Pulmonary emergency:

Acute Respiratory Failure
for medical students

นศพ. ปี 5 คณะแพทยศาสตร์ รพ. รามาธิบดี

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Acute Respiratory Failure

For medical students

Detajin Junhasavasvdikul

Aug 2013
Outline

- Introduction
- Basic physiology of respiration
  - Ventilation (Breathing)
  - Gas exchange
- Important causes of respiratory failure
  - Gas exchange failure
    - Pneumonia (CAP)
    - Acute PE
  - Ventilatory failure
    - Asthma
    - COPD
    - Hemoptysis
    - Other
Introduction

Acute respiratory failure (ARF)
Function of the lungs

- Get $O_2$ in
- Remove $CO_2$ out
- Regulate acid-base status (pH)
- Other
  - Heat dissipation
  - Metabolize some substance e.g. Angiotensin I $\rightarrow$ II
Disturbance of $O_2$, $CO_2$ or pH

$O_2$, $CO_2$

Receptor & Respiratory center

Lungs and Respiratory muscles

Dyspnea sensation
Disturbance of O₂, CO₂ or pH

Lungs and Respiratory muscles

Dyspnea sensation

Receptor & Respiratory center

Respiratory Failure

ผิดปกติ เกินกว่าที่ร่างกายจะ compensate ได้
Definition of Acute Respiratory Failure (ARF)

- Based on arterial $pO_2$ and $pCO_2$
- Differ between textbooks
- Arterial $pO_2 < 60$ mmHg

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    - Other
Basic Physiology of Respiration
Basic Physiology of Respiration

- Ventilation (Breathing)
- Gas exchange
Ventilation (Breathing)

- Breathing = Working
  “Work of breathing”
  - To bring air into and out of the lung

- Amount of work that must be done = “Load”
- Ability of respiratory muscle = “Capacity”
Ventilation (Breathing)

Normal: Excess muscle capacity (Reserve)
Impending respiratory failure: Load = Capacity
Ventilation (Breathing)

Increase load

Respiratory failure: Load > Capacity
Ventilation (Breathing)

Decrease capacity

Normal load

Loss of capacity (muscle power)
Respiratory failure: Load > Capacity
Ventilation (Breathing)

Drive: The demand of breathing

Load

Capacity
Ventilation (Breathing)

Increase drive

Drive

Load

Capacity
Ventilation (Breathing)

Decrease drive
Ventilation (Breathing)

วิธีปรับเทียบอีกวิธี ซึ่งสามารถ อธิบายเรื่อง Drive ได้ง่ายกว่า

Model:
“คนบอกของเดินไปตามถนน”
Ventilation (Breathing)

- Load
- Capacity
- Drive
Ventilation (Breathing)

Load
Capacity
Increase load

Drive

ยังพอเดินไหว
Ventilation (Breathing)

Load

Increase load

Capacity

Drive

Load เพิ่มมากกว่า ..
Ventilation (Breathing)

Respiratory failure: Load > Capacity

.. ไม่ไหวแล้ว

Increase load
Ventilation (Breathing)

Load

Normal load

Drive

↓ Capacity

คนแก่ ง้อกแกง
Ventilation (Breathing)

- Drive
- Load
- Capacity

เพิ่ม Load เพียงเล็กน้อย..
Ventilation (Breathing)

Respiratory failure: from ↓ Capacity
Ventilation (Breathing)

Load

Normal load

Capacity

Drive

เดินไปเรื่อยๆ ไหวอยู่
Ventilation (Breathing)

Basic Physiology of Respiration

Load
Normal load

Capacity

Drive

ถูกหมายวิงไส้
Ventilation (Breathing)

Respiratory failure: from \( \uparrow \) Drive
Ventilation (Breathing)

Basic Physiology of Respiration

Respiratory failure: from \( \downarrow \) Drive

Load

Capacity

Normal load

ไม่ยอมเดิน

misomเดิน
Ventilation (Breathing)

Model นี้ เอามาเทียบกับระบบหายใจ ...

Drive

Load

Capacity
Ventilation (Breathing)

Basic Physiology of Respiration

Drive

Capacity

Load
Ventilation (Breathing)

“Resistive load” (Airway resistance)

Load แบ่งย่อยได้ 2 อย่าง

“Elastic load”

Load

Drive

Capacity

Basic Physiology of Respiration
Gas exchange

- Alveoli (Parenchyma)
- Interstitial tissue
- Pulmonary vasculature

* Simplified model, actual mechanisms are much more complicated
Gas exchange

V/Q mismatch: Impaired gas exchange → decrease arterial pO₂ (Hypoxemia)

* Simplified model, actual mechanisms are much more complicated
Gas exchange

**Shunt**: Mixture of arterial & venous blood → decreases arterial pO\(_2\) (Hypoxemia)

* Simplified model, actual mechanisms are much more complicated
Gas exchange

Diffusion defect: Impair gas diffusion → Hypoxemia, especially when exercise

* Simplified model, actual mechanisms are much more complicated
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    - Asthma
    - COPD
    - Hemoptysis
    - Other
Important Causes of Respiratory Failure
Gas exchange failure

- Gas exchange failure
  - ↓ arterial pO₂
  - ↔ arterial pCO₂

CO₂ ละลายน้ำได้ดีมาก จึงไม่ค่อยมีปัญหาในการแลกเปลี่ยน

“Hypoxemic respiratory failure”
“Oxygenation failure”
“Respiratory failure type 1”

* Simplified model, actual mechanisms are much more complicated
Important Causes of Respiratory Failure

Gas exchange failure

- Normal Alveolar $O_2$
- Low arterial $O_2$

$\leftrightarrow pAO_2$ but $\downarrow paO_2$

$=$ Wide A-a gradient

V/Q mismatch, Shunt, Diffusion defect
Important Causes of Respiratory Failure

Ventilatory failure

- $\uparrow$ arterial $pCO_2$
- $\downarrow$ arterial $pO_2$

* Simplified model, actual mechanisms are much more complicated
Important Causes of Respiratory Failure

- Ventilatory failure
  - $\uparrow$ arterial $pCO_2$
  - $\downarrow$ arterial $pO_2$

“Hypercapnic respiratory failure”
“Respiratory failure type 2”
Important Causes of Respiratory Failure

Ventilatory failure

\[ \uparrow \text{pACO}_2 \quad \text{pAO}_2 \quad \Downarrow \text{paO}_2 \quad \text{Low arterial O}_2 \]

\[ \Rightarrow \text{Low Alveolar O}_2 \quad \propto \]

\[ \Downarrow \text{pAO}_2 \quad \Downarrow \text{paO}_2 \quad = \text{Normal A-a gradient} \]
Comparison: Types of respiratory failure

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Oxygenation failure (Type 1)</th>
<th>Ventilatory failure (Type 2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \text{paO}_2 )</td>
<td>( \downarrow )</td>
<td>( \downarrow )</td>
</tr>
<tr>
<td>A-a gradient</td>
<td>Wide</td>
<td>Normal</td>
</tr>
<tr>
<td>( \text{paCO}_2 )</td>
<td>( \leftrightarrow ) or ( \downarrow )</td>
<td>( \uparrow )</td>
</tr>
</tbody>
</table>
Mixed type 1 & Type 2?

