alphabetische Reihung der Handouts!

Institut für Intensivmedizin

Schulungsprogramm Intensivmedizin USZ
Corona-Einsätze von Medizinstudierenden 6. Studienjahr UZH

BASIC's on Intensive Care Medicine

Teil 1/3 – sämtliche teilnehmenden Medizinstudierenden 6. Studienjahr UZH
Mittwoch, 11. März 2020, Grosser Hörsaal Pathologie USZ, PATH D 22
Chair: KD Dr. Katja Auinger, Institut für Intensivmedizin

08:00–08:30 **Begrüssung, Einführung**

08:30–09:15 **Acute respiratory failure**
- Pathophysiology of acute hypoxemic and hypercapnic respiratory failure
- Arterial blood gas analysis from the respiratory point of view
- Oxygen therapy

09:15–09:45 **Airway management**
- Intubation of the critically ill patient

09:45–10:00 Pause

10:00–10:45 **Mechanical ventilation**
- Basics
- Vent modes (Pressure controlled and volume controlled ventilation)

10:45–11:15 **Mechanical ventilation**
- Settings
- Trouble shooting

11:15–11:30 Pause

11:30–12:15 **Basics on automated ventilation**

12:15–13:00 Pause

13:00–13:30 **Transport of the critically ill patient**
13:30–14:00 **Interpretation of arterial blood gas analyses from the metabolic point of view**
14:00–14:30 **Assessment of the critically ill patient**

14:30–15:00 Pause

15:00–15:30 **Basic on shock**
15:30–16:00 **Hemodynamic monitoring**
16:00–16:30 **Arrhythmias**

16:30–17:00 Pause

17:00–17:30 **Sedation/Analgesia and Nutrition in short words**

Kontakte
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- Organisatorisches: Dr. Lorenzo Käser, Ressort Lehre USZ, lorenzo.kaeser@usz.ch, 044 255 30 35

USZ Universitäts Spital Zürich

Acute respiratory failure

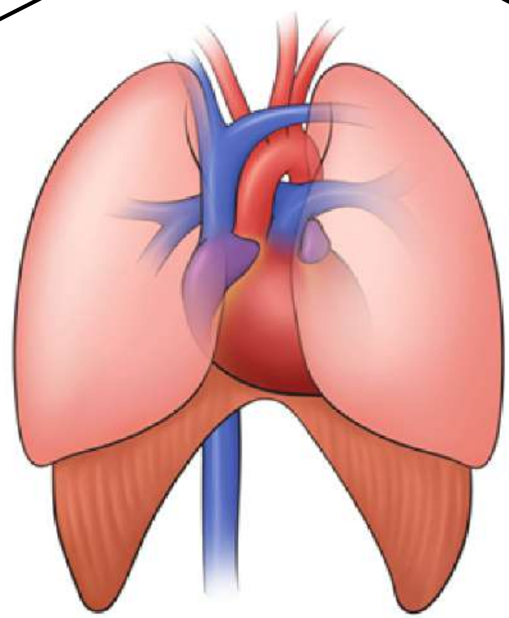
Continued development of BASIC Collaboration courses is supported by unrestricted educational grants from:

Definition

Acute respiratory failure occurs when the pulmonary system is no longer able to meet the metabolic demands of the body

Hypoxaemic respiratory failure

$PaO_2 \leq 8$ kPa
when breathing room air



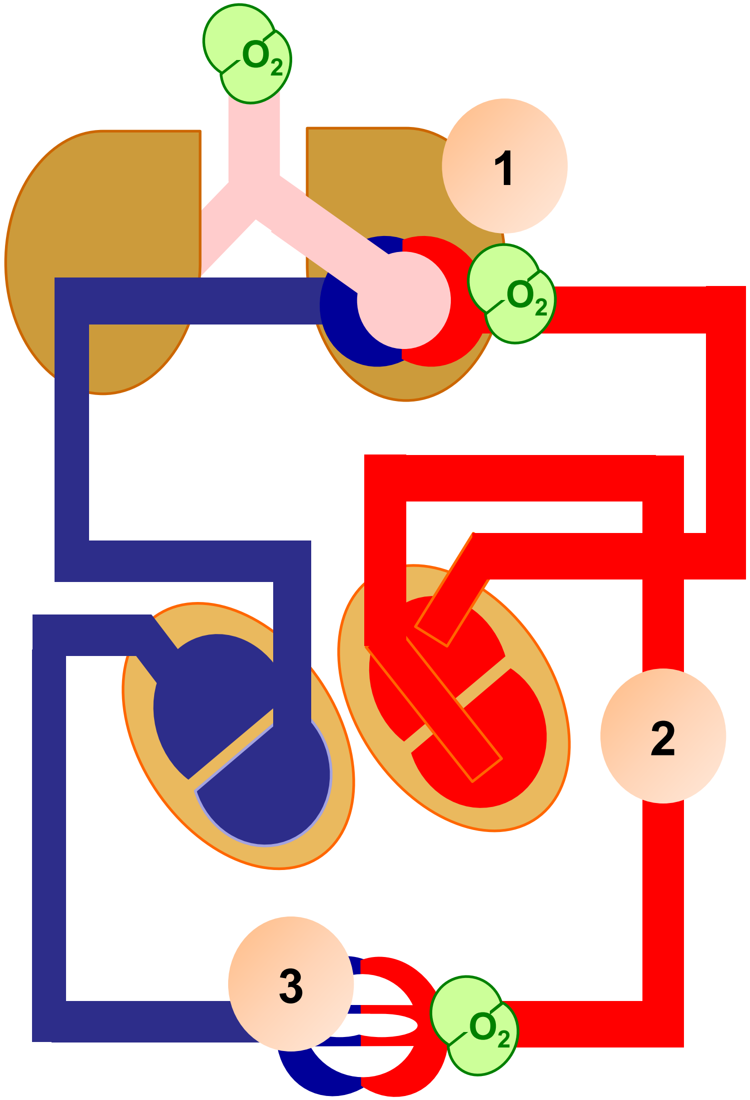
Hypercapnic respiratory failure

$PaCO_2 \geq 6.7$ kPa

combination of both

Basic phyisology

Oxygen



Continuous delivery of oxygen from inspired air to tissue cells

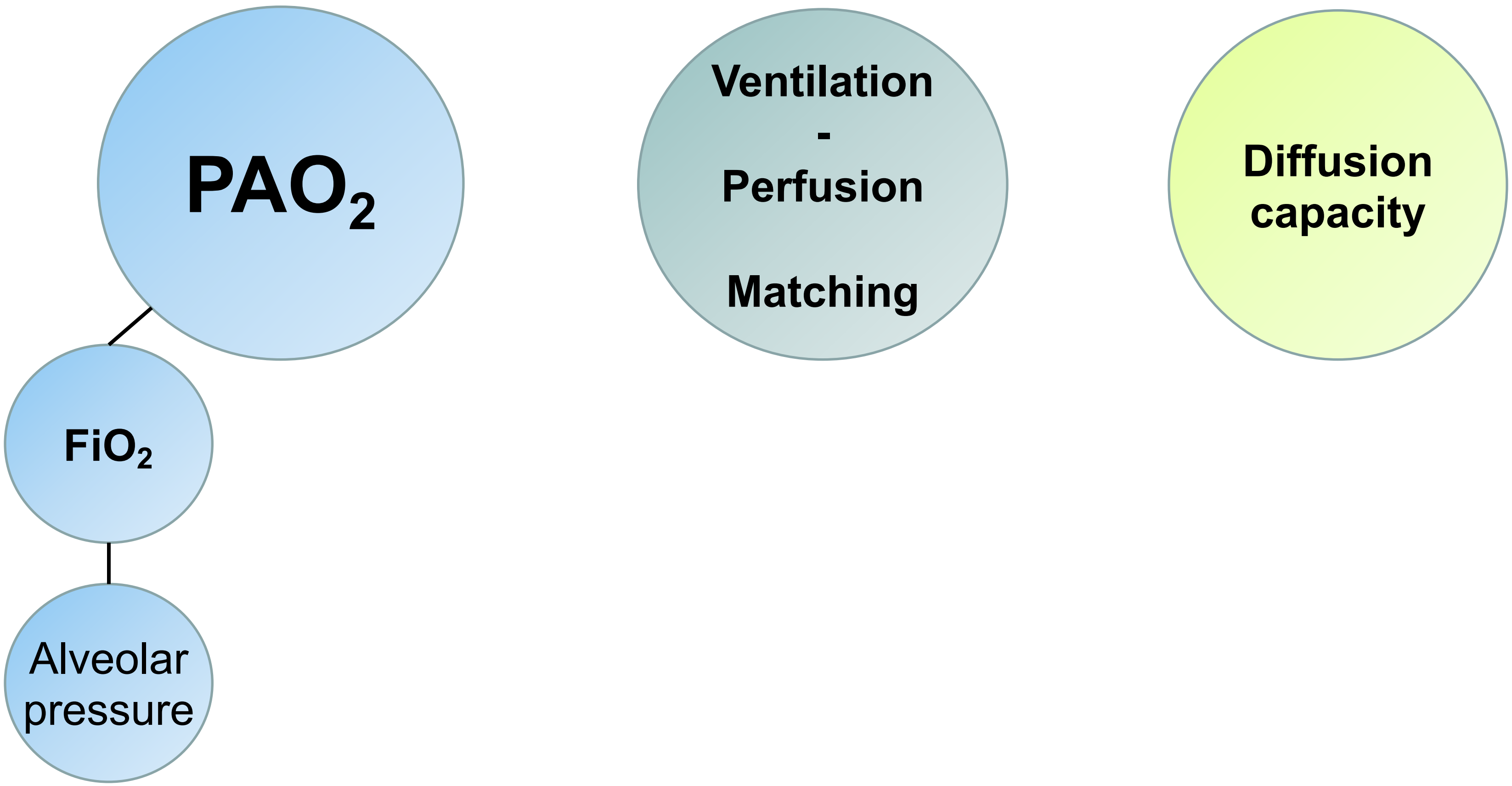
3 sequential events

1. **Uptake** of oxygen from alveolar air into the lungs
2. **Transport/delivery** of oxygen in blood from lung to tissues
3. **Release** of oxygen from blood to tissues

Oxygen uptake

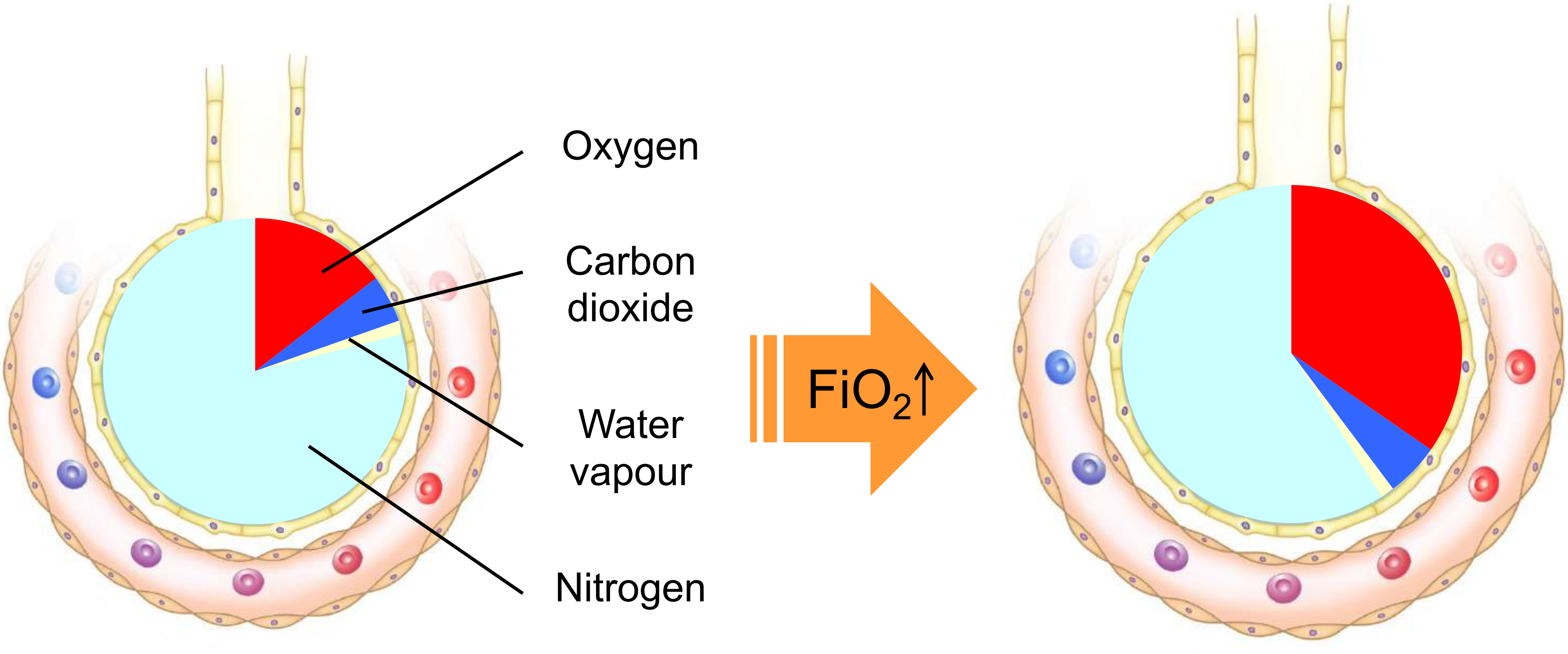
Key parameter: PaO_2

PaO_2 in arterial blood is the result of oxygen uptake via diffusion through the alveolo-capillary membrane from the lungs to the blood



