in order to treat Cheyne-Stokes respiration; they include oxygen delivery, various pharmaceutical treatments aimed to stabilize the ventilatory system and other pharmaceutical treatments aimed to improve the left ventricular ejection fraction. However, none of them was effective.

OBJECTIVES: This review summarizes some of the current knowledge regarding Cheyne-Stokes respiration pathophysiology, prevalence, prognostic implication and available treatments.

Normal respiration

Adaptive Servo-Ventilation (ASV) Time (s Biot's respiration What is adaptive servo-ventilation (ASV)? aka ataxic respiration - Periodic breathing: -ASV is an exciting breakthrough created by the ResMed Company specifically for the treatment of central as well as obstructive hyperphoea (or normophoea) and aphoes Poor prognosis Neuron damage apneas. How does ASV work? Time (s) -ASV is a new form of positive airway pressure unit that continuously monitors the patient's breathing pattern in exquisite detail. Kussmaul breathing -Whenever it detects significant reductions or pauses in Metabolic addosis (Diabetes breathing, it intervenes with just enough support to maintain the patient's breathing at 90% of what had been normal for that Ketones (Diabetic ketoacidosi individual just prior to the decrease in breathing. Uremia S = Sepsis 5 = Salkylater -Then, when the patient's breathing problem ends, the machine M = Methanol "backs out" gently. A = Aldehydes (U) L = Lactic acid/Lactic acidosis Also, when the patient's breathing is stable, ASV provides just enough pressure support to help maintain airway patency; thereby providing an approximate 50% reduction in the work of breathing. The machine is subtle in its interventions...and it continuously Chevne-Stokes respiration adjusts itself to meet the patient's needs in a manner that will Periodic breathing: Gradual hyperphoea/hypophoea and Aphoea feel normal for that patient at that point in time: which renders it Sie en Hypoxemia Orug comfortable. ypoperfusion of the brain (respiratory cent ASV is the ultimate "smart machine"

Question 18.

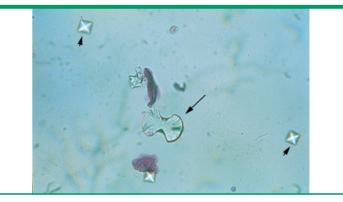
• D; Intravenous fomepizole and hemodialysis

- 55 year-old man
- Altered MS with rapid shallow breathing
- GCS: 7
- Anion gap metabolic acidosis
- Osmolar gap metabolic acidosis
- Elevated creatinine and lactic acid
- Urine with envelope-shaped crystals

Manage ethylene glycol ingestion

Found down: suspicious for toxic ingestion! Simple metabolic acidosis on ABG Anion gap metabolic acidosis= 36 Osmolal gap= 105 **Elevated creatinine** Elevated lactate (don't stop looking)

Calcium oxalate crystals in the urine



Urine sediment showing both dumbbell-shaped calcium oxalate monohydrate (long arrow) and envelope-shaped calcium oxalate dihydrate (short arrows) crystals. Although not shown, the monohydrate crystals may also have a needle-shaped appearance. The formation of calcium oxalate crystals is independent of the urine pH.

Courtesy of Frances Andrus, BA, Victoria Hospital, London, Ontario.

Differential diagnosis of an elevated plasma osmolal gap

With anion gap metabolic acidosis

Ethylene alvcol indestion

Methanol indestion

End-stage chronic kidney disease (GFR <10 mL/min) without regular dialysis

Diabetic ketoacidosis

Alcoholic ketoacidosis

Lactic acidosis

Formaldehyde ingestion

Paraldehyde ingestion

Without metabolic acidosis

Ethanol or isopropyl alcohol ingestion

Diethyl ether indestion

Infusion of nonconductive glycine, sorbitol, or mannitol solutions

Severe hyperproteinemia

Severe hyperlipidemia

Treatment for Ethylene Glycol Poisoning

Indications for antidotal therapy with fomepizole or ethanol

Documented serum methanol or ethylene glycol concentration >20 mg/dL (methanol SI equivalent 6.2 mmol/L; ethylene glycol SI equivalent 3.2 mmol/L)