- **Hypoxic respiratory failure (Type 1)**
  - Hypoxemia → Increase “drive”

- **Respiratory muscle fatigue**
  - Drive > Capacity

- **Ventilatory failure (Type 2)**

**Pneumonia**:
- SatO$_2$ 88%, pO$_2$ 56 mmHg
- Tachypnea: RR 30 /min
- pCO$_2$ 25 mmHg

- Use of accessory muscle
- Abdominal paradox
  - .. Still delay intubation

- RR 10 /min
- pCO$_2$ 70 mmHg
- Comatose
Gas exchange failure

Important causes of respiratory failure
Pneumonia
Pneumonia

Gas exchange failure

V/Q mismatch → Shunt

¬ Ventilation (\(\dot{V}\))

Perfusion (\(\dot{Q}\))

¬ Simplified model, actual mechanisms are much more complicated
Community acquired pneumonia (CAP)

- NOT an immunocompromised host (HIV, transplanted, neutropenia, immunosuppressed)
- NOT recently D/C from hospital (within 3 weeks)
CAP - Diagnosis

- **Acute** onset (< 2 weeks)
- **Hx / PE** - **S/S of LRI** (3 of 5)
  - Fever
  - Cough
  - Dyspnea
  - Pleuritic chest pain
  - Crepitation / signs of consolidation
- **CXR** : New infiltration

Thai guideline 2544
Supportive care
- Correct hypoxemia → O2
- Mechanical ventilator if the patient has ventilator failure
- Adequate hydration and secretion drainage

Selecting “Site of care”
- OPD
- IPD
- ICU

Specific care: ATB selection
Site of care selection

- Patient’s severity / Risk of mortality
  - Clinical parameters
  - Criteria / Score
When to admit?

Clinical parameters

- Patient profiles: Age > 65
- Past hx: Co-existing Dz
  - Malignancy, CRF, CHF, liver Dz, CVA
- Physical exam:
  - RR > 30, SBP < 90, PR > 125, Mental status change, etc.
- Lab:
  - PaO2 < 60, Hct < 30, pH < 7.35, Presence of pleural effusion, etc.
When to admit?
Criteria / Score

- CURB-65 จำ่าง่ายสุด
- Pneumonia severity index (PSI) score
CURB-65 score

- Confusion
- BUN > 19 mg/dL
- Respiratory rate ≥ 30 /min
- Blood pressure < 90/60 mmHg
- Age ≥ 65

Admit if score ≥ 2
เราระบุ CURB-65 score

- 60 years-old man, old CVA and HT, bed-ridden ไม่ทำตามสั่งอยู่เดิม
- T 40 c, HR 110/min, BP 95/65 (Baseline 150/90), RR 20/min, Air-hunger
- Sat O2 = 88% (O2 mask 10 lpm) BUN 15 mg/dL

Admit? CURB-65 = 0
CURB-65

- มีข้อจำกัด
- เป็นเพียงแนวทางพิจารณา ไม่ใช่กฎเกณฑ์ตายตัว
- Clinical judgment by individual case ยังสำคัญ
When to admit ICU?

- CURB-65 score $\geq 3$
- Other defined criteria
ICU admission criteria

<table>
<thead>
<tr>
<th>Major criteria</th>
<th>Minor criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Need for mechanical ventilation</td>
<td>BP drop despite aggressive fluid</td>
</tr>
<tr>
<td>- Septic shock</td>
<td>Multilobar disease</td>
</tr>
<tr>
<td>SpO2 &lt; 90% with O2</td>
<td>P/F ratio &lt;= 250</td>
</tr>
<tr>
<td>WBC &lt; 4000 /mcL</td>
<td>Confusion</td>
</tr>
<tr>
<td>Subtemp &lt; 36 c</td>
<td>BUN &gt; 20 mg/dL</td>
</tr>
<tr>
<td>Plt &lt; 100,000 /mcL</td>
<td>RR &gt; 30/min</td>
</tr>
<tr>
<td>Multilobar disease</td>
<td>WBC &lt; 4000 /mcL</td>
</tr>
</tbody>
</table>

1 Major or 2 Minor (THAI)

3 Minor (IDSA/ATS 2007)

Thai guideline 2001

IDSA / ATS guideline 2007

จำได้ถึงจำ.. ถ้าไม่ได้ถึงใช้ Clinical judgment
# Classification by severity

<table>
<thead>
<tr>
<th>CURB-65 score</th>
<th>Site of Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>(≥ 3)*</td>
<td>ICU</td>
</tr>
<tr>
<td>2</td>
<td>IPD (Ward)</td>
</tr>
<tr>
<td>1</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>OPD</td>
</tr>
</tbody>
</table>

* or by clinical judgment / other criteria
ATB selection

- Possible pathogen
- Common pathogen
- Disease modifier
- Severity of patients

ยิ่งคนไข้หนัก ยิ่งต้องให้ ATB เยอะ ไว้ก่อน กันหน่อยว่า
Common pathogen

- **S. pneumoniae**
- Atypical pathogen
  - *Chlamydophila pneumoniae* 
    *(Chlamydia)*
  - *Mycoplasma pneumoniae*
  - *Legionella pneumophila*
- Viruses
  - *H. influenzae* 
    *(in chronic lung disease, <5%)*
ATB consideration - Disease modifier

- Drug resistance *S. pneumoniae* (DRSP)
  - Age > 65 y
  - B-lactam within 3 months
  - Alcoholism
  - Exposure to children in day care
  - Multiple medical comorbidities

