胸部與呼吸系統常見問題之評估

國軍左營分院 胸腔內科主治醫師 翁銘偉

健康評估的資料來源

- ⇒臨床觀察(clinical observation)
- ⇒理學檢查(physical examination)
- ⇒病患主訴(patient's complaints)
- ⇒實驗檢查值(Lab. Data)
- ⇒目前接受之治療(current treatment)
- ⇒醫療同仁提供資訊
- ⇒其它

運用身體評估的先決條件

- 能自體表辨認各器官構造位置
- 了解各器官構造生理活動之原理
- 熟悉身體評估的手技
 - 視觸叩聽
- 能判斷問題的嚴重性
- 適當轉介

心肺功能的評估

- 肺:
 - Ventilation, diffusion
- 心:
 - Pumping, perfusion
 - 運送 O2、CO2、養分、代謝廢物、分泌物

組織氧合與心肺功能

- 通氣、換氣 (Ventilation):
 - 氣之吸入與呼出 (肺)
- 擴散 (Diffusion):
 - (氣體)移進移出微血管
- 攜帶 (Carry): Hgb,
 - CaO2=1.34 X Hgb X SaO2 + 0.003XPaO2
- 灌流 (Perfusion):
 - 微血管充盈的程度 (以攜 O2 與 CO2)
- 氧氣需求 (O2 demand):
 - BMR
 - Disease severity
 - Physical Activity

肺部功能不足常見之症狀與徵候

- Dyspnea
- SOB
- Orthopnea
- Cyanosis

肺臟常見之疾病與其問題性質

• Rib fructure, 呼吸肌麻痺	• 影響胸闊擴張
 Bronchitis, Broncheoectasis, asthma, (COPD) 	• 影響呼吸道通暢
 Pneomothorax, Pleural effusion, Emphysema, Pneumonia, Pulmonary 	•影響肺泡擴張
edema, ARDS,	•影響肺泡氣體擴散

大綱

- (A) 視診 (Inspection)
- (B) 觸診 (Palpation)
- (C) 叩診 (Percussion)
- (D) 聽診 (Auscultation)
 - (1)技巧
 - (2)正常呼吸音
 - (3)不正常呼吸音 (Adventitious Breath Sounds)
 - (4) Voice Sounds

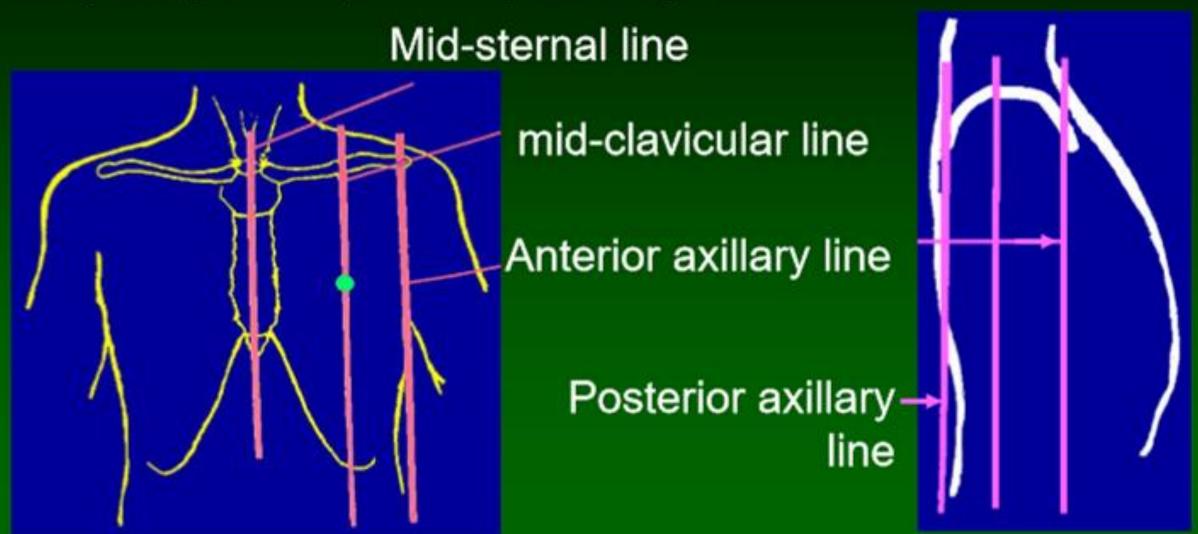
(A) 視診(1): 有無呼吸窘迫(respiratory distress)?

- (1) 黑青 (cyanosis)?
- (2) 端坐呼吸 (orthopnea) ?
- (3) 使用呼吸輔助肌?
- (4) Paradoxical breathing?
- (5) 縮嘴吐氣 (pursed lip breathing)?

Inspection

- Rate, rhythm, effort of breathing (expiratory phase)
- Listen to obvious abnormal sounds
- Retraction and use of accessory muscle
- Asymmetry, deformity, or increase A-P diameter
- Trachea at midline? Neck veins
- Nasal alae, purse lips

Suprasternal notch, Sternal (Louis) angle, Infrasternal angle, xyphoid proess, scapula, ribs, intercostal space, spinal process, costolspinal angle



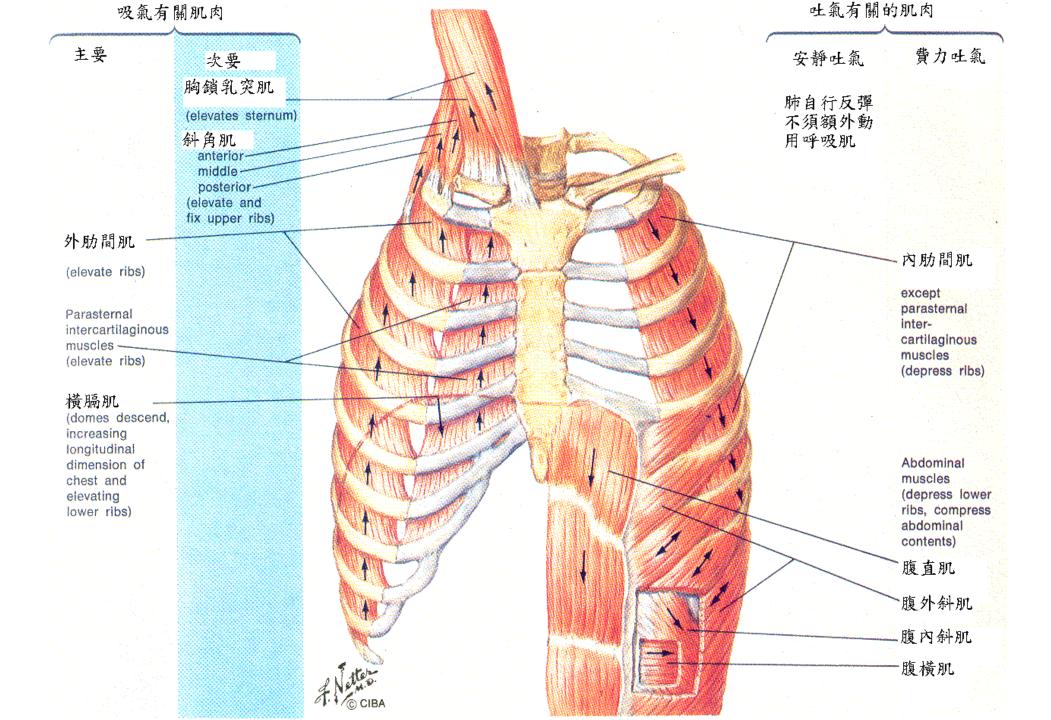


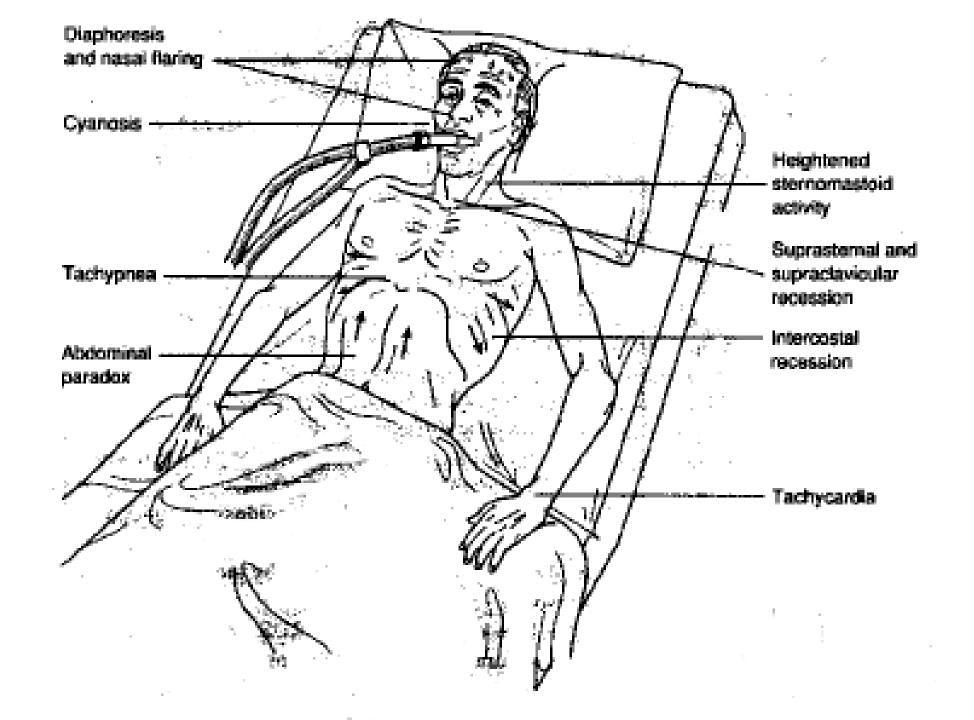
Superior vena cava syndrome

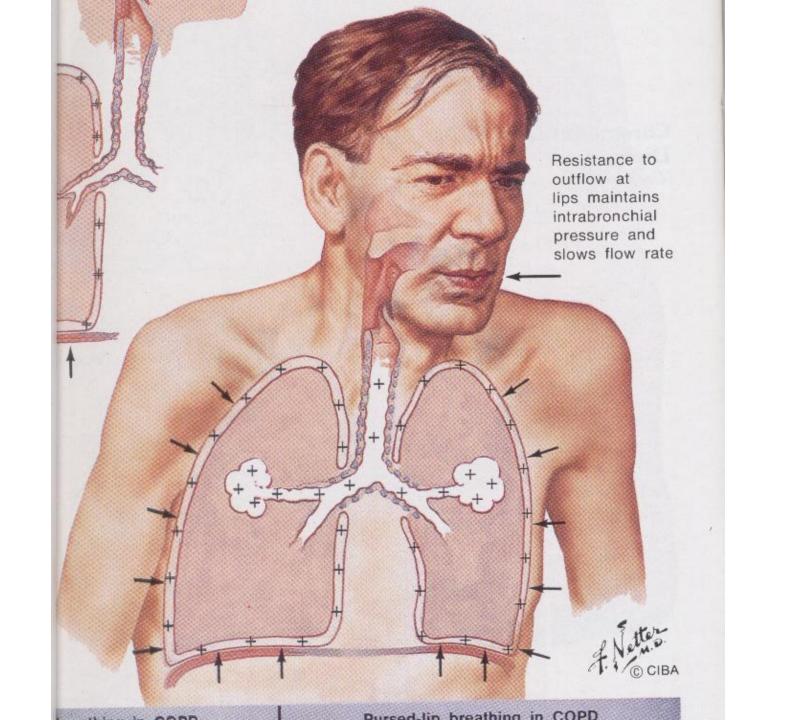
SVC obstruction due to mediastinal tumor, lymphadenopathy, etc.



Department of Internal Medicine





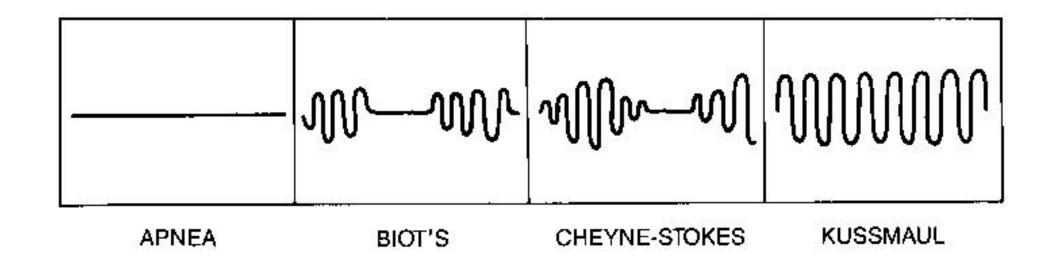


(A) 視診(2)呼吸次數

- 不要讓病人知道你正在量呼吸次數 (可以手假把脈搏分散病人注意力,同時以眼睛注視胸部起伏,在心中默數次數)
- 至少要數半分鐘
- 正常人次數在 12-16/min

(A) 視診 (3)呼吸型態

- (1) Apnea(停止呼吸):Cardiac arrest
- (2) Biot's (不規則呼吸間隔著一長段停止呼吸): IICP、呼吸抑制藥物、腦受傷(通常是延腦)
- (3) Cheyne-Stokes (漸強→漸弱→停止→漸強→漸弱→停止…): 呼吸抑制藥物、腦受傷(通常是大腦)、CHF
- (4) Kussmaul (深而快):代謝性酸中毒



Inspection of breathing rhythm

Normal

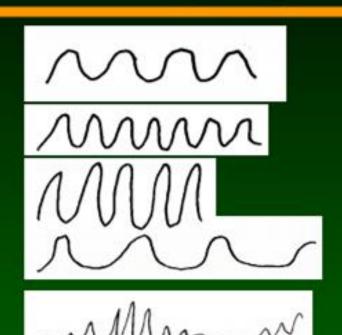
Tachypnea

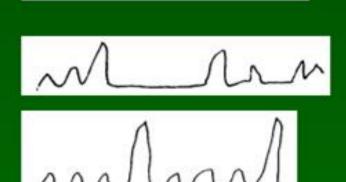
Hyperpnea

Bradypnea

Cheyne-Stokes breathing

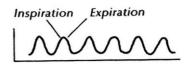
Biot's (ataxic)
breathing
Sighing respiration





Visual Examination of the Chest Breathing Patterns

Rate, Depth, Regularity



Normal

Adults:12-20/min Infants: 44/min



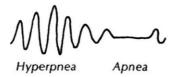
Ataxic breathing

Biot's breathing
Irregularly irregular
e.g., brain medullary injury



Tachypnea

Rapid, shallow breathing



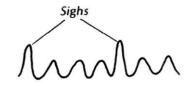
Cheyne-Stokes breathing

Regular rate, irregular depth **MAY** be normal e.g., heart failure



Hyperypnea
Rapid, deep breathing
Hyperventilation
Kussmaul breathing

(metabolic acidosis)



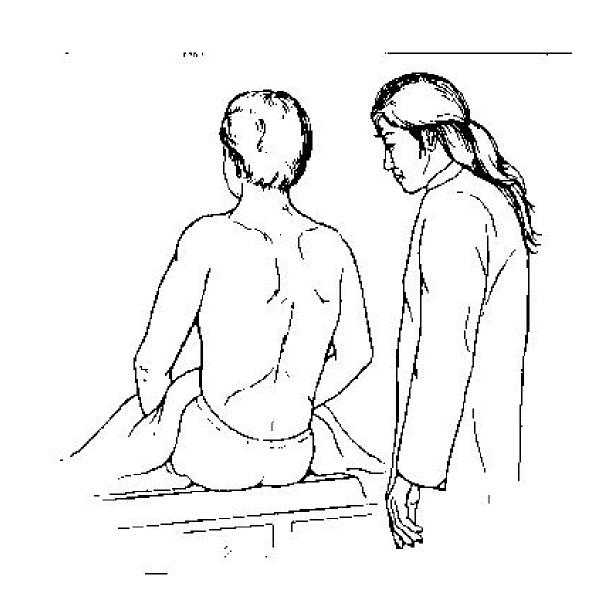
Sighs

Hyperventilation syndrome 1 sigh per 200 breaths

 $\mathcal{N}\mathcal{M}$

Bradypnea

(A) 視診 (4)胸廓外型



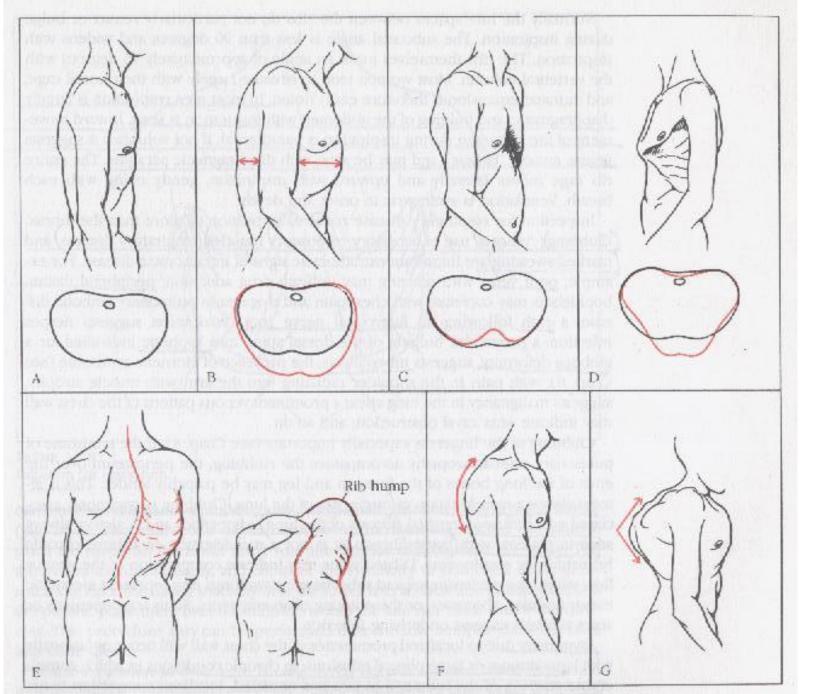
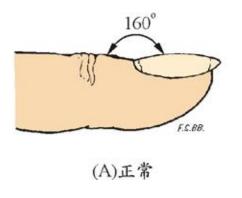


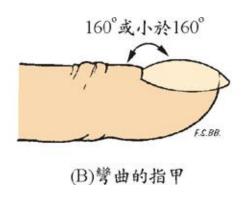
FIGURE 11-7

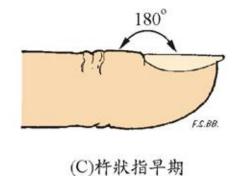
(A) 視診(5) 手指

- 杵狀指 (clubbing fingers):
- 通常代表長期缺氧的結果。可見於各種胸腔疾病(肺癌、支氣管 擴張、肺纖維化等等),也可見於心臟病(CHF、亞急性心內膜 炎)或肝病(肝癌、肝硬化)

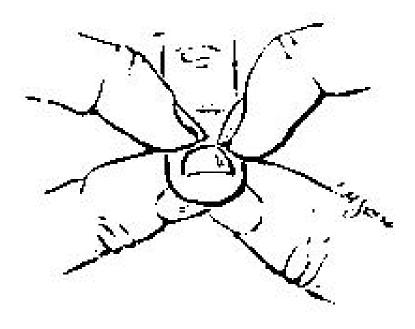
杵 狀 指



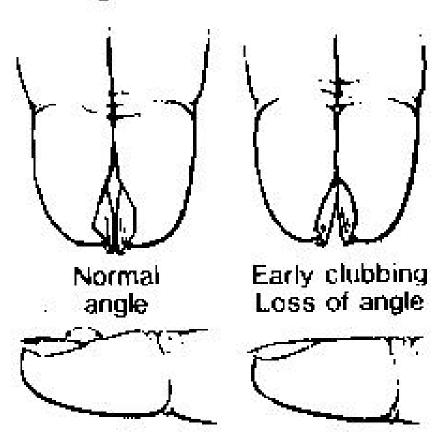




Testing for fluctuation at the base of the nail

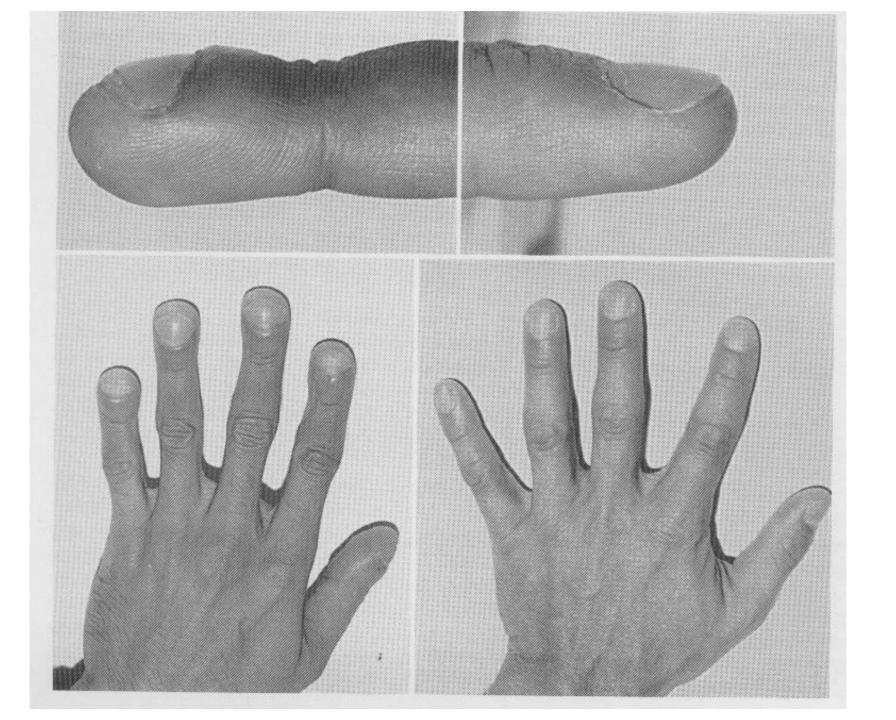


Testing for loss of normal angle

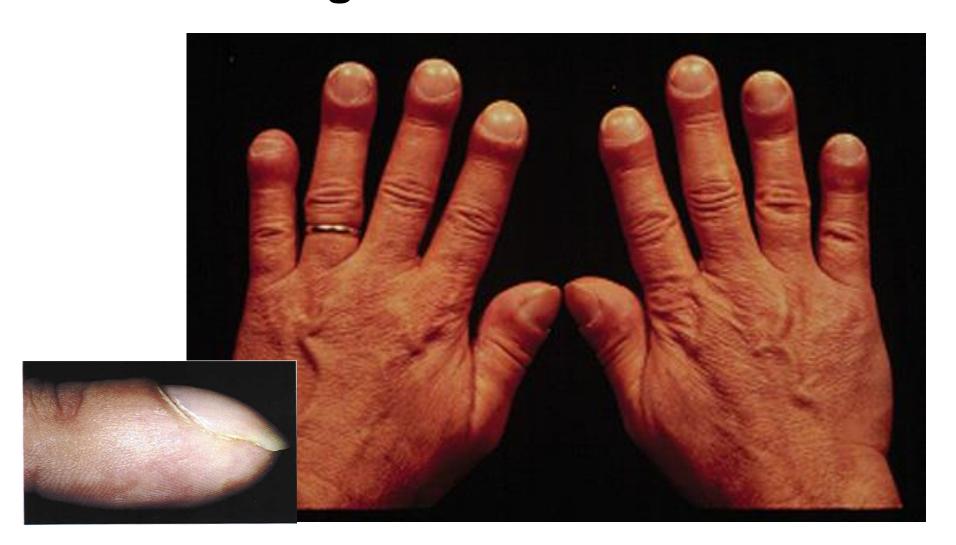


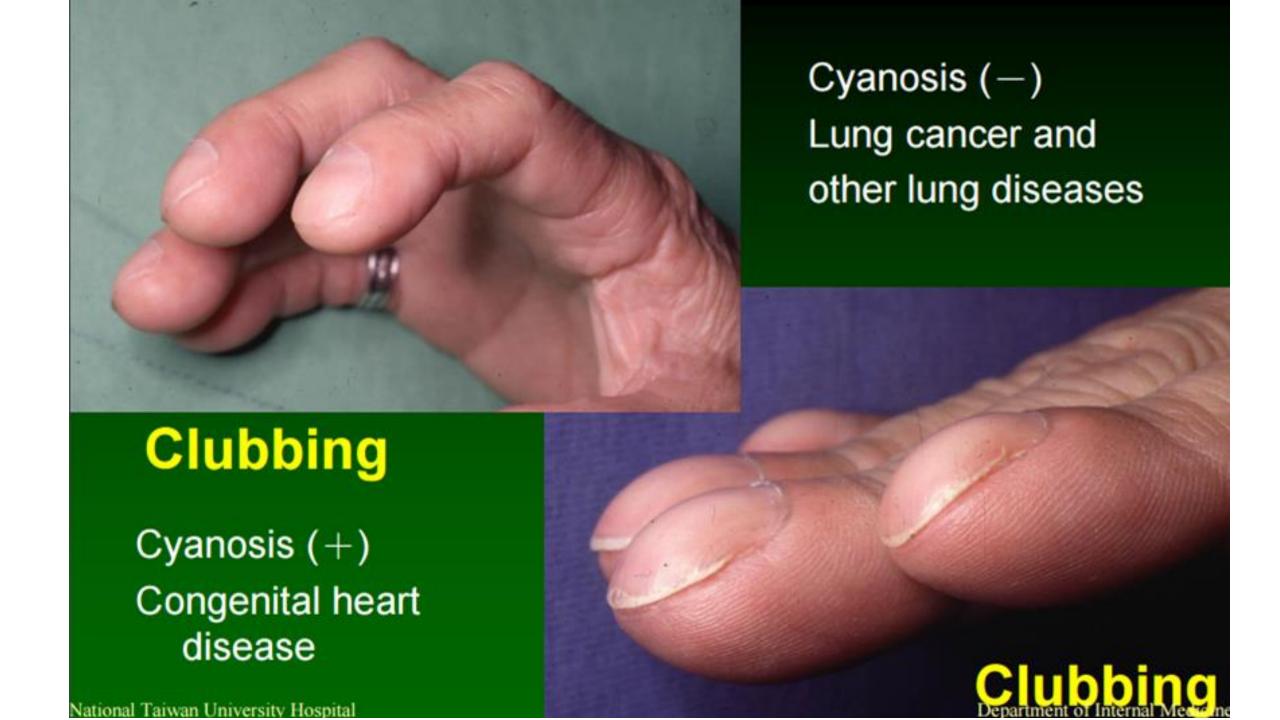
Late clubbing





Systemic Signs of Pulmonary Disease Clubbed Fingers





Palpation

- Identification of tenderness or deformity (palpating ribs and sternum)
- Assessment of observed abnormalities eg: subcutaneous nodule, hemangioma
- Assessment of respiratory expansion
- Assessment of tactile fremitus

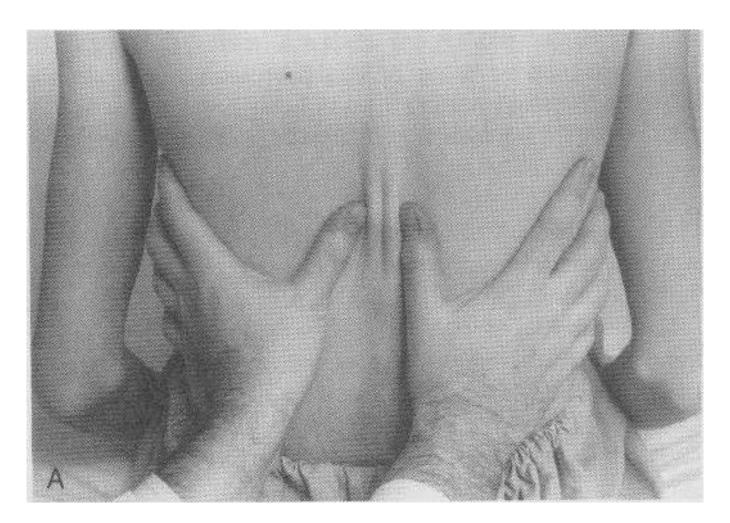
(B) 觸診 (1): 胸腔擴張度

- 正常人可擴張 4-6cm
- 注意對稱性
- 擴張不足: 阻塞性肺病、肺纖維化、肌肉無力、僵直性脊髓炎

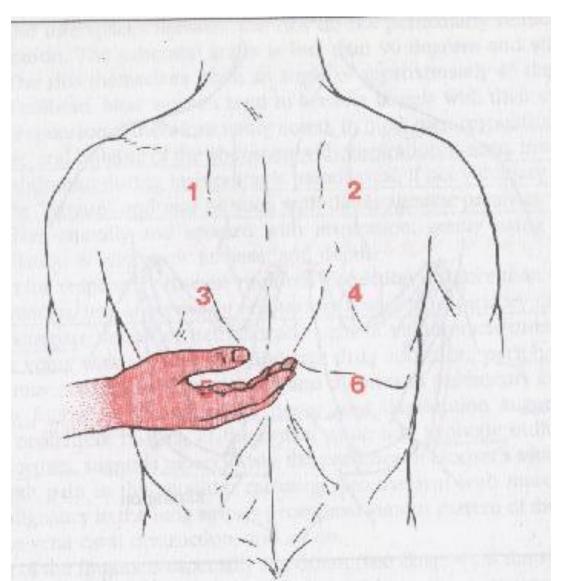
(B) 觸診 (2):觸覺震顫

- 觸覺震顫 (tactile fremitus) = 經由呼吸空氣在胸腔表面所產生的 震動
- · 語聲震顫 (vocal fremitus) = 經由說話在胸腔表面所產生的震動
- 增加震顫: 肺炎、肺塌陷
- ·減少震顫:肋膜積水、氣胸、單側氣管阻塞、COPD、胸壁太厚 (肌肉或脂肪)