Oxygen uptake: PAO_2 - FiO_2

Alveolar pressure = $P_AO_2 + P_ACO_2 + P_AH_2O + P_AN_2$



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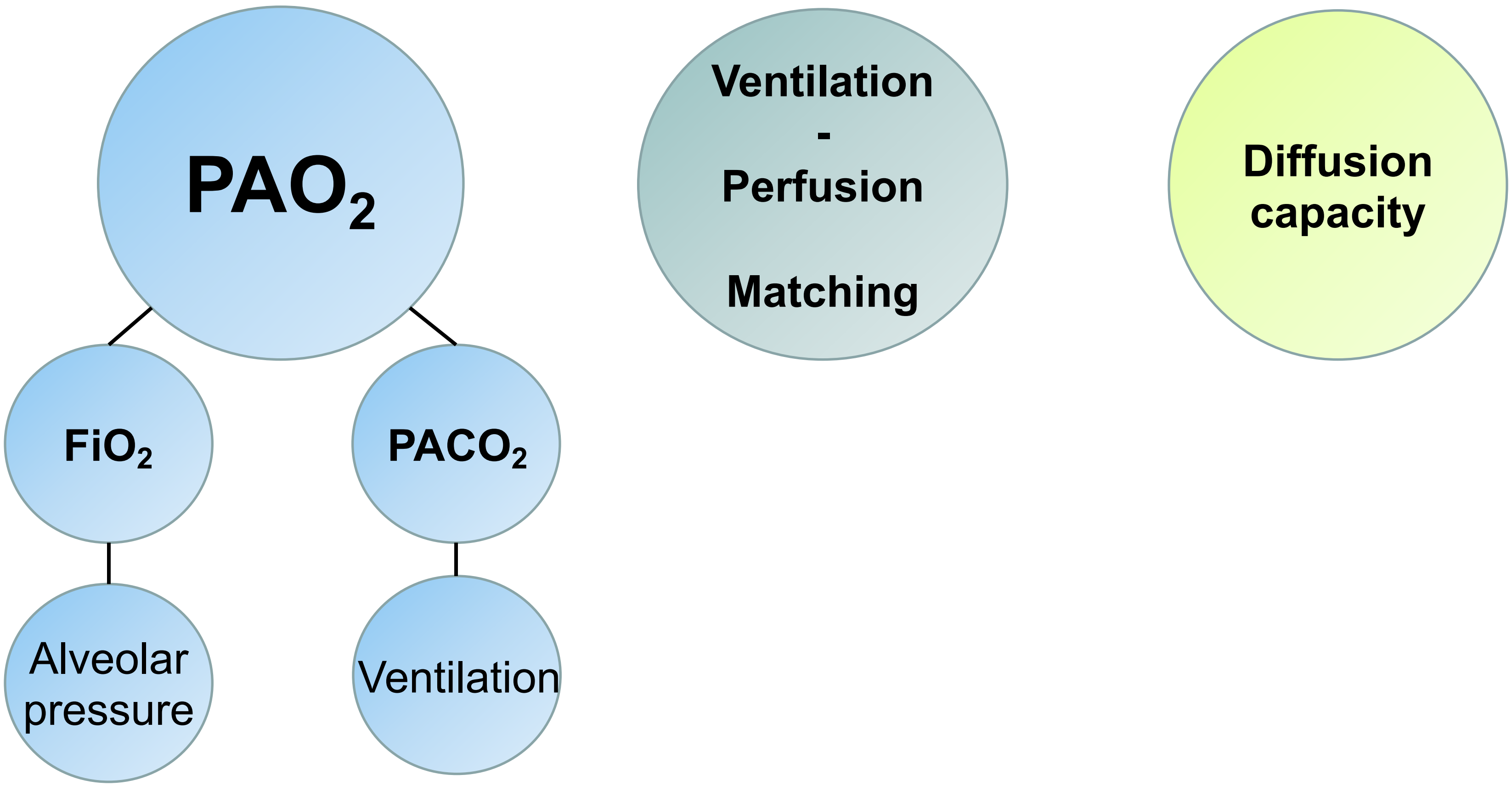
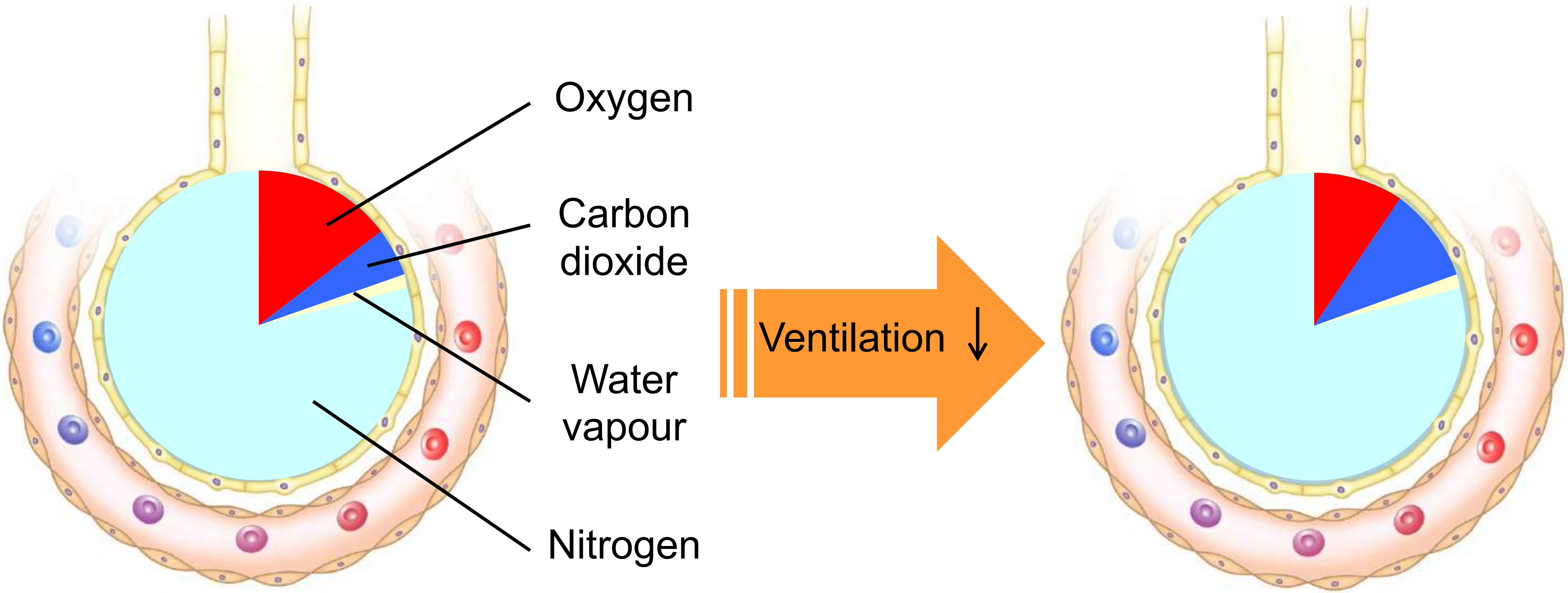


Oxygen uptake: PAO_2 - $pACO_2$

Alveolar pressure $= P_AO_2 + P_ACO_2 + P_AH_2O + P_AN_2$



<u>A-a gradient</u>

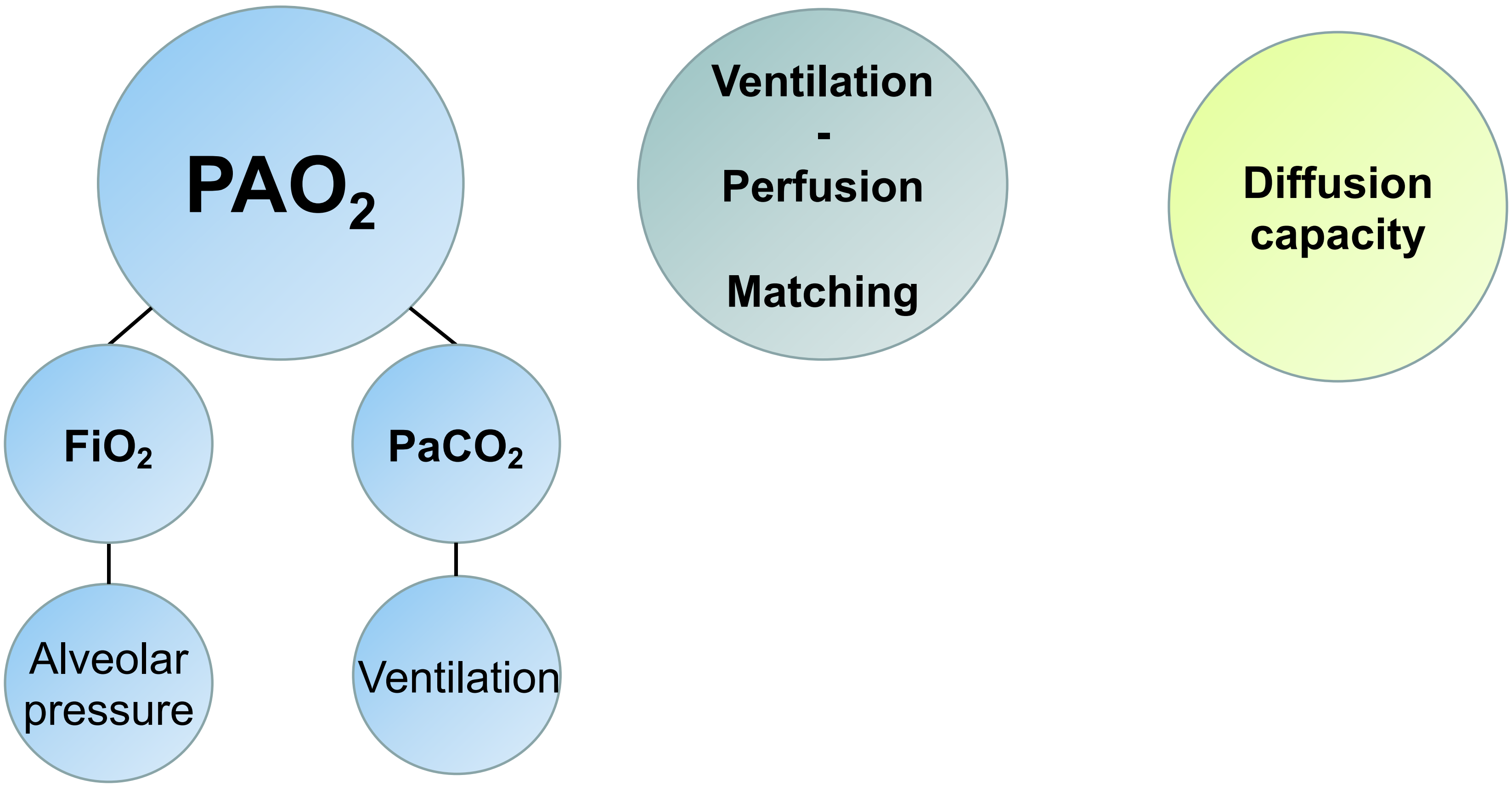
normal: hypoxia due to hypercapnia

increased: hypoxia due to shunt or diffusion abnormality

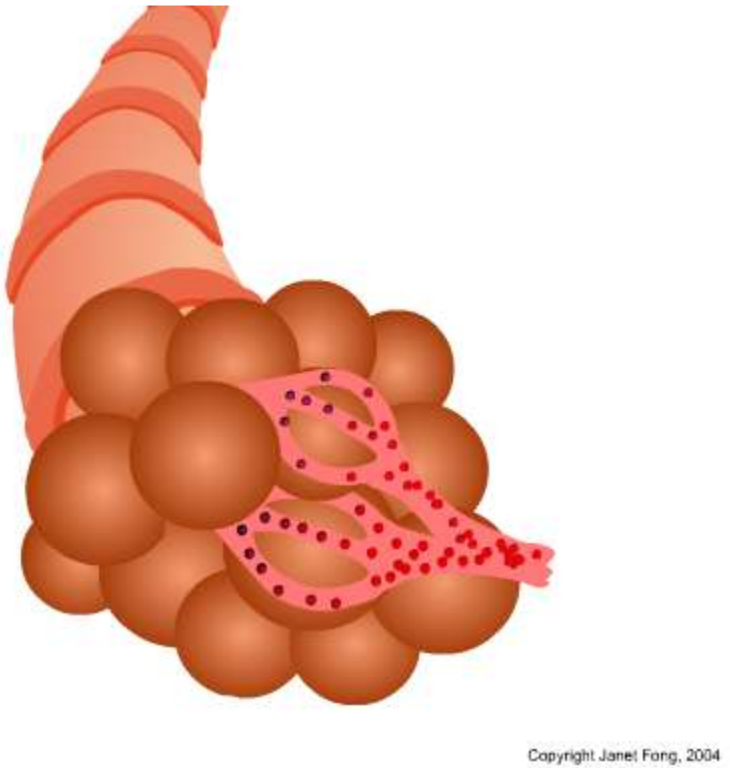
Oxygen uptake

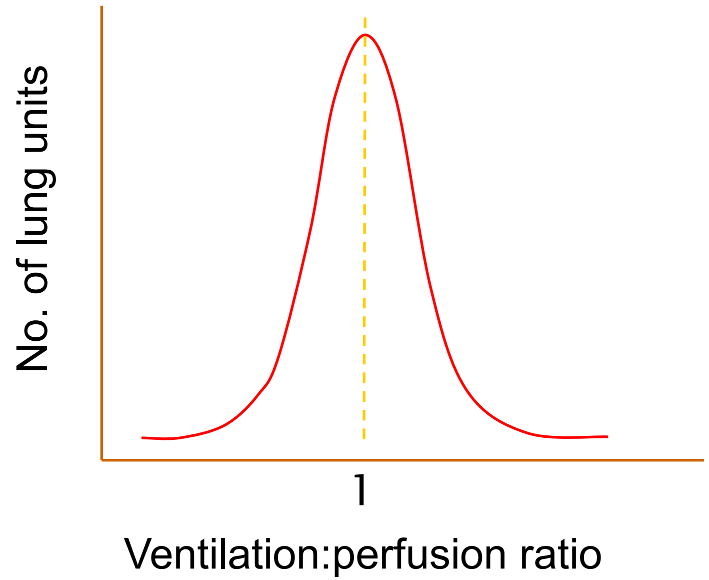
Key parameter: PaO_2

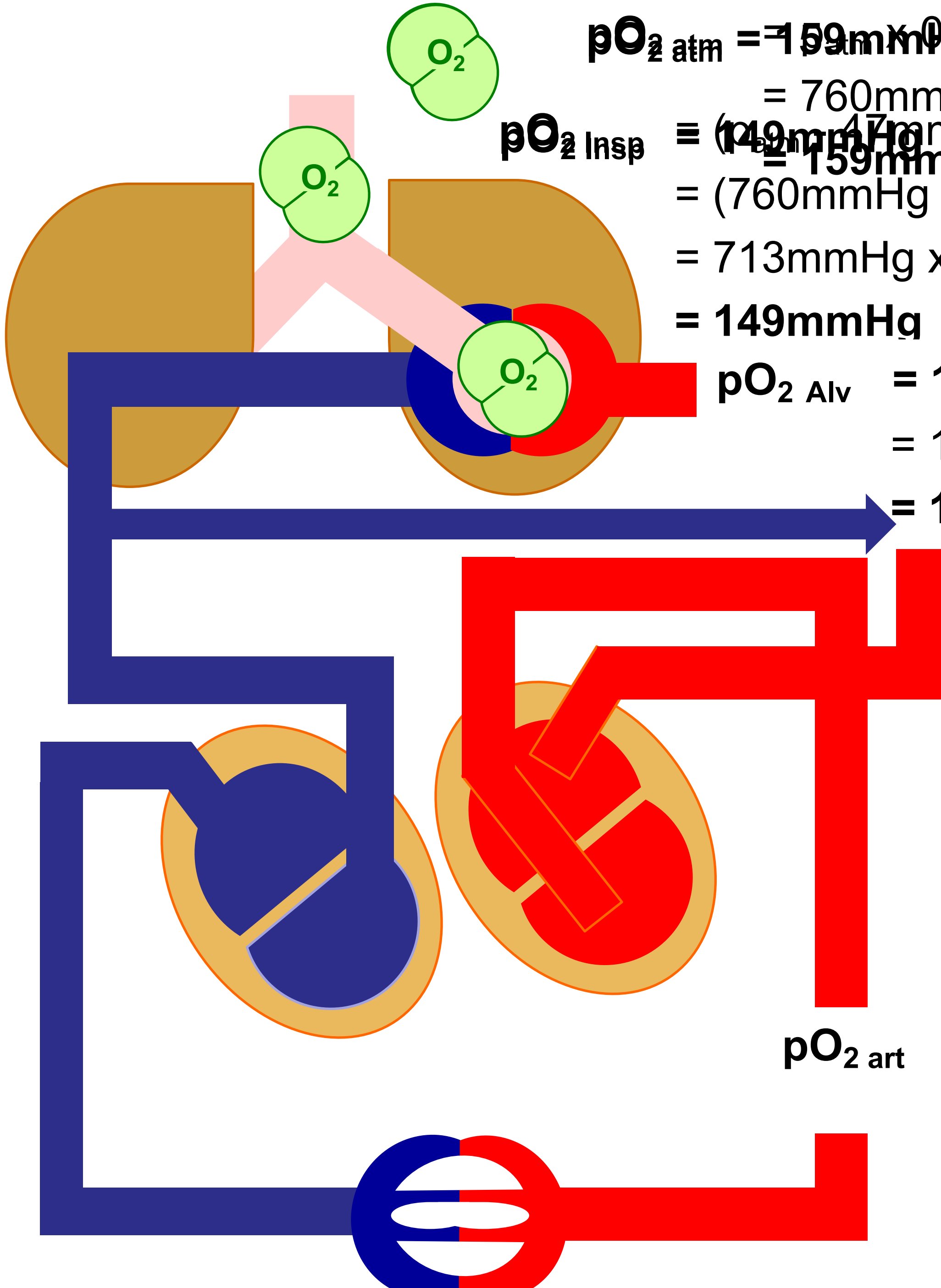
PaO_2 in arterial blood is the result of oxygen uptake via diffusion through the alveolo-capillary membrane from the lungs to the blood