DRSP in Thailand: Still response to high-dose B-lactam
ATB consideration - Disease modifier

- Enteric Gram-Negative Bacilli
  - Cardiopulmonary Disease
  - Recent Antibiotics
  - Nursing home residence
  - Multiple medical comorbidities
ATB consideration - Disease modifier

- **P. aeruginosa**
  - Structural lung Dz
  - Broad-spectrum ATB > 7 d in the past month
  - Corticosteroids (>10 mg pred.)
  - Severe malnutrition
ATB consideration - Disease modifier

- **P. aeruginosa**

  "4s"

1. **S1** – Structural lung: Bronchiectasis
2. **S2** – Broad Spectrum ATB: for Seven days within “Sam-Sib (30) wan (วัน)”
3. **S3** – Steroid: prednisolone “Sib” (10) mg
4. **S4** – Starvation / Severe malnutrition
Classification by severity

**CURB-65 score**

- **0**
- **1**
- **2**
- **≥ 3**

**Site of Treatment**

- **OPD**
- **IPD (Ward)**
- **ICU**
### Classification by severity

<table>
<thead>
<tr>
<th>CURB-65 score</th>
<th>Site of Treatment</th>
<th>Modified Pathogens</th>
<th>CAP classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥ 3</td>
<td>ICU</td>
<td>+ DRSP, Enteric GNB + S. aureus + P. aeruginosa</td>
<td>4B</td>
</tr>
<tr>
<td></td>
<td></td>
<td>+ DRSP, Enteric GNB + S. aureus</td>
<td>4A</td>
</tr>
<tr>
<td>2</td>
<td>IPD (Ward)</td>
<td>+ DRSP, Enteric GNB +/- Anaerobes</td>
<td>3B</td>
</tr>
<tr>
<td></td>
<td></td>
<td>+ DRSP, Enteric GNB</td>
<td>3A</td>
</tr>
<tr>
<td>1</td>
<td>OPD</td>
<td>+ DRSP, Enteric GNB</td>
<td>2</td>
</tr>
<tr>
<td>0</td>
<td></td>
<td>Normal</td>
<td>1</td>
</tr>
</tbody>
</table>
# CAP classification

<table>
<thead>
<tr>
<th>CAP classification</th>
<th>Patients characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>4B</td>
<td>ICU, + risk of <em>P. aeruginosa</em></td>
</tr>
<tr>
<td>4A</td>
<td>ICU, No risk of <em>P. aeruginosa</em></td>
</tr>
<tr>
<td>3B</td>
<td>IPD, + cardiopulmonary disease / modifying factors</td>
</tr>
<tr>
<td>3A</td>
<td>IPD, No cardiopulmonary disease</td>
</tr>
<tr>
<td>2</td>
<td>OPD, + cardiopulmonary disease / modifying factors</td>
</tr>
<tr>
<td>1</td>
<td>OPD, No cardiopulmonary disease</td>
</tr>
</tbody>
</table>
Antibiotics

- **CAP 1 (OPD)**
  - Macrolide
    Roxithromycin, Clarithromycin, Azithromycin
  - OR -
  - Doxycycline

Macrolide เดี่ยว

(ไม่นิยม)
Antibiotics

- CAP 2 (OPD, cardiopulmonary dis)
  - Oral B-lactam AND Macrolide
    - e.g. Amoxy/Clavulanate + Clarithromycin
  - OR
  - Respiratory (Newer)
    - Fluoroquinolone
      - e.g. Levofloxacin, Moxifloxacin

B-lactam + Macrolide

- OR -

GNB, DRSP

Atypical

FQ

หลักเลี้ยง ถ้ายังสงสัย TB ..จะทำให้ Partial treat, Delay Dx
Antibiotics

- **CAP 3A & 3B (IPD)**
  - (Macrolide alone for 3A)
  - OR -
  - **IV B-lactam**
  - AND Macrolide (Oral for 3A)
  - OR -
  - **IV Respiratory (Newer)**
  - Fluoroquinolone

Ceftazidime + Co-trimoxazole for *B. pseudomallei*
Antibiotics

- CAP 4A (ICU, No *P. aeruginosa*)
  - IV B-lactam AND IV Macrolide
  - OR -
  - IV B-lactam AND IV Respiratory Fluoroquinolone

ควรมียา 2 ตัวเสมอ

Ceftazidime + Co-trimoxazole for *B. pseudomallei*
Antibiotics

CAP 4B (ICU, + *P. aeruginosa*)

- IV anti-pseudo. B-lactam*
  AND
  IV anti-pseudo. Respiratory Fluoroquinolone

- OR -

- IV anti-pseudo. B-lactam*
  AND Aminoglycosides
  AND non-anti-pseudo FQ (or Macrolide)

*Carbapenem may also be considered
Further cases exercise

- ผู้ป่วยชาย 75 ปี มา ER Dx Pneumonia CAP3a กำลังจะ Admit หำนเป็น Extern อยู่เวรดึกคนเดียว จะสั่ง order lab อะไรบ้าง?
Further cases exercise

- [ ] ผู้ป่วยคนนี้ได้ IV Amoxy / clavulanate + Azithromycin หลังจากนั้น 2 วัน ไข้ลง แต่แพทย์ตรวจดีส่ง CXR ให้ใหม่ ปรากฏ Infiltration ไม่ลดลง ทำเนื่องเป็น Extern บังเอิญติดวันหยุดสางราตร์ Staff ไม่มา 5 วัน ทำเนื่องดีติเอง .. จะทำอะไรต่อ

a) ยาเดิม รออีก 5 วัน ให้ Staff มาราตร์ก่อน
b) เปลี่ยนเป็น Oral ATB
c) Step up ATB เป็น Piperacillin/Tazobactam
d) Refer รพ.จังหวัด
e) คุยกับญาติ End of life care
ผู้ป่วยคนเดิม .. จะวางแผน Discharge เมื่อใด? คุณลุงอยากกลับบ้านแล้ว เพราะหลานมาจาก กทม. จะมาเยี่ยมช่วงสงกรานต์?
Pneumonia - Further reading

- Thai CAP guidelines (2544)
Acute Pulmonary Embolism
Gas exchange failure

Acute Pulmonary Embolism

- V/Q mismatch (and shunt)

* Simplified model, actual mechanisms are much more complicated
Acute pulmonary embolism