(B) 觸診 (1): 胸腔擴張度



(B) 觸診 (2):觸覺震顫



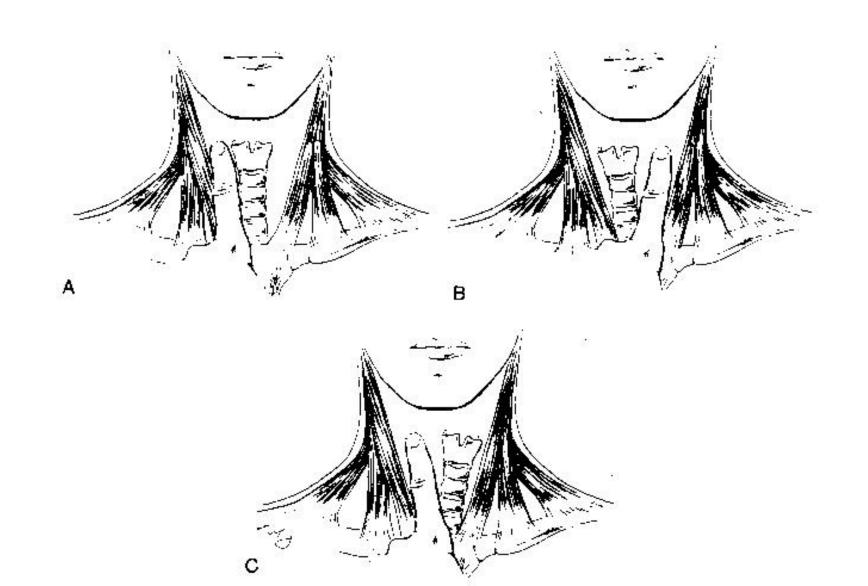


Palpation of thoracic expansion

Voice "ah" then:

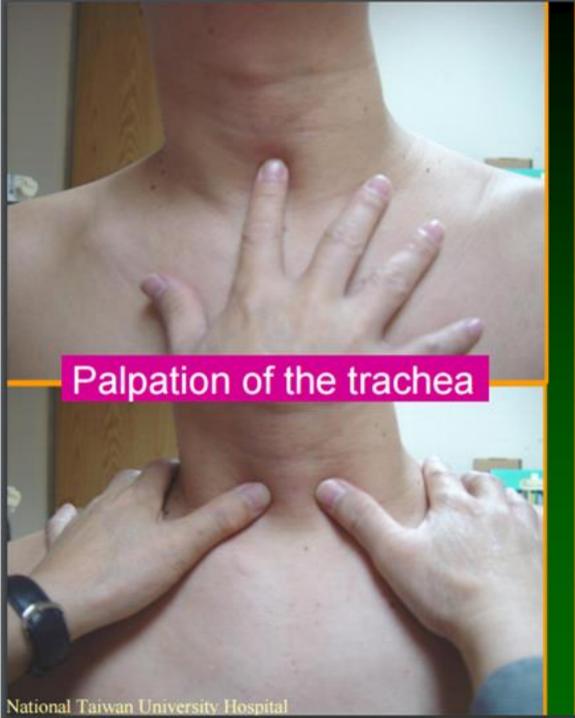
- Vocal fremitus with a stethoscope
- Tactile fremitus with hands

(B) 觸診(3):氣管位置



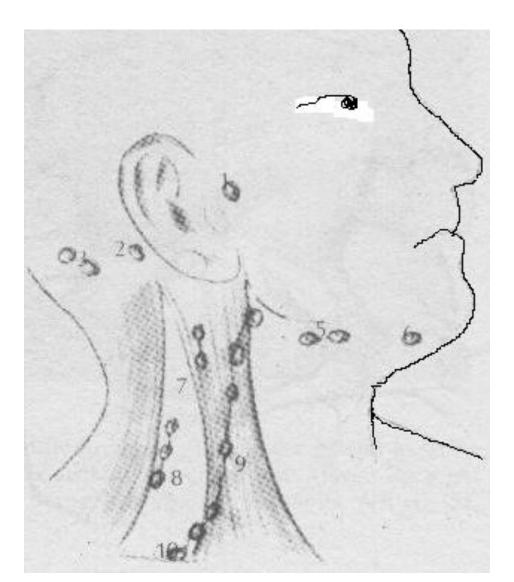
(B) 觸診(3):氣管位置(續)

- A: 用食指估計氣管與兩側胸鎖乳突肌間的距離,有否對稱
- B: 當右肺塌陷時,氣管被拉向右側
- C: 當右肺有壓力性氣胸、大量肋膜積水時,氣管被推向左側

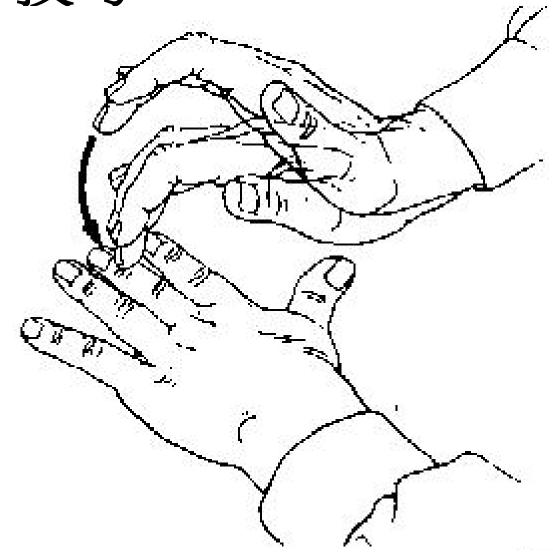




(B) 觸診(4) 頭頸部淋巴節

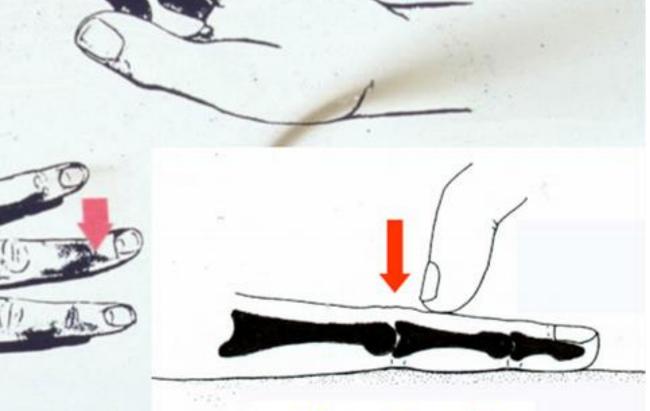


(C) 叩診: 技巧



Percussion

With same force and same direction when comparing right and left sides.



Chest wall

Turner R, Blackwood R

Sonorous Percussion

 Resonance, Hyperresonance, Tympany, Dullness, Flatness

Definite Percussion

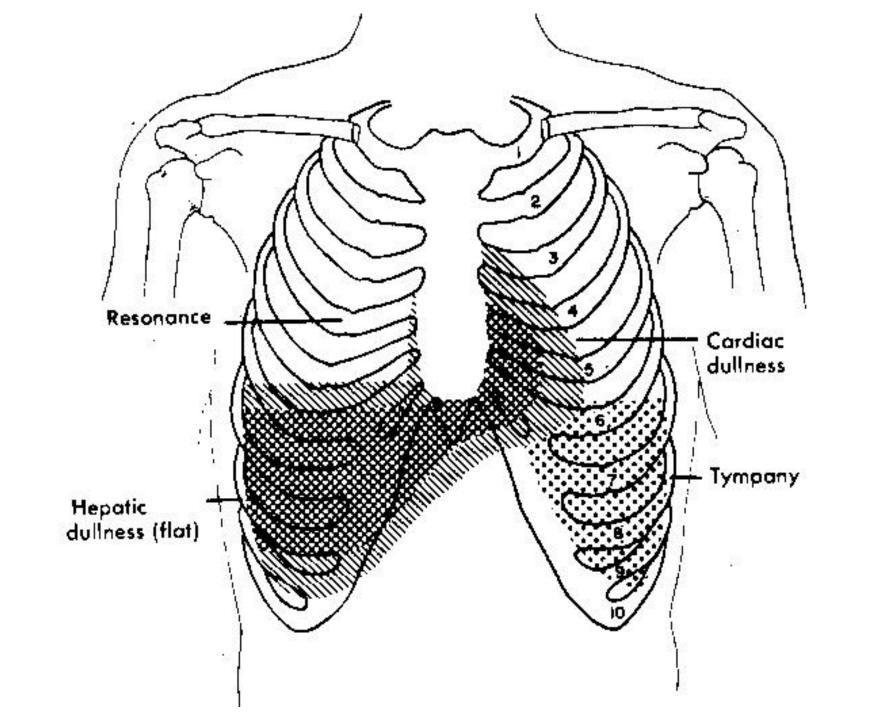
Outline lung-heart, liver, spleen borders

表 8-1 叩診聲音之特色 (Characteristics of Percussion Notes)

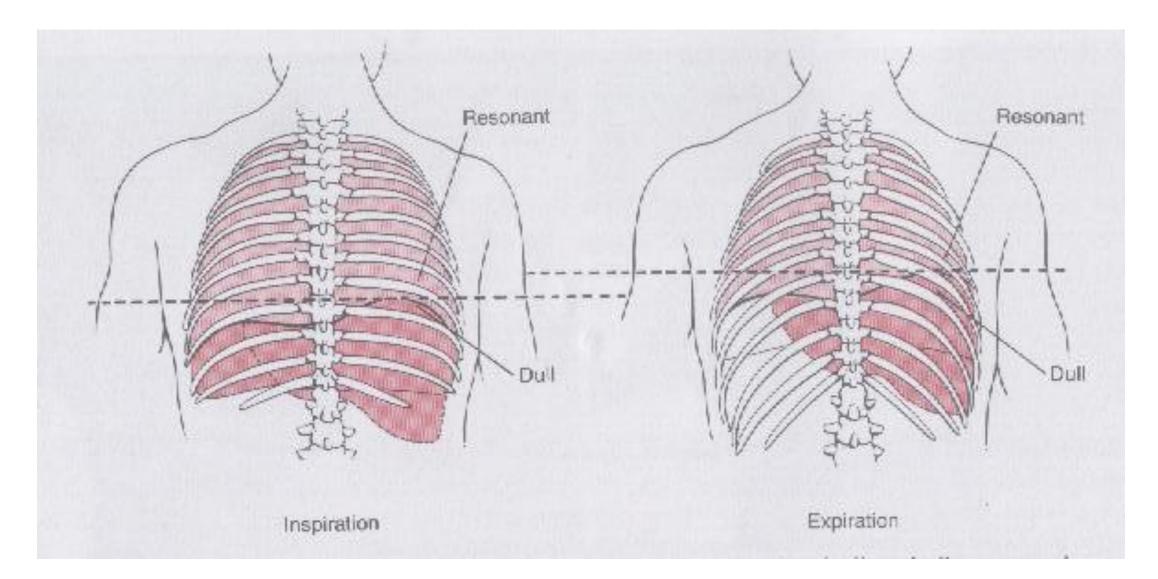
	振幅	音調	音質	音長	位置實例
共鳴音 (resonant)	中響	低沈	清而中空	中度	正常的肺組織外表叩診音
UUUUU 過度共鳴音 (hyperresonant)	較響	較低	隆隆聲	較長	正常的兒童肺組織外表 若在成人,表示有異常多量空氣 在其中,如肺氣腫
鼓音 (tympany)	響	高	如鼓之樂聲 (像定音鼓)	持續最長	充滿空氣之内臟,如胃小腸
濁音 (dull)	微響	高	東西被包著打的音色	短	較密實之器官,如肝、心、脾
實音 (flat)	甚輕	高	急斷音,完全沈鈍之音	極短	無空氣的組織,如大腿骨的肌肉、骨頭或腫瘤外表叩診音

叩診音依性質可分為五種

- 1. 實音(Flatness):音質沉鈍,為叩擊實質組織的回音,如肌肉、骨頭等。
- 2. 濁音(Dullness):亦稱鈍音,音質像東西被包著打的碰擊聲,為叩擊充滿血液之器官時的回音,如肝臟、脾臟等。
- 3. 鼓音(Tympany):音質如鼓聲,為叩擊充滿空氣之肌質器官的回音,如胃、小腸等。
- 4. 過度共鳴音/過度反響音(Hyperresonance):音質如隆隆聲,為兒童正常的肺外表叩診聲;若於成人,則表示肺中充滿異常量的空氣,如肺氣腫、肺癌等。
- 5. 共鳴音/反響音(Resonance):音質中空,為叩擊充滿空氣之組織的回音,如肺臟。



(C) 叩診:評估橫膈移動



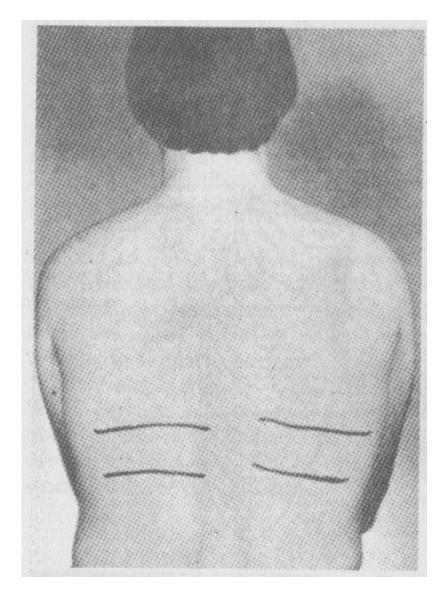
(C) 叩診:評估橫膈移動

• 正常女人:3 cm; 男人:5-6cm

• 移動減少:

➤兩側:COPD、肌無力

▶單側:膈神經麻痺



Diaphragmatic Excursion



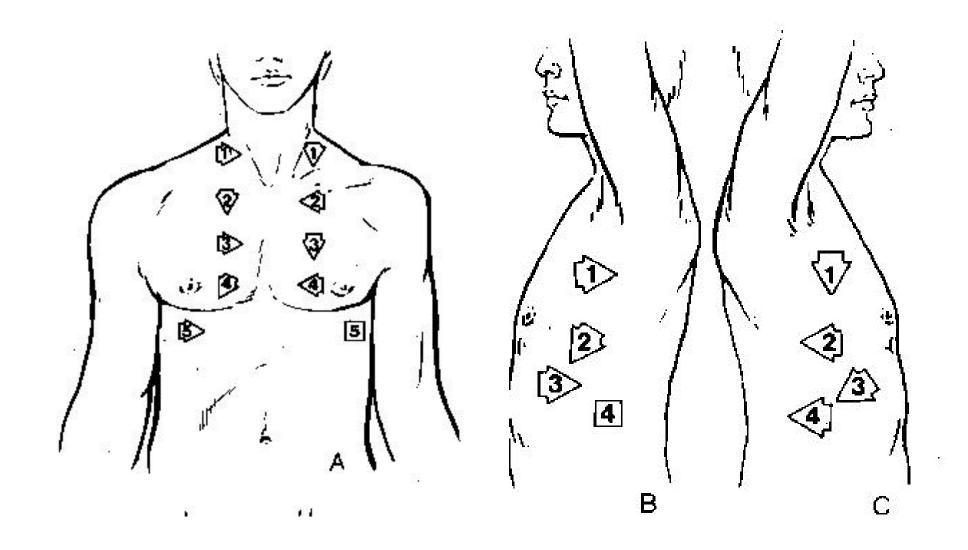
正常情形下, 吸、呼之間肺下 爆應可昇降二個脊柱之高度

T10 spinous process (呼氣)

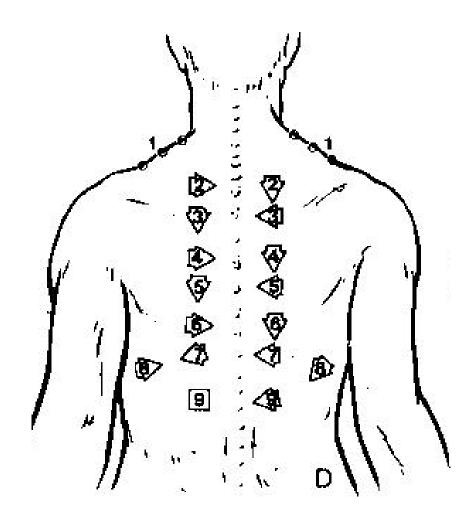
T12 Inspiration (吸氣)

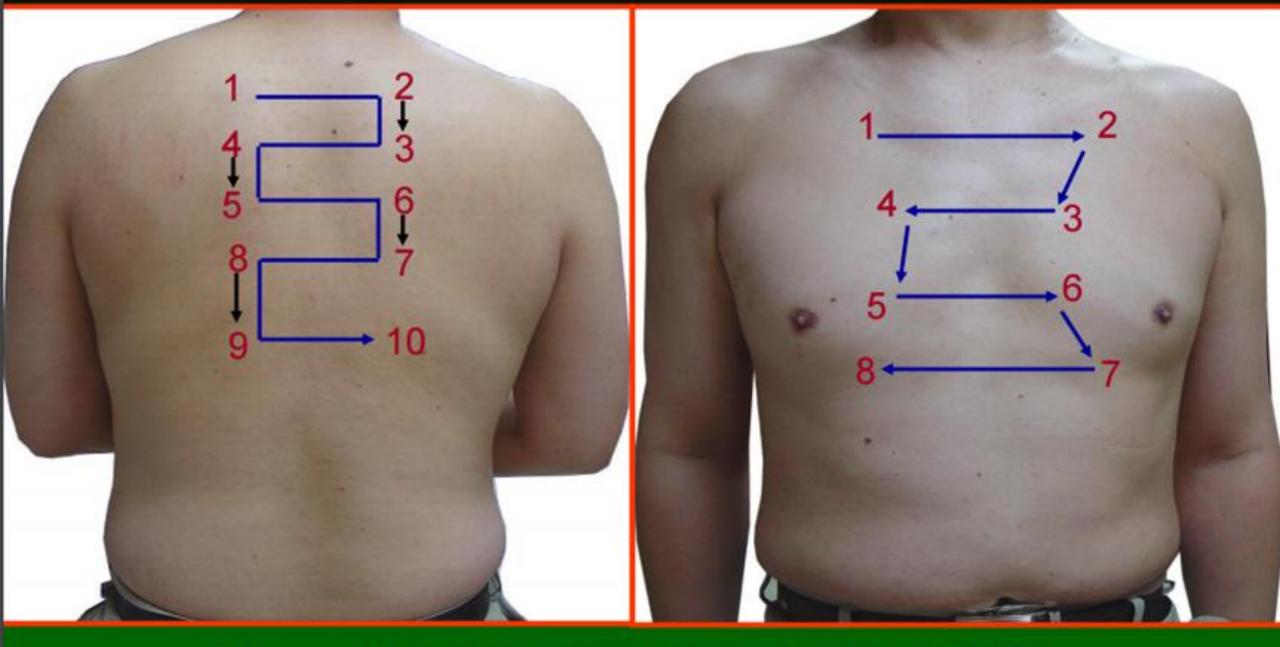
從背後叩診

(C) 叩診:順序



(C) 叩診:順序



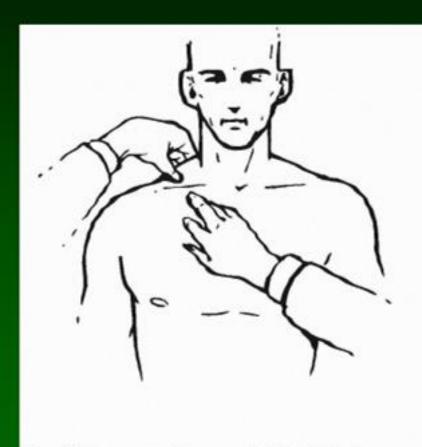


The order of percussion and auscultation

National Taiwan University Hospital

Department of Internal Medicine

Percussion of Apex

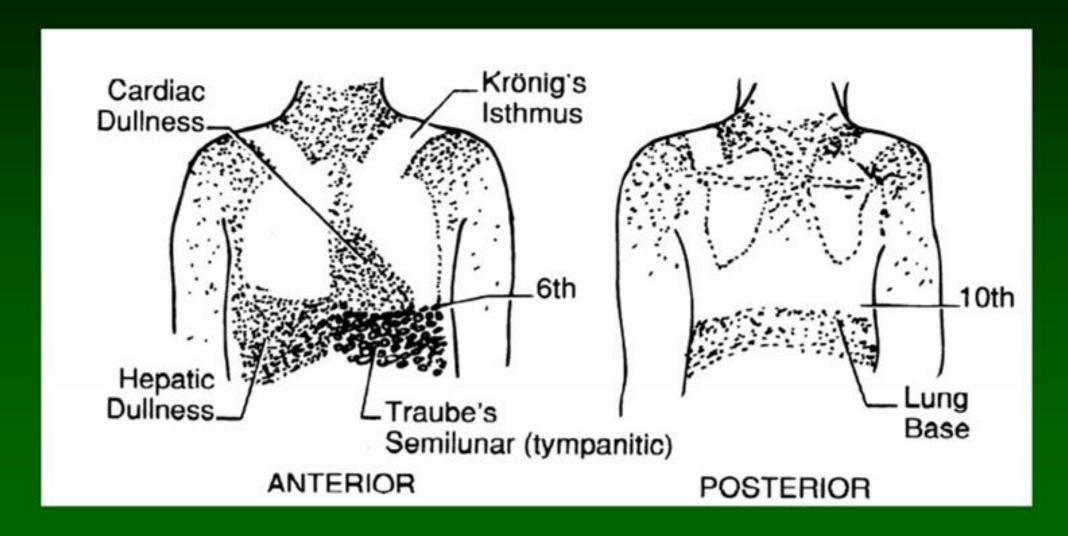


A. Percussion of Right Apex



B. Percussion of Left Apex

Percussion



(D) 聽診:沒有聽診器時



(D) 聽診:聽診器的發明人

- Rene Theophil Laennec (1781-1826)
- 法國醫生
- 發明聽診器並首先描述正常與不正常的呼吸音





8–4

聽診器:膜面(左);鐘面(右)

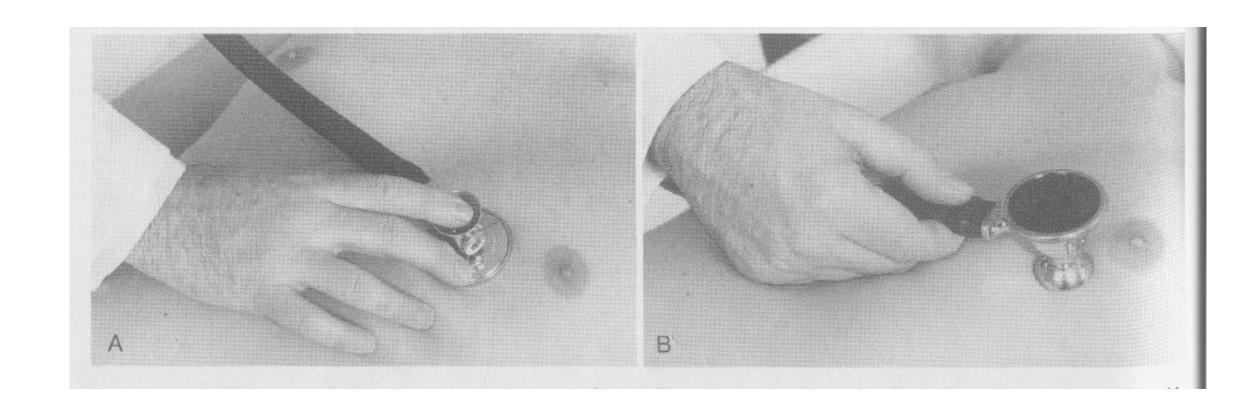
聽診(Auscultation)

- 聽診是利用聽覺去感受個案身體所發出之聲,檢查者除了用耳朵 聽外,身體大部分的聲音需藉助聽診器。
- 聽診器的選擇: 耳塞的斜度應向前方朝著鼻子,管子長度要有30-36公分,選擇有兩個聽筒的,且應用酒精棉球消毒。
- 1. 膜面(Diaphragm): 適合聽診音調較高的聲音,如腸音、呼吸、正常的心音。緊貼於病人身上。
- 2. **鐘面(Bell)**:深而中空的杯狀,適合聽診**音調較低**的聲音,如不正常的心音或**心雜音**。輕放在病人身上。

(D) 聽診: 技巧

- Bell: for low-pitched sounds; 輕置於皮膚上
- Diaphragm: for high-pitched sounds; 緊緊地壓在皮膚上聽(呼吸音多屬 high-pitch,皆以diaphragm聽)
- 安靜的環境
- 請病人張口以較深且快的方式呼吸
- 不可隔著衣服聽

(D) 聽診: 技巧



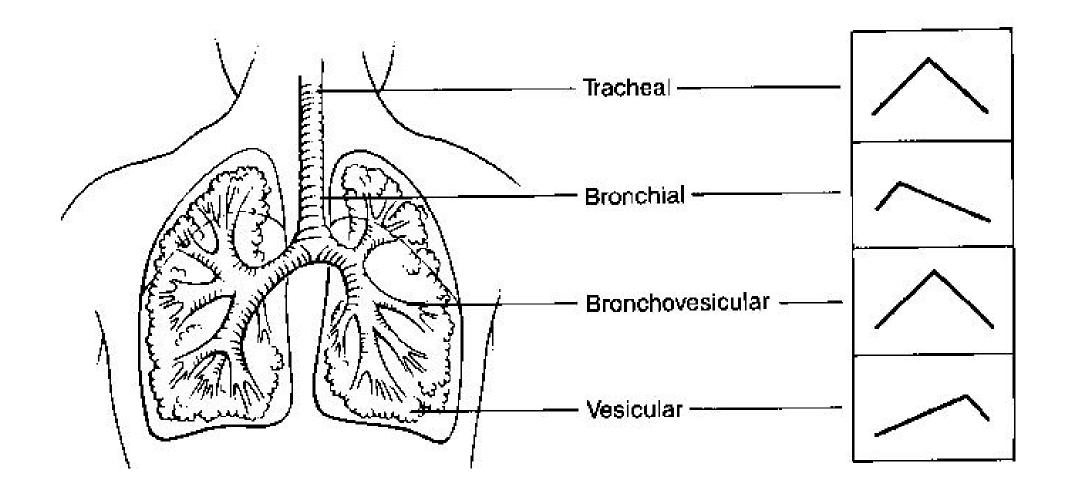
(D) 聽診:正常呼吸音

	Tracheal	Bronchial	Broncho- vesicular	vesicular
聲音強度	非常大聲	大聲	中等	小聲
Pitch	Very high	High Moderate		Low
I:E Ratio	1:1	1:3	1:1	3:1
特性描述	Harsh	Tubular	Rustling,but tubular	Gentle rustling
正常位置	胸腔外氣管	胸骨柄	大支氣管	大部分肺周邊

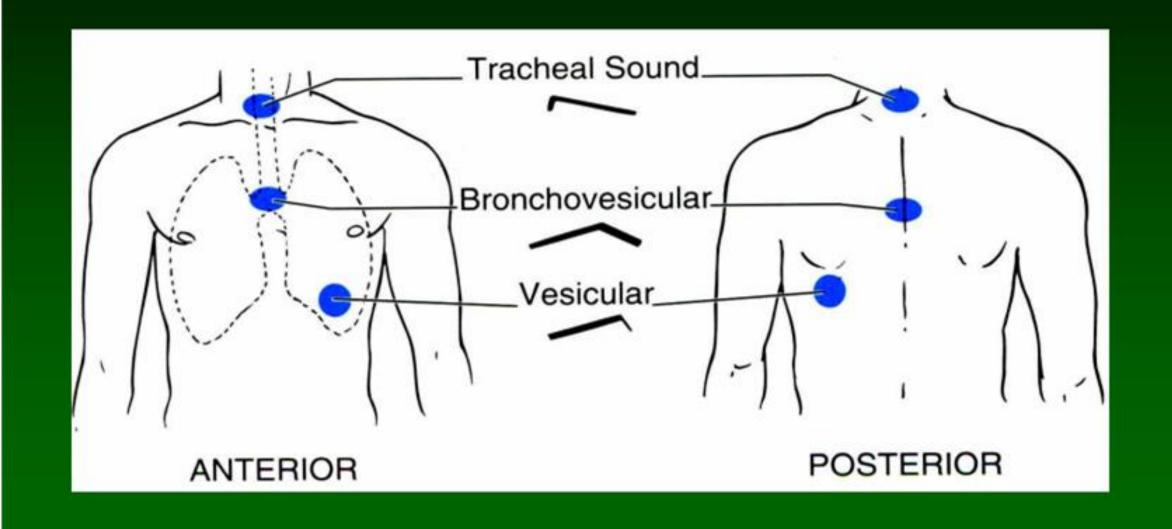
Auscultation of the Chest Breath Sound Characteristics