Oxygen uptake: Ventilation-perfusion matching







$pO_{2\ atm}$ = 159mmHg

= P_{atm} x 0.21

= 760mmHg x 0.21

= 159mmHg

$pO_{2\ Insp}$ = 149mmHg

= (P_{atm} - 47mmHg) x 0.21

= (760mmHg - 47mmHg) x 0.21

= 713mmHg x 0.21

= 149mmHg

$pO_{2\ Alv}$ = 100mmHg (pCO_2/R)

= 149 - (40/0.8)

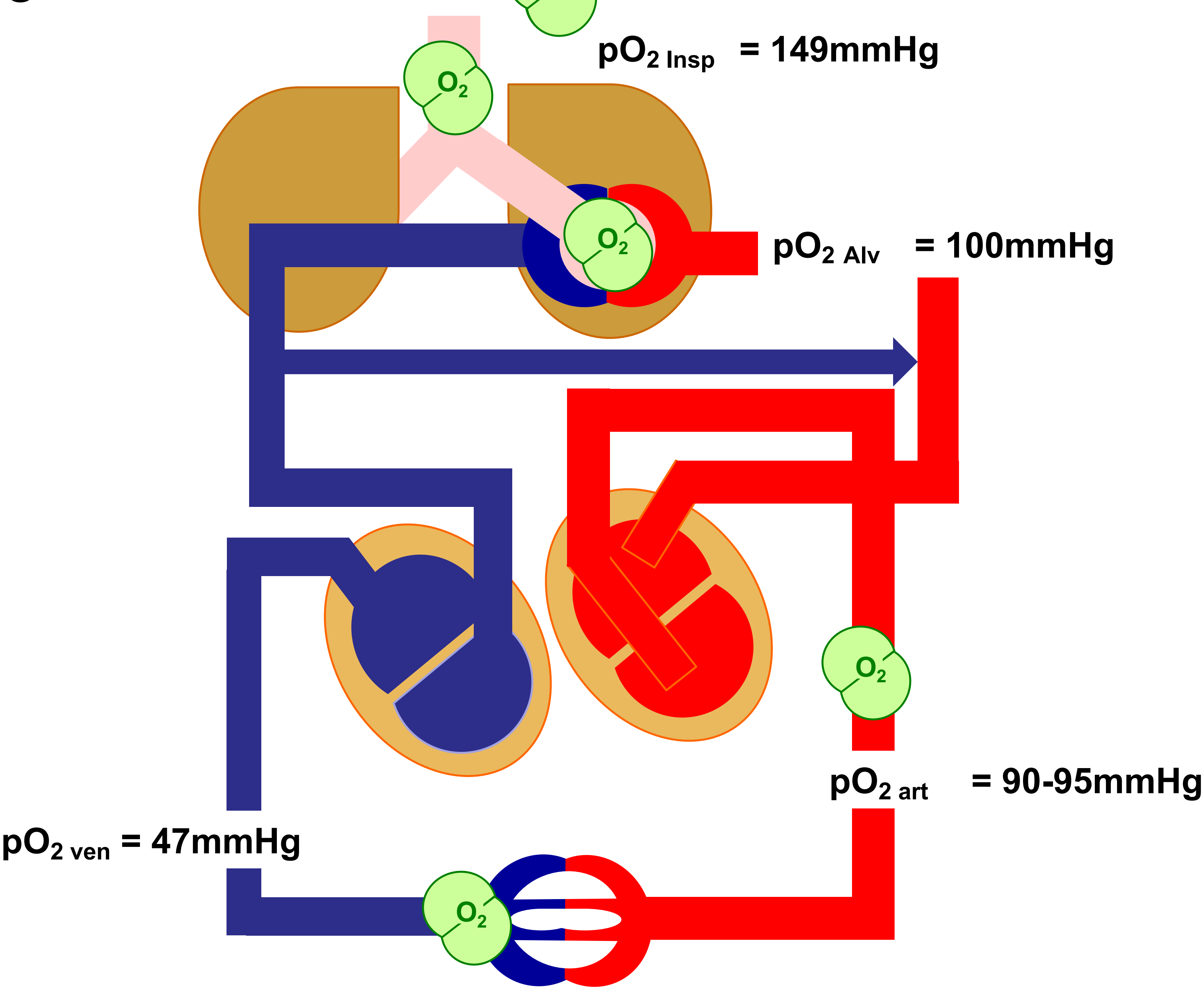
= 100mmHg

$pO_{2\ art}$ = 90-95mmHg

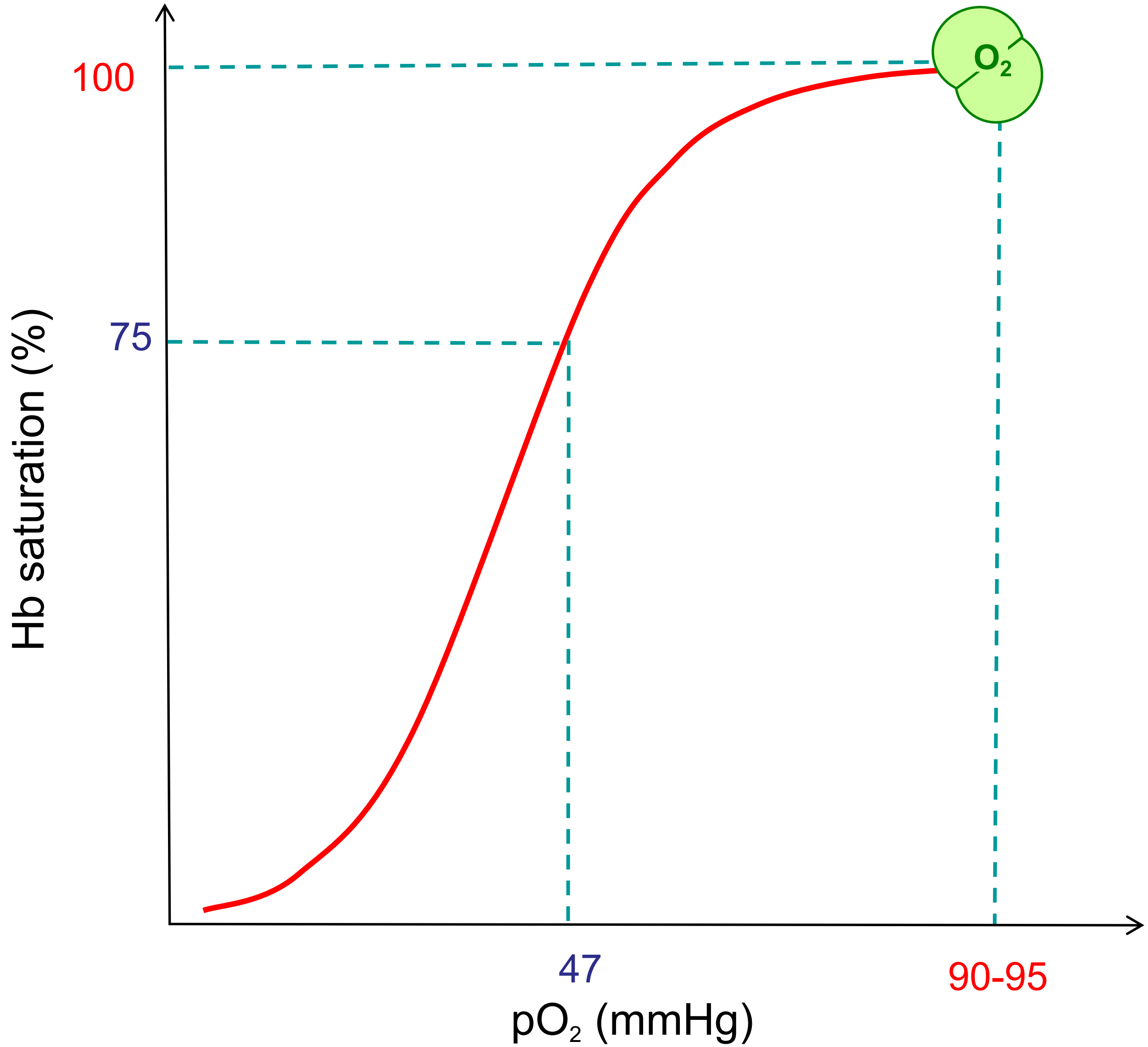
= 90-95mmHg

Oxygen cascade

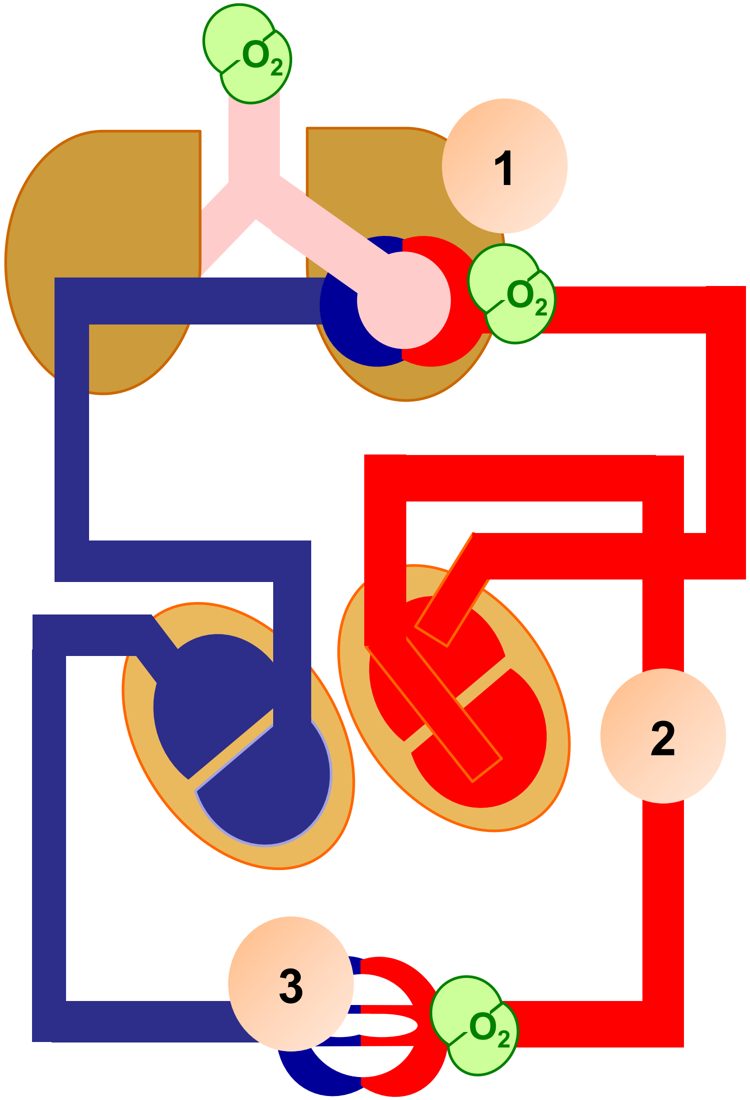
Oxygen cascade



Pulse oximetry



Oxygen