- Obstruction of pulmonary vessels by thrombus, fat, air, tumor
- Thromboembolism is the most common
- DVT and/or PE → Venous Thromboembolism (VTE)
PE and DVT are the same entity

- In a group of patients with PE
  - 79% will have DVT
- In a group of patients with DVT
  - 50% will have PE

Tapson VF. Acute pulmonary embolism.
Incidence of PE

- Norwegians countries: 1-2 cases/1000 persons-year (Up to 10 in elderly > 75 y)
- Thai: DVT 24-61% post ortho. surgery PE 12% post ortho. surgery

Goldhaber SZ, Bounaumeaux H. 2012 May 12;379(9828):1835-46
Mortality of PE

- Untreated:
  All-cause Mortality 30-50% in 1 y

- Admitted and treated:
  All-cause mortality 8.2-17.4% (at 3 mo)
  Mostly in the first 12-14 days

Risk factors for VTE

<table>
<thead>
<tr>
<th>Transient</th>
<th>Permanent</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Strong</strong></td>
<td><strong>Moderate</strong></td>
</tr>
<tr>
<td>- Fracture, major trauma</td>
<td>- Active cancer</td>
</tr>
<tr>
<td>- Hip / Knee replacement</td>
<td>- Inherited thrombophilia</td>
</tr>
<tr>
<td>- Major general surgery</td>
<td></td>
</tr>
<tr>
<td>- Spinal cord injury</td>
<td><strong>Weak</strong></td>
</tr>
<tr>
<td></td>
<td>- Age</td>
</tr>
<tr>
<td><strong>Moderate</strong></td>
<td></td>
</tr>
<tr>
<td>- Hormone replacement therapy</td>
<td></td>
</tr>
<tr>
<td>- Oral contraceptives</td>
<td></td>
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<tr>
<td>- Pregnancy / Post-partum</td>
<td></td>
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<tr>
<td>- Previous VTE</td>
<td></td>
</tr>
<tr>
<td><strong>Weak</strong></td>
<td></td>
</tr>
<tr>
<td>- Bed rest &gt;3 days</td>
<td></td>
</tr>
<tr>
<td>- Prolonged air travel</td>
<td></td>
</tr>
</tbody>
</table>
Symptoms

- Dyspnea
- Pleuritic chest pain
- Hemoptysis
- Acute cor-pulmonale
  (Syncope / Shock)

Pulmonary infarction
Signs

- Tachypnea
- Tachycardia
- Elevated JVP
- Signs of DVT

http://www.stoptheclot.org/learn_more/blood_clot_symptoms__dvt.htm
Investigations

- **CXR**: Not specific
  - Cardiac enlargement
  - Enlarged PA
  - Plate atelectasis / Elevated diaphragm
  - Effusion
  - Lung oligemia (Westermark’s sign)
  - Pleural-based opacity (Hampton’s hump)
Hampton’s hump
Westermark’s sign

Investigations : ABG

- Hypoxemia (75%)
- PIOPED study
  (Stein et.al. Chest. 1995)
  - Linear correlation of A-a gradients with severity
  - 11% had normal A-a gradients

ระวัง A-a gradient ปกติ ไม่ Rule out!
Investigations: EKG

- Nonspecific abnormality in 70%
- Massive PE:
  - S₁Q₃ and Inverted T₃
  - RBBB
Right ventricular strain?

T-wave inversion in the right precordial leads (V1-V3)

S1Q3T3

S-waves in lead I

Q-waves in lead III

Inverted T-waves in lead III
D-dimer

- A degradation product of cross-linked fibrin
- Increase in patients with acute VTE
- Highly sensitive (with ELISA and some automated turbidimetric assays)

Generation of D-dimer from cross-linked fibrin

Fibrinogen

Thrombin cleavage

Fibrin

Fibrinopeptide A & B

Fibrin polymer

Factor XIIIa cross-linking

Crosslinked Fibrin

Plasmin cleavage

Fibrin Degradation Products

D-dimer

Image source: http://ahdc.vet.cornell.edu/sects/coag/test/Ddimer.cfm
D-dimer threshold

- Level < 500 mcg/L (ng/mL) rules out VTE in patients with low or intermediate probability

- The 3-month thromboembolic risk was 0.8% with the combination of PE “unlikely” (dichotomised Wells’ score) and a normal D-dimer test (Tinaquant assay)

- Age-adjusted diagnostic threshold may increase accuracy

D-dimer threshold

- Level < 500 mcg/L (ng/mL) rules out VTE in patients with low or intermediate probability.
- The 3-month thromboembolic risk was 0.8% with the combination of PE unlikely (dichotomised Wells score) and a normal D-dimer test (Tinaquant assay).
- Age-adjusted diagnostic threshold may increase accuracy.

“ปลาทู Test”

“ปลาทู Test” เป็น Test ที่ใช้ทดสอบว่าสัตว์ที่อยู่ในกล่องเป็นแมว

อธิบายเรื่องสถิติ ง่ายๆ ดังนี้
“ปลาทู Test”

แมว 98% ถ้าเจอ “ปลาทู Test” จะออกจากกล่องมากินปลาทู (Sensitivity 98%)
“ปลาทู Test”

มีกล่องหนึ่ง ทำ “ปลาทู Test” แล้วไม่มีตัวอะไรออกมา
“ปลาทู Test”

มีกล่องหนึ่ง ทำ “ปลาทู” ไม่มีตัวอะไรออกมา

แต่เงาที่เหมือนแมว หางก็เหมือนแมว ร้องก็ร้องเมี้ยวๆ

เมี้ยว เมี้ยว...