	Duration of sounds	Intensity of Expiratory Sounds	Pitch of Expiratory Sounds	"Normal" Location
Vescicular	Inspiration > Expiration	Softer	Relatively low	Both lung fields
Broncho- vescicular	Inspiration = Expiration	Intermediate	Intermediate	1 st & 2 nd interspaces anteriorly; between scapulae
Bronchial	Inspiration < Expiration	Loud	Relatively high	Over manubrium (?)
Tracheal	Inspiration = Expiration	Very Loud	Relatively high	At sternal notch

(D) 聽診:正常呼吸音



Auscultation



Tracheal Sound



• 聲音來源: R.A.L.E. Repository

Bronchial Sounds

- 正常情況下只出現在胸骨柄位置
- 當它在肺部較週邊的地方被聽到,可能表示肺部有實質化(例如肺炎)



• 聲音來源:McGill University

Bronchovesicular Sounds

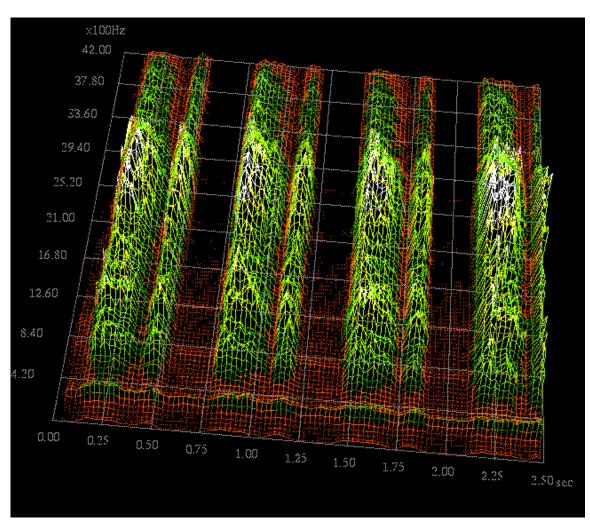
- 正常情況下只出現在大支氣管第一、二肋間)或背後兩肩胛骨間
- 當它在肺部較週邊的地方被聽到,可能表示肺部有實質化(例如肺炎)



• 聲音來源:Loyola University

Vesicular Sound





• 聲音來源: McGill University

呼吸音變小聲

- 肺氣腫 (肺泡數目減少)
- 横膈麻痺(呼吸肌無力)
- 支氣管完全阻塞 (沒有氣進出)
- 氣胸、肋膜積水、肋膜增厚 (聲音傳導受阻)

呼吸音變大聲

• 肺實質化(consolidation)(如肺炎、肺塌陷、肺癌等)(因為實質化以後的肺較先前充滿空氣的肺更利於聲音傳導,所以更大聲)

Adventitious (偶然的;外來的) Breath Sound

有以下幾種,都屬於不正常的呼吸音

- Wheeze = Sibilant (發絲音的,作絲聲的。) rhonchus
- Rhonchus (乾囉音)= Sonorous (響亮的,洪亮的)rhonchus
- Crackle (劈啪聲,爆裂聲) = Rale (水泡音;羅音)
- Friction (摩擦,阻力) rub = Pleural rub
- Stridor

Wheeze



- 連續高音,音頻>400Hz,近似吹口哨音,**常出現在吐氣時**(但也可能跑到吸氣時)
- 由於氣流快速通過變狹窄的極小支氣管所發出的
- 可見於:氣喘、肺水腫、心衰竭、支氣管炎等疾病

• 聲音來源: McGill University

Rhonchus



- 連續低音,音頻<200Hz,近似打鼾或呻吟聲,常出現在吐氣時
- 由於氣流通過變窄後的較大支氣管所形成
- 可見於氣喘病、支氣管炎等病

• 聲音來源: McGill University

Crackle



- 不連續音,常出現在吸氣期,類似在耳鬢搓揉頭髮的「嗶嗶啵啵」聲 (捻髮音)
- 聽見 crackle 時,令病人咳嗽將痰清出後,再聽。有時 crackle 就會自然不見了。
- •成因:由於氣道內異常積水,吐氣時肺泡會塌陷,但吸氣時又會再被撐開。當許多肺泡不同時被撐開,就會發出crackle聲
- 可見於心衰竭、肺積水、肺炎、肺纖維化
- 聲音來源: McGill University



Friction Rub

- •成因:當肋膜表面因發炎物堆積、或變厚、或長癌時,兩層肋膜間隨著呼吸而彼此滑動的動作就會出現阻力,此謂之Friction rub。
- 通常在吸氣末和吐氣初聽到
- 較常出現的位置是腋下或是後面的肺基底部。

• 聲音來源: Loyola University

Stridor



- 是一種特別大聲的 wheeze,可以在離病人稍遠的地方、不用聽診器就聽到。
- 通常是因為上呼吸道狹窄造成的。

• 聲音來源:R.A.L.E. Repository

Voice Sounds

- 是用聽診器聽診時病人正在說話所聽到的異常聲音。有以下三種:
- Egophony
- Broncho phony
- Whispered pectoriloquy



Egophony



若聽診的部位有實質化(consolidation)(如肺炎、肺塌陷、肺癌等)→令病人發出「伊」的聲音,從聽診器聽起來會變得像在唸「A」的聲音,此謂之 egophony

• 聲音來源: McGill University





- 正常聽診時若病人說話,從聽診器聽到的聲音較小聲、也較不清晰
- 但是若聽診的部位有實質化(consolidation)(如肺炎、肺塌陷、肺癌等)→說話的聲音將反常地大聲且清晰,此謂之 Bronchophony

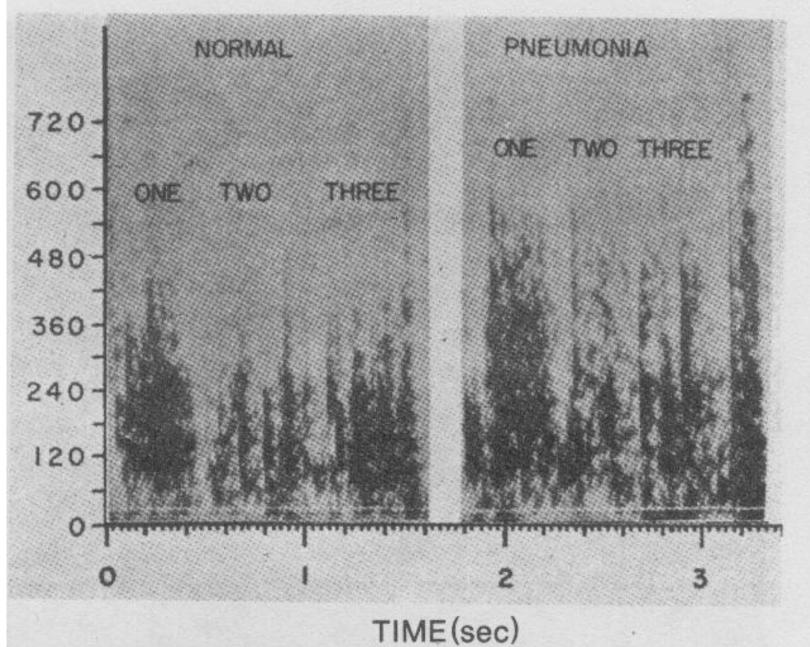
• 聲音來源: McGill University



Whispered Pectoriloquy

- 聽診時若病人以近乎耳語的小聲說話,正常時應當幾乎無法被聽診器聽見。
- 但是若聽診的部位有實質化(consolidation)(如肺炎、肺塌陷、肺癌等)→小聲耳語的聲音將變得異常地大聲且清晰,此謂whispered pectoriloquy。

• 聲音來源: Loyola University



e 5-24. Whispered pectoriloquy. Note that in pneumonia, the spectrogram indicates

Asthma

病因:Bronchospasm,支氣管狹窄

視:cyanosis ?, RR↑,使用呼吸輔助肌

觸:T/F↓

叩:正常or Hyperresonace(chronic)

聽:Wheeze

語音聽診→消失or減弱

Atelectasis

病因:肺泡膨脹不全、塌陷

視:咳嗽、患側胸廓擴張不佳、患部肋間肌內縮

觸:氣管微偏向患側、患側胸廓擴張不全、

T/F減弱或消失

叩: Dullness or flatness

聽:無呼吸音、語音聽診減弱或消失

臨床應用:臥床病人及麻醉後病人尤其要留意之Atelectasis聽診

Bronchitis

病因:氣管樹發炎、有部份阻塞及分泌物或收縮

視:嚴重時呼吸快、發紺;咳嗽、黏性痰液

觸:T/F正常或增強

口: resonance

聽:吸氣→rales;呼氣→rhonchi、wheeze

Emphysema

病因:肺泡異常擴張、膨脹及肺泡破壞,空氣 滯留氣管內

視:桶狀胸、使用呼吸輔助肌、活動時呼吸困 難、RR↑、pink puffer

觸:患側無胸廓擴張、T/F減弱或消失

叩: Hyperresonance 、横膈離軌度變小

聽: 吸氣晚期fine rales;呼氣期rhonchi

or wheeze

纖維性Pleural effusion 與 Pleuritis

病因: 肋膜發炎而產生滲出液積留於肋膜腔

視:RR↑

觸:T/F減弱或消失、患側胸廓擴張不佳、嚴重時氣管偏向健側

口 : Dullness

聽: Friction rub

備註-Friction rub音常見於心臟病患及肺癌病患

- Pleuritis時,有Friction rub音;Pleural effusion時,無Friction rub音,聽診時聲音變弱或消失
- 心臟疾病造成之肋膜積水為漿液性,癌症病變造成的肋膜積水為蛋白性,與肋膜間會形成粘黏與纖維化,才會聽診出 Friction rub音

Pneumonia c consolidation

病因:肺泡內空氣為水份或組織取代

視:RR↑、患側胸廓擴張不佳

觸:患側胸廓擴張變小、T/F增強、但當支氣

管阻塞時則減弱

叩: Dullness、Flatness

聽:Rales、語音聽診→支氣管語音、耳語音

Pneumothorax

病因:空氣滯留肋膜腔

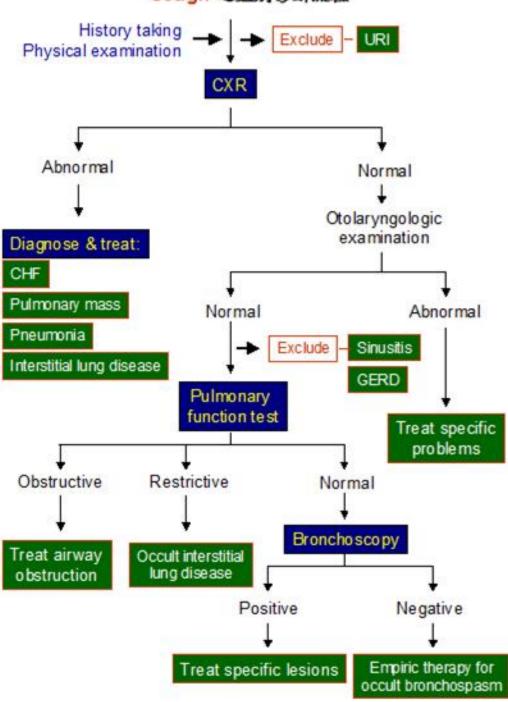
視:RR↑、患側胸廓擴張不佳、嚴重時會發紺、肋間肌膨出

觸:患側胸廓擴張變小、T/F消失、嚴重時氣管偏向 健側

□□ : Hyperresonance

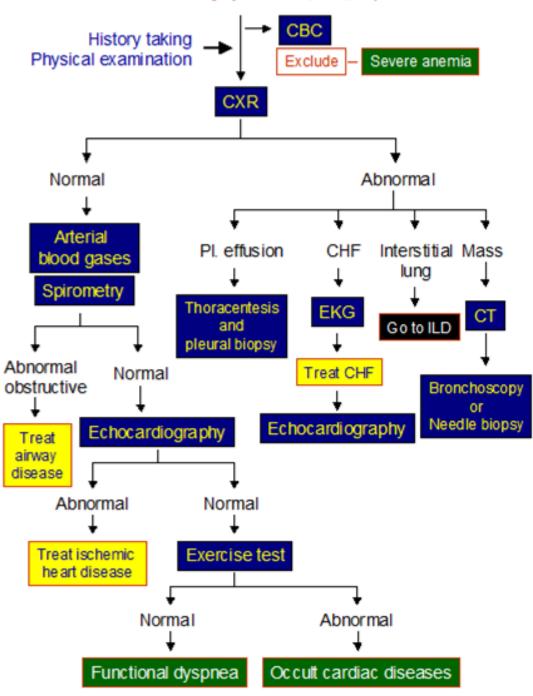
聽:患部Breathing sound 減弱

Cough 之鑑別診斷流程

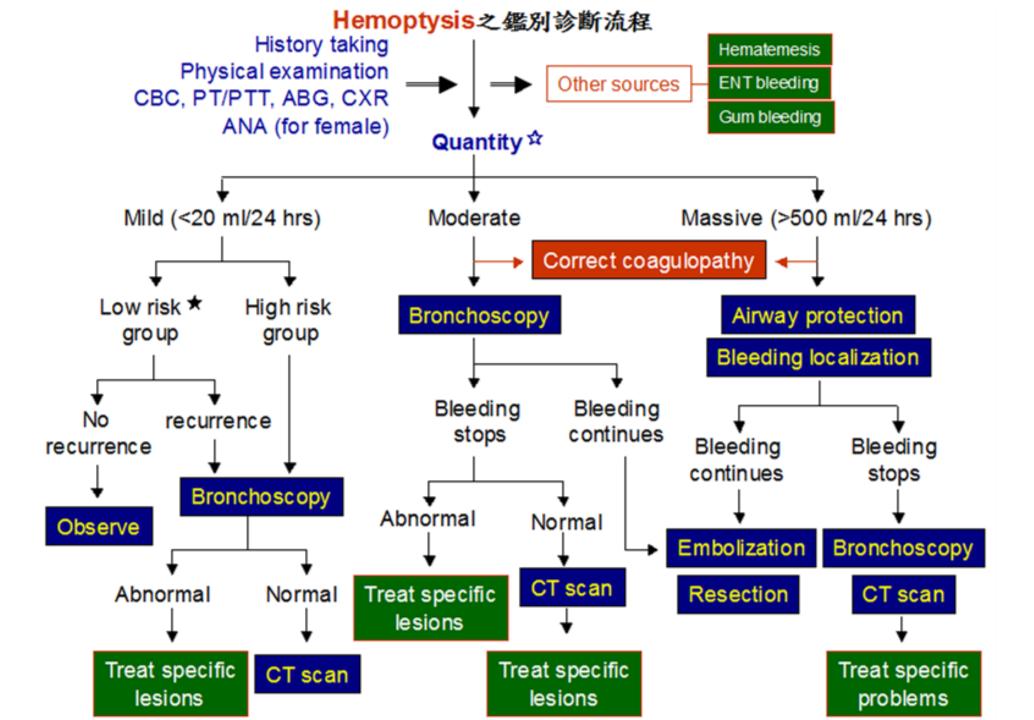


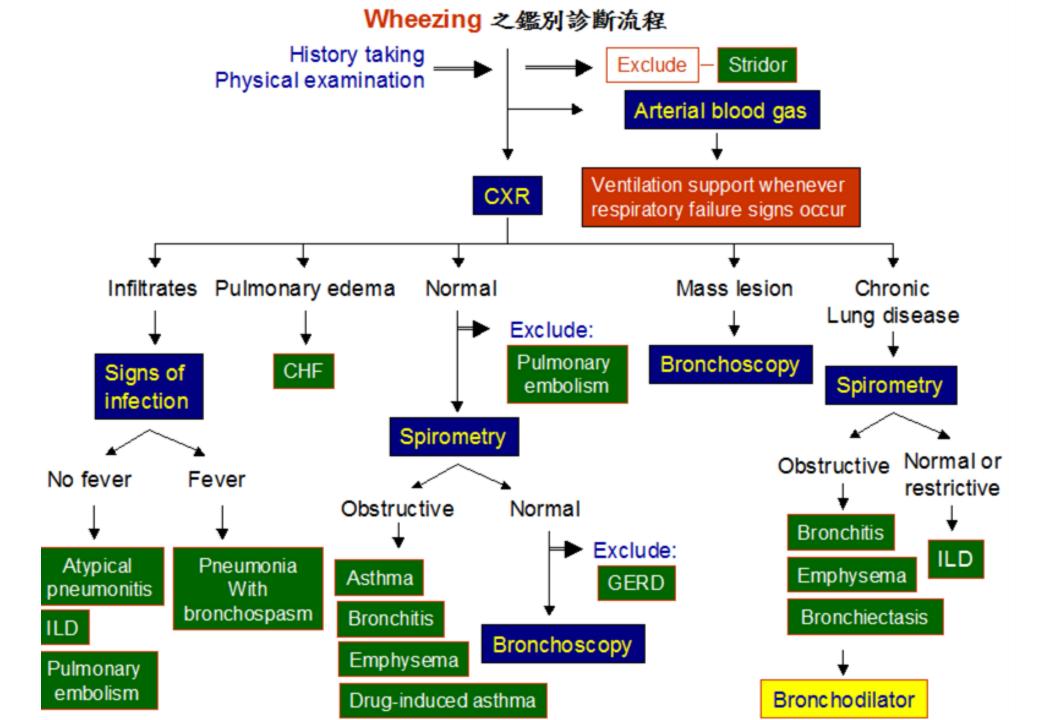
Acute Onset Dyspnea之鑑別診斷流程 History taking Ventilation support whenever Physical examination respiratory failure signs occur CXR Abnormal Normal Arterial blood gas Diffused Pneumo-Pleural Pneumonia infiltration Hypocapnia Normocapnia effusion thorax Hypercapnia ECG & **ECG** Echocardiography Airway diseases Asthma Normal Abnormal Abnormal Normal **Functional Pulmonary** Ischemic ALI or ARDS Cardiogenic embo lis m dyspnea heart diseases pulmonary edema Interstitial lung diseases

Chronic dyspnea 之鑑別診斷流程



Stridor之鑑別診斷流程 History taking ___ **⇒** Exclude − Wheezing Physical examination Acute or Subacute Chronic severe CXR Neck X-ray Subcutaneous epinephrine Inhaled racemic epinephrine CXR Abnormal Normal Risk of No risk of aspiration aspiration Lesion involving Laryngoscopy trachea Bronc hoscopy A chalasia Abnormal Normal Removal of Foreign body Vocal cord Bronchoscopy paralysis Fever No fever Neuromuscular disease Abnormal Normal Neoplasm **Epiglottitis** Laryngospasm Stricture Tracheitis Laryngeal edema Specific Functional Rheumatoid Ludwig's Tracheal hematoma treatment stridor arthritis angina Tracheal stenosis Diphtheria Laryngoscopy Abscess Specific Bronchoscopy treatment





Cough

Definition

- Cough is an protective mechanism that ensures the removal of mucus, noxious substances, and infectious organisms from the larynx, trachea, and large bronchi.
- Cough is an explosive expiration that provides a normal protective mechanism for clearing the tracheobronchial tree of secretions and foreign material.
- Cough interference with normal lifestyle, and concern for the cause of the cough, especially fear of cancer.

Mechanism

- Coughing may be initiated either voluntarily or reflexively
- As a defensive reflex it has both afferent and efferent pathways
- The cough starts with a deep inspiration followed by glottic closure, relaxation of the diaphragm, and muscle contraction against a closed glottis

Etiology

- An exogenous source (smoke, dust, fumes, foreign bodies)
- An endogenous origin (upper airway secretions, gastric contents).
- Any disorder resulting in inflammation, constriction, infiltration, or compression of airways can be associated with cough.
- Asthma is a common cause of cough.
- In a nonsmoker the most common causes of chronic cough are postnasal drip, asthma, and gastroesophageal reflux

Etiology

Acute cough (<3 weeks)

Is most often due to upper respiratory infection (common cold, acute bacterial sinusitis, and pertussis), serious disorders, such as pneumonia, pulmonary embolus, and congestive heart failure, can also present in this fashion.

Sub acute cough (between 3 and 8 weeks)

Is commonly post-infectious, resulting from persistent airway inflammation and/or postnasal drip following viral infection, pertussis, or infection with Mycoplasma or Chlamydia.

Chronic cough (>8 weeks)

In a smoker raises the possibilities of asthma, COPD or bronchogenic carcinoma, Eosinophilic Bronchitis, Esophageal Disease, Post Nasal Drip, ACEI, Smoking.

Common Causes of Chronic Cough

- Postnasal drip (38-87%)
- Asthma (14-43%)
- GERD (10-40%)
- Chronic Bronchitis (0-12%)
- More than one cause (24-72%)

Chronic cough (> 8 weeks)

Chronic Cough of Post-Nasal Drip

- PNDS is a symptom complex without objective findings.
- The diagnosis is by a history of the sensation of "something dripping into the throat," frequent throat clearing, nasal congestion or discharge.
- There is wide cultural diversity in reporting such symptoms by patients with "colds."
- In the USA, 50% with colds reported these symptoms, in the UK less than 25%, and in Latin America and India almost none.
- Cough may be the only manifestation of PNDS. There may be no symptoms of the "drip."
- PNDS is often seen due to Allergic Rhinitis, Non-Allergic Rhinitis, Vasomotor Rhinitis and Chronic Bacterial Sinusitis.

Chronic cough (> 8 weeks)

Asthma

- Asthma is a chronic inflammatory disease of airways characterized by increased responsiveness of the tracheobronchial tree to many stimuli.
- Physiologically there is a reversible narrowing of bronchi and clinically there are paroxysms of wheezing, cough, and dyspnea.
- If airway obstruction exists, reversibility is shown by > 12% \uparrow 200 cc in FEV1 after two puffs of a β 2-adrenergic agonist.

Chronic cough (> 8 weeks)

Gastro-esophageal disease (GED)

There are two main mechanisms of cough in GED:*

- Micro or macro-aspiration of esophageal contents into the tracheo-bronchial tree.
- Acid in the distal esophagus stimulating a vagally mediated esophageal-tracheobronchial cough reflex (GI symptoms may be absent).

Less Common Causes of Chronic Cough

- Bronchiectasis (0-5%)
- ACE inhibitor Rx
- Post-infectious
- Occult aspiration
- Lung Cancer
- Occult CHF
- Interstitial Pulmonary Fibrosis
- Occult infection (eg atypical mycobacteria)
- Foreign body

Industrial bronchitis Nasal polyps Problems with:

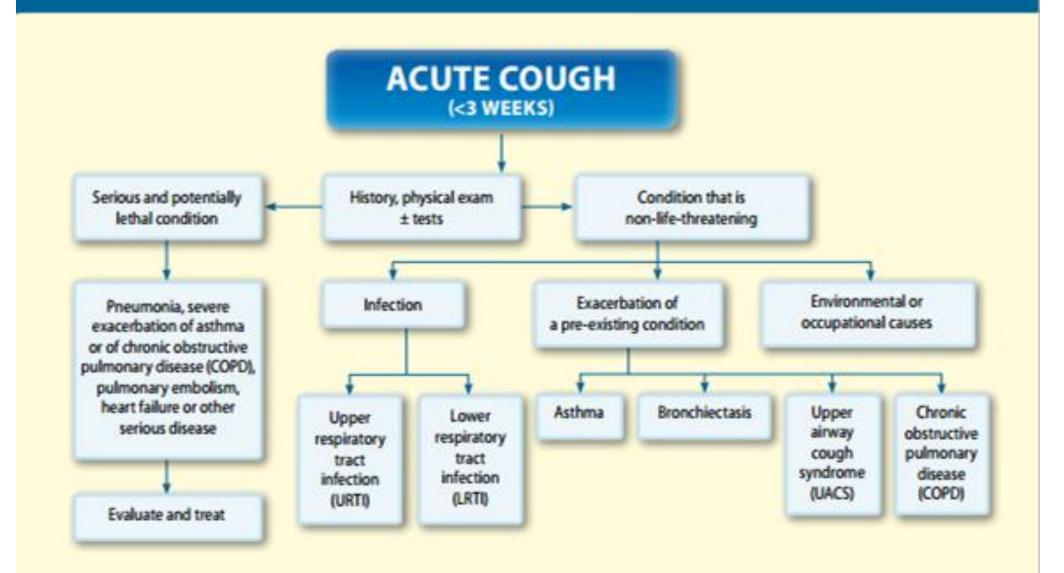
- Auditory canal
- Larynx
- Diaphragm
- Pleura
- Pericardium
- Esophagus

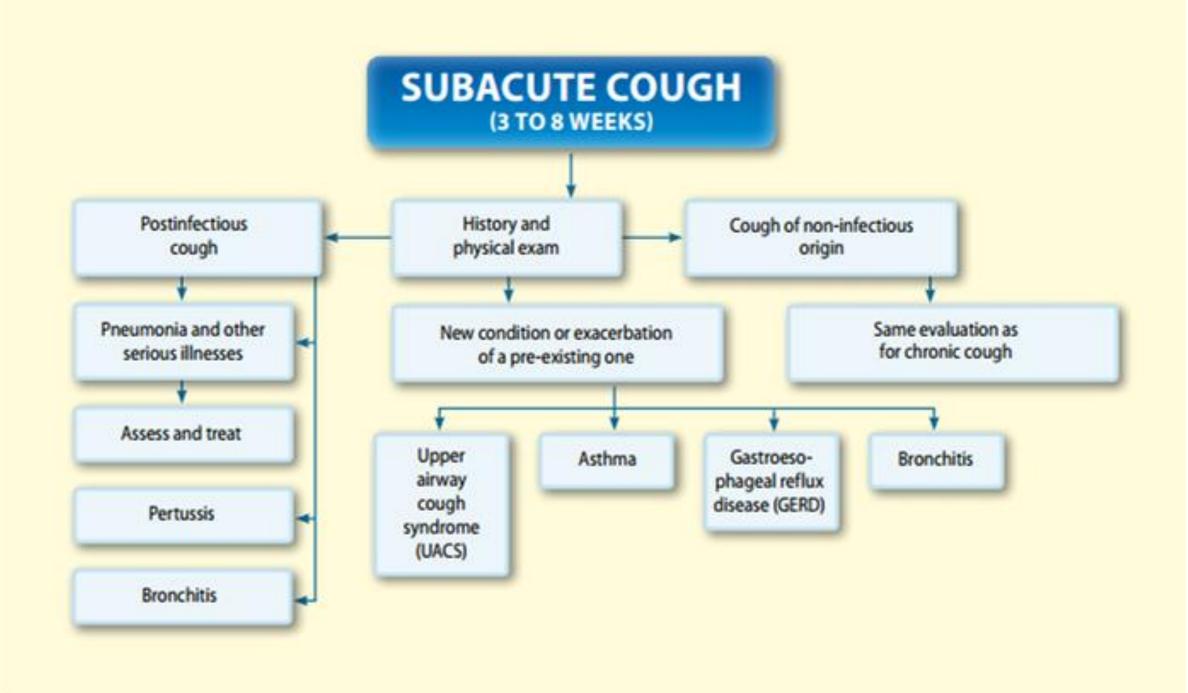
Psychogenic

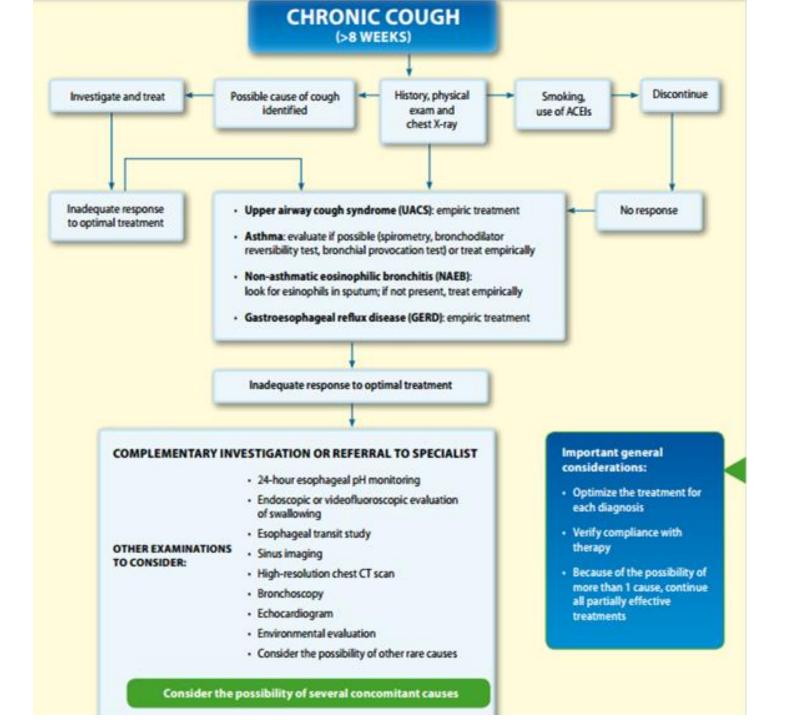
Approach to the Patient: Cough

- A detailed history
- Physical examination
- Chest radiography CXR
- Pulmonary function testing PFT
- Gross and microscopic examination of sputum
- High-resolution computed tomography (HRCT) or contrast CT
- Fiberoptic bronchoscopy