Continuous delivery of oxygen from inspired air to tissue cells

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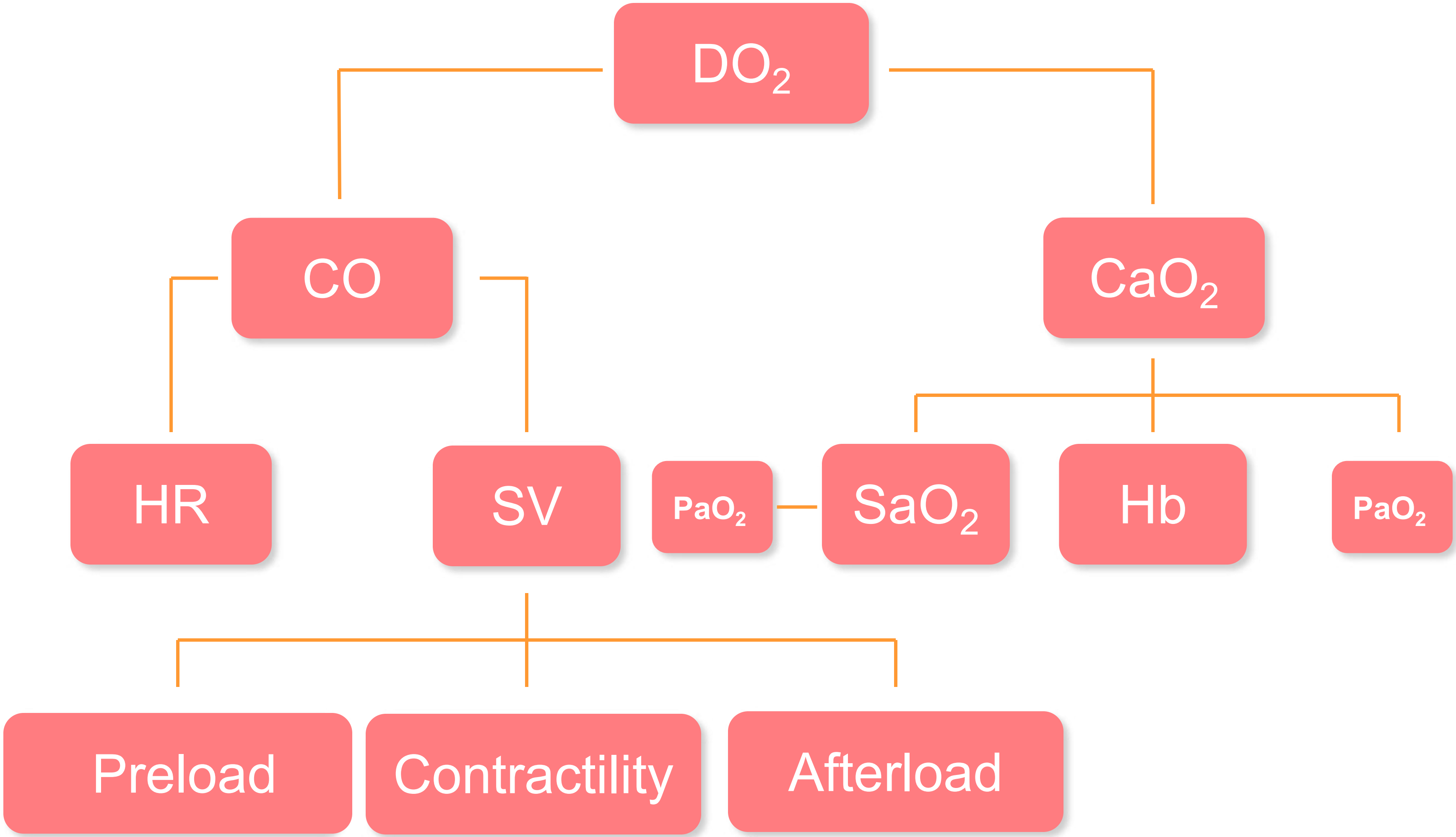
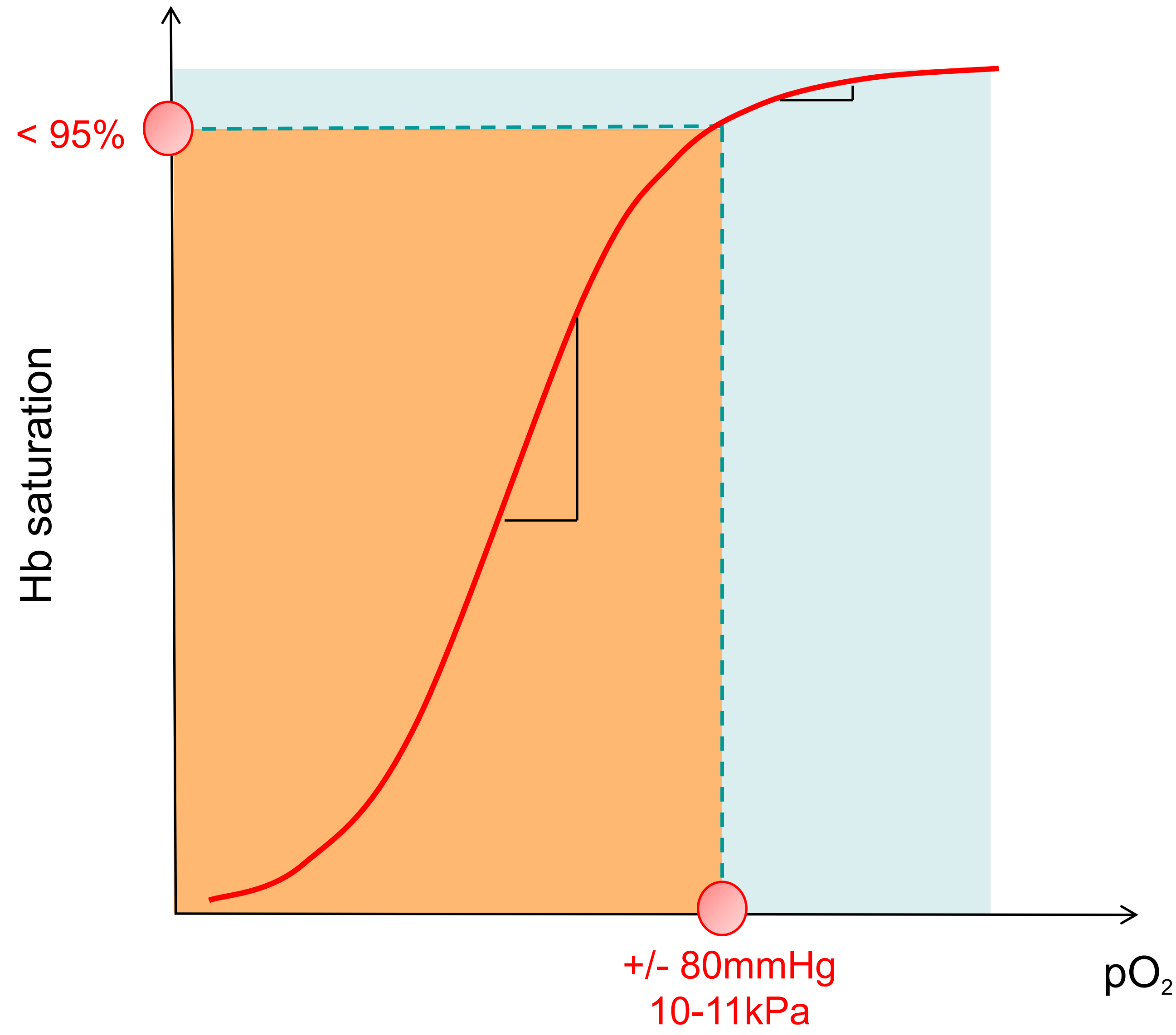
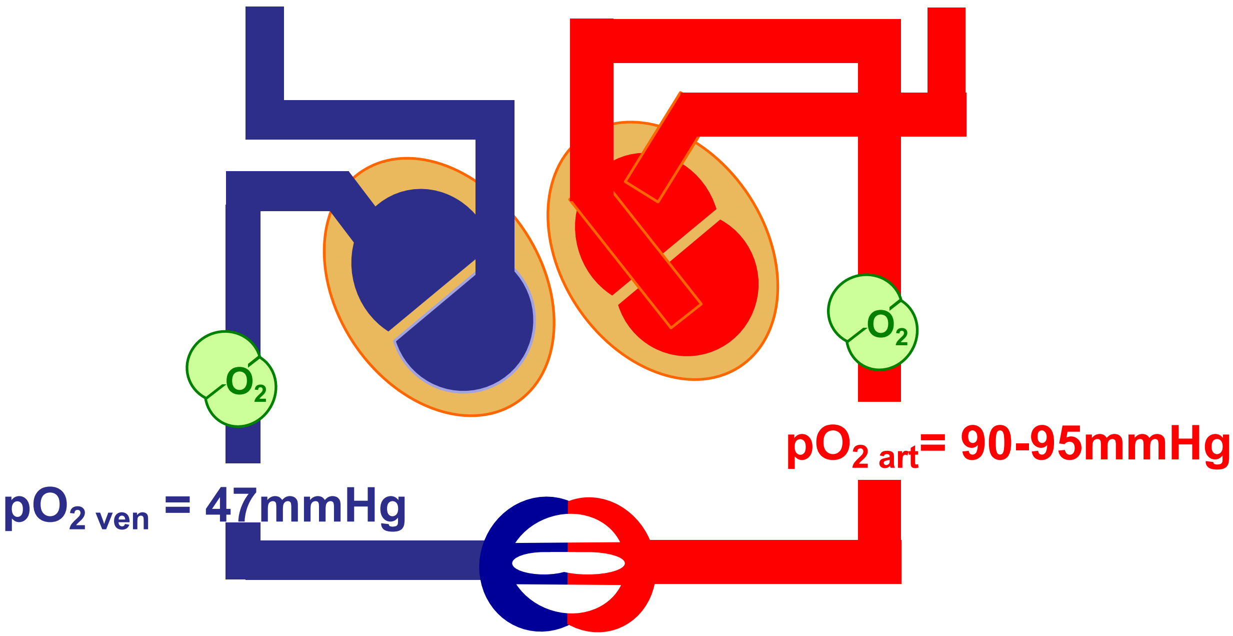
Determinants of oxygen delivery



Oxyhemoglobin dissociation curve



Oxygen delivery - oxygen consumption



CaO_{2ven}
$= (Hb \times SmvO_2 \times 1.39) + (pO_{2\ ven} \times 0.0031)$

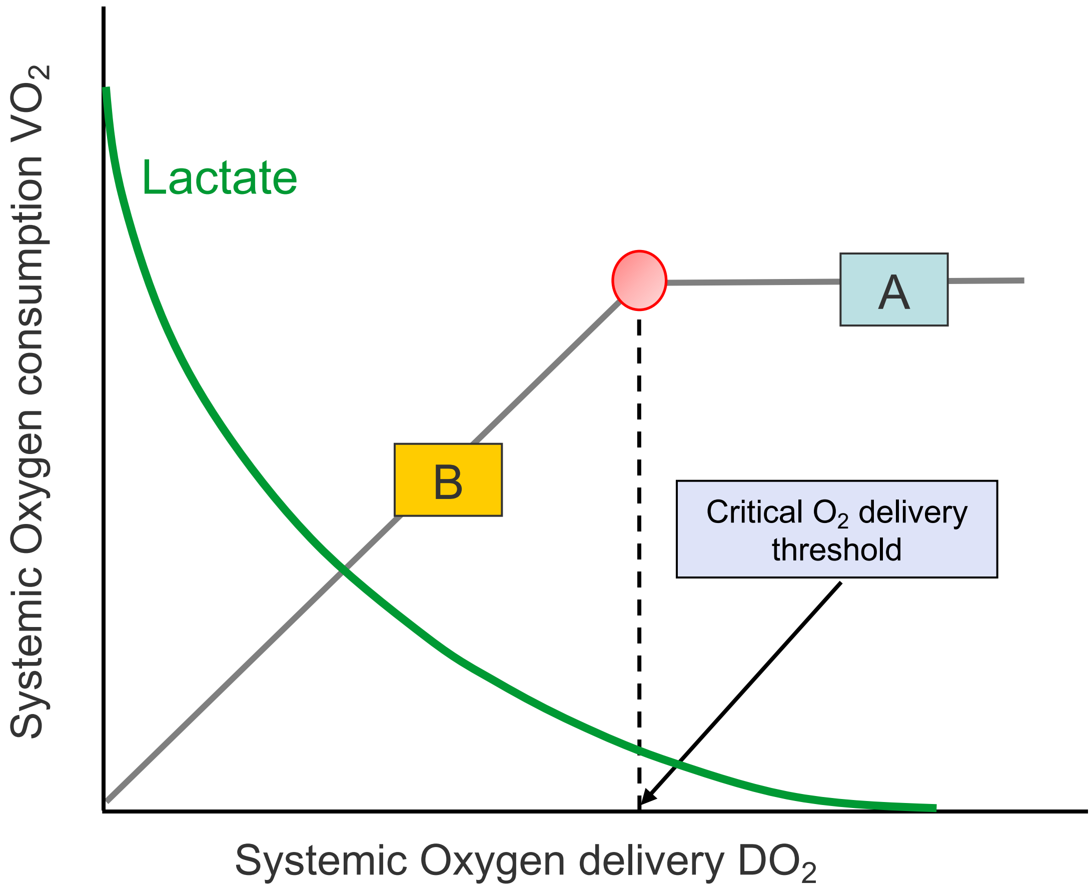
CaO_{2art}
$= (Hb \times SaO_2 \times 1.39) + (pO_{2\ art} \times 0.0031)$