ท่านสรุปว่า .. ตัวในกล่องไม่ใช่แมว เพราะ “ปลาทู Test” Negative ??
“ปลาทู Test”

มีกล่องหนึ่ง ทำ “ปลาทู Test” แล้วไม่มีตัวอะไรออกมา

HIGH Probability ที่จะเป็นแมว
Back to our “D-dimer”

- D-dimer is very sensitive
  - It has high “Negative predictive value” (NPV) for R/O PE
- But its NPV will drop in a case with high probability of PE

- DON’T use D-dimer in cases with high probability of PE
High probability or Low probability

- Score for probability prediction
  - Wells score
  - Revised Geneva score
Wells score for PE

<table>
<thead>
<tr>
<th>Factor</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous PE or DVT</td>
<td>+1.5</td>
</tr>
<tr>
<td>Heart rate &gt;100 beats per min</td>
<td>+1.5</td>
</tr>
<tr>
<td>Recent surgery or immobilisation</td>
<td>+1.5</td>
</tr>
<tr>
<td>Clinical signs of DVT</td>
<td>+3</td>
</tr>
<tr>
<td>Alternative diagnosis less likely than PE</td>
<td>+3</td>
</tr>
<tr>
<td>Haemoptysis</td>
<td>+1</td>
</tr>
<tr>
<td>Cancer</td>
<td>+1</td>
</tr>
</tbody>
</table>
### Wells score for PE (2)

#### Total score

<table>
<thead>
<tr>
<th>Score</th>
<th>Probability</th>
<th>%PE</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;2</td>
<td>Low</td>
<td>2-6</td>
</tr>
<tr>
<td>2-6</td>
<td>Intermediate</td>
<td>17-24</td>
</tr>
<tr>
<td>≥7</td>
<td>High</td>
<td>54-78</td>
</tr>
</tbody>
</table>

#### Modified Wells score

<table>
<thead>
<tr>
<th>Score</th>
<th>Probability</th>
<th>%PE</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤4</td>
<td>PE unlikely</td>
<td>8-13</td>
</tr>
<tr>
<td>&gt;4</td>
<td>PE likely</td>
<td>37-56</td>
</tr>
</tbody>
</table>

Diagnostic algorithm

Risk stratification (Vital signs)

Low risk

Wells score

Clinical assessment

Low / intermediate probability

D-dimer test

High probability

ไม่ต้องส่ง D-dimer

No PE

Imaging

Neg

Pos
Imaging

- Direct detection of PE
  - Pulmonary angiogram
  - CTA (MDCT)
  - V/Q scan
  - MRA / MRV

- Indirect detection of PE
  - Compressive U/S (for DVT)
CTA : meta-analysis and PIOPED II study

- **CTA** (mainly single detector) compares favourably with invasive pulmonary angiogram

- **MDCT** improve visualisation of segmental and subsegmental pulm a.

- **PIOPED II study** : sens 83% (76-92%) and spec 96% (93-97%)

• Railway sign

• Polo mint sign

CTA for acute PE
Ventilation / perfusion scan

- Alternative to MDCT in
  - MDCT not available
  - Patients with renal failure
  - Allergy to contrast dye
- A normal V/Q scan rules out PE, with a NPV of 97%
- High probability in V/Q scan has a PPV of 85 to 90%
- Diagnostic in only 30 to 50% of pts

Normal V/Q scan

http://www.thecorrect.com/medical-emergency/v-q-scan.html
High probability for PE

http://www.thecorrect.com/medical-emergency/v-q-scan.html
Imaging modalities in acute PE: Conclusion

1\textsuperscript{st} choice
- CTA (May combined with CTV or CUS)

2\textsuperscript{nd} choice (if contraindicated for CTA)
- Proximal CUS (Compression Ultrasound)
- V/Q scan

Alternative
- Angiography (Esp. if need endovascular treatment)
- MRA / MRV
Diagnostic algorithm

Risk stratification (Vital signs)

Low risk

High risk (BP drop, RV dysfunction)

Wells score

Clinical assessment

D-dimer test

Pos

Neg

Imaging

Low / intermediate probability

No PE

Stability?

Stable

Unstable

Bedside Echo, U/S

Prompt treatment, include thrombolysis

Prompt treatment, include thrombolysis
Treatment : anticoagulant

- Parenteral anticoagulation with low-molecular weight heparin, the pentasaccharide fondaparinux, or standard unfractionated heparin

  - Enoxaparin : 1.0 mg/kg SC bid or 1.5 mg/kg SC OD
  - Heparin (more prefer? in hypotensive patients)
    Load : 80 u/kg IV
    Maintenance : 18 u/kg/hour IV drip
    check PTT level q 4-6 h keep 1.5x of ULN

  +/− Initiation of Warfarin
Indication for thrombolytic drug

- Pulmonary embolism with cardiogenic shock (Grade 2C)
  2 = Weak recommendation
  C = Low-quality evidence

- A patient presents with systemic hypotension without shock: Frequently considered

- Submassive embolism: RV dilatation and hypokinesia without systemic hypotension – is still debated
Treatment: Thrombolytic drug

<table>
<thead>
<tr>
<th>Table 13 Approved thrombolytic regimens for pulmonary embolism</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Approved thrombolytic regimens for pulmonary embolism</strong></td>
</tr>
<tr>
<td><strong>Streptokinase</strong></td>
</tr>
<tr>
<td>250,000 IU as a loading dose over 30 min, followed by</td>
</tr>
<tr>
<td>100,000 IU/h over 12–24 h</td>
</tr>
<tr>
<td><strong>Accelerated regimen:</strong> 1.5 million IU over 2 h</td>
</tr>
<tr>
<td><strong>Urokinase</strong></td>
</tr>
<tr>
<td>4400 IU/kg as a loading dose over 10 min, followed by</td>
</tr>
<tr>
<td>4400 IU/kg/h over 12–24 h</td>
</tr>
<tr>
<td><strong>Accelerated regimen:</strong> 3 million IU over 2 h</td>
</tr>
<tr>
<td><strong>rtPA</strong></td>
</tr>
<tr>
<td>100 mg over 2 h</td>
</tr>
<tr>
<td>or 0.6 mg/kg over 15 min (maximum dose 50 mg)</td>
</tr>
</tbody>
</table>

rtPA = recombinant tissue plasminogen activator.
### Table 14 Contraindications to fibrinolytic therapy

**Absolute contraindications**
- Haemorrhagic stroke or stroke of unknown origin at any time
- Ischaemic stroke in preceding 6 months
- Central nervous system damage or neoplasms
- Recent major trauma/surgery/head injury (within preceding 3 weeks)
- Gastrointestinal bleeding within the last month
- Known bleeding

**Relative contraindications**
- Transient ischaemic attack in preceding 6 months
- Oral anticoagulant therapy
- Pregnancy or within 1 week post partum
- Non-compressible punctures
- Traumatic resuscitation
- Refractory hypertension (systolic blood pressure > 180 mmHg)
- Advanced liver disease
- Infective endocarditis
- Active peptic ulcer
Outline