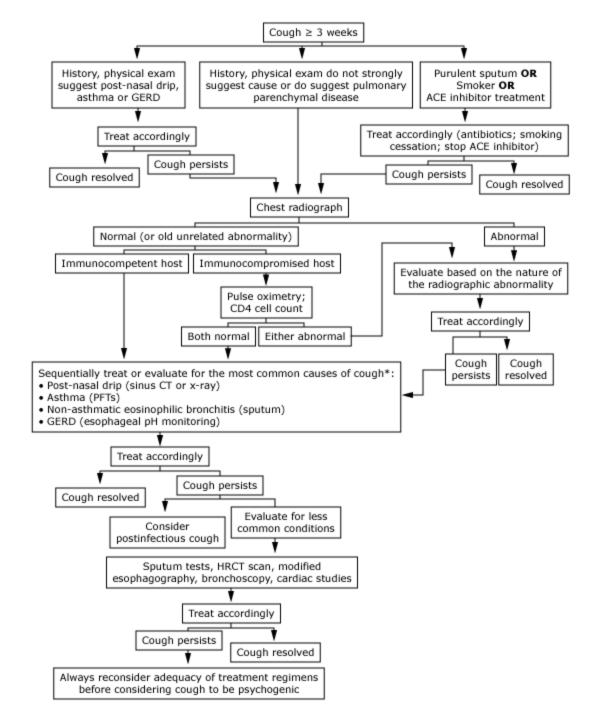
Assessment Algorithms

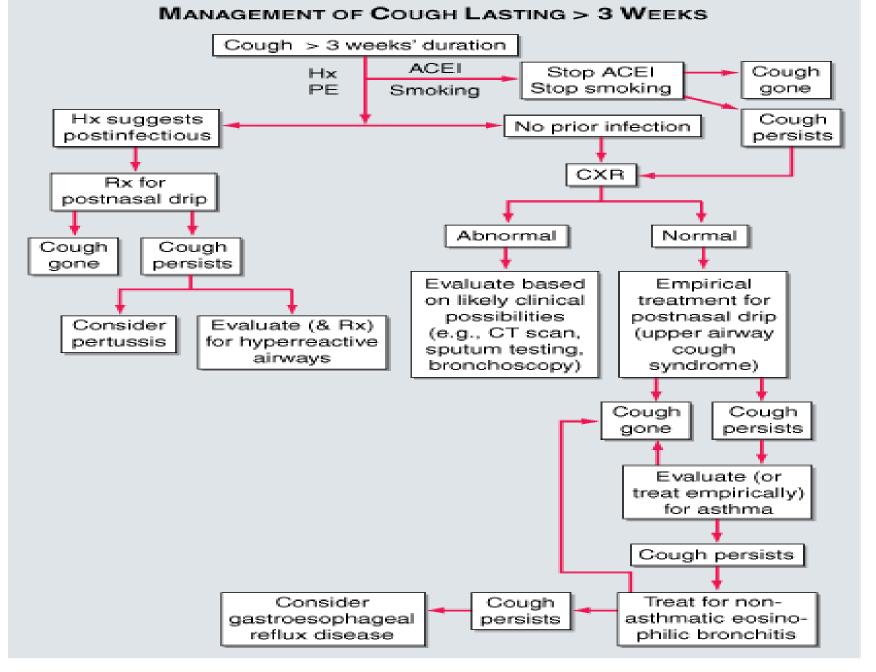






Algorithm For evaluation of subacute and Chronic cough





Source: Fauci AS, Kasper DL, Braunwald E, Hauser SL, Longo DL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 17th Edition: http://www.accessmedicine.com Copyright © The McGraw-Hill Companies, Inc. All rights reserved.

COUGH CHARACTERISTICS ACCORDING TO ETIOLOGY

Etiology	Characteristics					
Cough caused by medication	- Generally, it is non-productive and usually resolves within 4 weeks of stopping medication. - Affects 5-20% of patients receiving an angiotensin converting enzyme inhibitor (ACEI). However, it has not been reported with ARBs (angiotensin receptor blockers). - Beta-blockers may be implicated in chronic cough, often by aggravating underlying asthma.					
Upper airway cough syndrome (UACS)	Rhinosinusitis and other upper airways disorders are the most common etiologies of chronic cough. This syndrome used to be called postnasal drip syndrome. The symptoms are often subtle (e.g. sensation of secretions in the back of the throat or impression of imitation of the upper airways). Examination may reveal signs of pharyngitis. Sinus X-rays or CT scans may indicate signs of sinusitis. The cough will diminish with nasal corticosteroids, a first- or latest-generation antihistamine or nasal anticholinergic, depending on the cause. Nasal saline rinses may also be helpful.					
Asthma	Cough may be its only symptom. In the presence of suggestive symptoms, diagnosis should be confirmed by the demonstration of variable airway obstruction. The atopic history and ideally allergy	Parameter FEV ₂ : forced expiratory volume in 1 second PEF: peak expiratory flow measured with a	administration of a bronchodilator > >20% after 10 to 14 days of treatment with inhaled or oral conticosteroids			
	skin tests should be documented. - The cough will respond to asthma treatment, usually inhaled corticosteroids.	flow meter such as the Mini-Wright Methacholine challenge test: The PCso methacholine is the concentration required to cause a 20% drop in FEV ₃	or after repeated tests CP20 <8 mg/ml (Juniper method) The American Thorack Society considers that there is a grey zone between 4 and 16 mg/ml			
Gastroesophageal reflux disease (GERD)	Reflux can be the cause of the cough or its consequence. It may or may not cause typical symptoms such as heartburn or regurgitation. It is 1 of the 3 most common causes of chronic cough, and is responsible for approximately 25% of cases. It is recommended that the first diagnostic test be an evaluation of the response to empiric reflux treatment, such as a proton pump inhibitor. Esophageal pH monitoring can sometimes be useful; however, the pH can be normal if the reflux is nonacidic (alkaline reflux).					
Chronic bronchitis	 Cough secondary to tobacco dependence is often considered normal for smokers. In the absence of significant respiratory function abnormalities, it resolves 2 to 3 weeks after smoking cessation. In long-term smokers, the possibility of cancer and COPD should be excluded. Chronic bronchitis is defined as a cough with sputum expectoration for at least 3 consecutive months for at least 2 consecutive years. 					
Non-asthmatic eosinophi- lic bronchitis (NAEB)	A difficult diagnosis for the primary care physician to make. Patients present with bronchial eosiniphilia on sputum analysis, without bronchial hyperresponsiveness. The cough responds to inhaled corticosteroids, just as it does in asthma.					
Post-infectious cough	 A respiratory infection is often the cause of this acute or subacute cough. It accounts for about 15% of chronic cough cases; thus other diagnoses must be excluded. Generally the cough is non-productive and resolves in several weeks. Chest and sinus X-rays are usually normal. 					

Cough: Treatment

- Definitive treatment of cough depends on determining the underlying cause and then initiating specific therapy.
- Elimination of an exogenous inciting agent (cigarette smoke, ACE inhibitors) or an endogenous trigger (postnasal drip, gastro esophageal reflux).
- Empirical approach to treatment is with an antihistaminedecongestant combination, nasal glucocorticoids, or nasal ipratropium spray to treat unrecognized postnasal drip

Nonspecific therapy; Cough

- The cause of the cough is not known or specific treatment is not possible, and
- The cough performs no useful function or causes marked discomfort or sleep disturbance.

An irritative, nonproductive cough may be suppressed by an antitussive agent, which increases the latency or threshold of the cough center.

Such agents include codeine (15 mg qid) or nonnarcotics such as dextromethorphan (15 mg qid).

PRINCIPAL TREATMENTS FOR COUGH, ACCORDING TO ETIOLOGY

Possible causes	Treatments				
Acute or subacute cough (0-8 weeks)					
Upper respiratory tract infection (usually viral)	- First-generation antihistamines + oral decongestants - If subacute: among the therapeutic options, ipratropium and short-acting corticosteroids are good choices				
Acute sinusitis	First-generation antihistamines + oral decongestants + antibiotics as needed				
Asthma exacerbation	- Introduce or increase inhaled corticosteroids, inhaled 62-agonists - Oral corticosteroids if severe				
COPD exacerbation	Bronchodilators, oral corticosteroids, antibiotics				
Pertussis	Antibiotic (macrolide) – isolate for 5 days				
Chronic cough (>8 weeks)					
Asthma	Environmental control and education, bronchial anti-inflammatories, bronchodilators, regular follow-up, action plan				
Bronchiectasis	Bronchial toilet, bronchodilators*, treatment of secondary infections, surgery* if localized and infections are frequent				
Chronic bronchitis	Smoking cessation, avoid respiratory irritants, bronchodilators, corticosteroids*				
Non-asthmatic eosinophilic bronchitis	Inhaled corticosteroids				
UACS	First-generation antihistamines + oral decongestants or nasal ipratropium				
Allergic rhinitis	Topical nasal corticosteroids, latest-generation antihistamines				

Non-allergic rhinitis	Inhaled nasal corticosteroids, nasal ipratropium			
Chronic rhinosinusitis/Nasal polyposis	Inhaled nasal (oral*) corticosteroids, ENT assessment (surgery*)			
Gastroesophageal reflux disease	Diet, refrain from eating or drinking 2 hours before bedtime. Avoid alcohol, caffeine, smoking, theophylline, calcium channel blockers, anticholinergics and NSAIDs. Elevate the head of the bed by 10 to 15 cm. Lose weight if obese. Possible medications: proton pump inhibitors, antacids, H2-receptor antagonists, sucralfate, prokinetic agents			
Cough and pulmonary neoplasia	Treatment of the cause – smoking cessation – non-specific antitussives if too difficult			
Cough caused by medication	Stop the medication that is responsible			
Postinfectious cough	- May resolve on its own - First-generation antihistamines + oral decongestants, inhaled corticosteroids or nasal ipratropium may help			
Psychogenic or habit cough	Psychotherapy, non-specific antitussive treatment for a short period*			

^{*} Indicates a therapeutic option that may be useful in certain patients

Note: Non-specific antitussives may sometimes be used for a very short period if the cough is very debilitating and disrupts sleep.

Hemoptysis

Definition:

Hemoptysis:

Expectoration of blood from the respiratory tract

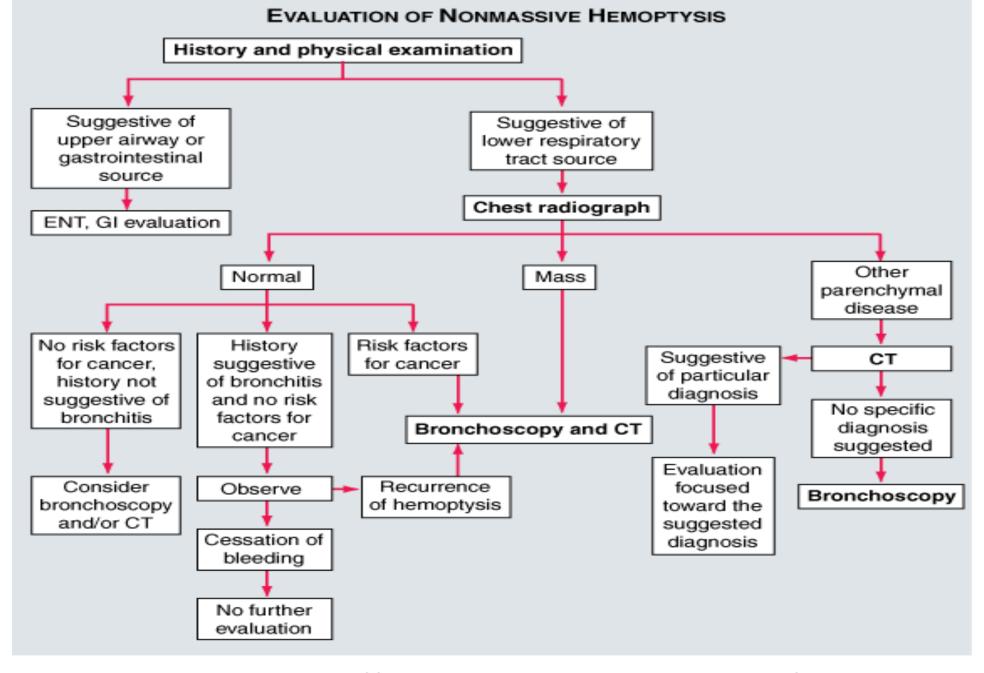
Massive hemoptysis:

Expectoration of >100–600 mL over a 24-h period

Etiology: Hemoptysis? Hematemesis?

 It is important to determine initially that the blood is not coming from alternative sites.

- ✓ Dark red appearance versus bright red appearance.
- ✓ An acidic pH, in contrast to the alkaline pH of true hemoptysis.



Source: Fauci AS, Kasper DL, Braunwald E, Hauser SL, Longo DL, Jameson JL, Loscalzo J: Harrison's Principles of Internal Medicine, 17th Edition: http://www.accessmedicine.com

Copyright @ The McGraw-Hill Companies, Inc. All rights reserved.

Differential Diagnosis(1)

Tracheobronchial source

- Neoplasm (bronchogenic carcinoma, endobronchial metastatic tumor, Kaposi's sarcoma, bronchial carcinoid)
- Bronchitis (acute or chronic)
- Bronchiectasis
- Broncholithiasis
- Airway trauma
- Foreign body

Differential Diagnosis(2)

Pulmonary parenchymal source

- Lung abscess
- Pneumonia
- Tuberculosis
- Mycetoma ("fungus ball")
- Goodpasture's syndrome
- Idiopathic pulmonary hemosiderosis
- Wegener's granulomatosis
- Lupus pneumonitis
- Lung contusion

Differential Diagnosis(3)

Primary vascular source

- Arteriovenous malformation
- Pulmonary embolism
- Elevated pulmonary venous pressure (esp. mitral stenosis)
- Pulmonary artery rupture

Miscellaneous/rare causes

- Pulmonary endometriosis (catamenial hemoptysis)
- Systemic coagulopathy or
- Use of anticoagulants or thrombolytic agents

Approach to the Patient

- History (acute, chronic, drugs...)
- Previous or coexisting disorders
- Physical examination
- Chest radiograph
- Lab. (complete blood count, a coagulation profile, Gram and acid-fast stains
- Fiberoptic bronchoscopy or Rigid FB.
- HRCT (suspected bronchiectasis)

Hemoptysis: Treatment

- The rapidity of bleeding
- Gas exchange
- Massive or blood-streaking
- Partially suppressing cough
- Isolation of the right and left mainstem bronchi by double-lumen endotracheal tubes
- Inserting a balloon catheter through a bronchoscope
- Laser phototherapy, electrocautery
- Bronchial artery embolization
- Surgical resection of the involved area of lung (for the life-threatening hemoptysis)



Clinical pathophysiology

O respiratory system dyspnea: includes discomfort related to disorders of the central controller the ventilatory pump the gas exchanger

Clinical pathophysiology

- cardiovascular system dyspnea.
 Includes
- cardiac diseases (eg, acute ischemia, systolic dysfunction, diastolic dysfunction, pericardial diseases)
- anemia

The language of Dyspnea

- Acute hypercapnia or restricted thoracic motion produces a sensation of "air hunger".
- Acute bronchoconstriction leads to a series of sensations from "chest tightness" to an increased "effort to breathe" to a sensation of "air hunger" as the degree of obstruction worsens. The sensation of "tightness" appears to be independent of the work of breathing

The language of Dyspnea

- Patients with COPD complain of an increased "effort to breathe" as well as a sensation of "unsatisfying breaths" or a sense that they "cannot get a deep breath".
- Cardiovascular deconditioning is characterized by "heavy breathing".

Dyspnea: Different Diagnosis

- Airway disease: Upper airway obstruction, Asthma, COPD
- Lung parenchyma: Pneumonia,
 Interstitial lung disease, malignancy,
 non-cardiogenic lung edema
- Pulmonary vascular disease: Pulmonary embolism, Arterio-venous malformation, vasculitis, veno-occlusive disease

Dyspnea: Different Diagnosis

- Neuromuscular disease
- Cardiovascular disease: low cardiac output (CHF), right to left shunt, pulmonary hypertension
- Anemia
- Psychological problem: anxiety, hyperventilation
- Other systemic conditions: sepsis, metabolic acidosis.

History Taking

- Frequency
- Intensity
- Duration
- Daily activity
- Predisposing factor
- Associated symptom
- Past History

History Taking: Associated symptom

- Cough
- Sputum: character
- Chest pain
- Fever
- Strider

History Taking: Predisposing Factor

- Weather/Season
- Posture
- Day and night
- Exercise

History Taking: Past Hx

- Occupation
- Drug
- Allergy
- Smoking
- Underlying disease

BEHAVIORAL CONSIDERATIONS

- Anxiety, anger, pain, and depression may be associated with dyspnea intensity out of proportion to the physiologic impairment.
- Increased ventilation associated with anxiety, anger or pain may push an individual with <u>a</u> limited pulmonary reserve at baseline closer to his or her ventilatory limits and increase respiratory discomfort for any given activity.

Physical Examination

- 1. Respiratory rate, blood pressure, pulse rate, cyanosis, clubbing finger
- 2. Chest palpation, percussion, auscultation (crackle, wheeze, heart murmur, S3)
- 3. General appearance, peripheral edema

Lab exam and Image

- Pulse oximeter
- * ABG
- CxR
- **EKG**
- Lung function test
- Ventilation perfusion scan

Approach of Acute Dyspnea

DD of Acute Dyspnea

pulmonary

- *pulm edema
- *asthma/COPD
- *PE
- *pneumonia
- *pneumothorax

non-pulmonary (cardiac)

- *arrhythmias
- *acute MI
- *myocardial ishemia

Dx of exclusion: anxiety

Initial Management

- 1. Secure the airway
- 2. Oxygen therapy
- 3. Reduces ventilatory demand: bed rest, anti-pyretics in febrile patient, sedative agent in anxiety patient, painkiller use.

Initial Management

- 4. Bronchodilator and/or steroid (airway disease)
- 5. Diuretics (pulmonary edema)
- 6. "MONA " therapy (acute coronary syndrome)
- 7. Treat underlyling disease

Oxygen Therapy

- O2 canula (FiO2 24-40%): no work when open mouth breathing
- Venturi mask
- Aerosol mask
- Non-rebreathing mask (FiO2 > 90%)

Mechanical Ventilation

- Indicated while respiratory failure developed
- NIPPV (BIPAP)
- Traditional MV via endotracheal/tracheostomy tube

Approach to the Patient with Chest pain

- Goal of evaluation:
 - 1. determine the diagnosis
 - 2. assess the safety of the immediate Mx
- Past medical history provides valuable information for DDx

Causes of chest pain- cardiac

- Stable angina
- Rest or unstable angina
- Myocardial infarction
- Pericarditis
- Aortic stenosis
- Aortic dissection
- Pulmonary embolism
- Pulmonary hypertension

Causes of chest pain- noncardiac

- Pneumonia, pleurisy
- Pneumothorax
- Esophageal reflux
- Boerhaave's syndrome
- Peptic ulcer
- Gallbladder disease
- Musculoskeletal disorder
- Herpes zoster
- Anxiety states

Considerations in the assessment

1. Chest pain due to acute, potential life- threatening condition?

ACS/ AMI, Aortic dissection Pulmonary embolism Pneumothorax

2. Chest pain due to chronic condition likely to lead to Pulmonary hypertension serious complications?

Aortic stenosis
Pulmonary hypertension
Stable angina

3. Other acute condition need specific Tx?

Pericarditis
Pneumonia
Herpes zoster...

4. Other chronic condition...

Acute chest discomfort

- First assess respiratory & hemodynamic status
 - → if compromised, stablize pt first
 - → if emergent Mx not needed, start taking Hx, PE, +/- Lab
 - always r/o potential life-threatening conditions first

History

- Onset
 - sudden /acute
 - gradual
- Duration
 - 10-20min
 - > 30min
 - prolonged
- Aggravating/ relieving factor
 - exercise, cold
 - position change
 - SL NTG

- Location / radiation
 - retrosternal
 - to jaw, shoulder
 - to back
- Qaulity
 - sharp / burning
 - dull, pressure, squeezing
 - pleuritic
- associated symptoms
 - cold sweating
 - dyspnea

Physical exam

- Blood pressure / pulsation, four limbs
- Auscultation
 - diminished breathing sound / rales
 - friction rub
 - murmurs, S3, S4
- palpation
 - tender point
 - Murphy's sign / epigastric / RUQ tenderness

Lab tests

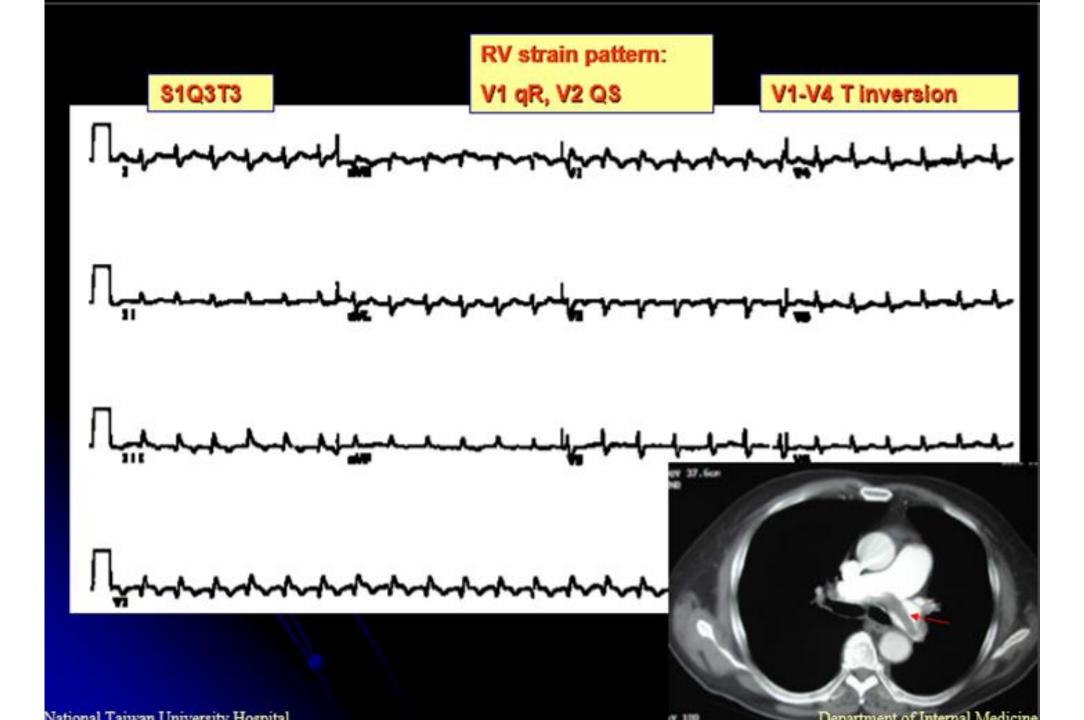
- ECG
- CXR
- Cardiac markers: CK, CK-MB, Tnl
 - singles negative value do not have high sensitivity for AMI or predicting Cx -> should not discharge pt only based on single negative result
- Chest CT
- Cardiac echo

Clinical features - AMI

- History
 - Acute/ sudden onset; often > 30min
 - Pressure / squeezing
 - Retrosternal, radiate to jaw/ neck/ shoulder
- PE:
 - may be totally normal in AMI
 - signs of HF: S3, S4, basal rales;
 - signs of low cardiac output / perfusion
- Lab:
 - ECG: ST elevation, new LBBB
 - CK/ CK-MB/ Tnl

Clinical features – Pulm embolism

- History
 - sudden onset
 - substernal or region of pulm infarct
 - pleuritic or angina-like
 - pain may ↑ if deep breathing
- PE:
 - friction rub over the region of pulm infarct
 - signs of acute RV failure: BP ↓, JVE, P2 ↑
 - signs of DVT: lower leg painful swelling
 - signs of desaturation
- Lab:
 - ECG / CXR / Chest CT/ Lung scan



Clinical features – aortic dissection

- History
 - sudden onset
 - anterior of chest, radiate to back
 - sharp, tearing pain
- PE:
 - four limb pulsation / BP
 - murmur of AR (involving aortic root)
 - neurologic deficit
 - signs of tamponade
- Lab:
 - CXR / Chest CT



F1: 12.100 730



Clinical features – pneumothorax

- History
 - sudden onset
 - pleuritic pain
 - lateral to side of pneumothorax
- PE:
 - respiratory distress, desaturation
 - hyperinflation, tympanic on percussion
 - decreased breathing sound
 - hypotension if tension pneumothorax
- Lab:
 - CXR

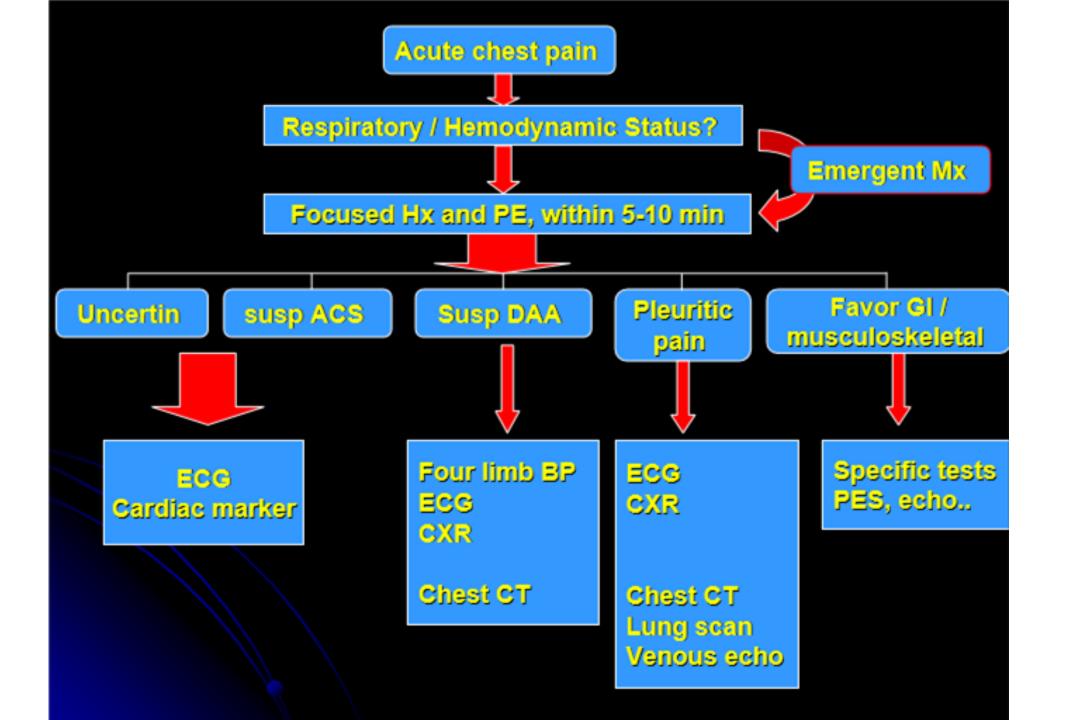




Clinical features - Pericarditis

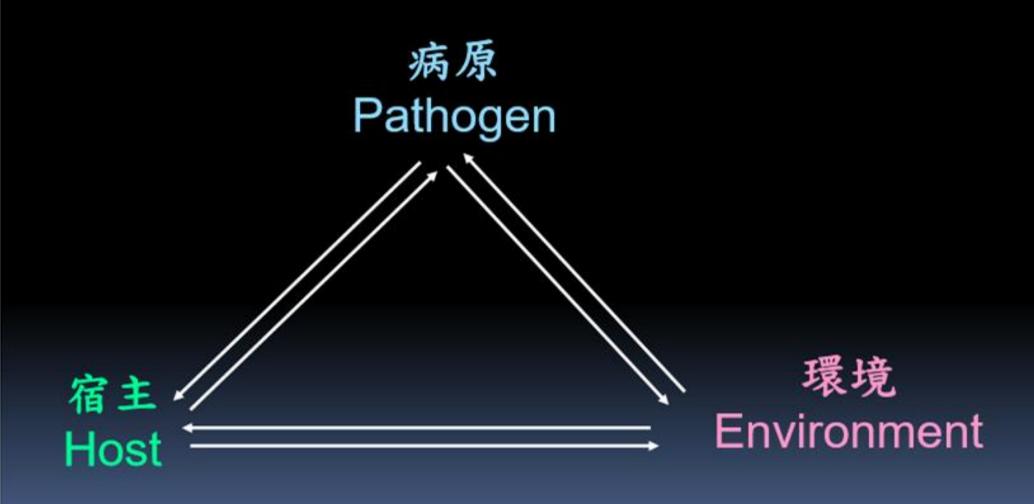
- History
 - lasts many hours to days; may wax and wane
 - sharp pain
 - pain ↑ if deep breathing / supine position
 - ↓ if sitting up and leaning forward
- PE:
 - pericardial friction rub
 - signs of tamponade if much fluid amount:
 JVE, pulsus paradoxus, hypotension
- Lab:
 - ECG\\ CXR
 - Cardiac echo