. Blut ⟶

VO_2= 4.1ml O_2/dL Blut x 50 = 205mlO_2/min

DO_2= 19.8ml O_2/dL Blut x 50 = 990mlO_2/min

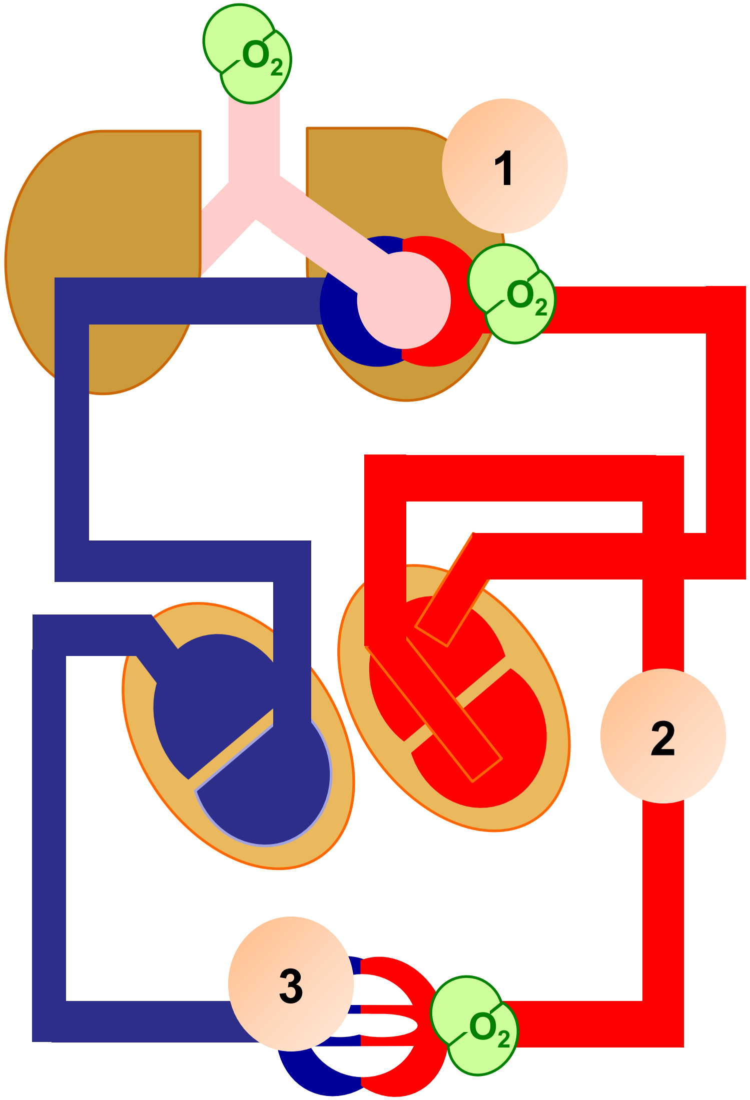
Relationship between DO_2 and VO_2



A = delivery independent phase

B = delivery dependent phase

Oxygen



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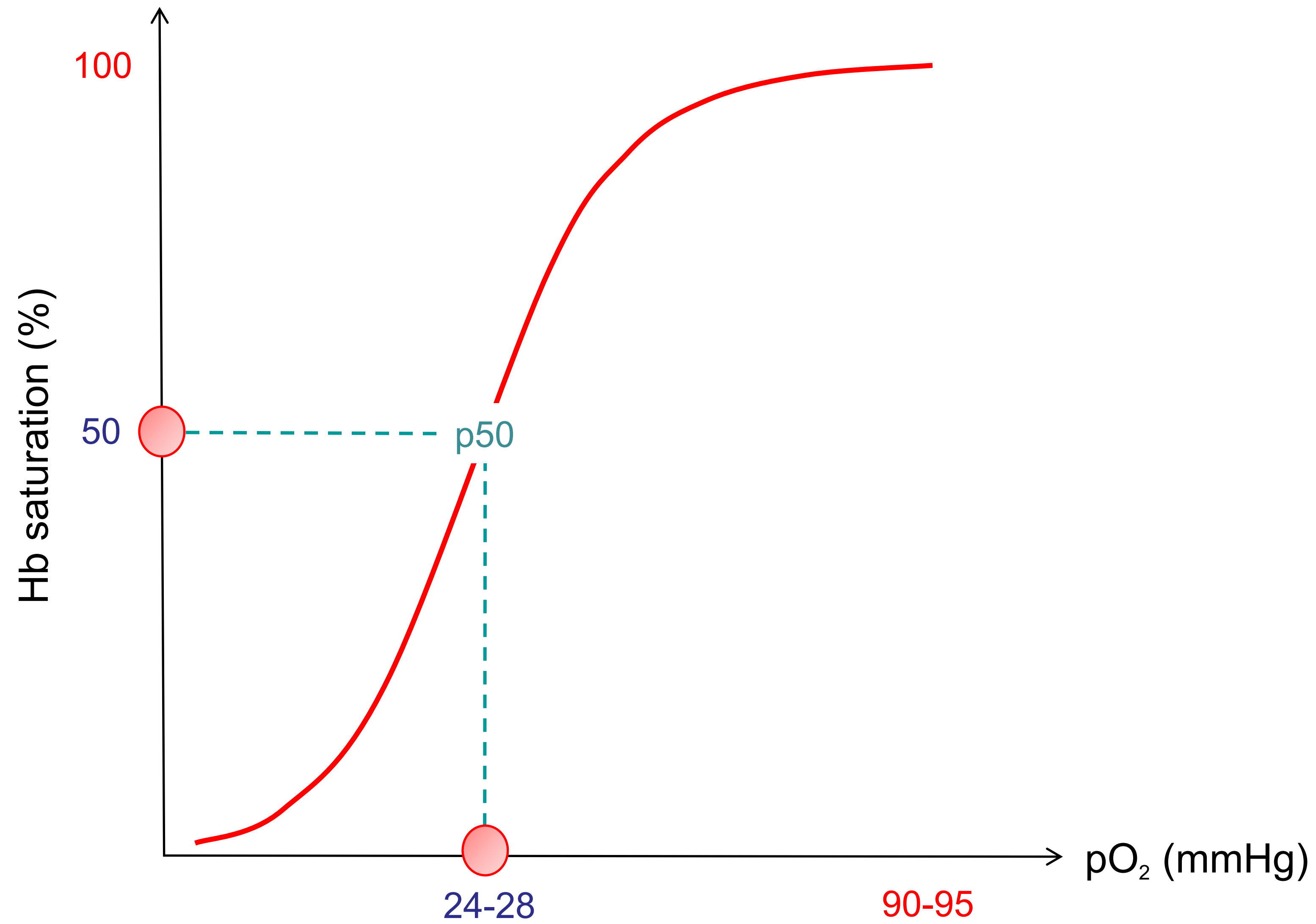
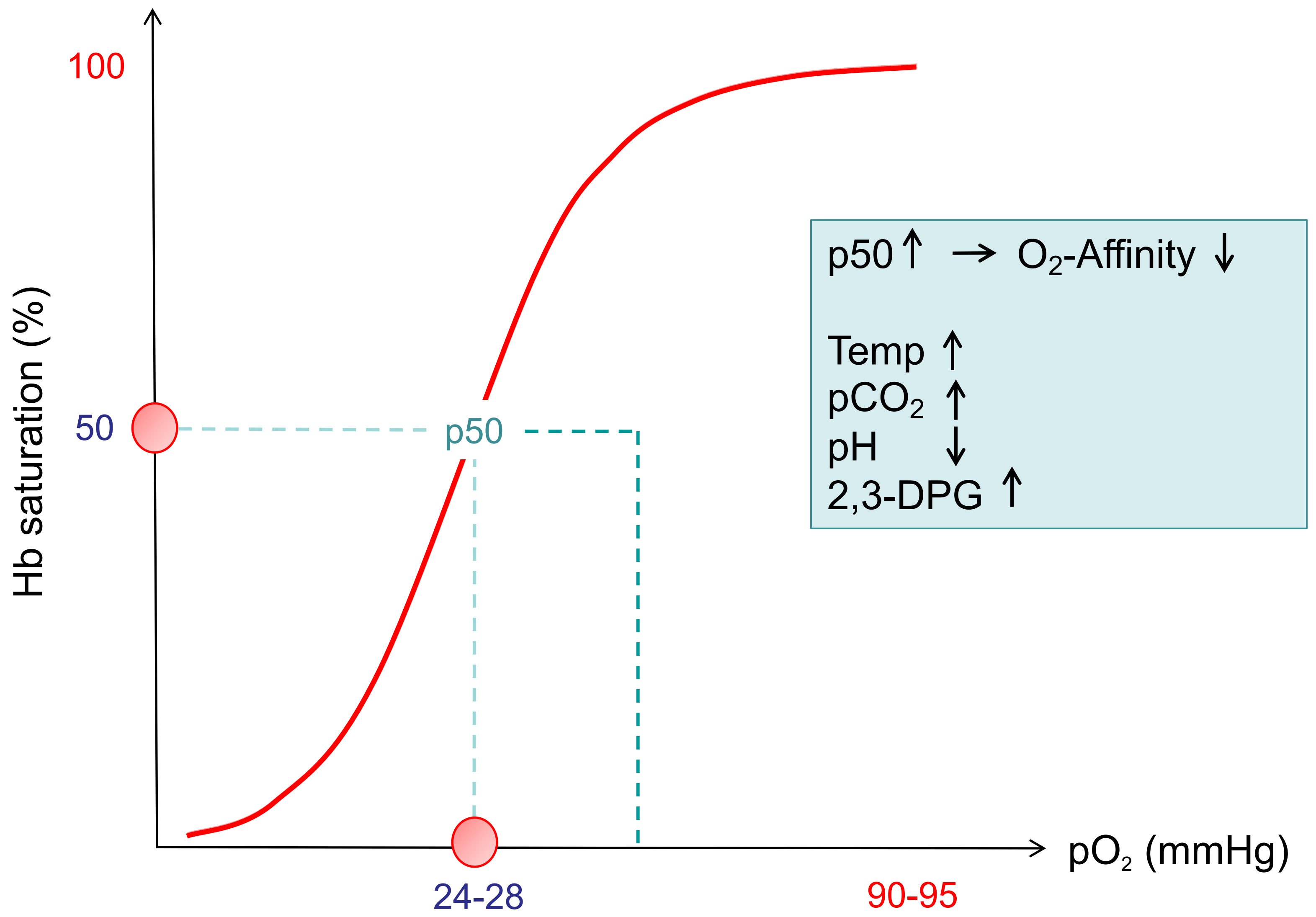
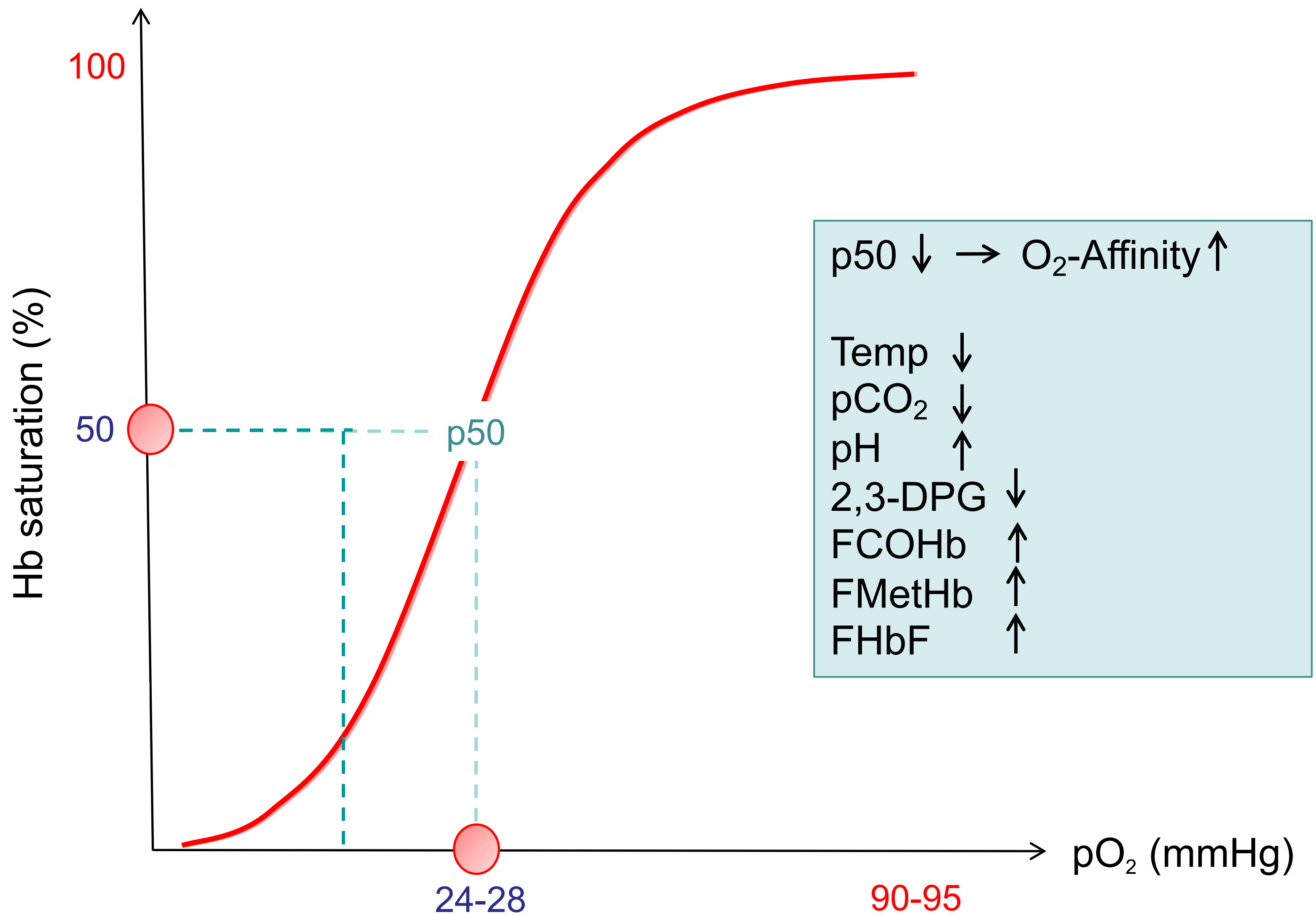
Oxygen release



Oxygen release



Oxygen release



Oxygen status and blood gas analysis

Blutgas Ergebnis

pH	6.885	
pCO_2	44.1	mmHg
pO_2	319*	mmHg
$cHCO_3^-(P,st)_c$	8.5	mmol/L
$cBase(Ecf)_c$	-24.8	mmol/L
sO_2	98.3	%

Oxymetrie Ergebnis

ctHb	138	g/L
sO_2	98.3	%

* 42.5kPa

Oxygen status and blood gas analysis

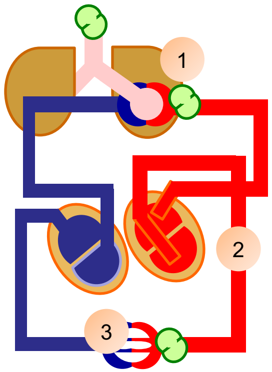
lutgas Ergebnis		
pH	6.885	
pCO_2	44.1	mmHg
pO_2	319	mmHg
$cHCO_3^-(P,st)_c$	8.5	mmol/L
$cBase(Ecf)_c$	-24.8	mmol/L
sO_2	98.3	%
xymetrie Ergebnis		
ctHb	138	g/L
sO_2	98.3	%
FO_2Hb	58.9	%
FCOHb	37.9	%
FHHb	1.0	%
FMetHb	2.2	%

Hemoglobin		
	Oxyhemoglobin	FO_2Hb
	Carboxyhemoglobin	FCOHb
	Deoxyhemoglobin	FHHb
	Methemoglobin	FMetHb

$$sO_2(\%) = \frac{FO_2Hb}{FO_2Hb + FHHb} \times 100 = 98.3\%$$

$$O_2Hb(\%) = \frac{FO_2Hb}{FO_2Hb + FHHb + FCOHb + FMetHb} \times 100 = 58.9\%$$

Oxygen status and blood gas analysis

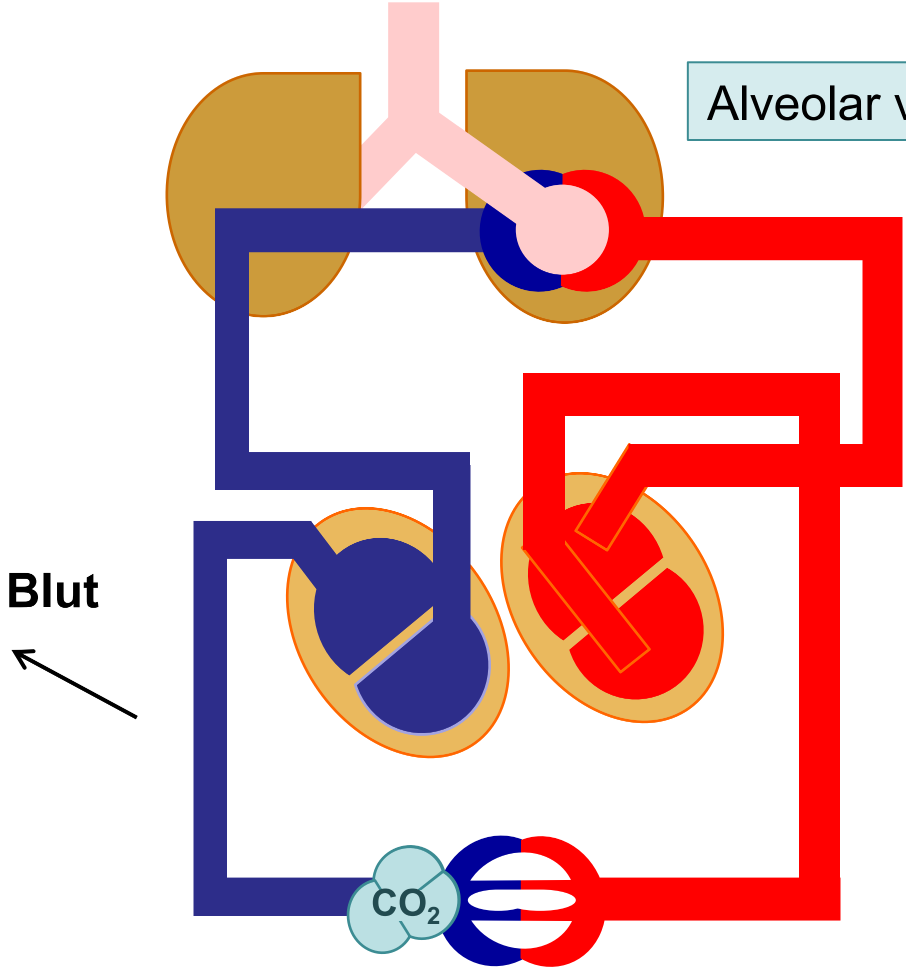


Continuous delivery of oxygen from inspired air to tissue cells

3 sequential events	Surrogate marker	
1 Uptake of oxygen from alveolar air into the lungs	PaO_2	
2 Transport/delivery of oxygen in blood from lung to tissues	CaO_2	Lactate
	Hypoxaemia	Hypoxia
3 Release of oxygen from blood to tissues	p50	• hypoxemic • ischemic • anemic • histotoxic