- Introduction
- Basic physiology of respiration
  - Ventilation (Breathing)
  - Gas exchange
- Important causes of respiratory failure
  - Gas exchange failure
    - Pneumonia (CAP)
    - Acute PE
  - Ventilatory failure
    - Asthma
    - COPD
    - Hemoptysis
    - Other
Ventilatory failure

Important causes of respiratory failure
Asthma - Acute asthmatic attack

Asthma cigarettes in 1800s – an anti-muscarinic alkaloid
Asthma - Acute asthmatic attack

- Pathophysiology of asthma
  - Airway inflammation
  - Bronchial hyperresponsive
  - Variable airflow obstruction
  - Symptoms of dyspnea
Acute asthmatic attack (Exacerbation of asthma)

- Episodes of **progressive** increase in shortness of breath, cough, wheezing, or chest tightness, or some combination of these symptoms

- Airflow limitation, measured by PEF or FEV1, is a more reliable severity index than symptoms
Acute asthmatic attack

- Spasm
- Swelling
- Secretion

Airway obstruction
↓
↑ Resistive load
↓
Respiratory failure

http://www.nhlbi.nih.gov/health/health-topics/topics/asthma/
Pathophysiology of acute asthma

- Airway obstruction
- Uneven distribution of ventilation
- Increased work of breathing
- V/Q mismatch
- Increased wasted ventilation
- Hyperinflation

Early asthmatic attack: Hypoxemia (V/Q mismatch)

- Hypoxemia and hypercarbia
- Respiratory acidosis and metabolic acidosis

- Increase O2 consumption and CO2 production
Management of acute asthmatic attack

- (Dx and) evaluation of severity
- Supportive Rx
  - O2 supplement
  - Bronchodilator
  - Ventilatory support (ETT)
  - Other adjunctive Rx
- Specific Rx
  - Steroid
  - (Bronchodilator)
- Re-evaluate clinical response
Evaluation of severity

- History
  (Indicate more severe disease)
  - Hx previous intubation
  - Hx admission from asthma within 1 year
  - Use of B2 agonist > 1 canister / mo
  - Current steroid use
    (Systemic / Inhaled)
Evaluation of severity (2)

- Physical exam
  - Signs of impending respiratory failure
    - Unable to supine
    - Incomplete sentences
    - Accessory muscle use
    - Abdominal paradox
  - More severe:
    - Unconscious
    - Air-hunger
    - RR < 12 / min
    - BP drop

Immediate Intubation!
Evaluation of severity (3)

- Laboratory
  - PEF**: 
    (< 100 Liter/min = Severe)
  - ABG
  - CXR
  - To find the cause in severe case e.g. pneumonia
Perception of severity

Patients perception of asthma : poor

Objective assessment of airway obstruction ✔
Supportive Rx

- Oxygen supplement
- Bronchodilator
- Ventilatory support
- Other adjunctive Rx
Oxygen supplement

- Keep Sat O2 just ≥90%
- Low-flow O2 and titrate against pulse oximeter
- Should be humidified

ถ้าอากาศแห้ง จะ Bronchospasm ได้ง่าย

**Bronchodilator**

- **Inhaled** short-acting bronchodilator
  - Short-acting Beta-2 agonists (SABA) ***
  - Combined short-acting anti-cholinergic and SABA

- **Systemic bronchodilator = Adjunctive therapy**, NOT the first-line management
Beta2 agonist

salbutamol (Ventolin)
Fenoterol and Ipratropium

Berodual
pMDI with spacer versus NB

Volumetric spacer

Handheld Nebulizer
ตาม Thai asthma guidelines 2555 นอกจากกรณีที่พ่น salbutamol 3 ครั้งแล้วยังไม่ Improve ยังมีกรณีใดอีกที่จะเลือกใช้ Fenoterol + Ipratropium?
Ventilatory support

- Ventilatory failure (Late consequence) from respiratory muscle fatigue
- ETT is recommended
  - Non-invasive ventilator (CPAP, BiPAP) is risky due to the flip-flop nature of asthma (Pt can get worse all of a sudden)
  - NIV may be considered case-by-case by an expert under close monitoring!
Adjunctive therapy

- Anticholinergic
- Inhaled corticosteroid
- Magnesium
- Adrenaline
- Intravenous salbutamol
- Methylxanthines (Aminophylline)
Specific Rx

- Systemic corticosteroid *****
- Reduce admission and relapse rate
- IV or Oral corticosteroids
  - IV dexamethasone
    4-10 mg IV q 6h
  - Oral prednisolone
    30-60 mg/day

จะให้ Steroid ไปนานเท่าใด กี่วัน ตาม Thai asthma guidelines 2555?
Re-evaluate clinical response

Acute asthma

Evaluation

Bronchodilator MDI via volumetric spacer

Systemic corticosteroid is administrated in patients whose severity factors are identified

Re-evaluation & PEFR

Discharge

Admit
PEF - General concepts

- Controlled asthma
  - PEF ≥ 80% predicted

- Patients with acute attack stable enough to D/C home
  - PEF ≥ 70% predicted
  - No wheezing and acceptable HR & RR

OPD เคส Stable asthma

เคส ER ที่ acute attack มา จะยอม Accept ค่า PEF ต่ำบางข้างหน่อย
Acute Asthma Clinical Practice Guideline

1. Unconscious
   - Air hunger
   - RR < 12/min
   - Unstable hemodynamic

2. Hx of Intubation
   - Hx of steroid (inhaled, oral)
   - Admission in 1 year
   - Salbutamol + 1 ranitodine

Assessment 1
- Intubation

Assessment 2
- Initial PEF
- 1st dose SABD
  (+steroid if any of B, D)

Assessment 3
- A+C+D + PEF

Assessment 4
- A+C+D + PEF

1. PEF > 70%
   - Discharge

2. PEF > 70%
   - tany of C

3. PEF 50-70%
   - tany of C

4. PEF < 50%
   - Admit ICU

Initial PEF
- 2nd, 3rd dose SABD as needed
  (q 15-20 min with interval assessment)

** Administering oxygen via appropriate route and monitoring O₂ saturation throughout.

## At any time, presence of any of 'A' should lead to immediate intubation and ICU admission.

* S1, S2, S3: Short-acting bronchodilator (SABD) administration with 4 puffs of salbutamol MDI (100 μg)
  q 15-20 min via volumetric spacer.
* S2, S3: 2nd or 3rd SABD is needed only when PEF < 70% or PEF > 70% plus any of 'C'.
* S4: 4th SABD prefer combination of β₂ agonist + ipratropium bromide. Assessment: 30-60 min later.
* Systemic steroid eg, dexamethasone 6 mg, q 6 hr. OR oral prednisolone 40 mg, should be given if
  presence of any of 'B' by history, or any of 'A' or 'D' at any time during this ER visit.