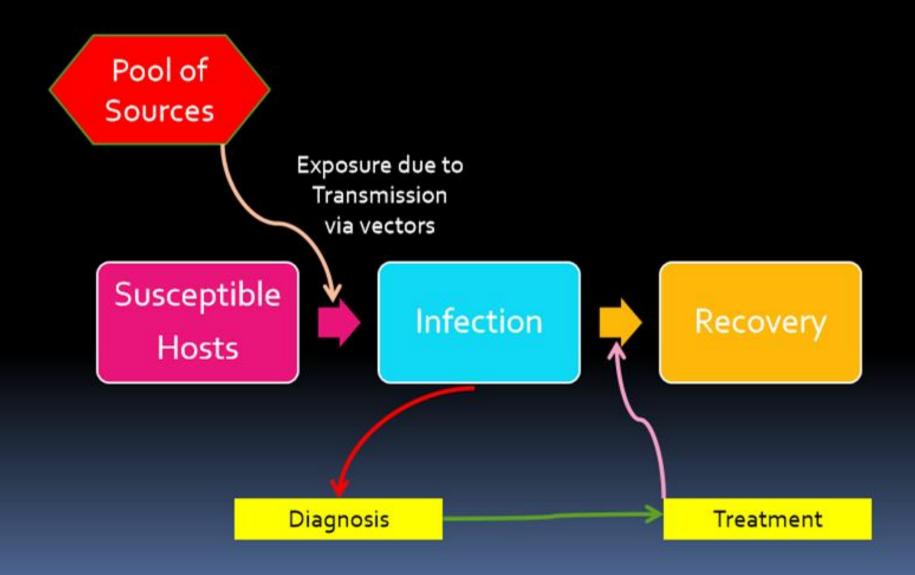


INFECTIOUS DISEASES

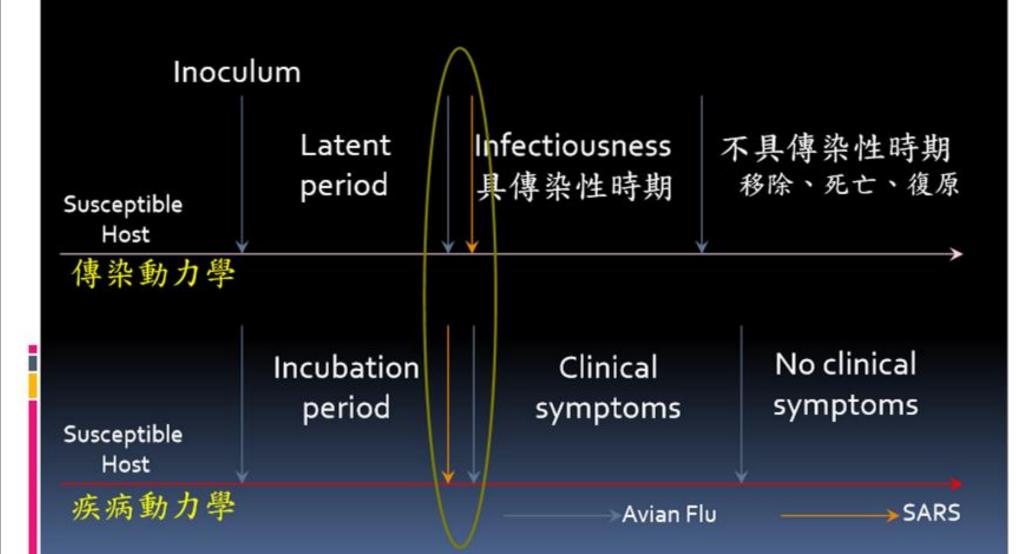
Infectious Diseases到底有什麼不一樣?



Basic Model of Infectious Diseases



Time-line of Infectious Diseases



Managing Infectious Diseases

- 減少susceptible host:
 - The concept of R_o
- Blocking transmission
- Diagnosis
 - History, physical examination
 - Laboratory diagnosis
- Effective treatment:
 - Initiation of effective antimicrobial agents
 - Evaluation of the clinical response

Reducing Susceptible Hosts

- Community settings
 - □ 基礎再生數(R_o)決定疾病的持續流行與否
 - 一個可傳染宿主在其可傳染期間於一個完全易 感受的族群中預期可產生的新感染宿主個數
 - <1: 流行將不繼續

Vaccination

每次接觸的 傳播機率

X可傳染期間

isolation

PPE PEP

Treatment

Reducing Susceptible Hosts

- Healthcare setting:
 - Removal of special risk factors
 - Invasive procedure and therapy
 - Catheterization
 - CVCs, urinary catheter, draining tube, etc....
 - Immunuosuppressive agents
 - DM control
 - etc...
 - Standards of operations
 - Good nutrition
 - Prophylaxis

Blocking Transmissions

- Source control:
 - prions, virus, bacterial, fungus, parasite
 - Quarantine, isolation, disinfection
- Transmission blockade:
 - Airborne, droplet, contact, vector-borne
 - Social distance
 - Isolation precaution
 - Vector control:
 - Dengue fever, malaria, etc...

Diagnosis

- History:
 - Symptoms, onset temple
 - Fever pattern:
 - Not a good indicator
 - Exposure history:
 - Travel, occupation, sexual exposure, pet, residential
 - Family history
 - Preexisting medical / surgical conditions
 - Both diseases and medication

Evaluation of Fever

- Cause of fever:
 - Infectious diseases
 - Environment factors
 - Air condition, heat stroke, hot light
 - Drug fever:
 - Antimicrobial agents, anti-convulsants
 - Endocrine diseases:
 - Adrenal insufficiency, hyperthyroidism

Evaluation of Fever

- Cause of fever (continued):
 - Neoplasm:
 - Hematology: leukemia, lymphoma
 - Solid tumor: RCC, HCC, central necrosis
 - Inflammatory diseases:
 - Rheumatology / autoimmune diseases
 - Pancreatitis, chemical induced inflammation
 - Miscellaneous

Physical & Laboratory Examinations

- PE:
 - □ 從頭到尾別放過
- Laboratory examination:
 - Hemogram with differential count
 - BCS, remember ALP/γGT, LDH
 - Blood cultures, body fluid/secretion cultures
 - PCR and rapid test (antigen based)
 - Sometimes CRP or procalcitonin
 - Helpful to differentiate bacterial infection from others

The Role of Physicians

- Sensitivity / specificity means:
 - Nothing when the disease prevalence is low
 - Low positive predictive value
- How to face?
 - P_{post}=P_{prior} * [sensitivity/(1-specificity)]
 - Pfinal=Pprior_1*Pprior_2*Pprior_3*-----Pprior_n

Rothman & Greenland. Modern Epidemiology

Hemogram and Differential Count

Anemia:

- Chronic ill related
- Hemolysis, especially acute infection

WBC count:

- Leukocytosis: common bacterial infection
 - Cytokine storm
- Leukopenia: atypical bacterial, virus
 - Severe sepsis

Platelet count:

- Thrombocytosis: chronic inflammation / diseases
- Thrombocytopenia: atypical infection, but also severe sepsis
- Pancytopenia: Look out hemophagocytosis

New Marker for Bacterial Infection

- Procalcitonin:
 - Prehormone of calcitonin
 - Secreted by C cell of thyroid while hypercalcemia
 - Also secreted by liver and mononuclear cells of peripheral blood while stimulation of endotoxin and sepsis-related cytokine
 - Secreted within 4 hours, peak at 8 hours

Image Studies

- Plain film:
 - Sinus, CXR, KUB, etc...
- CT scan
- MRI
 - Bone and soft tissue
- Nuclear medicine
 - Bone scan
 - Ga-67 whole body inflammatory scan

Biopsy and Tissue Cultures

- The last step, but sometime it is the only method to obtain right diagnosis
- Earlier is better:
 - If too late:
 - Pathogens may not be easily found
 - Pathology become the final-common-pathway ones
 - Worse host condition to tolerate biopsy procedure
- Well explanation
- Dedicate management of the specimen

Usual Diagnosis

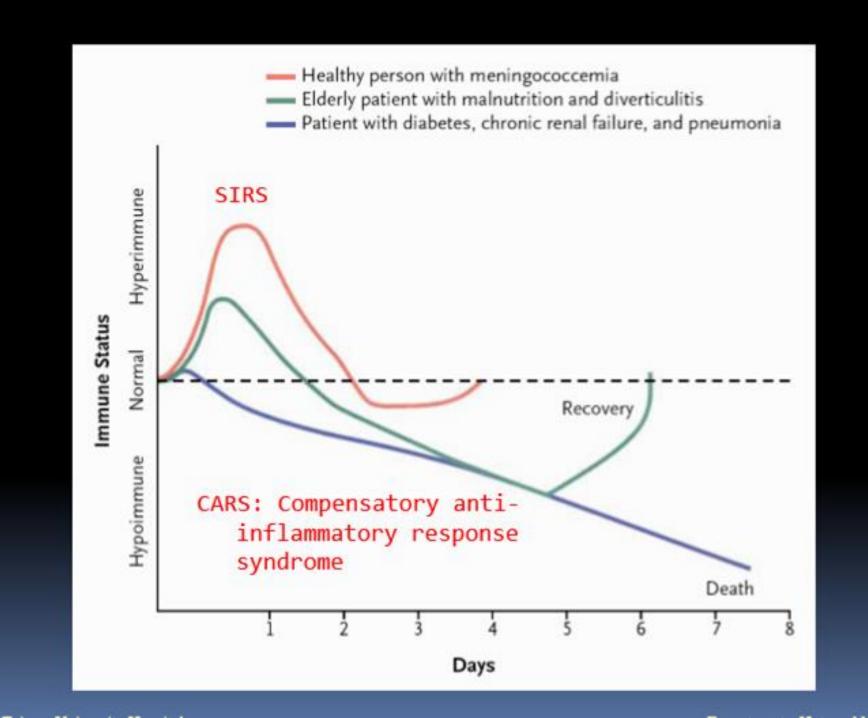
- Community settings:
 - Respiratory tract infection
 - Urinary tract infection
 - Skin and soft-tissue infection
 - Intra-abdominal infection
- Healthcare setting:
 - Bloodstream infection
 - Lower respiratory tract infection
 - Urinary tract infection
 - Surgical site infection

Principles of Treatment

- Removal of infection focus
- Timely effective antimicrobial agents
 - Bactericidal v.s. bacteriostatic
 - Empirical therapy is therefore important
 - Local epidemiology
 - Epidemiology of causative pathogen
 - Epidemiology of drug susceptibilities
 - Definite therapy while the result of microbiologic workup being available
 - Tolerability of host: adverse effects
 - Microenvironment and penetration

Treatment: Corticosteroid

- Only when poorly response to
 - Fluid resuscitation and vasopressor
 - No ACTH stimulation test: 不準
- Not every kind of steroid
 - Hydrocortisone is preferred, avoid dexamethasone
 - Adding 50 μg/day fludrocortisone if not hydrocortisone
- Taper steroid when vasopressor no longer needed



Epidemiology of Causative Pathogens

CAP (1): Lauderdale TL, et al. Resp Med 2005;99:1079-86

Table 2 Etiologic agents of 168 adult community acquired pneumonia patients requiring hospitalization in Taiwan.

Etiologic agent	Diagnostic category, No. (%)*		Total
	Definite	Probable	
Streptococcus pneumoniae	12 (7.1)	28 (16.1)	40 (23.8)
Haemophilus influenzae	2 (1.2)	6 (3.6)	8 (4.8)
Staphylococcus aureus	3 (1.8)	0	3 (1.8)
Beta-Streptococci	2 (1.2)	0	2 (1.2)
Gram-negative bacilli			
Klebsiella pneumoniae	5 (3.0)	3 (1.8)	8 (4.8)
Escherichia coli	3 (1.8)	0	3 (1.8)
Mycoplasma pneumoniae	24 (14.3)	:	24 (14.3)
Chlamydia pneumoniae	12 (7.1)	:	12 (7.1)
Legionella pneumophila	2 (1.2)	:	2 (1.2)
Influenza A virus	11 (6.5)	:	11 (6.5)
Respiratory syncytial virus	2 (1.2)	:	2 (1.2)
Adenovirus	2 (1.2)	:	2 (1.2)
Parainfluenza virus	2 (1.2)	:	2 (1.2)
Mycobacteria tuberculosis	2 (1.2)	:	2 (1.2)
Total no. of patients (%) [†]	73 (43.5)	36 (21.4)	99 (58.9)

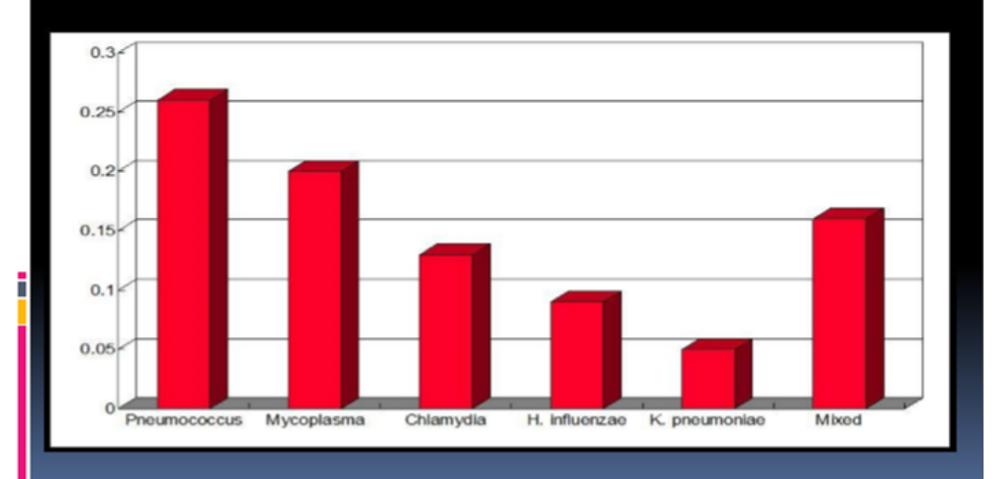
^{*}Values for mixed infections are included for each of the infecting organisms.

[†]Due to the presence of multiple organisms in some patients, the total number of patients is lower than the sum of patients for each agent.

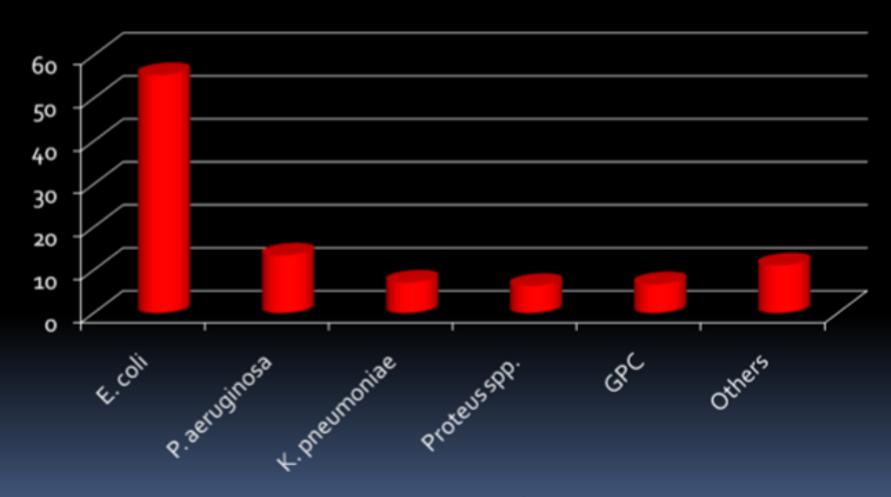
Diagnostic category not available for specific pathogen.

Etiology of CAP (2):

Yen MY, et al. J Formos Med Assoc 2005;104:724-30



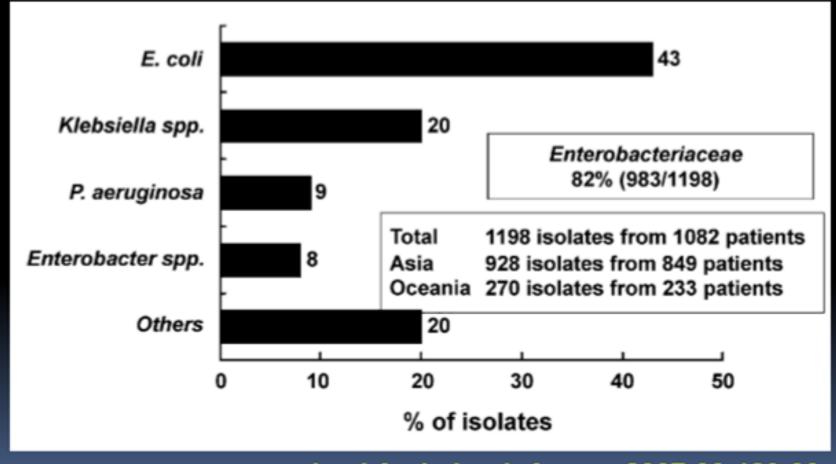
Etiology of Community UTI



Lau SM, Peng MY, Chang FY. JMII 2004;37:185-91.

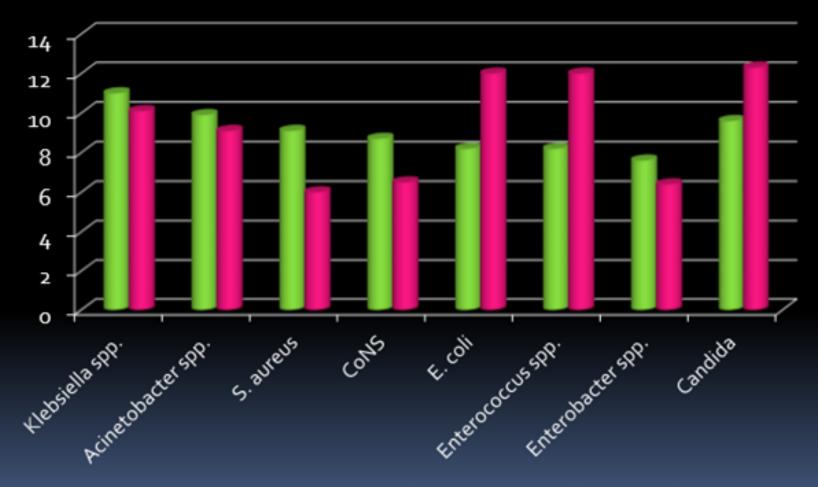
Etiology of Complicated IAI

IAI, SMART Study, Asia-Pacific area



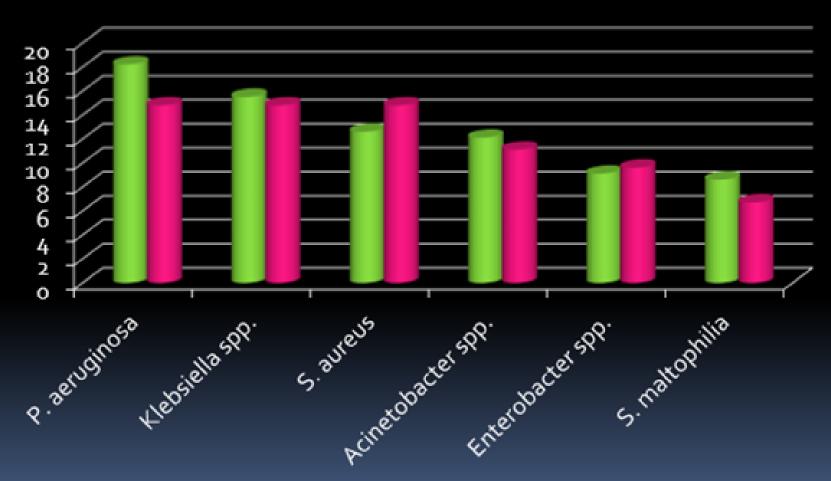
Int J Antimicrob Agents 2007;30:129-33

Bloodstream Infection, NTUH



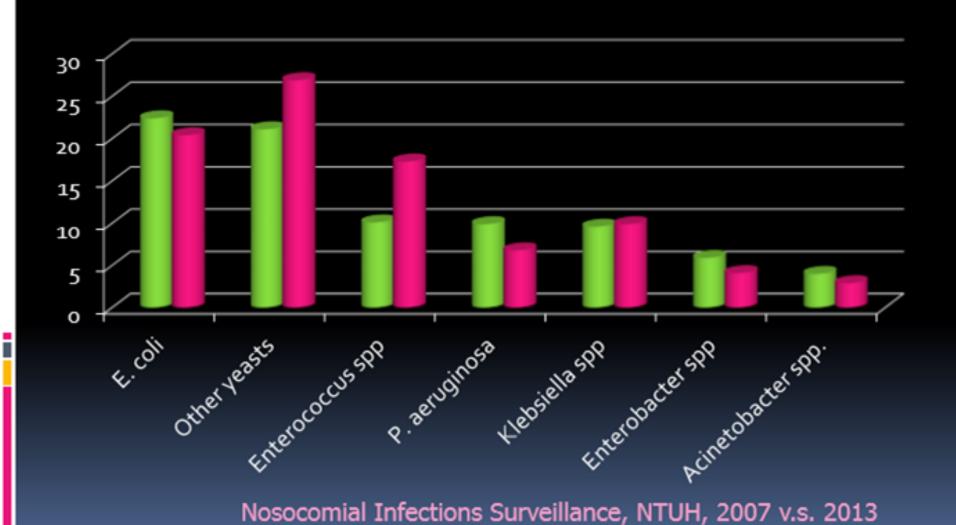
Nosocomial Infections Surveillance, NTUH, 2007 v.s. 2013

Respiratory Tract Infection, NTUH

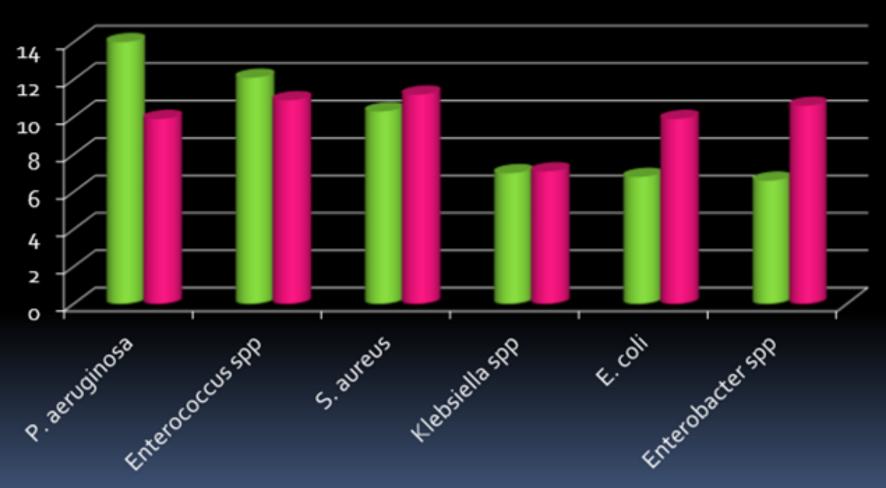


Nosocomial Infections Surveillance, NTUH, 2007 v.s. 2013

Etiology of Nosocomial UTI



Etiology of Nosocomial SSI



Nosocomial Infections Surveillance, NTUH, 2007 v.s. 2013

Drug Susceptibility Tests

- Why we need?
 - Using in vitro data to predict in vivo effectiveness
- Methods:
 - Disk diffusion method: most common used
 - Minimum inhibitory concentration (MIC):
 - Agar dilution method
 - Broth (micro- or macro) dilution method
 - E-test
 - Detection of enzymes or genes for resistance
 - Serum bactericidal test (SBT)

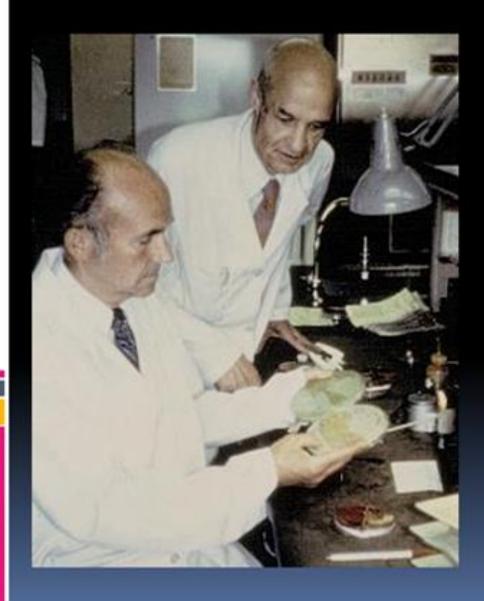
Purpose of Susceptibility Test

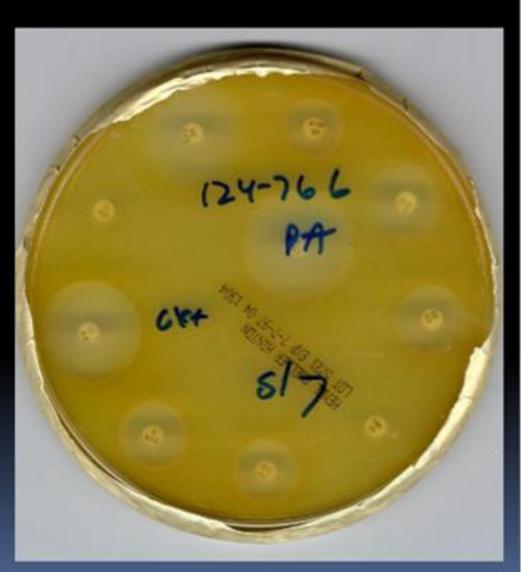
Predicting therapeutic efficacy

FO, favorable outcome; UFO, unfavorable outcome

Test	Normal host	Host with endocarditis, meningitis or immunodeficiency
Disk diffusion		
Susceptible (FO)	Excellent	Fair
Resistant (UFO)	Fair, except UTI	Excellent
MIC		
Susceptible (FO)	Excellent	Fair
Resistant (UFO)	Fair	Excellent

Disk Diffusion (Kirby-Bauer) Method





Disk Diffusion (Kirby-Bauer) Method

- Finally, based on correlation with MIC
 - S(usceptible): predict ≥85% clinical success
 - R(esistant): predict < 60% clinical success</p>
 - I(ntermediate): between S & R
- Only some bacteria have definitive interpretive standards

Minimum Inhibitory Concentration

- Not routinely available at any clinical laboratory
- E-test method is more easy to perform but more expensive
- Should be ordered only by ID specialists
- For deep seated infection:
 - Osteomyelitis, infective endocarditis, meningitis
- Not more than three agents in every request

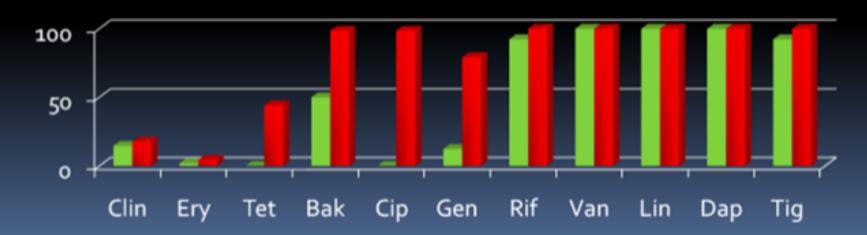
Serum Bactericidal Test

- More like in vivo environment:
 - Serum resistance to microorganism: humeral factors
- Both peak and trough time-points are checked
- Indication:
 - Infective endocarditis
 - Osteomyelitis
 - Meningitis
 - Other deep seated infections suggest by ID specialists