Carbon dioxide

$55ml CO_2/100ml$ **Blut**



$VCO_2 = 250ml/min$

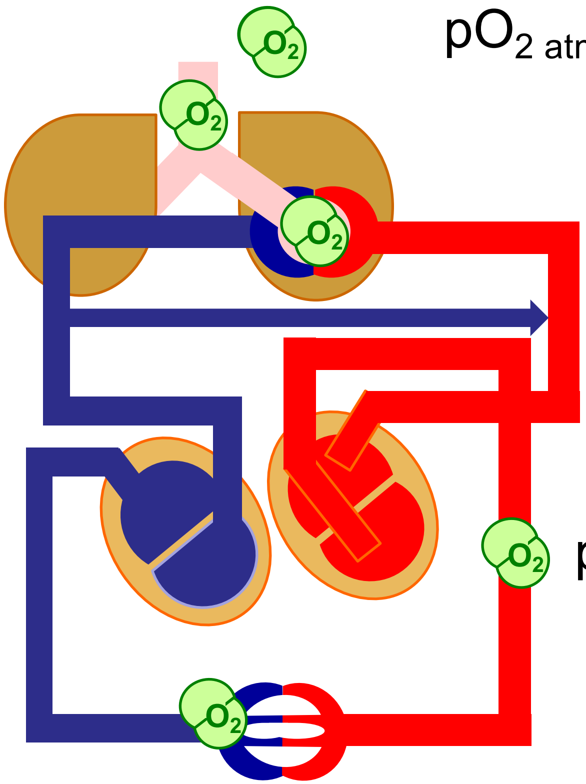
Alveolar ventilation = RR x (V_T-V_D)

- Largely dependent on alveolar ventilation
- Anatomical dead space constant but physiological dead space depends on ventilation-perfusion matching

Pathophysiology

Hypoxaemic respiratory failure

- Low inspired pO_2

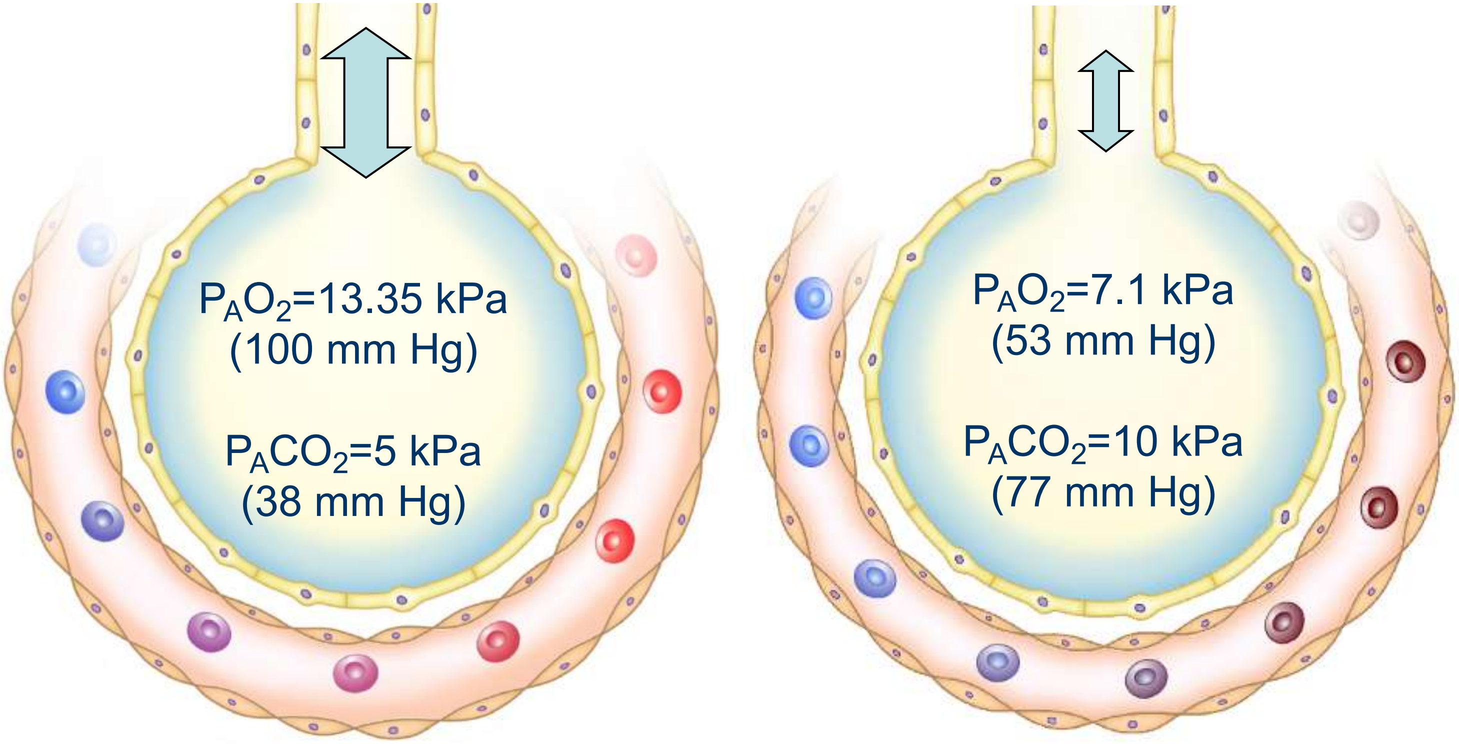


	Zurich	Everest
	$pO_{2\,atm} = p_{atm} \times 0.21$	$pO_{2\,atm} = p_{atm} \times 0.21$
	$= 760mmHg \times 0.21$	$= 253mmHg \times 0.21$
	$= 159mmHg$	$= 53mmHg$
	$pO_{2\,art} = 95mmHg$	$pO_{2\,art} = 24.6\ mmHg$ *

* Grocott, NEJM 2009

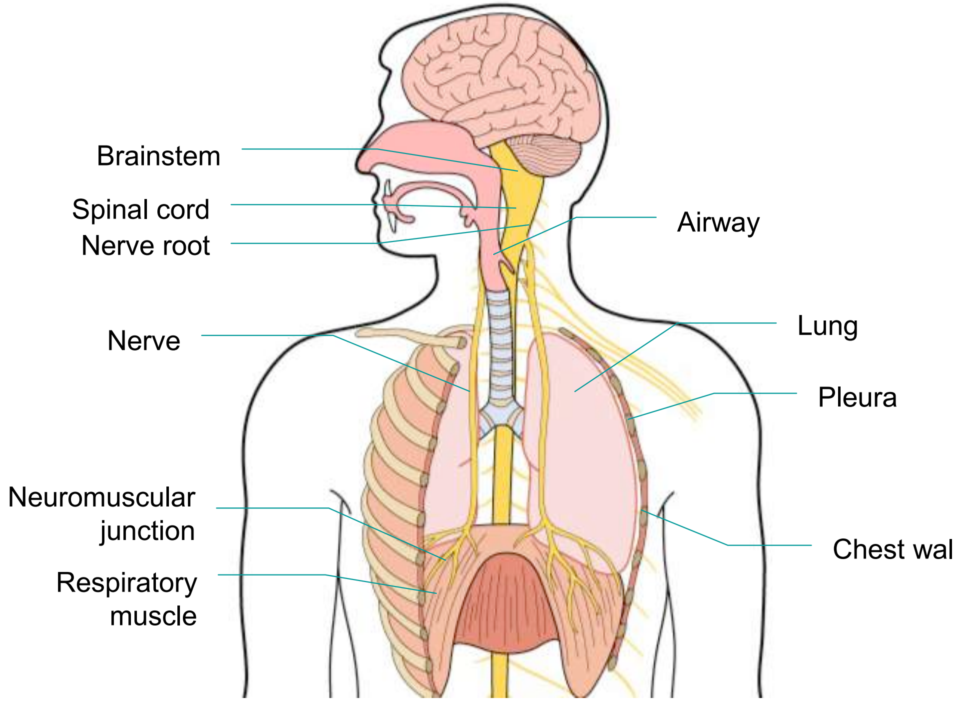
Hypoxaemic respiratory failure

- Low inspired pO_2
- Hypoventilation



Hypoxaemic respiratory failure

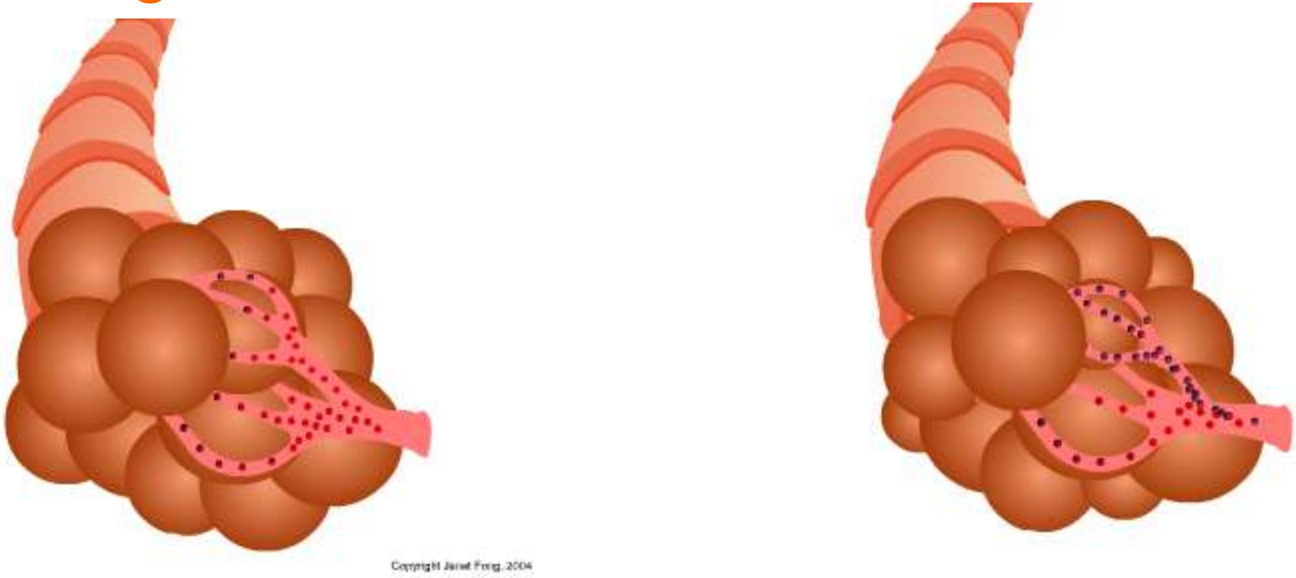
- Low inspired pO_2
- Hypoventilation



Sites at which disease may cause hypoventilation

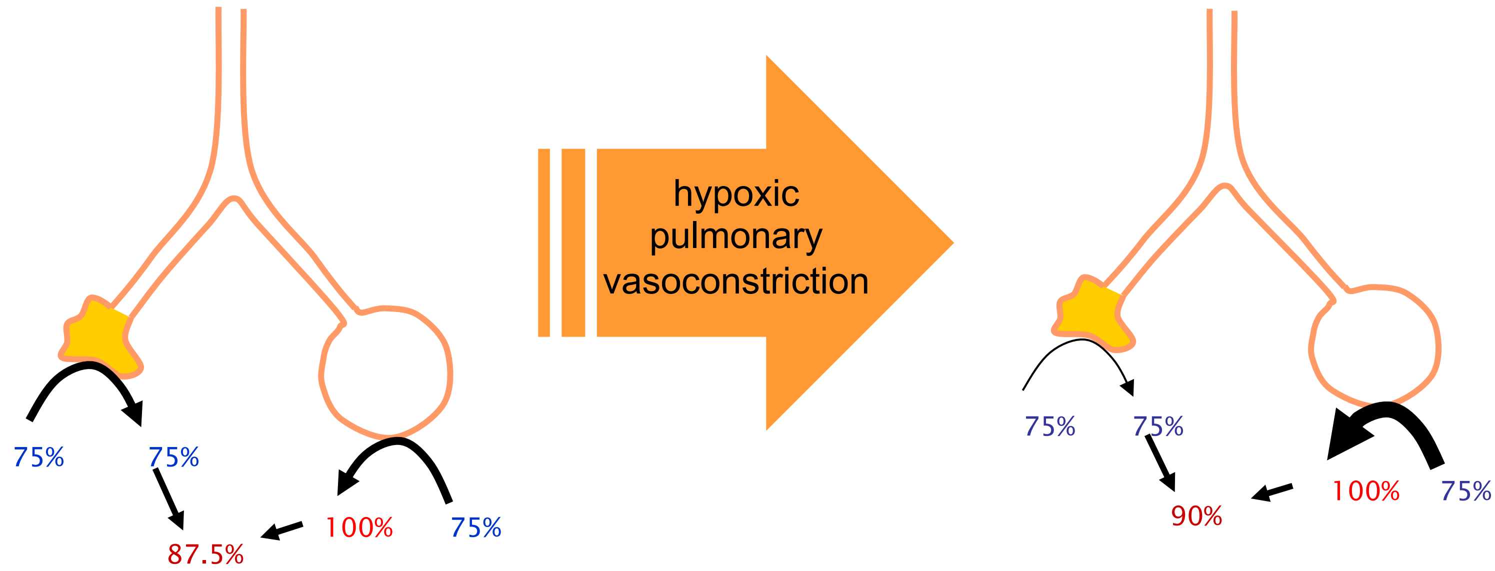
Hypoxaemic respiratory failure

- Low inspired pO_2
- Hypoventilation
- Ventilation-perfusion mismatch
  - Shunting



Hypoxaemic respiratory failure

- Low inspired pO_2
- Hypoventilation
- Ventilation-perfusion mismatch
  - – Shunting



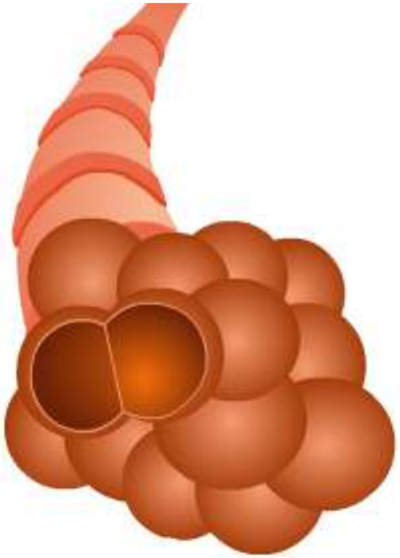
Hypoxaemic respiratory failure

- Low inspired pO_2
- Hypoventilation
- Ventilation-perfusion mismatch
  - Shunting: Intra-pulmonary
    - Pneumonia
    - Pulmonary oedema
    - Atelectasis
    - Collapse
    - Pulmonary haemorrhage or contusion