Documented recent history of ingesting toxic amounts of methanol or ethylene glycol and serum osmol gap >10

OR

Strong clinical suspicion of methanol or ethylene glycol poisoning and at least two of the following:

- a) Arterial pH <7.3
- b) Serum bicarbonate <20 meg/L (mmol/L)

c) Osmol gap >10

d) Urinary oxalate crystals present

- We recommend immediate hemodialysis be performed in the setting of a known methanol or ethylene glycol ingestion when the following conditions are
 present (Grade 1B):
 - · Metabolic acidosis, regardless of drug level
 - Elevated serum levels of methanol or ethylene glycol (more than 50 mg/dL [SI equivalent: methanol 15.6 mmol/L; ethylene glycol 8.1 mmol/L]), unless arterial pH is above 7.3
 - · Evidence of end-organ damage (eg, visual changes, renal failure)

We recommend treatment with hemodialysis if the clinician suspects a toxic alcohol ingestion in a patient with a severe, unexplained anion gap metabolic acidosis and significant osmolal gap (Grade 1B). We generally refrain from using hemodialysis to treat patients with elevated ethylene glycol concentrations, provided their serum pH is near normal, their renal function is normal, and <u>fomepizole</u> is given. (See <u>'Hemodialysis'</u> above.)

Question 19.

B; Intravenous fluid bolus

- 74 year-old woman
- Sepsis due to UTI
- Hypotensive
- Tachycardic
- Tachypneic
- No urine output x 6 hours

Manage shock in a hospitalized patient

4 Types of Shock:

- 1. Hypovolemic
 - External or internal bleeding
- 2. Cardiogenic
 - Tachyarrhythmia, Bradyarrhythmia, AMI, Acute valvular problem
- 3. Distributive
 - Septic, Anaphylactic, Acute neurogenic, Acute adrenal crisis
- 4. Obstructive
 - Tamponade, Tension pneumothorax, Atrial myxoma, Pulmonary embolus

Restore perfusion:

- Fluids, 500 cc boluses (30 cc/kg)
 - Continue until BP acceptable, tissue perfusion acceptable, CHF, or failure to augment perfusion
- Vasopressors
- Inotropes
- RBC transfusion

Steps in Management:

- Assess/Stabilize respiration
- Assess Perfusion
 - Signs of inadequate perfusion
 - SBP < 90 or \downarrow by 40 mm Hg
 - MAP < 70 mm Hg
 - Tachycardia > 90 bpm
 - Cool, vasoconstricted skin
 - Obtunded/restless
 - Oliguria/Anuria
 - Lactate > 4 mmol/L
- Central venous access
- Early goal directed therapy:
 - CVP 8-12
 - MAP ≥65 mm Hg
 - − Urine output \geq 0.5 cc/kg/hr
 - SCV02 ≥ 70%

Question 20.

• C; Norepinephrine

- 78 year-old woman with Alzheimer's dementia
- Septic, altered, in the ICU, presumed urinary source
- Antibiotics, 30 cc/kg fluid bolus given
- Remains hypotensive with HR 100 and lethargic

Manage septic shock

Steps in Management:

- Assess/Stabilize respiration
- Assess Perfusion
 - Signs of inadequate perfusion
 - SBP < 90 or \downarrow by 40 mm Hg
 - MAP < 70 mm Hg
 - Tachycardia > 90 bpm
 - Cool, vasoconstricted skin
 - Obtunded/restless
 - Oliguria/Anuria
 - Lactate > 4 mmol/L
- Central venous access
- Early goal directed therapy:
 - CVP 8-12
 - MAP ≥65 mm Hg
 - − Urine output \geq 0.5 cc/kg/hr
 - SCV02 ≥ 70%

Restore perfusion:

- Fluids, 500 cc boluses, 30 cc/kg
 - Continue until BP acceptable, tissue perfusion acceptable, CHF, or failure to augment perfusion
- Vasopressors: Norepinephrine preferred agent
- Inotropes
- RBC transfusion

Treat the source of infection:

- Identify the septic focus
- Broad spectrum antibiotics (after cultures if possible)
- Surgery necessary?