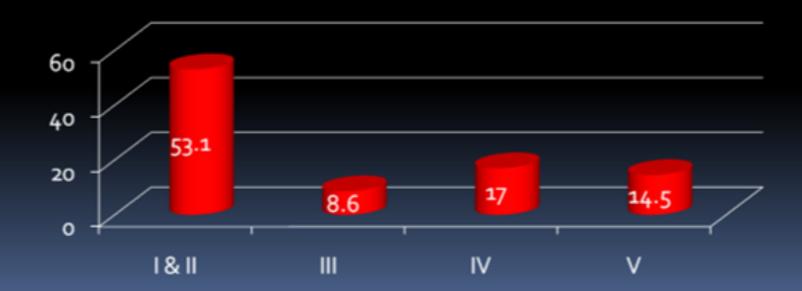
Interpretation of SBT Results

Peak titer	Trough titer	Interpretation
1:2	1:2	Inadequate
1:4 ~ 1:16	1:4	Intermediate
≥ 1:32	≥ 1:8	Adequate

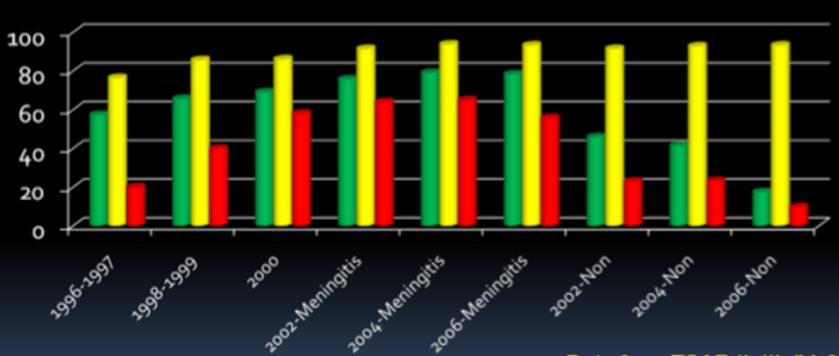
- S. aureus
 - For methicillin susceptible ones:
 - Resistant to penicillin
 - For methicillin resistant ones:



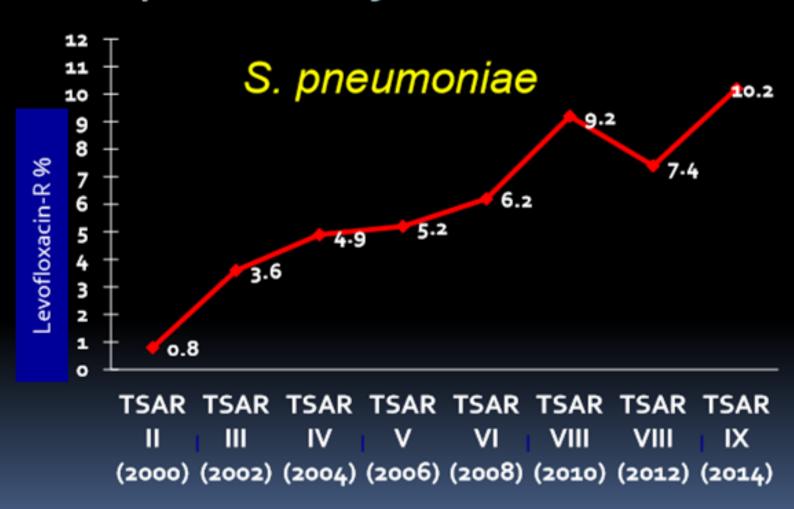
- Streptococcus pyogenes (Group A):
 - Still susceptible to nature penicillin
 - Look out the resistance to erythromycin



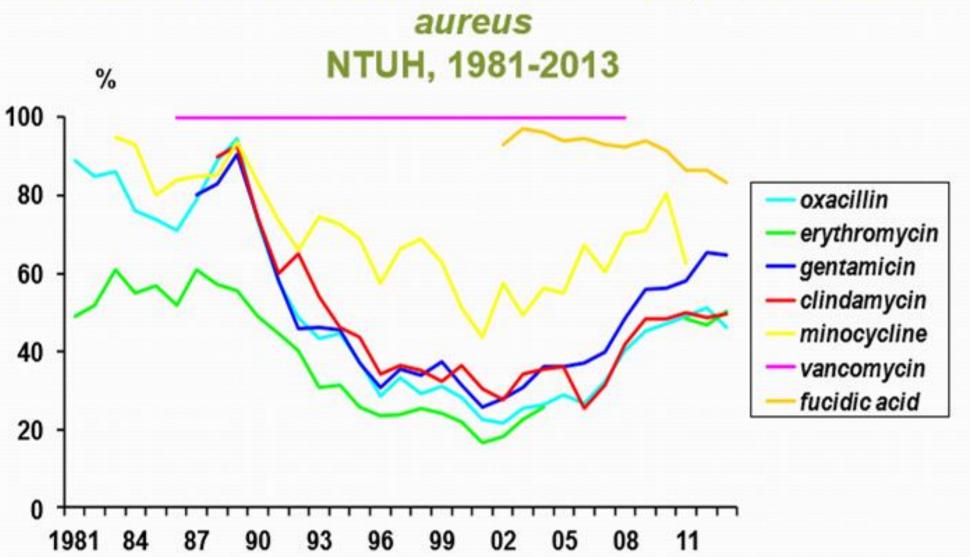




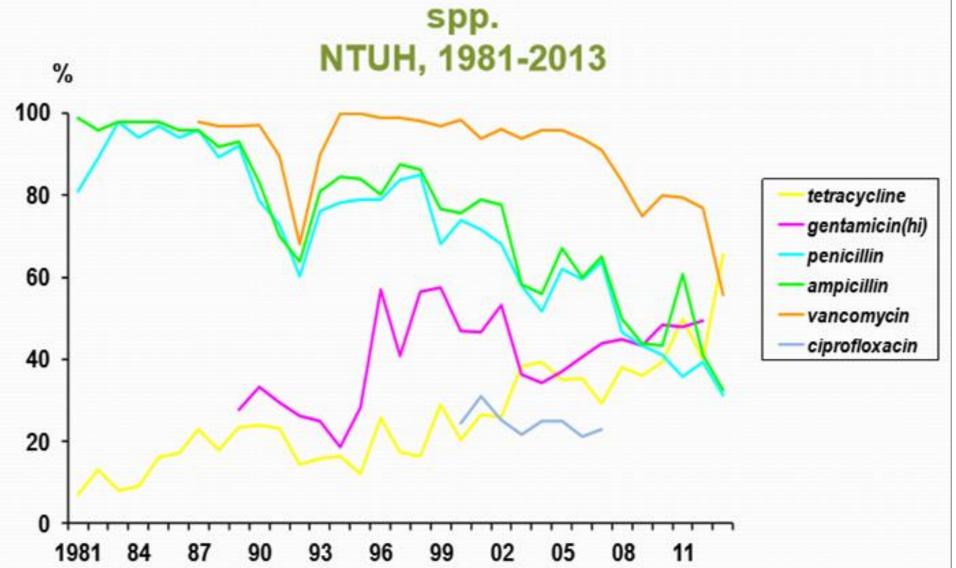
Data from TSAR II, III, IV, & V
Hsueh PR, et al. J Clin Microbiol 1999;37:897-901;
Fung CP, et al. J Antimicrob Chemother 2000;45:49-55;
Hsueh PR, et al. Antimicrob Agents Chemother 2000;44:1342-5
Siu LK, et al. Microb Drug Resist 2002:8:201-8.



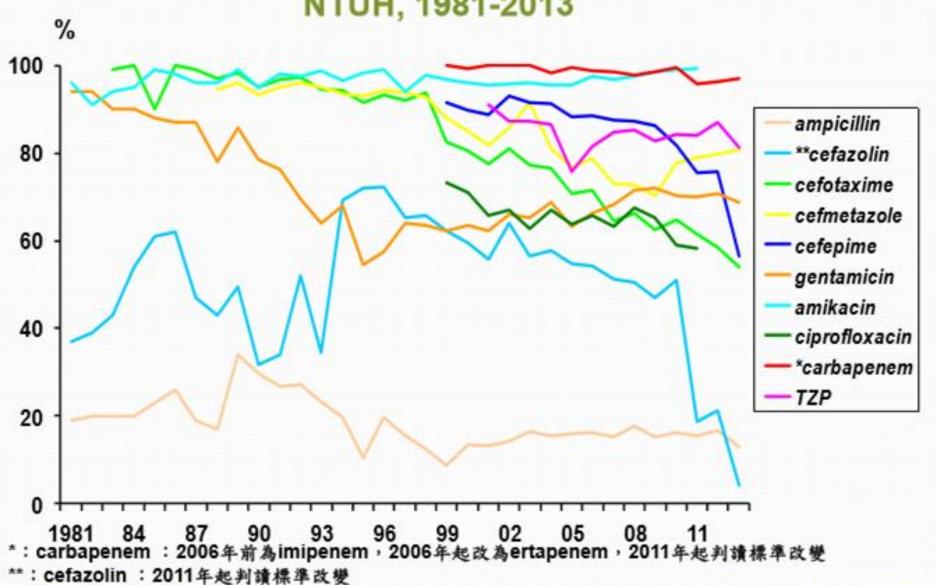
Antimicrobial Susceptibility of Health Care-Associated Strains of Staphylococcus



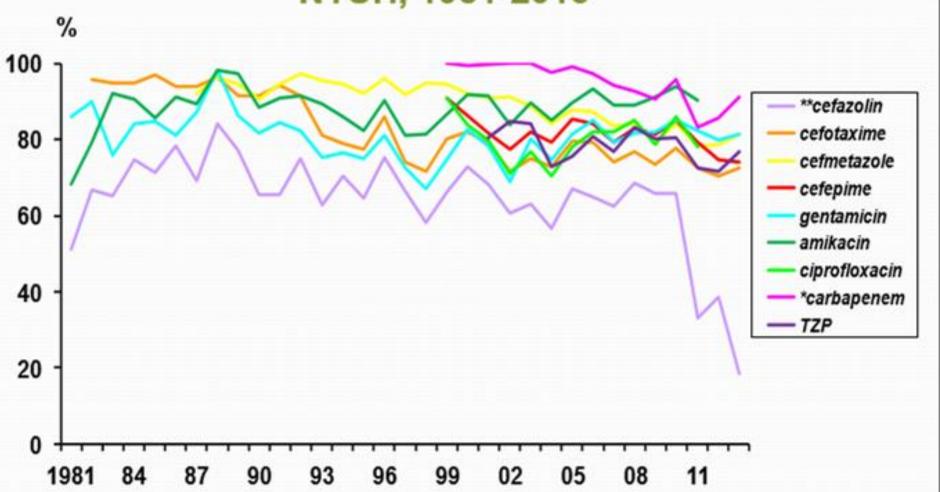
Antimicrobial Susceptibility of Health Care-Associated Strains of Enterococcus



Antimicrobial Susceptibility of Health Care-Associated Strains of *E.coli* NTUH, 1981-2013



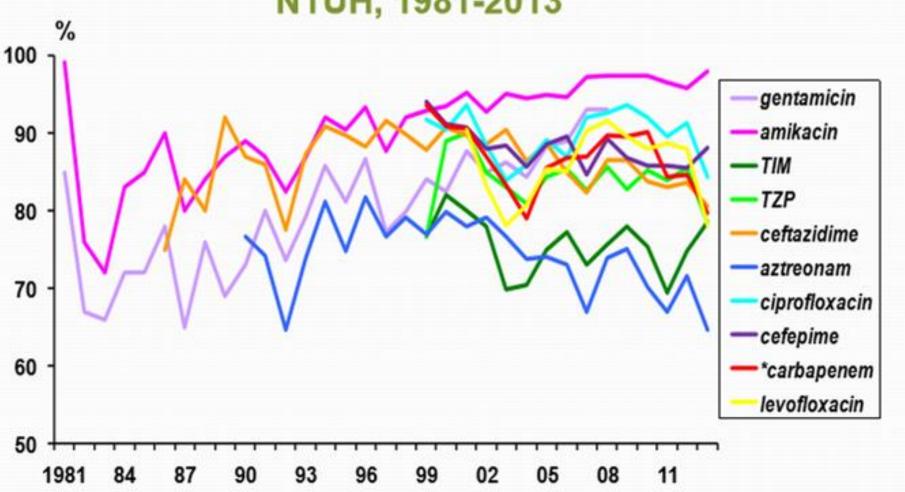
Antimicrobial Susceptibility of Health Care-Associated Strains of *Klebsiella* spp. NTUH, 1981-2013



^{*:} carbapenem: 2006年前為imipenem, 2006年起改為ertapenem, 2011年起判讀標準改變

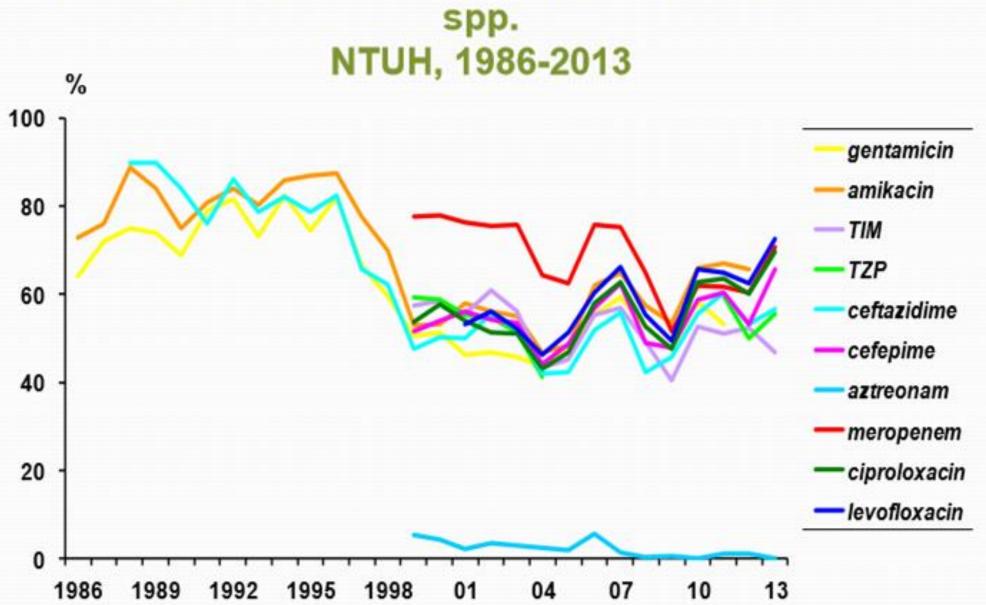
**: cefazolin: 2011年起判讀標準改變

Antimicrobial Susceptibility of Health Care-Associated Strains of Pseudomonas aeruginosa NTUH, 1981-2013



^{*:} carbapenem: 2004年前為meropenem, 2005年起為imipenem

Antimicrobial Susceptibility of Health Care-Associated Strains of Acinetobacter spp.



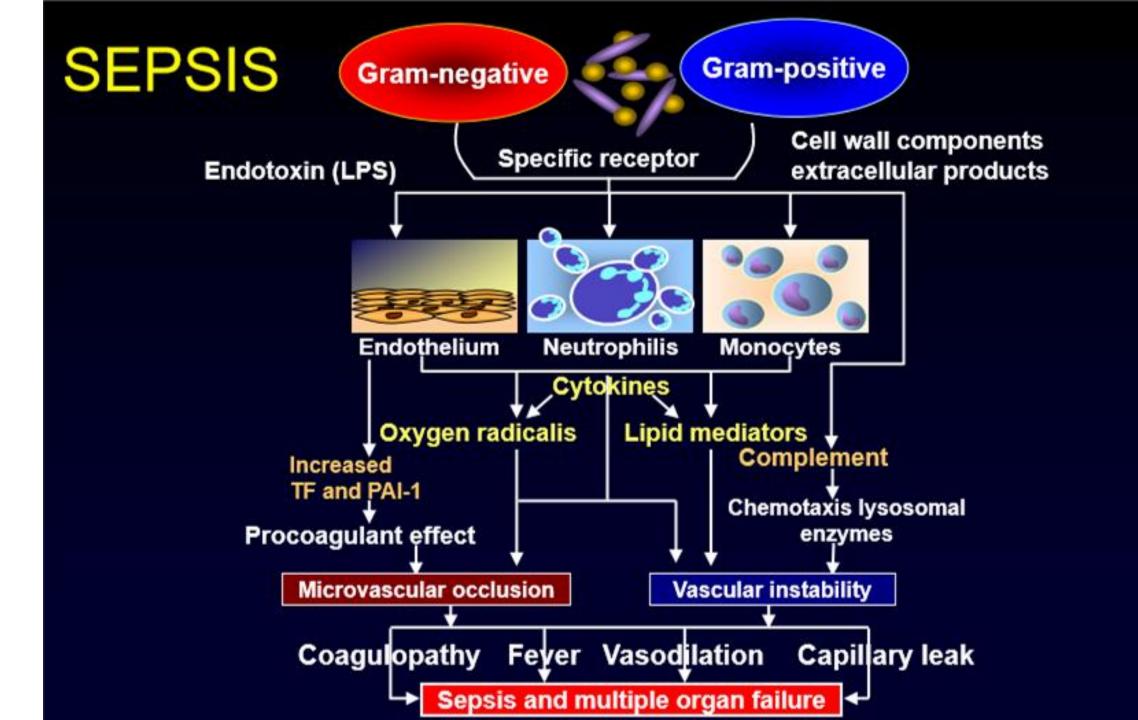
Collateral Damage due to TX: Emergence of Resistance

- Only using antimicrobial agents when needed
- De-escalating therapy
- Short course therapy
- Heterogeneity of antimicrobial agents

Evaluation of Treatment Response

- Subjectively, more well-being:
 - Appetite, toxic feeling
- Defervescence
- Recovery of leukocytosis / leukopenia
- Improvement of local inflammation
 - Skin, U/A, CXR, etc...
- Normalizing of CRP
- Normalizing of other previous abnormal data

Update in Sepsis Management-2015



Homeostasis is Lost in Sepsis



†Coagulation †Inflammation

Homeostasis

Increased PAI-1
Increased TAFI
Reduced Protein C
(endogenous
activated Protein C
inhibits PAI-1)

↓Fibrinolysis

Category of Sepsis Syndrome

- SEPSIS is defined as a documented or suspected infection with two of the followings: Systemic inflammatory response syndrome (SIRS)
- Fever or hypothermia
- Heart rate >90 /min
- Tachypnea
- Altered mental status
- Edema
- Organ dysfunction...

- Hyperglycemia
- Leukocytosis, leukopenia or immature form 10%
- Lactate > 1 mmol/L
- Decrease Capillary refilling
- Elevation of CRP or Procalcitonin

Category of Sepsis Syndrome (cont.)

Severe sepsis

 Severe sepsis is defined as sepsis associated with organ dysfunction, hypoperfusion, hypotension

Septic shock

 Acute circulatory failure as persistent arterial hypotension (SBP <90 mmHg, MAP <70, or a reduction in SBP of >40 mm Hg from baseline) despite fluid resuscitation

Infection --- Severe Sepsis

Increasing inflammation

High risk of infection

Infection

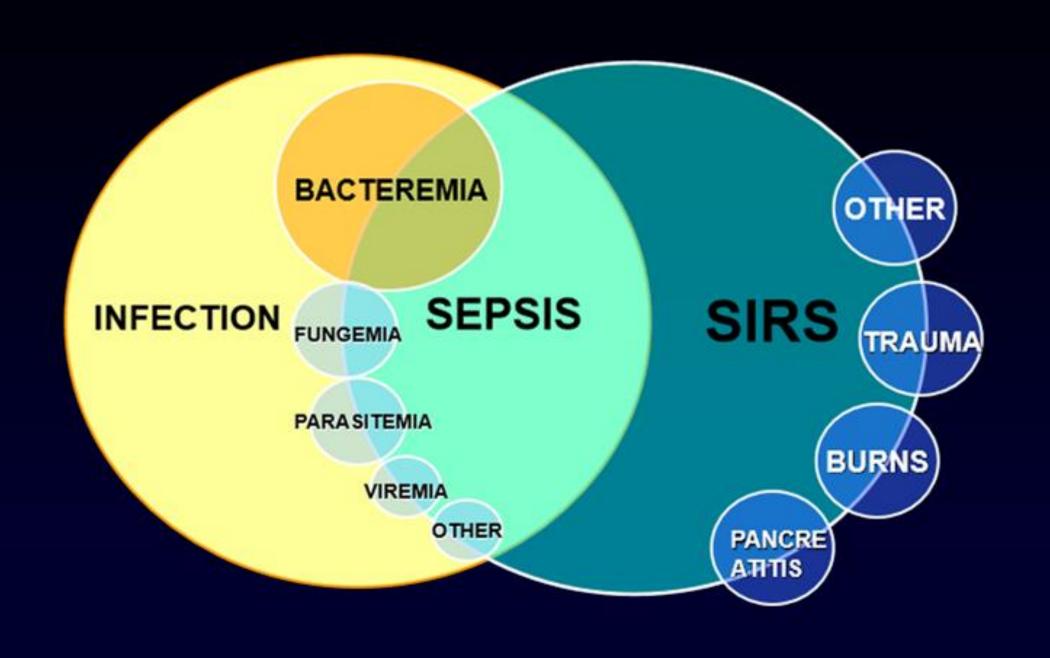
SIRS plus infection

Septic shock

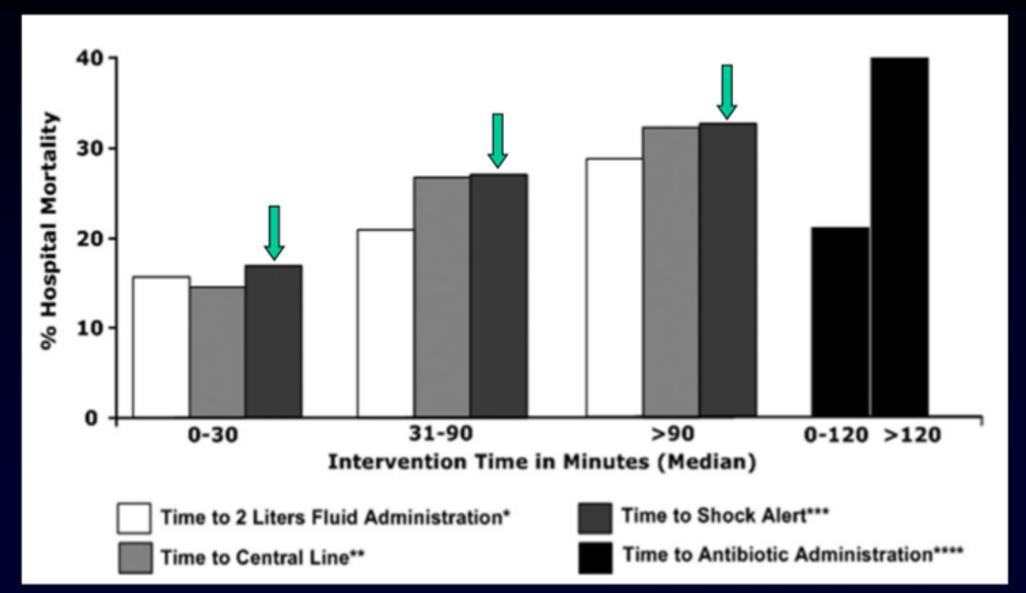
Prophylactic
Antibiotics,
Immune stimulants

Antibiotics, antitoxins

Coagulation Inhibitors antioxidants Anti-inflammatory
Cytokines,
Inflammatory
Cytokine antagonists



Time is life-saving in sepsis management



Surviving Sepsis Campaign (SSC) Guidelines for Management of Severe Sepsis and Septic Shock

Goal: To reduce the mortality of severe sepsis/septic shock by 25% in 2009

Revised in 2007 Published in Jan, 2008 Published in Feb, 2013

Dellinger PR, Levy MM, et al. Surviving Sepsis Campaign International Guideling 2012

Crit Care Med 2013 Feb;41(2):580



For additional information, please go to: www.survivingsepsis.org. IMPROVING OUTCOMES IN SEVERE SEPSIS

AACN ESCMID
ACCP SIS
ACEP ESICM Surviving
ANZICS ISF
ATS SCCM Sepsis
Campaign

European Society of Intensive Care Medicine (ESICM) www.esicm.org

International Sepsis Forum (ISF) www.sepsisforum.org

Society of Critical Care Medicine (SCCM) www.sccm.org An International Effort

Surviving Sepsis Campaign

- Sepsis resuscitation bundle (< 6hrs)
- Sepsis management bundle (6~24 hrs)

SSC Resuscitation Bundles, revised, May 2015

TO BE COMPLETED WITHIN 3 HOURS:

- 1) Measure lactate level
- 2) Obtain blood cultures prior to administration of antibiotics
- 3) Administer broad spectrum antibiotics
- 4) Administer 30 mL/kg crystalloid for hypotension or lactate 4mmol/L

TO BE COMPLETED WITHIN 6 HOURS:

- 5) Apply vasopressors (for hypotension that does not respond to initial fluid resuscitation) to maintain a mean arterial pressure (MAP) ≥ 65 mm Hg
- 6) In the event of persistent hypotension after management
 - re-assess volume status and tissue perfusion by either two (CVP, ScvO2, echocardiography, Passive leg raising test for fluid responsiveness)
- 7) Re-assess lactate, if initial elevated

Sepsis Management Bundle

- within 24 hrs of sepsis onset

- Low dose steroid for septic shock
- Glucose control maintained <180 mg/dl
- Lung protective strategy for mechanical ventilation

SCC 2013 Diagnosis and Antimicrobial Therapy

Diagnosis

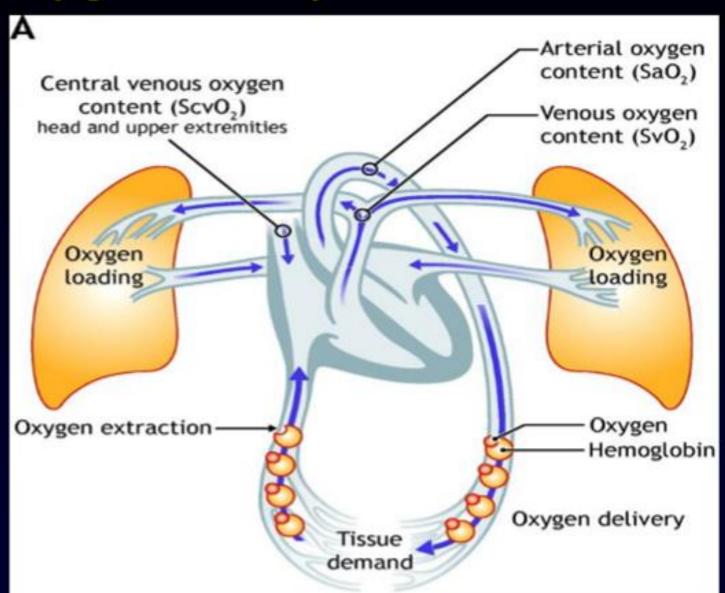
- Cultures before abx if no significant delay (> 45 mins) in the start of abx (1C).
- At least 2x blood cultures (both aerobic and anaerobic bottles) with at least 1 drawn percutaneously and 1 drawn through each vascular access device, unless the device inserted <48 hrs (1C).