    Intra-cardiac
    - Any cause of right to left shunt
      - eg Fallot's, Eisenmenger,
      - Pulmonary hypertension with patent foramen ovale

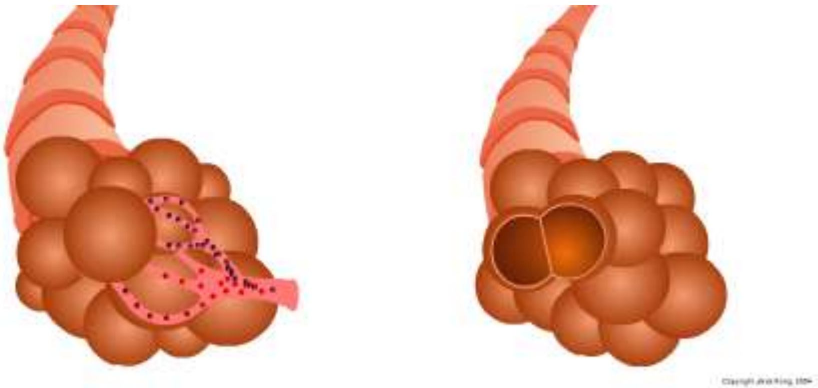
Hypoxaemic respiratory failure

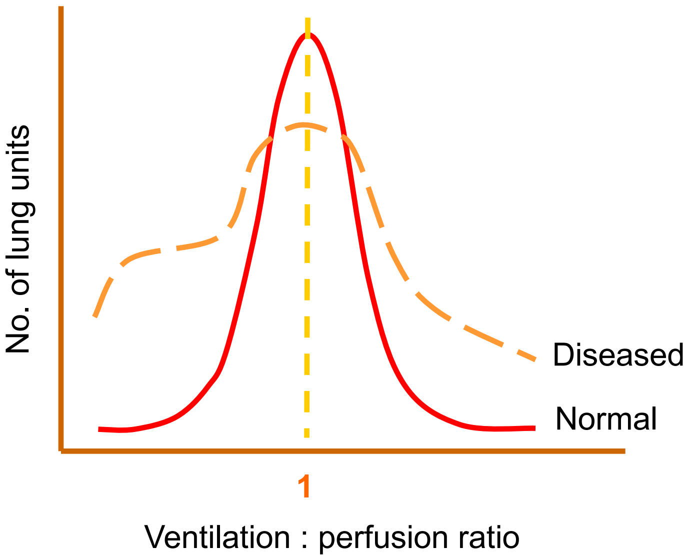
- Low inspired pO_2
- Hypoventilation
- Ventilation-perfusion mismatch
  - Shunting
  - Dead space ventilation



Hypoxaemic respiratory failure

- Low inspired pO_2
- Hypoventilation
- Ventilation-perfusion mismatch
  - Shunting
  - Dead space ventilation





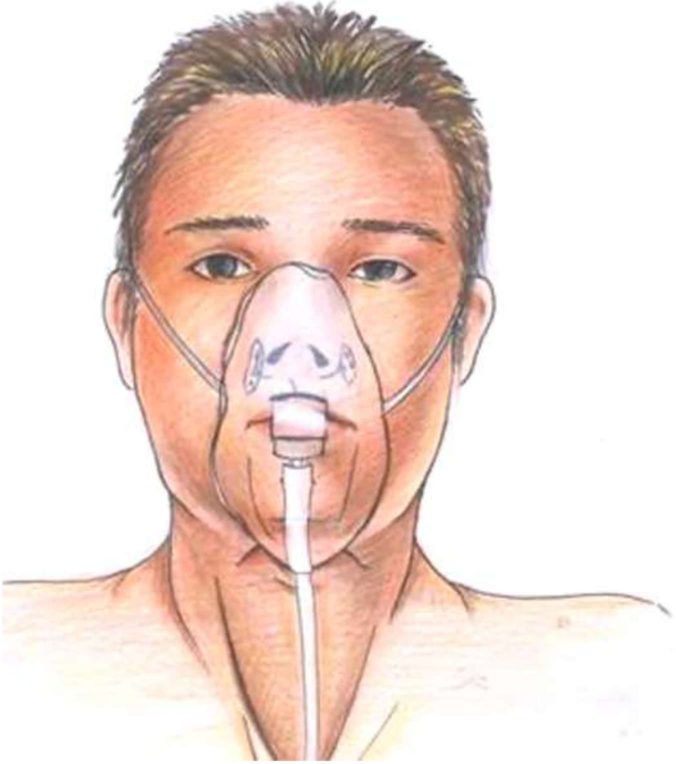
Hypoxaemic respiratory failure

- Low inspired pO_2
- Hypoventilation
- Ventilation-perfusion mismatch
  - Shunting
  - Dead space ventilation
- Diffusion abnormality

Respiratory monitoring

Clinical

- Respiratory compensation
  - Tachypnoea
  - Accessory muscles
  - Recesssion
  - Nasal flaring



Clinical

- Respiratory compensation
- Sympathetic stimulation
  - ↑HR
  - ↑BP (early)
  - sweating

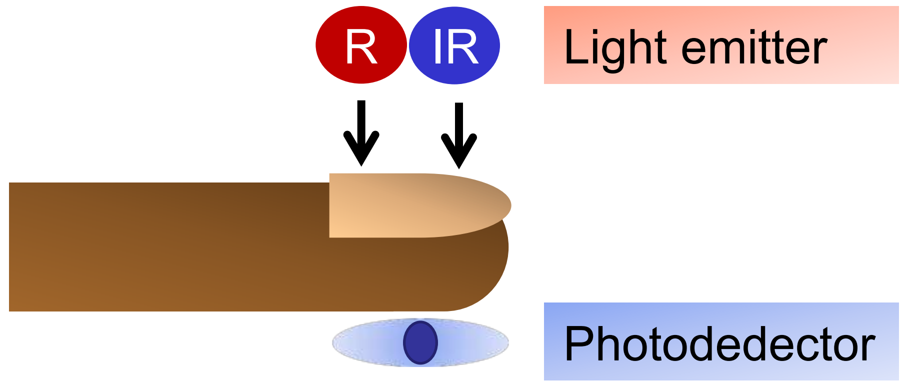
Clinical

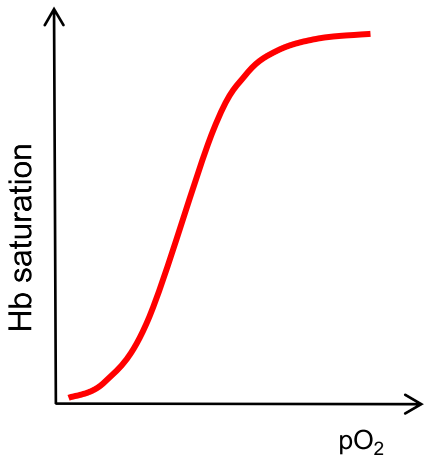
- Respiratory compensation
- Sympathetic stimulation
- Tissue hypoxia
  - Altered mental state
  - ↓HR and ↓BP (late)

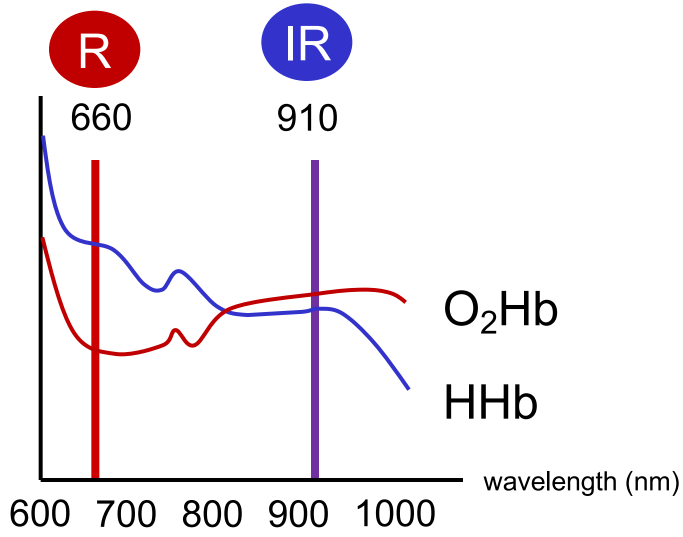
Clinical

- Respiratory compensation
- Sympathetic stimulation
- Tissue hypoxia
- Haemoglobin desaturation
  - Cyanosis (FHHb > 50g/L)