§ Discharge medication included inhaled salbutamol q 8 hr. PLUS oral prednisolone 30 mg/d if any ER
  visit in the past week, or systemic steroid given during this ER visit.

§§ Schedule patient to OPD follow up next 3-6 days.

§§§ Prefer ICU admission. Intermediate care unit admission will be the 2nd priority.
After D/C

- Follow-up at OPD within 5-7 days after D/C
- Re-evaluate controller drugs
Asthma - Further reading

- Thai asthma guidelines (2555)
COPD - Acute exacerbation
COPD - Acute exacerbation

Global Initiative for Chronic Obstructive Lung Disease

GLOBAL STRATEGY FOR THE DIAGNOSIS, MANAGEMENT, AND PREVENTION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

UPDATED 2013
Is it COPD?

- **History**
  - Risk factor:
    - Tobacco (usually >10 pack-year)
    - Household wood smoke (เตาถ่าน)
  - Age: Usually > 40 year
  - Symptoms:
    - Chronic cough
    - Hx exacerbation

- **Physical examination**
- **Lab**
Is it COPD?

- **History**

- **Physical examination**
  - Signs of air trapping: Increase AP diameter, Shortened cricosternal distance, Hoover’s sign
  - Hypertrophy of accessory muscles
  - Expiratory wheezing [Not specific]

- **Lab**
COPD - Physical examination

- [http://meded.ucsd.edu/clinicalmed/lung.htm](http://meded.ucsd.edu/clinicalmed/lung.htm)
- [http://www.meddean.luc.edu/lumen/meded/mech/cases/case8/image_f.htm](http://www.meddean.luc.edu/lumen/meded/mech/cases/case8/image_f.htm)

มักผอมแห้ง .. เพราะใช้พลังงานทั้งหมดไปกับการหายใจในแต่ละวัน (Pink puffer type)
Is it COPD?

- History
- Physical examination
- Lab
  - CXR or CT scan
  - Previous pulmonary function test
    Post bronchodilator FEV1/FVC < 0.70
CXR

Hyperaeration

Flat diaphragm

11 posterior rib

Tubular heart shape
DEFINITION

An exacerbation of COPD is an acute event characterized by a worsening of the patient’s respiratory symptoms that is beyond normal day-to-day variations and leads to a change in medication.
COPD-AE Management

- Assessment of severity
- Supportive Rx
  - Controlled O2 supplement
  - Bronchodilator
  - Ventilator support
- Specific Rx
  - Systemic corticosteroid
  - ATB if indicated
Assessment of Severity

Stage IV: Very Severe COPD, the most important sign of a severe exacerbation is a change in the mental status of the patient and this signals a need for immediate evaluation in the hospital.

<table>
<thead>
<tr>
<th>Medical History</th>
<th>Signs of Severity</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Severity of FEV$_1$</td>
<td>• Use of accessory respiratory muscles</td>
</tr>
<tr>
<td>• Duration of worsening or new symptoms</td>
<td>• Paradoxical chest wall movements</td>
</tr>
<tr>
<td>• Number of previous episodes (exacerbations/hospitalizations)</td>
<td>• Worsening or new onset central cyanosis</td>
</tr>
<tr>
<td>• Comorbidities</td>
<td>• Development of peripheral edema</td>
</tr>
<tr>
<td>• Present treatment regimen</td>
<td>• Hemodynamic instability</td>
</tr>
<tr>
<td></td>
<td>• Signs of right heart failure</td>
</tr>
<tr>
<td></td>
<td>• Reduced alertness</td>
</tr>
</tbody>
</table>
Assessment of Severity (2)

- **Pulse oximetry and arterial blood gas measurement.** A PaO₂ < 60 mm Hg and/or SaO₂ < 90% with or without PaCO₂ > 50 mmHg when breathing room air indicate respiratory failure.

\[
\text{PaO}_2 < 60 \text{ mmHg} \quad (\text{Moderate hypoxemia})
\]

or

\[
\text{PaCO}_2 > 50 \text{ mmHg}
\]
Supportive Rx - Oxygen

- Adequate levels of oxygenation (PaO$_2$ > 60 mm Hg, or SaO$_2$ > 90%)
- CO$_2$ retention can occur insidiously with little change in symptoms
- ABG checked 30-60 minutes later
  - Check for CO$_2$ retention or acidosis

Keep O2 sat 90-92%
Supportive Rx - Bronchodilator

- Beta-2 agonists (1st choice)
- Anti-cholinergic (Add on)

Air-driven drug nebulization is safer than O2 driven
Supportive Rx - Ventilatory support

- Non-invasive ventilation is **USEFUL** in selected cases of AE- COPD with respiratory failure
  - Success rate 80-85%
  - Decrease intubation rate and mortality (Evidence level A)

ถ้าเป็นแค่ COPD : NIV (CPAP/BiPAP) มีประโยชน์
Ventilatory support - NIV

"25, 35, 45" Rule for NIV

- RR > 25 /min
- pH < 7.35 (But not <7.25)
- pCO2 > 45 mmHg (But not >60)

Table 5.7. Indications for Noninvasive Mechanical Ventilation

At least one of the following:
- Respiratory acidosis (arterial pH ≤ 7.35 and/or PaCO2 ≥ 6.0 kPa, 45 mm Hg)
- Severe dyspnea with clinical signs suggestive of respiratory fatigue, increased work of breathing, or both, such as accessory muscle use, intercostal muscle use, and/or tracheal tug (But not ≤ 7.25)

เลือกเกณฑ์ Severe พอควร แต่ไม่น่าเกินไป
Important issue of NIV

- Mask fitting
- Exhalation port must be identified
- Know the limitation - always standby for ETT
- Don’t use NIV in:
  - Alteration of conscious
  - Large amount of secretion
  - Severe pathology eg. Multilobar pneumonia, ARDS

Consult senior residents if you are unsure!!!
### Table 5.8. Indications for Invasive Mechanical Ventilation