Antimicrobial Therapy

- Effective therapy ASAP, but no more than within 1hr of recognition of septic shock (1B) and severe sepsis without shock (1C)
- Initial one or more drugs that have activity against all likely pathogens (1B)
- Reassess daily for potential de-escalation (1B)
- Low procalcitonin level to assist in the discontinuation of empiric abx (2C)

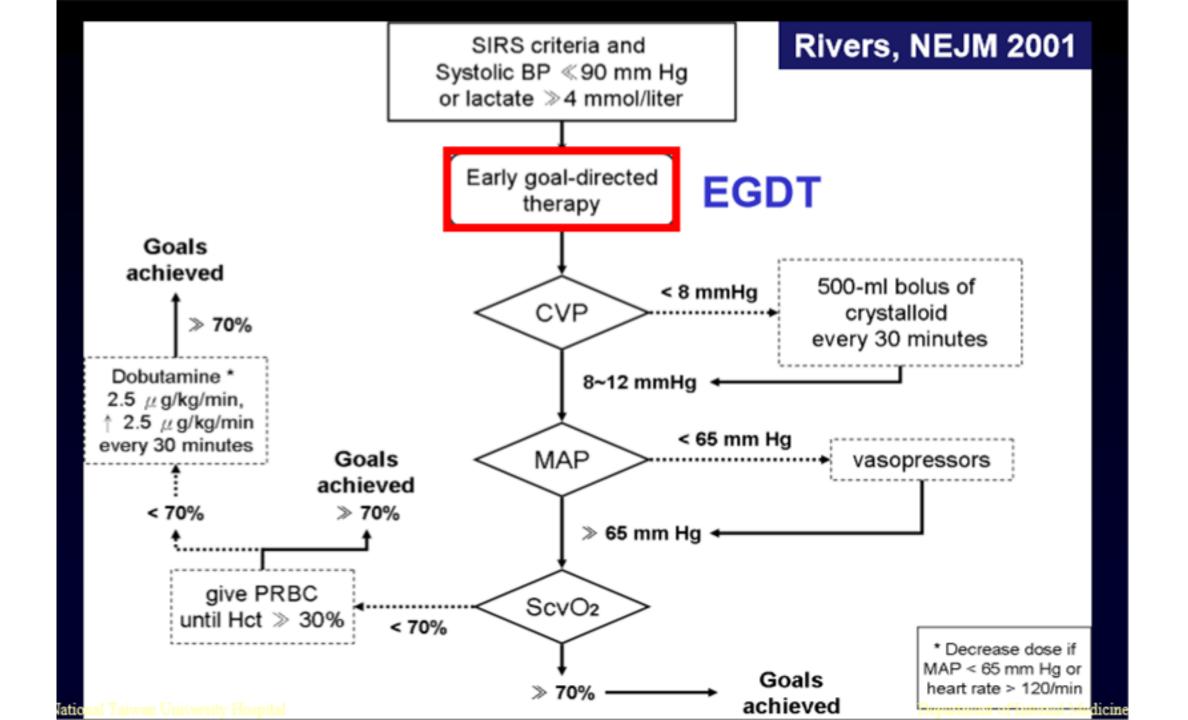
SCC 2013 Initial Resuscitation and Infection Issues

Oxygen transport and utilization

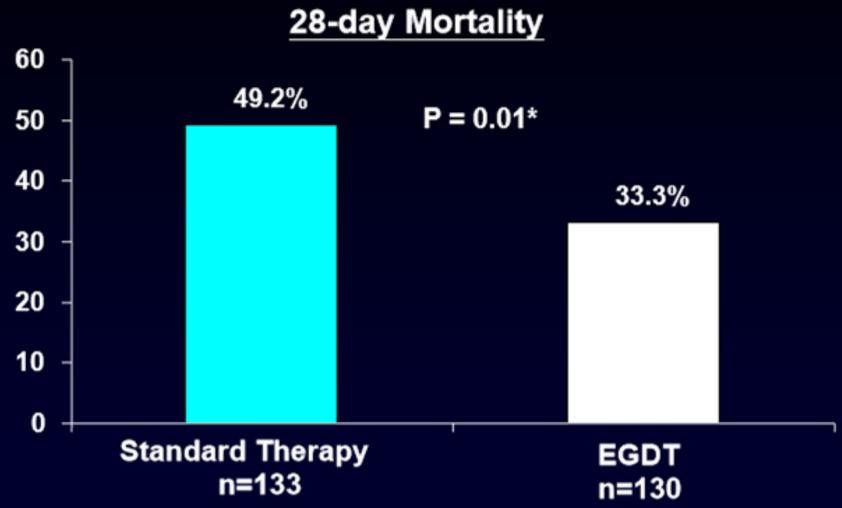


Interpretation of ScvO₂

ScvO2 > 75%	Normal extraction	
	O2 supply > demand	
75% > ScvO2 > 50%	Compensatory extraction	
	Increasing O2 demand or decreasing O2 supply	
50% > ScvO2 >30%	Exhaustion of extraction	
	Beginning of lactic acidosis, O2 supply < O2 demand	
30% > ScvO2 > 25%	Severe lactic acidosis	
ScvO2 < 25%	Cellular death	



Early Goal-Directed Therapy (EGDT)



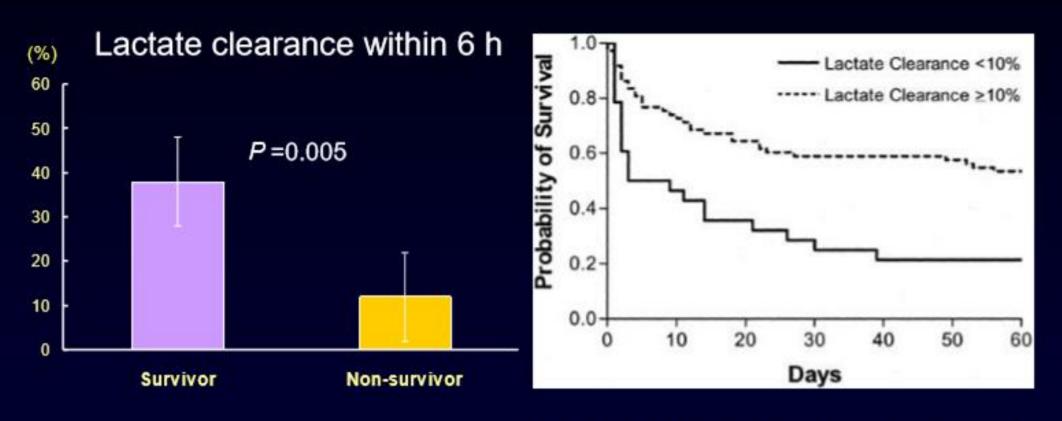
*Key difference was in sudden CV collapse, not MODS

River E. N Engl J Med 2001;345:1368.

Initial Resuscitation

- Go on EGDT (1C)
 - Rivers et al. ↓15.9% absolute reduction in 28-day mortality rate.
 - 中国危重病急救医学 2010. 17.7% absolute reduction in 28-day mortality rate.
 - A large number of observation studies.

Early lactate clearance



(Nguyen et al. Crit Care Med 2004;32:1637)

Initial Resuscitation

- For pts with lactate elevation, target lactate normalization (2C)
- Early quantitative resuscitation based on lactate clearance (\$\psi\$> 10%) non-inferior to target ScvO2> 70%.

Jones AE, JAMA 2010.

Fluid Therapy

- Crystalloid as the initial fluid of choice (1B) crystalloid = colloid
- Avoid hydroxyethyl starches (HES) for fluid resuscitation of severe sepsis and septic shock (1B)
- Albumin when require substantial amounts of crystalloid (2C)
- Initial fluid challenge in pts with septic shock with minimum 30ml/kg of crystalloids (1C)
 1000ml crystalloid or 300-500ml colloid

Therapeutic goals of volume support in critically ill patients

PCWP

CVP

Hemoglobin

Albumin

DO₂

12-15 mmHg

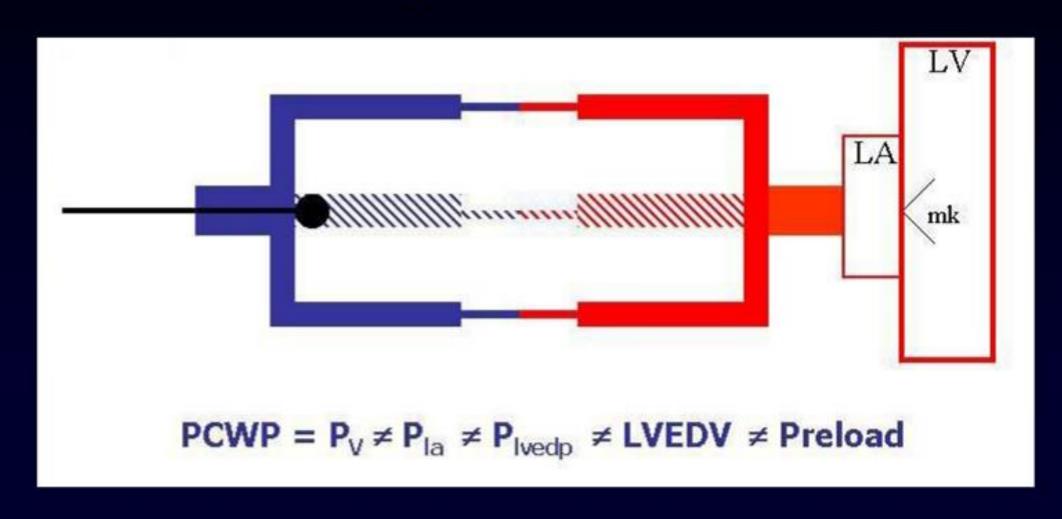
8-12 mmHg

8 g/dl

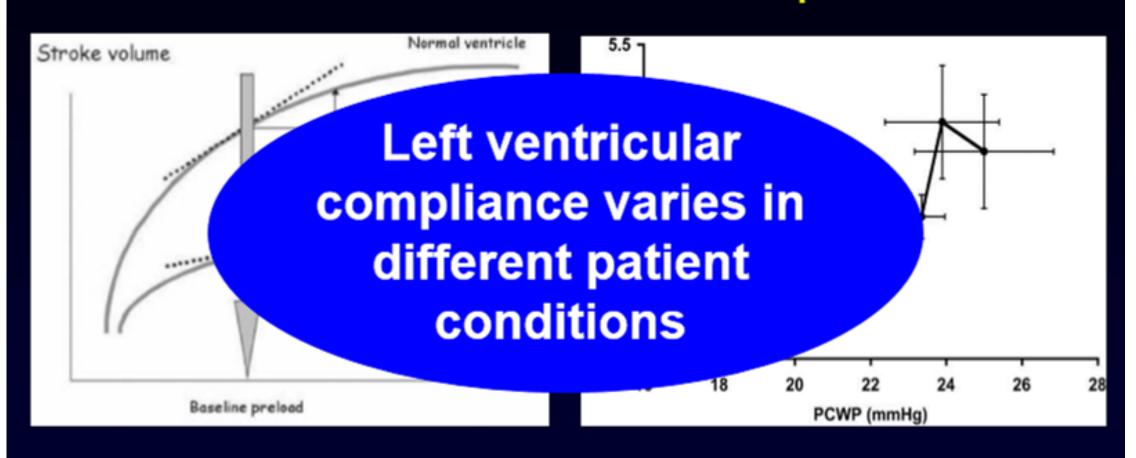
2.5 g/dl

450-600 ml/min/m²

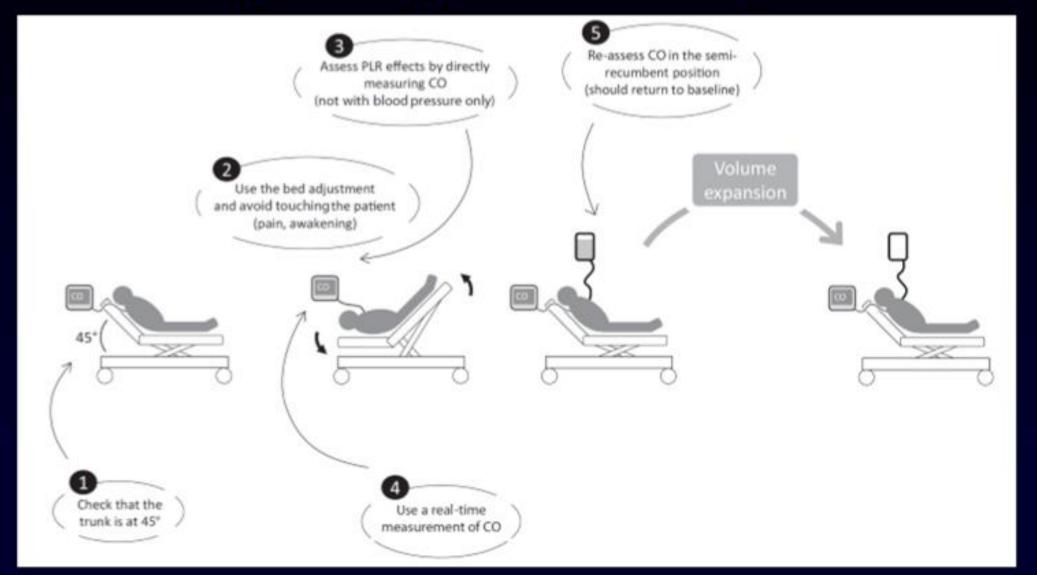
CVP/PAOP level dose not reflect the actual preload condition



CVP/PAOP level dose not reflect the actual cardiovascular response



Passive leg raising for fluid responsiveness

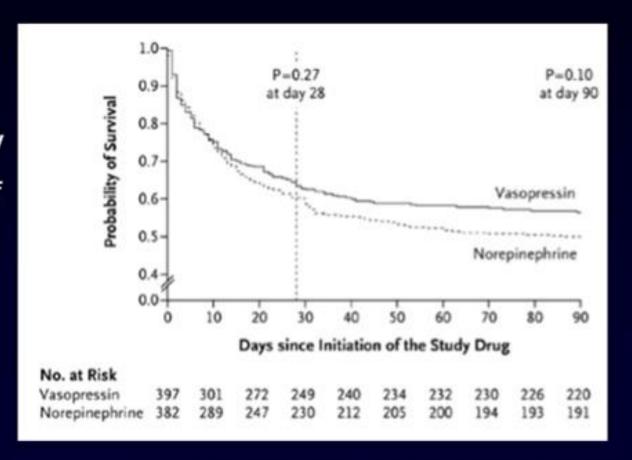


SCC 2013 -Hemodynamic Support and Adjuvant Therapy

Vasopressin or Norepinephrine

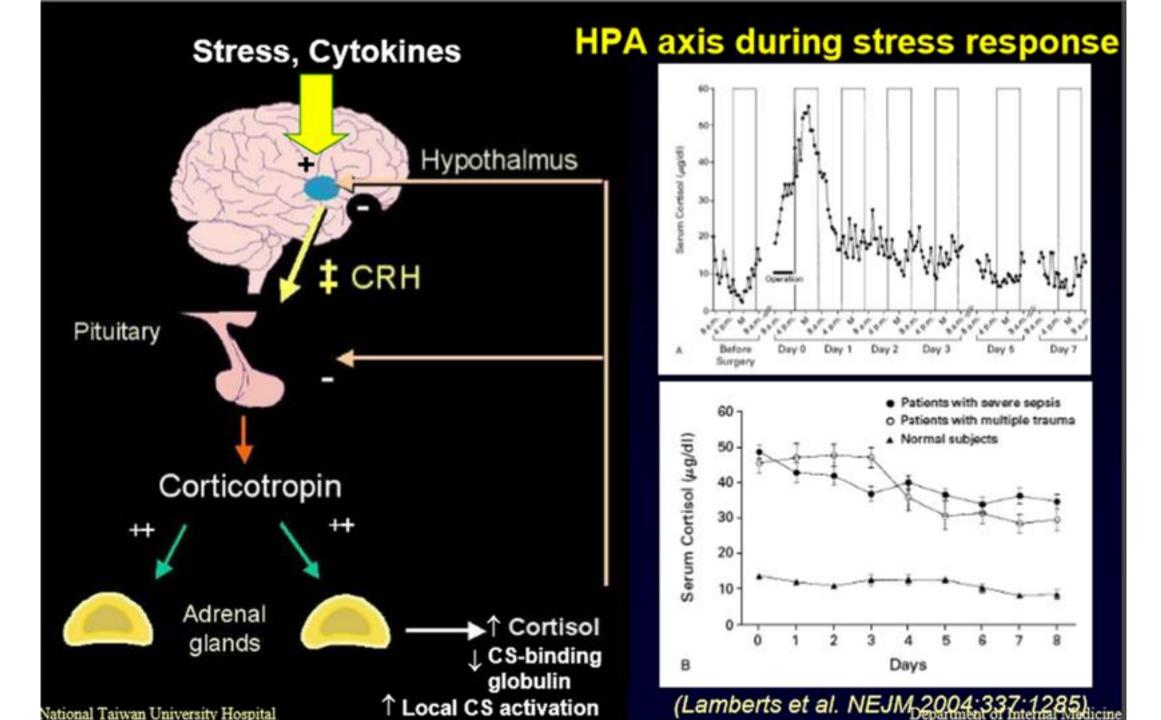
VASST (Vasopressin And Septic Shock Trial)

- 778 septic shock
- Vasopressin = NE in 28d and 90d mortality
 - Lower 90d mortality of VP group in less severe septic shock

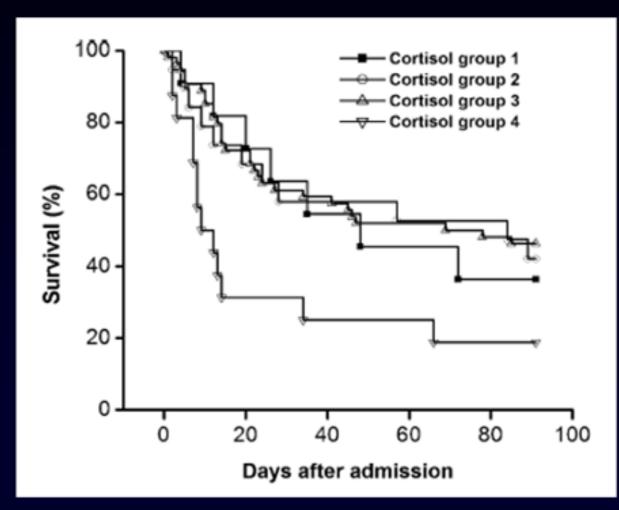


Vasopressors

- Target MAP 65mmHg (1C)
- Norepinephrine as the first choice (1B)
- Epinephrine add to and potentially substituted for norepinephrine (2B)
- Vasopressin 0.03 U/min can be added to NE (UG, VASST trial)
- Low dose vasopressin not recommended as the single initial vasopressors (UG)
- Dopamine as alternative agent, only when
 - low risk of tachyarrhythmias and
 - Absolute or relative bradycardia (2C)



Cortisol Level and Sepsis Mortality



Morning cortisol level

Group 1: \leq 345 nmol/L

Group 2: 345~552 nmol/L

Group 3: 552~1242 nmol/L

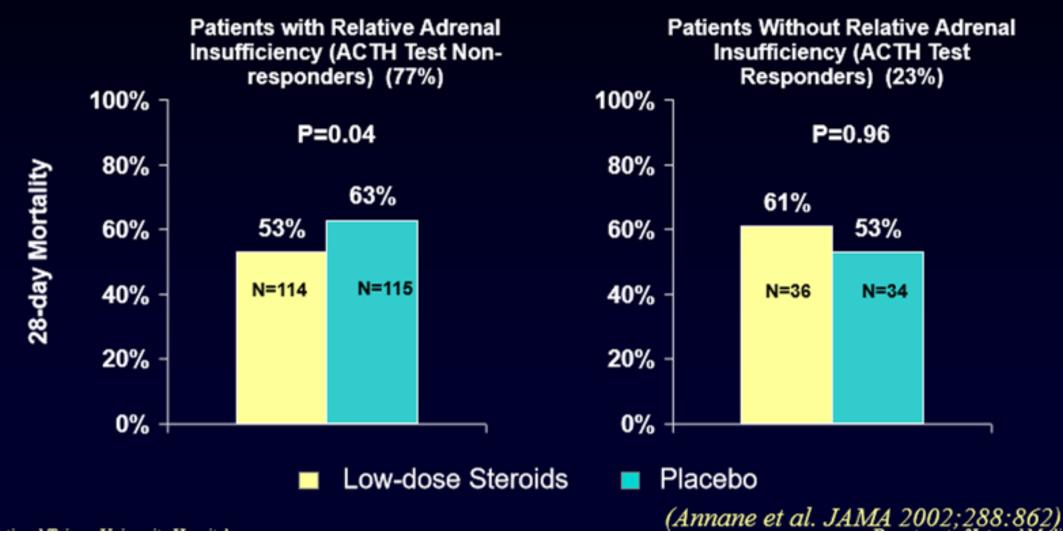
Group 4: ≥1242 nmol/L

Prognostic classification in septic shock based on cortisol levels and cortisol response to corticotropin

	Baseline cortisol level (μg/dL)	∆ max (μg/dL)	28 d mortality (%)
Good	≤ 34	> 9	26
Intermediate	≤ 34 > 34	≤ 9 > 9	67
Poor	> 34	≤ 9	82

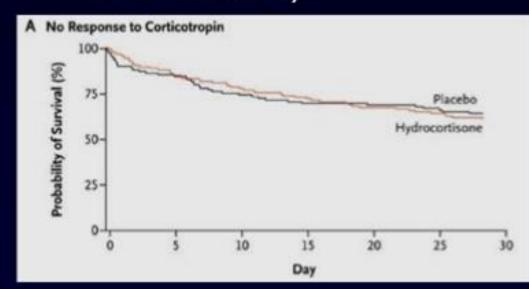
Low Dose Steroids and Septic Shock

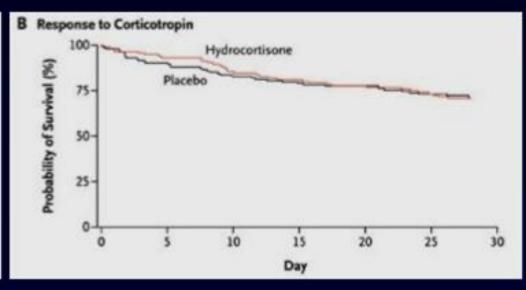
Relative adrenal insufficiency: Δmax < 9 μg/dL, 30-60 min after test



Corticosteroid of Septic Shock (CORTICUS) study

- The largest RCT (n=499) of steroid in pts with septic shock
- Hydrocortisone did not improve survival or reversal shock (but hasten the reversal of shock in patients whose shock was reversed)



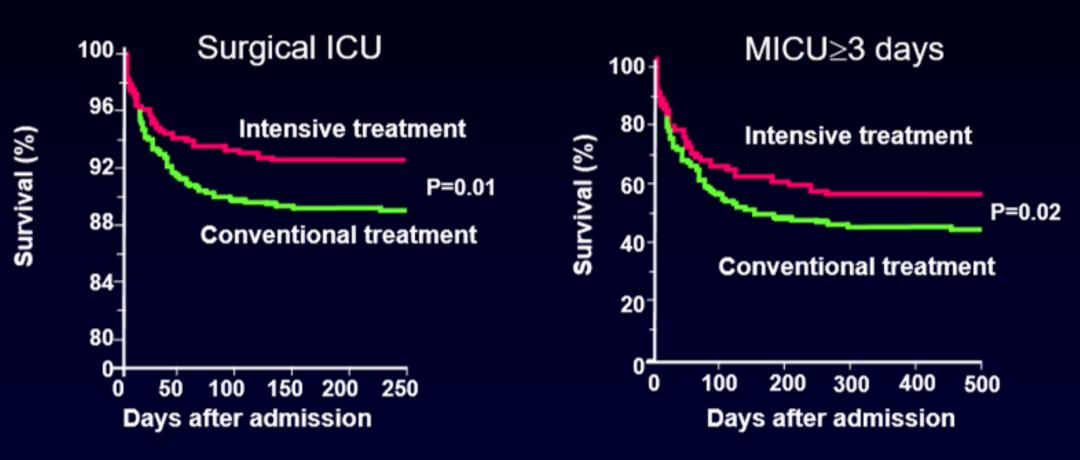


(Sprung et al. N Engl J Med 2008;358:111)

Corticosteroids

- NOT use if adequate fluid resuscitation and vasopressor therapay are able to store hemodynamic stability. If not achievable
 → iv hydrocortisone at a dose of 200 mg/day (2C)
- Not using ACTH stimulation test (2B)
- Tapered when vasopressors no longer required (not abrupt cessation) (2D)
- Not use in the absence of shock (1D)
- When given, use continuous infusion (2D)
 - Better glucose control

The Role of Intensive Insulin Therapy in the ICU



Berghe G, et al. N Engl J Med 2001;345:1359-67 Berghe G, et al. N Engl J Med 2006;354:449-61

Intensive versus Conventional Glucose Control in Critically III Patients

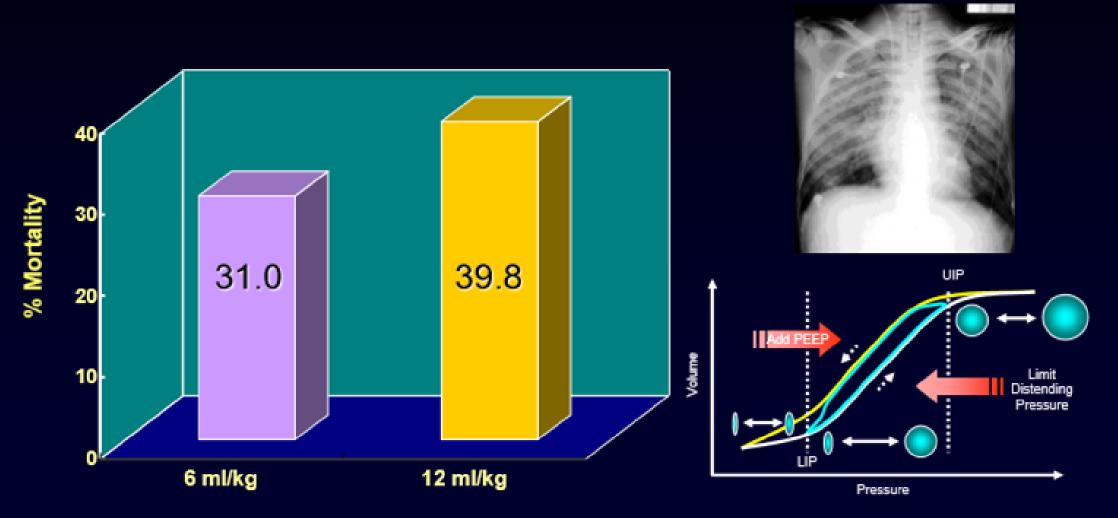
The NICE-SUGAR Study Investigators*

- 6104 patients (randomization), 3054 with intensive (blood sugar 80-108), 3050 with conventional control (BG ≤180)
- 27.5 % mortality in intensive and 24.9% in conventional (odds ratio for intensive control, 1.14; 95% C.I, 1.02 to 1.28; P = 0.02).
- Severe hypoglycemia in intensive (6.8% vs 0.5%)
- Treatment effect: no difference in surgical and medical ICU

Glucose Control

- Protocolized approach when blood glucose > 180 mg/dL. Goal ≤180mg/dL rather than 110 mg/dL (NICE-SUGAR trial) (1A)
- Blood glucose be monitored q1-2h until glucose values and insulin infusion rates stable, then q4h thereafter (1C)

Lung Protective Ventilation ARDSnet Mechanical Ventilation Protocol



(The ARDS Network. N Engl J Med 2000; 342:1301)

Mechanical Ventilation

- Target 6mL/kg PBW in pts with sepsis-induced ARDS (1A, vs. 12mL/kg)
- Plateau pressure be measured, be ≤ 30 cmH2O (1B)
- PEEP to avoid atelectotrauma (1B)
- Higher rather than lower PEEP (2C)
- Recruitment (2C)
- Prone in sepsis-induced ARDS with P/F<100 (2B)
- Head of the bed elevated 30-45° (1B)
- NIV in sepsis-induced ARDS (2B)
- Weaning protocol, SBT (1A)
- Against routine use of the PAC (1A)
- Conservative fluid strategy (1C)
- In the absence of bronchospasm, not using β2-agonist for tx of sepsis-induced ARDS(1B)

SCC 2013 -Support Therapy of Severe Sepsis

Blood Products

- RBC transfusion only when Hb <7g/dL to target 7-9 g/dL (1B) except
 - Myocardial ischemia
 - Severe hypoxemia
 - Acute hemorrhage
 - Ischemic heart disease
 - EGDT
- Not using EPO for severe-sepsis-related anemia (1B)
- Not using FFP to correct coagulopathy without bleeding or planned invasive procedure (2D)
- Not using antithrombin for the tx of severe sepsis and septic shock

<5k

5-30k

- In severe sepsis, PLT transfusion when (2D)
 - <10k (absence of bleeding)</p>
 - <20k (significant risk of bleeding)</p>
 - ≥50k (active bleeding, surgery, or invasive procedure)

Sedation, Analgesia, NMBA

- Minimize continuous or intermittent sedation (1B)
 - ↓MV duration, ↓ICU length of stay
- NMBAs be avoided in septic pts without ARDS due to prolonged neuromuscular blockage following discontinuation. If must be maintained→ intermittent bolus as required or continuous infusion with train-of-four monitoring. (1C)
- Short course NMBA (<48hrs) (2C)

Prophylaxis

- VTE pharmacoprophylaxis (1B)
- Mechanical prophylaxis for DVT (2C)
- Stress ulcer prophylaxis for pts with severe sepsis/septic shock with bleeding risk factors (1B)
 - PPI rather than H2RA (2D)
 - No risk factors→ no prophylaxis (2B)

Risk factors:

- 1. Coagulopathy
- 2. MV > 48hrs
- 3. Possibly hypotension

Nutrition

- Oral or enteral feeding as tolerated rather than IV glucose or complete fasting within 48hrs (2C)
- Avoid mandatory full caloric feeding in the 1st wk, but rather low dose feeding (~500 Kcal/day), advancing only as tolerated (2B)
- IV glucose + enteral nutrition better than TPN in 1st wk (2B)

Setting Goal of Care

- Discuss goals of care and prognosis with patients and families (1B).
- Incorporate goals of care into treatment and end-of-life care planning, utilizing palliative care principles where appropriate (1B).
- Address goals of care as early as feasible, but no later than within 72 hours of ICU admission (2C).

Compliance with bundle elements

	Initial Quarter Achieved, %	Final Quarter Achieved, % ^a	p Value Compared With Initial	Remaining Quarters Achieved, %	p Value Compared With Initial
Initial care bundle (first 6 hrs of presentation)					
Measure lactate	61.0	78.7	≤.0001	72.5	≤.0001
Blood cultures before	64.5	78.3	≤.0001	76.3	≤.0001
antibiotics					
Broad-spectrum antibiotics	60.4	67.9	.0002	67.0	≤.0001
Fluids and vasopressors	59.8	77.0	≤.0001	71.1	≤.0001
CVP >8 mm Hg	26.3	38.0	≤.0001	33.9	≤.0001
Scvo ₂ >70%	13.3	24.3	≤.0001	21.7	≤.0001
All resuscitative measures	10.9	21.5	≤.0001	21.1	≤.0001

- Compliance with each bundle element increased over time.
- Compliance with ALL elements increase over time, but remained low.

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812

MAY 1, 2014

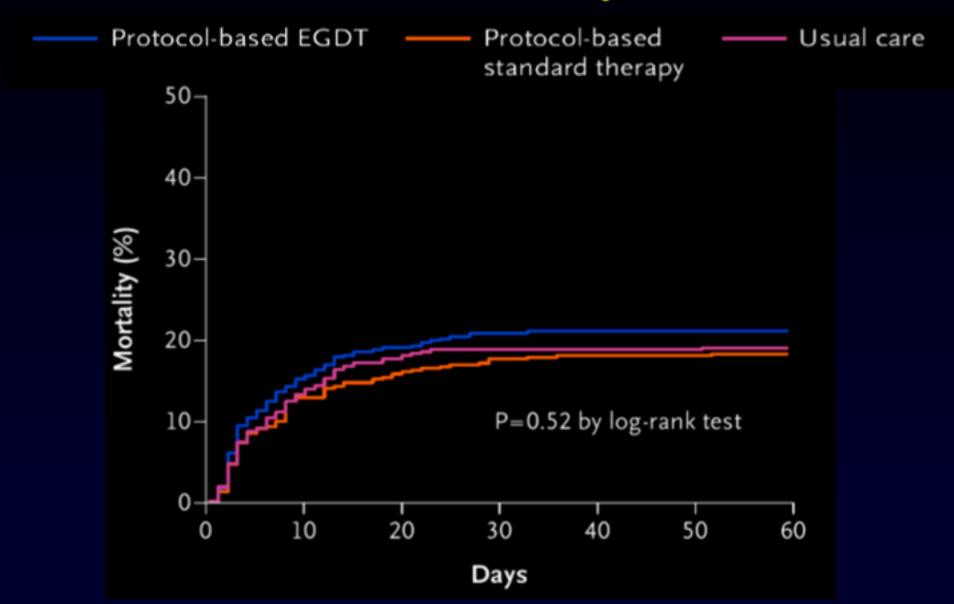
VOL. 370 NO. 18

A Randomized Trial of Protocol-Based Care for Early Septic Shock

The ProCESS Investigators*

- Protocol-based EGDT (Mortality,21%, 92/439)
- Protocol-based standard therapy (18.2%, 81/445)
 - not necessarily required CVC, inotropes
- Usual care (18.9%, 86/456)

ProCESS Trial: Primary Outcome



Comparisons among recent RCTs for EGDT

RCT		Rivers, 2001	ProCESS 2014	ARISE 2014	ProMISe 2015
Country, No. of ho	spital	USA, 1	USA, 31	ANZ, 51	UK, 56
No. of patient (EG	DT/UC)	130/133	439/456	793/798	625/626
ED stay to randon	n, hrs	1.3 ± 1.7	3	2.8	2.5
APACHE II	EGDT	21.4	20.8	15.4	18.7
	UC	20.4	20.7	15.8	18.0
Lactates	EGDT	7.7	4.8	4.4	5.2
	UC	6.9	4.9	4.2	5.1
IV fluid, mL	EGDT	4981	5059	4479	4116
in first 6 hrs	UC	3499	4362	4304	3987
CVC, %	EGDT	100	93.6	90.0	92.1
	UC	100	57.9	61.9	50.9
Antibiotics, %	EGDT	86.3	97.5	100	100
	UC	92.4	96.9	100	100
hospital mortality 60d,	EGDT	44.3	21.0	14.5	25.6
	UC	56.9	18.9	15.7	24.6
By 90 d, %	EGDT UC	_	31.9 33.7	18.6 18.8	29.5 29.2

Appraisal of Current RCTs in Sepsis

- ProCESS, ARISE, ProMISe
- Use of ScvO2 measurement and targeting therapies are not mandated
- Lower mortality rate, only applying in academic center
- Better performance of usual care arm
 - each of these institutions were systematically changed by the SCC over a decade

Closing Remarks

- Definition of sepsis syndrome
- Early recognition of sepsis and prompt intervention
- Aggressive therapy with fluid resuscitation timely
- Treatment of sepsis targeting on the pathophysiologic mechanisms
- Bundle care with favorable outcomes
- Evolving evidenced based medicine

Take Home Message:共勉

- 學而不思則惘,思而不學則怠
 - Evidence-base: please thinking external validity
 - No existing evidence: please thinking
 - 舉一隅不以三隅反者,則不復也

孔丘 論語

- 在上了許多課、讀了許多書之後
 - □ 超越前人,有了自己(融會貫通)

温瑞安 大俠蕭秋水

For patient: only love

Avedis Donabedian

Thanks for Your Attentions!

A 60-year-old Man with Progressive Dyspnea for Days

Q1: How do you approach a patient with dyspnea?

(History; Physical examination)

Analysis the symptom: LQQOPERA

- Location
- Quality
- Quantity/time course
 - Duration
 - Intermittent / persistent
- Onset mode
 - Hyperacute, acute, subacute, chronic
- Precipitation factors
 - Orthopnea; exertional
- Exaggerating factors
- Relieving factors
- Accompanying symptoms
 - Fever, cough, sputum, dizziness/headache

Systemic Approach

- Integument (skin, hair and nails) (INT)
- HEENT (HT)
- 3. Respiratory (RS)
- 4. Cardiovascular (CV)
- Gastrointestinal (GI)
- Genitourinary (GU)
- 7. Metabolic/endocrine (ME)
- 8. Hematologic (HE)
- Neuropsychiatry (NP)
- 10. Musculoskeletal (MS)

Present illness

2013/11

- Progressive dyspnea for 2 weeks
 - exertional (+), no orthopnea
 - Intermittent low grade fever
- Mild back pain

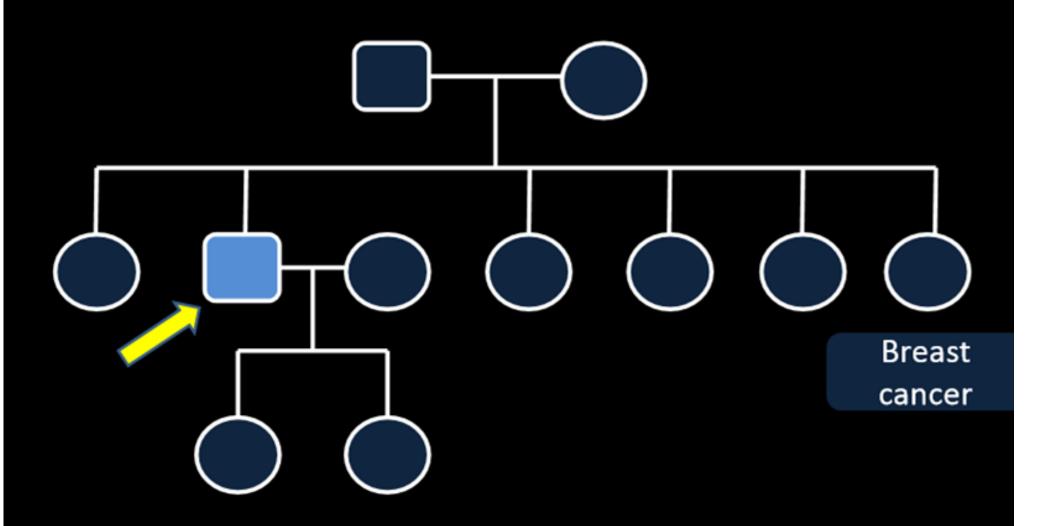
2013/11/15

- High fever 39.1~39.8C
- Fatigue and malaise
- Dry cough and progressive dyspnea for days → ER

Past History

- Occupation: 電子工廠職員
- Alcohol/betel nut/smoking: 0.5PPD for 30 years, quitted for 3 years
- Allergy: Tazocin {Skin rash}
- Medication ADR: denied
- Travel history: denied
- Contact and cluster history: denied
- Admission history: For PPU
- Previous surgery: PPU s/p subtotal gastrectomy & B-II

Family history



Physical examination

- BH: 172 cm, BW: 55kg, BMI: 18.6
- Appearance: ill-looking appearance
- Consciousness: clear and alert, E4V5M6
- T/P/R: 39.8/152/22, BP: 151/97 mmHg
- SpO2: 95% at ambient air
- HEENT:
 - Conjunctiva: mild pale, sclera: anicteric
 - Pupil: isocoric, 2.5mm/2.5mm, Light reflex:+/+, EOM: full
 - No injected throat, no oral ulcer/thrush
- Neck: Supple, goiter(-), carotid bruit(-), LAP(-) JVE(-)
- Chest: Symmetric expansion, no crackles or wheeze
- Heart: Regular heart beat, no murmur

Physical examination

- Abdomen: Flat, soft, Superficial vein engorgement(-) tenderness(-), Rebound tenderness(-) Bruit(-), Bowel sound: normo-active Liver: impalpable Spleen: impalpable
- Back: CV angle knocking tenderness(-)
- Extremity: Warm, pitting edema(-), clubbing(-)
- Skin: Petechiae(-), ecchymoses(-), wound(-), telangiectasia on hands and feet(-)
- Lymphadenopathy: Axilla (-), Inguinal (-)
- Peripheral pulse: all intact

Neurological examination

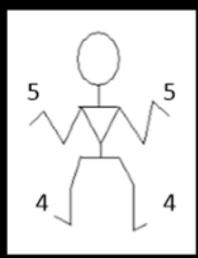
- Orientation: to person, to place, to time
- Language and speech: Intact
- Cranial nerves: intact
- Motor

Muscle tone: spasticity (-), rigidity (-)

Muscle atrophy: (-)

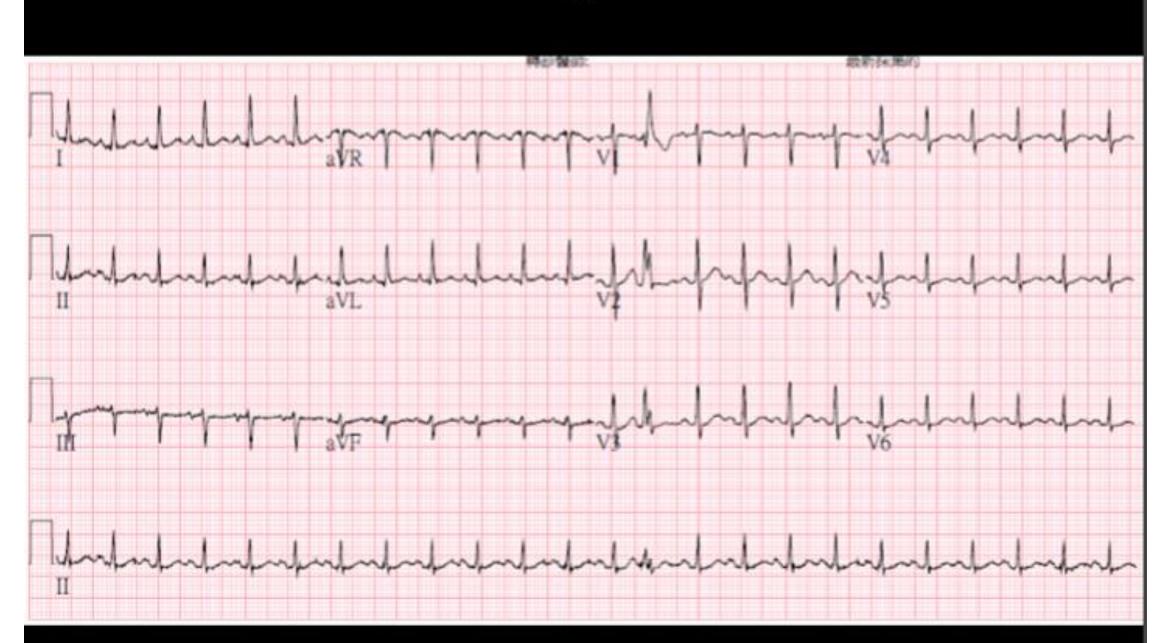
Babinski sign -/ -, Clonus -/-

- DTR: four limbs 2+
- Sensory system: intact
- Vestibular system and coordination: not performed
- Gait: no deviation
- Autonomic function:
 - Urinary incontinence(-), stool incontinence(-)



Q2: Systemic Approach to this CXR





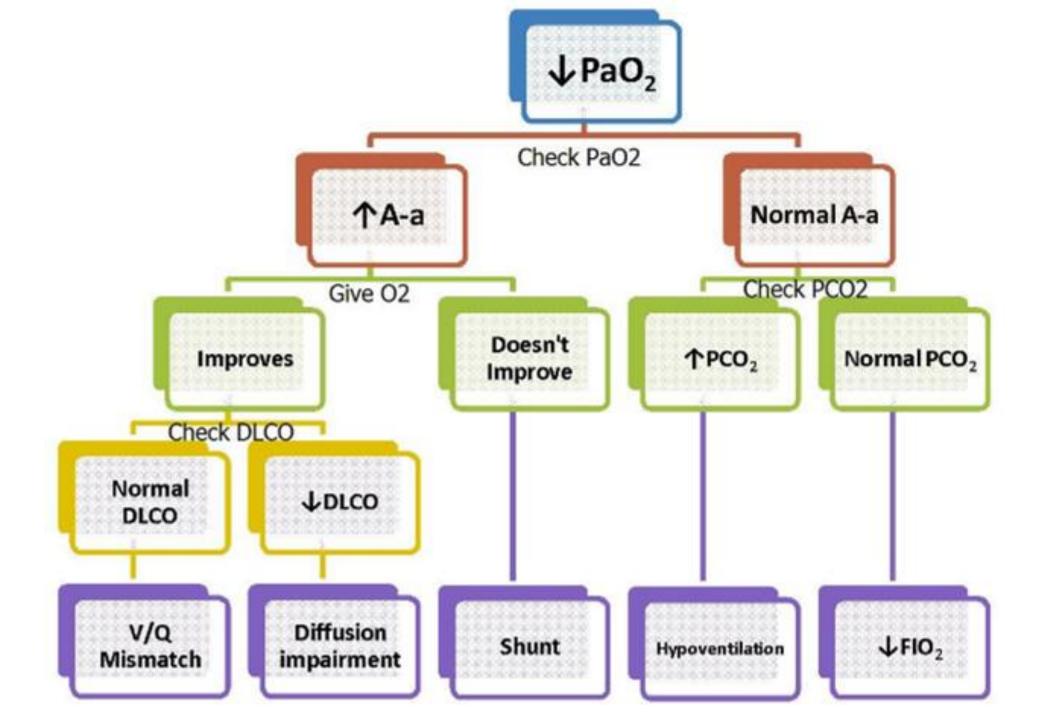
Q4-1: What lab study is crucial for this patient?

Laboratory studies (2013/11/15)

ABG

рН	PaCO2	PaO2	HCO3
	mmHg	mmHg	mmol/L
7.45	30.4	81	20.2

Q4-2: Please interpret this ABG



pН	PaCO2	PaO2	НСО3
	mmHg	mmHg	mmol/L
7.45	30.4	81	20.2

Under room air / sea level, A-a gradient = 150 - (1.25 x PaCO2) - PaO2 = 31

Normal Gradient Estimate = (Age/4) + 4= 19

SpO2 improved under N/C → V-Q mismatch

Laboratory studies(2013/11/15)

CBC

WBC	RBC	Hb	Hct	MCV	RDW	PLT
K/uL	M/ul	g/dL	%	fL	%	K/ul
12.6	4.64	12.5	36.7	85		202
Blast	Promyl.	Myelo.	Meta	Band	Seg.	
Diast	i romyn	myere.	meta	Dana	Jeg.	
%	%	%	%	%	%	
0	0	0	0	0	88.4	
Eos.	Baso.	Mono.	Lym.	Aty.lym	Plasma cell	Normo bl.
%	%	%	%	%	%	%
0	0.1	4.2	7.3	0	0	6.0

Laboratory studies (2013/11/15)

BCS

T-bil	ALT	Cre	Na	К	CRP
mg/dL	U/L	mg/dL	mmol/L	mmol/L	mg/dL
0.69	30	1.1	129	4.5	20.40

Laboratory studies (2013/11/15)

Urinalysis

Sp.Gr.	pН	Protein	Glu	Ketone	ОВ	Bil	Uro bil
		mg/dL	mg/dL	mg/dL	mg/dL	mg/dL	mg/dL
1.025	6.0	70(1+)	-	-	-	-	2.0

RBC.	WBC	Epith	Cast	Crystal	Nit.	Bac	Color	Turbid
/HPF	/HPF	/HPF	/LPF					
2-5	10-20 (2+)	2-5	-	1	1	1	Yellow	1

Q5:Tentative Diagnosis?

Problem List

- Miliary pulmonary lesion
 - Suspected miliary TB
 - Ruled out miliary carcinomatosis

Chronic respiratory alkalosis with hypoxemia

Problem List

- Miliary pulmonary lesion
 - Suspected miliary TB
 - Ruled out miliary carcinomatosis

Chronic respiratory alkalosis with hypoxemia

→ Negative pressure isolation Later, sputum AFS: 3+ Start HERZ

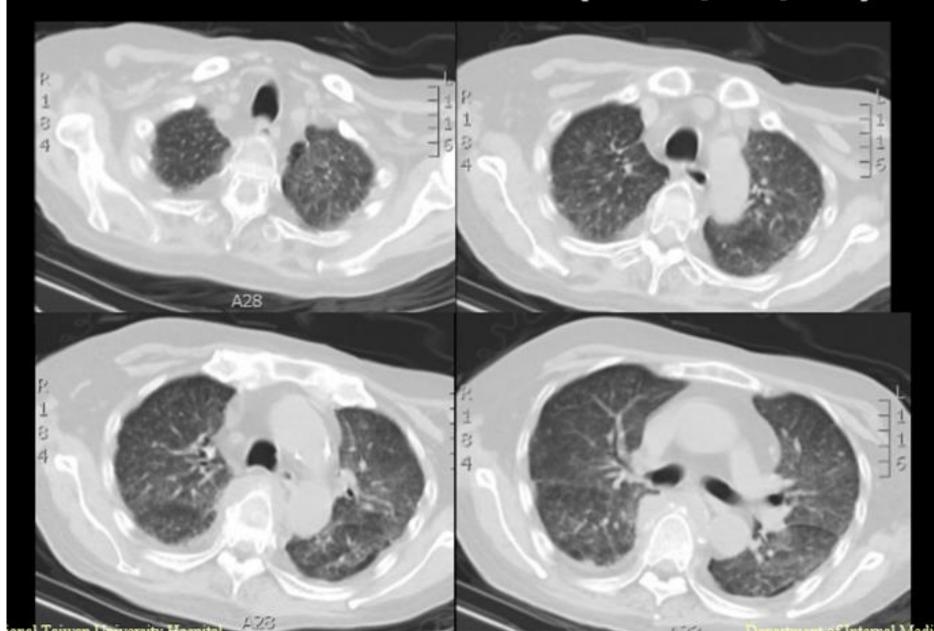
However...

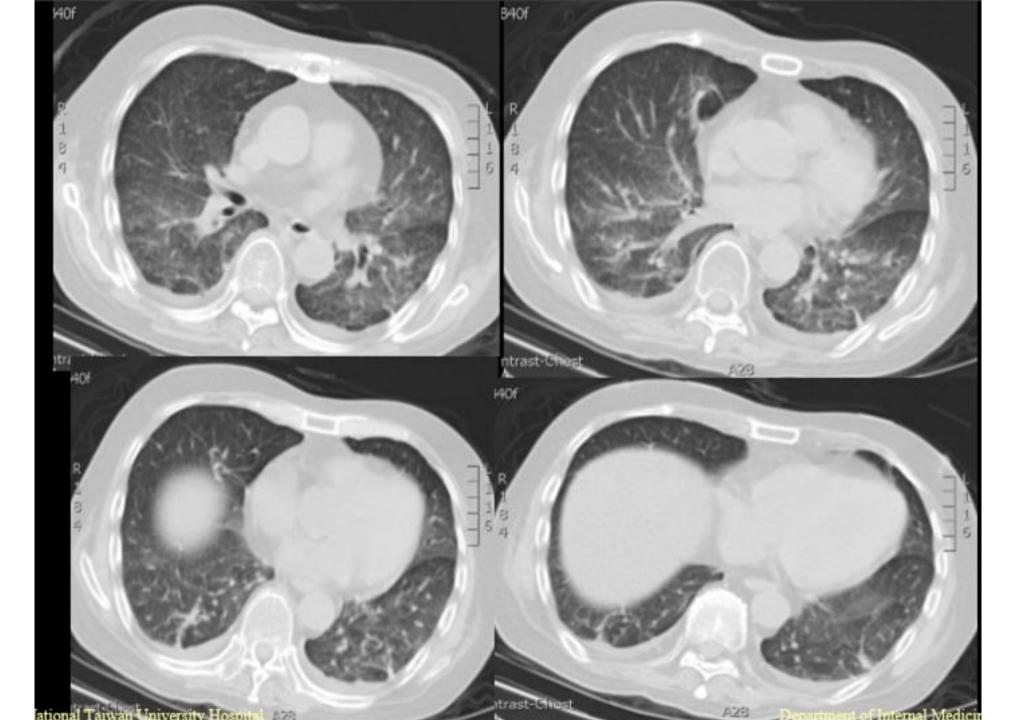
Bilateral lower extremities paraplegia

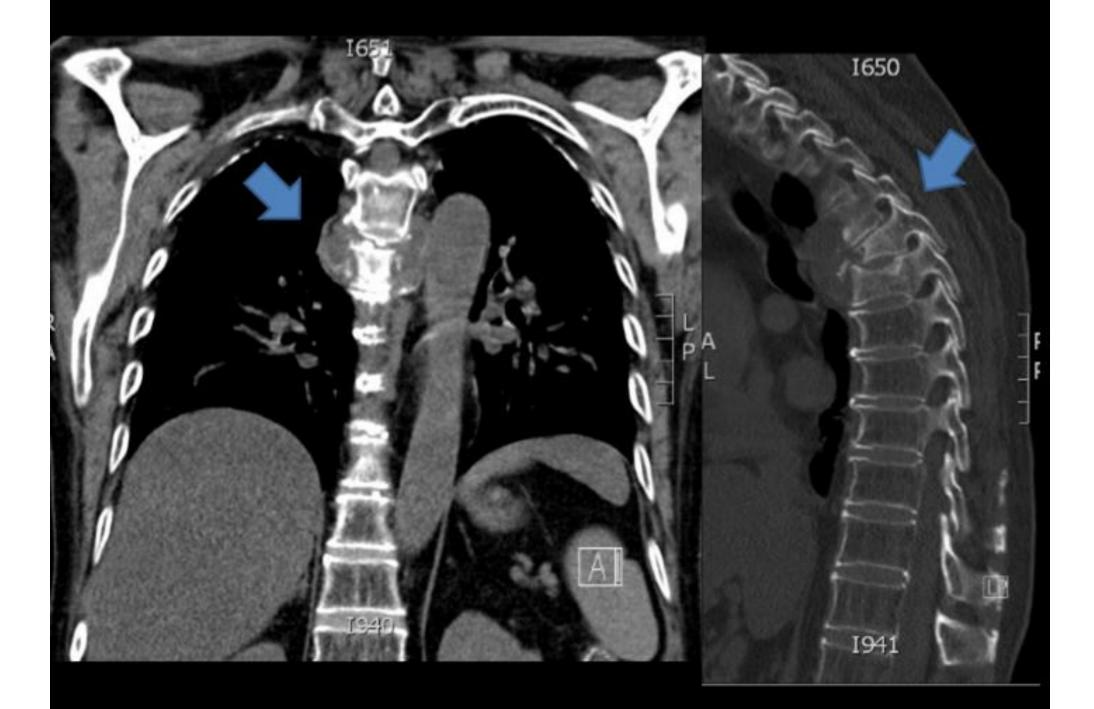
Sensory loss, urine incontinence

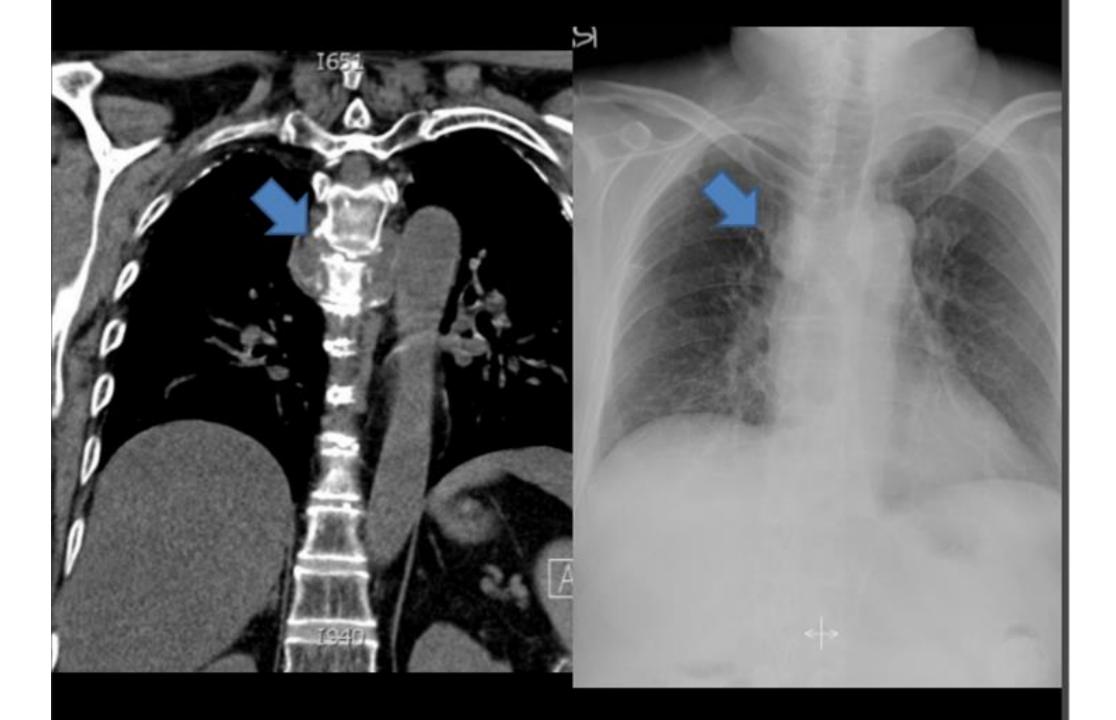
Q6: What is your impression and management?

Chest non-contrast CT (2013/11/15)

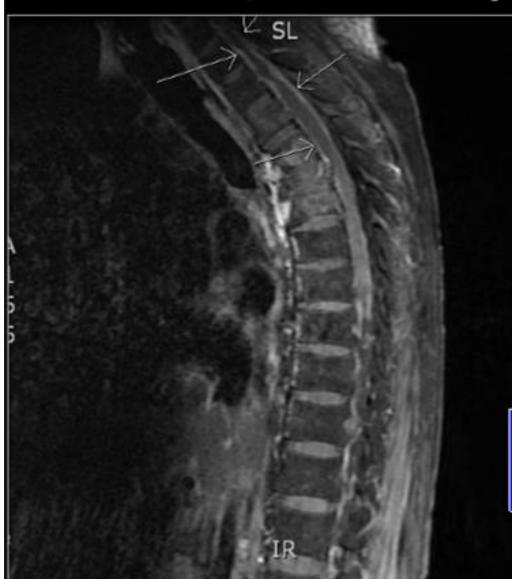








T-L Spine MRI (2013/11/18)



- Infectious spondylodiskitis at T4-T6 levels
- T5-T6 pathological compression fractures
- Paraspinal abscesses
- Extensive epidural phlegmons
- Cord compression and edema

- → Steroids
- → Consult for surgery

Even worse...

- Progressive dyspnea
- O2 demand increased to mask 10L/ 98%

pН	PaCO2	PaO2	HCO3
	mmHg	mmHg	mmol/L
7.14	59.9	56	20.5

 Q7: What is your impression and management?



Q8: When do you need to intubate a patient?

Endotracheal Intubation

- To protect airway
- To maintain oxygenation
- To maintain ventilation

Do not rely only on ABG

Anticipated need for intubation

Final diagnosis

- 1. Disseminated tuberculosis
- 2. Infectious spondylodiskitis at T4-T6 levels with pathological compression fractures at T5 and T6, and with paraspinal abscesses, causing cord compression, suspected tuberculosis of the spine
- Healthcare-associated pneumonia, status post endotracheal tube with mechanical ventilator(2013/11/18-)