- Unable to tolerate NIV or NIV failure
- Respiratory or cardiac arrest
- Respiratory pauses with loss of consciousness or gasping for air
- Diminished consciousness, psychomotor agitation inadequately controlled by sedation
- Massive aspiration
- Persistent inability to remove respiratory secretions
- Heart rate < 50 min\(^{-1}\) with loss of alertness
- Severe hemodynamic instability without response to fluids and vasoactive drugs
- Severe ventricular arrhythmias
- Life-threatening hypoxemia in patients unable to tolerate NIV
Specific Rx: Glucocorticosteroids

- 30 to 60 mg of oral prednisolone daily for 10-14 days is effective and safe (20-30 mg for 5-7 days in mild cases)
- Dexamethasone 5-10 mg IV q 6 h
- Shorten recovery time, improve lung function and arterial hypoxemia, reduce the risk of early relapse treatment failure and LOS
The Anthonisen’s Winnipeg criteria

(1) Worsening of dyspnea
(2) An increase in sputum volume
(3) Sputum purulence

- Type I exacerbations have all of above symptoms
- Type II exacerbations have two of the three symptoms
- Type III exacerbations are characterized by at least one of these

Specific Rx: ATB
ATB in AE COPD

- ATB for Pts with all three cardinal symptoms (Type I exacerbation) (Evidence B)
- ATB for PTs with 2 cardinal symptoms (one of these must be “sputum purulence”) (Evidence C)
COPD - Further reading

Thai COPD CPG (2553)
Hemoptysis
Blood in the airway
- Airway obstruction $\rightarrow$ Ventilatory failure
- Alveolar flooding / alveolar hemorrhage
  - Impaired gas exchange (V/Q mismatch)

Hemoptyisis

มีทั้ง Ventilatory failure และ Gas-exchange failure
Hemoptysis

- Hemoptysis: originate from
  - Bronchial arteries 90%
  - Pulmonary artery 5%
- Massive hemoptysis
  - More than 600 mL per 24 hours
  - No universal consensus
Major causes of hemoptysis

Not all hemoptysis = Active TB!
### Major causes of hemoptysis

**Etiologies of massive hemoptysis in several series**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Bronchiectasis</td>
<td>51 percent*</td>
<td>25 percent</td>
<td>20 percent</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>73 percent</td>
<td>16 percent</td>
<td></td>
</tr>
<tr>
<td>Bronchogenic carcinoma</td>
<td>5 percent</td>
<td>12 percent</td>
<td>15 percent</td>
</tr>
<tr>
<td>Aspergillosis</td>
<td>0</td>
<td>12 percent</td>
<td></td>
</tr>
<tr>
<td>Pneumonia</td>
<td>4 percent</td>
<td>5 percent</td>
<td>23 percent</td>
</tr>
<tr>
<td>Bleeding diathesis</td>
<td>0</td>
<td>0</td>
<td>15 percent</td>
</tr>
<tr>
<td>Other</td>
<td>10 percent</td>
<td>5 percent</td>
<td>20 percent</td>
</tr>
<tr>
<td>Undefined</td>
<td>8 percent</td>
<td>19 percent</td>
<td>0</td>
</tr>
<tr>
<td>&quot;Bronchitis&quot;</td>
<td>0</td>
<td>5 percent</td>
<td>7 percent</td>
</tr>
</tbody>
</table>

* All patients with bronchiectasis had tuberculosis.
Major causes of Hemoptysis

**Infectious:** Tuberculosis (Active and sequelae), fungal ball, lung abscess

**Inflammatory:** Bronchitis, (Infected) Bronchiectasis

**Neoplastic:** Bronchogenic carcinoma, bronchial adenoma, hemangioma

**Immune disorders:** Wegener granulomatosis and Goodpasture syndrome

**Pulmonary vascular disorders:** PE, AVM, Mitral valve disease (e.g. MS), PA catheter

**Miscellaneous:** Catamenial, pneumoconiosis, Coagulopathy, Anticoagulant therapy, Cocaine, Foreign body, Endobronchial tumors, Broncholith, Myxoma, DIC

Adapted from Baum’s Textbook of respiratory medicine
Diagnosis

- **CXR**: 1st step (Normal in 20-30% of patients)
- **CT scan**: For certain diagnoses such as Bronchiectasis
  - Much higher yield than CXR (sensitivity of 82-97% vs 37%).
- **Bronchoscopy**: Localize the site of bleeding in 64-67% of patients
Wegener granulomatosis present with hemoptysis

Poorly differentiated non small cell lung cancer with hemoptysis
Management

- Exclude other causes:
  - Hematemesis, Epistaxis

- Non massive hemoptysis
  - Depend on etiology of hemoptysis

- Massive hemoptysis
  - Airway protection
  - Stabilization of the patient
  - Selective intubation
  - Other strategies
Airway protection

- Trachea & main bronchus has total volume about 150 mL
  - > 150 mL of blood at a time can suffocate the patient to death

- Bleeding-side down (Prevent aspiration into the good side)
Airway protection

- Large size ETT (size 8.0 or larger)
- Unilateral intubation* (Lt mainstem intubation in cases with Rt-side lesion)
  - May be difficult due to more obtuse angle of Lt bronchus
  - Rotate tube to the left (90 degrees) after pass through vocal cords and advance until resistance is met

Rt-sided intubation generally **NOT** recommended

- This will occlude RUL bronchus
A double-lumen ETT, allows for the lumen of the bleeding side to be clamped and selective ventilation of the non-bleeding side.
Clinical monitoring

- Monitor V/S, Pulse oximetry, BP
- Can detect early stage of shock
- Should know that 600 ml in patient with BM 70 kg → 15% of TBW
- Give large-bore IV access and fluid resuscitation
- Any coagulopathy should be corrected.
- A patient with massive hemoptysis should be observed in the ICU, even if not intubated
Intervention for massive hemoptysis

- Therapeutic bronchoscopy
- Bronchial artery embolization
- Surgery

http://gwinnettmmedicalcenter.kramesonline.com/3,S,82445
http://www.autosuture.com
Other causes of ventilator failure

- Increase Load
  - Pneumothorax
  - Massive effusion
  - FB aspiration

- Decrease capacity
  - Myasthenia gravis
  - Botulinum / Cobra toxin
  - Severe myopathy

- Increase drive
  - Metabolic acidosis
  - Severe methemoglobin

- Decrease drive
  - Opioid intoxication
  - Brain stem stroke
Any question?
Thank you for your attention