Pulse oximetry







$$SpO_2\ (\%) = \frac{FO_2Hb}{FO_2Hb + FHHb}$$


123
80
40
87%
HR=95

Treatment

Treatment

- Treat the cause

- Supportive treatment

Oxygen therapy

Non-invasive ventilation

Mechanical ventilation

Treatment

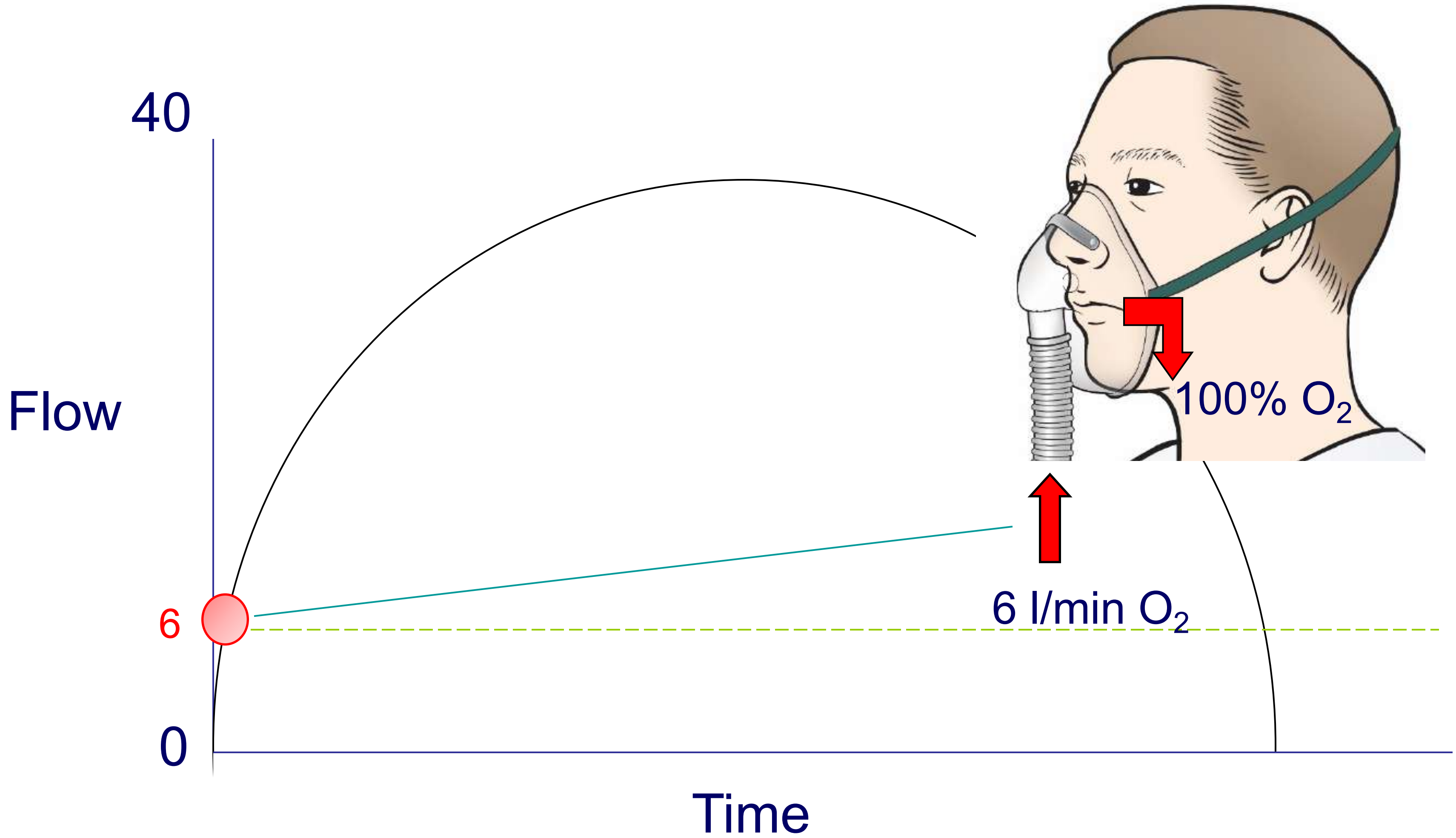
Supportive treatment: Oxygen therapy

Oxygen therapy → Variable performance devices

Oxygen therapy → Fixed performance devices

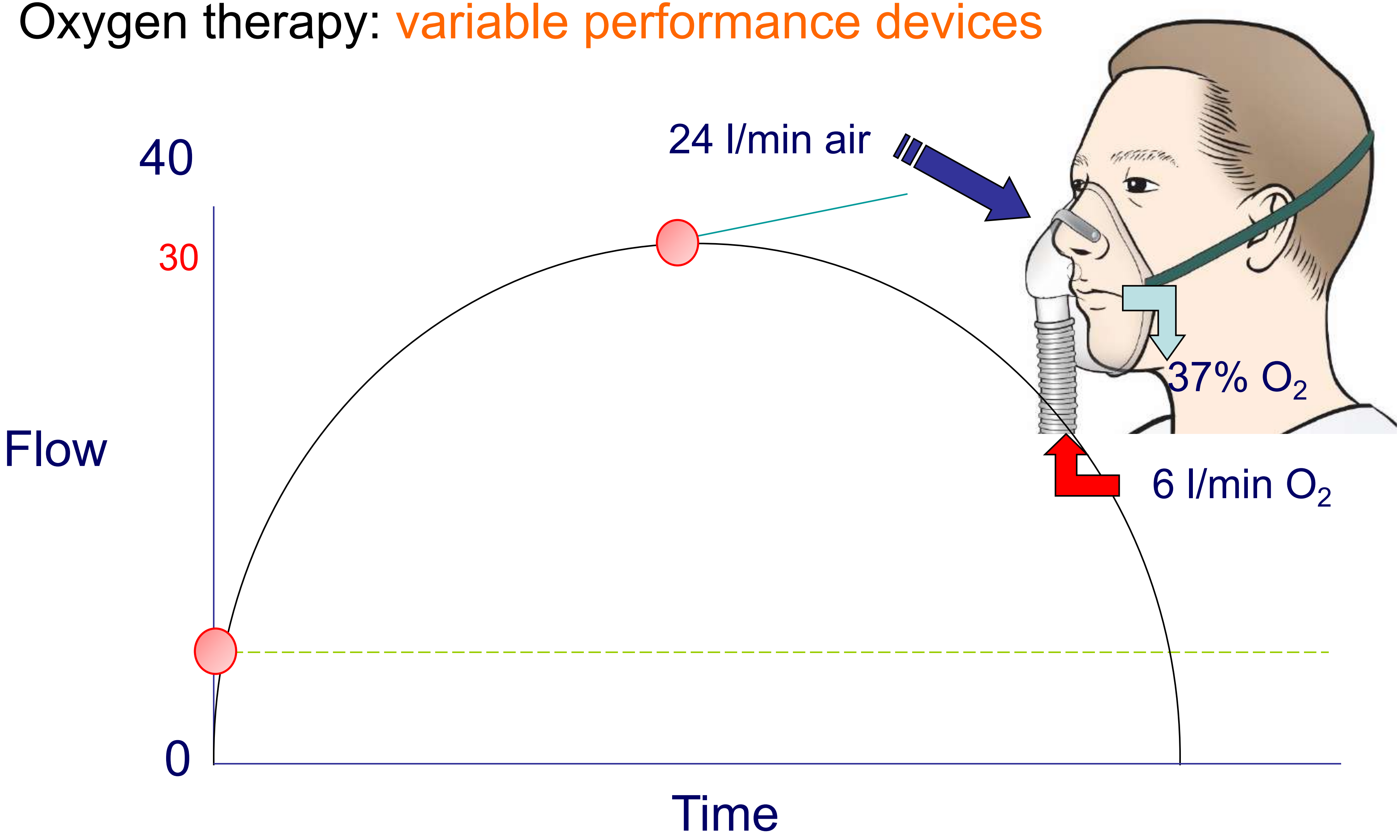
Treatment

Oxygen therapy: variable performance devices



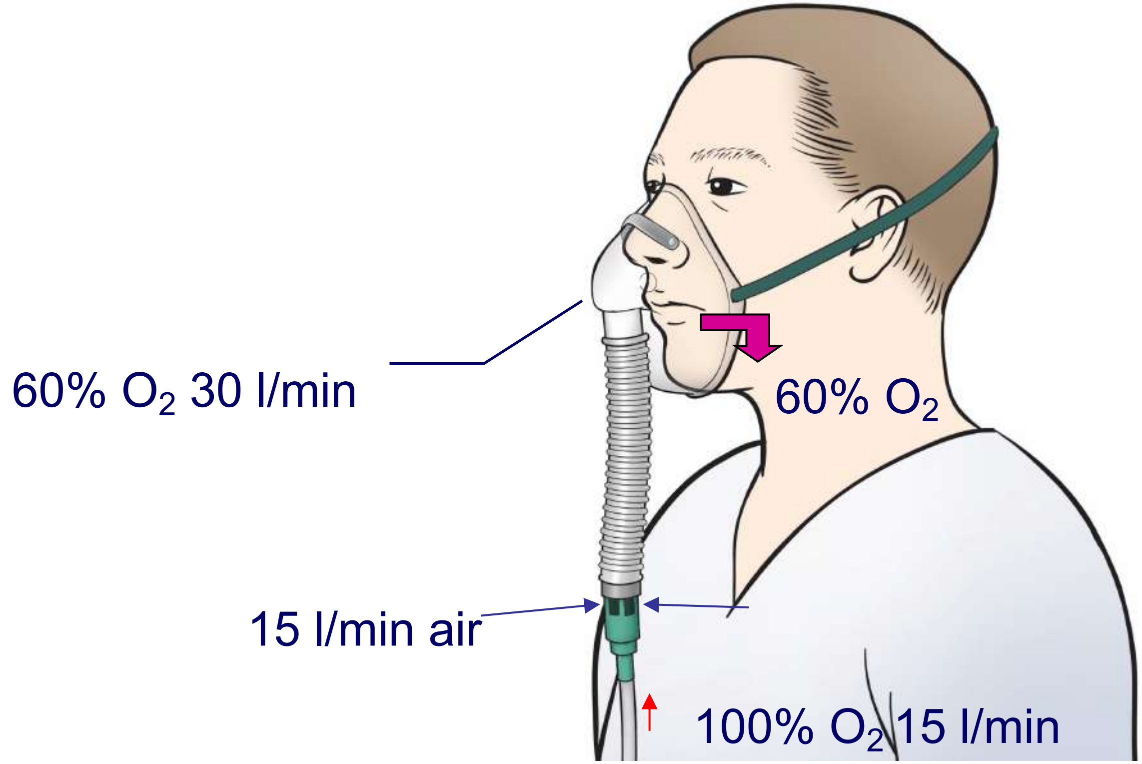
Treatment

Oxygen therapy: variable performance devices



Treatment

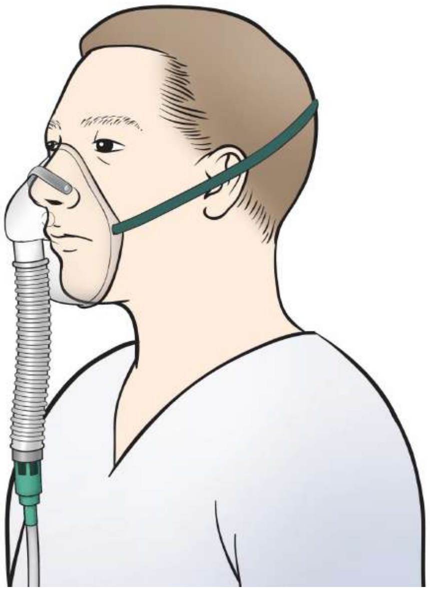
Oxygen therapy: fixed performance devices



Treatment

Oxygen therapy: fixed performance devices

Venturi mask

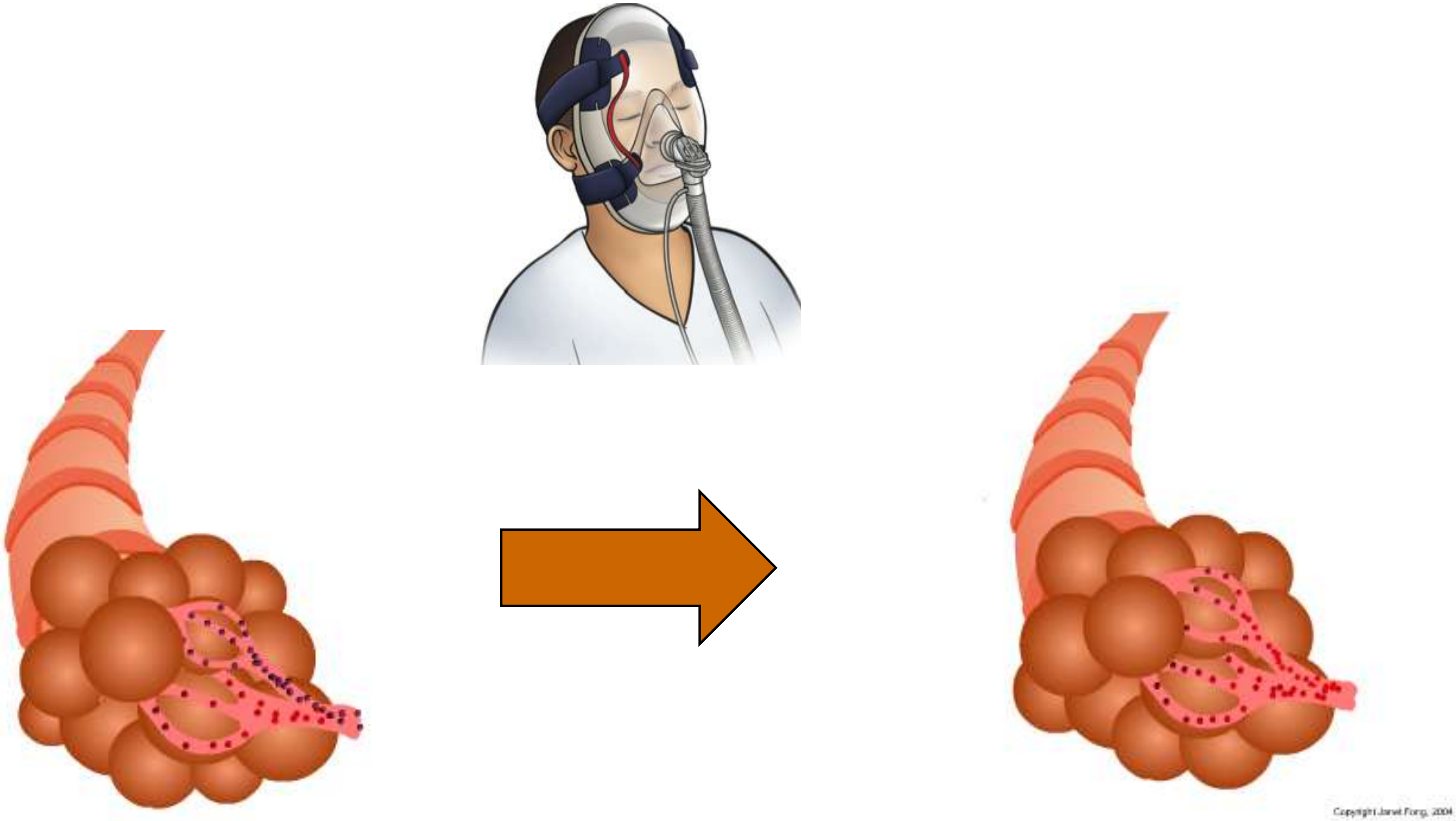


Non-rebreather face mask

Bag valve resuscitator

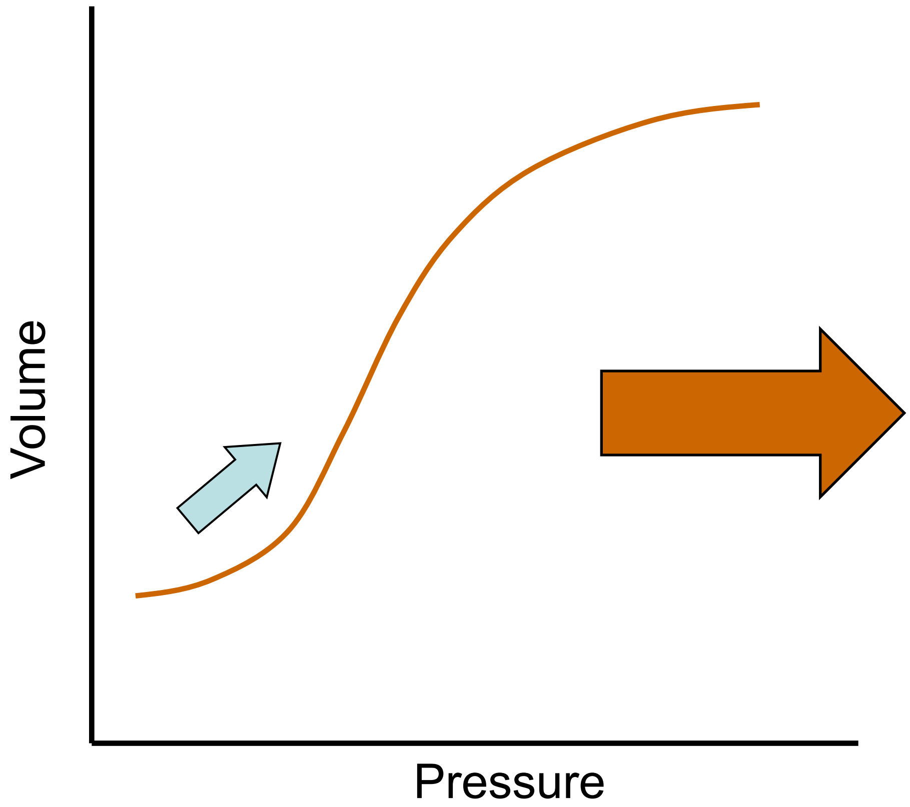
Treatment

Supportive treatment: Non invasive ventilation



Treatment

Supportive treatment: Non invasive ventilation



- reduces shunt by recruiting partially collapsed alveoli
- reduces work of breathing

Treatment

Supportive treatment: mechanical ventilation

Decision to ventilate

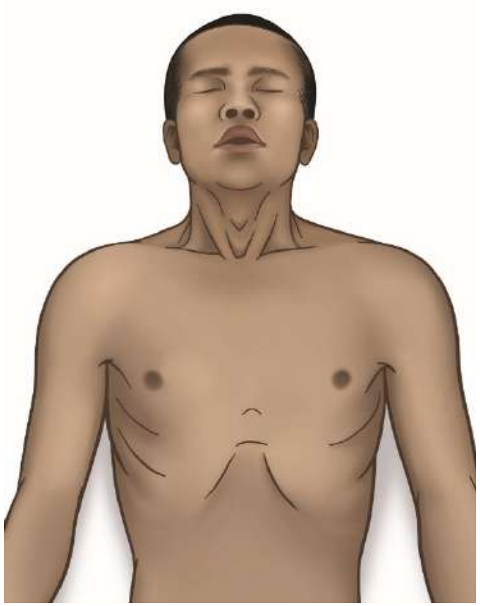
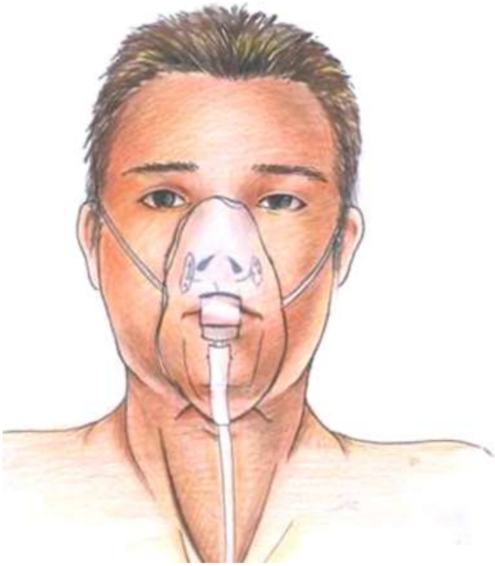
- complex
- multifactorial
- no simple rules

Treatment

Supportive treatment: mechanical ventilation

Factors to consider

- Severity of respiratory failure

Treatment

Supportive treatment: mechanical ventilation

<u>Factors to consider</u>

- Severity of respiratory failure
- Cardiopulmonary reserve

Treatment

Supportive treatment: mechanical ventilation

Factors to consider

- Severity of respiratory failure
- Cardiopulmonary reserve
- Adequacy of compensation
  - ventilatory requirement

Treatment

Supportive treatment: mechanical ventilation

Factors to consider

- Severity of respiratory failure
- Cardiopulmonary reserve
- Adequacy of compensation
  - ventilatory requirement
- Expected speed of response
  - underlying disease
  - treatment already given

Treatment

Supportive treatment: mechanical ventilation

Factors to consider

- Severity of respiratory failure
- Cardiopulmonary reserve
- Adequacy of compensation
  - ventilatory requirement
- Expected speed of response
  - underlying disease
  - treatment already given
- Risks of mechanical ventilation

Treatment

Supportive treatment: mechanical ventilation

Factors to consider

- Severity of respiratory failure
- Cardiopulmonary reserve
- Adequacy of compensation
  - ventilatory requirement
- Expected speed of response
  - underlying disease
  - treatment already given
- Risks of mechanical ventilation
- Non-respiratory indication for intubation

Summary

Summary

- Basic physiology
  - Oxygenation
  - Ventilation
- Pathophysiology
  - Hypoxemic respiratory failure
- Respiratory monitoring
- Treatment
  - Oxygen therapy, non-invasive ventilation, mechanical ventilation
- Cases

Airway management

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Case

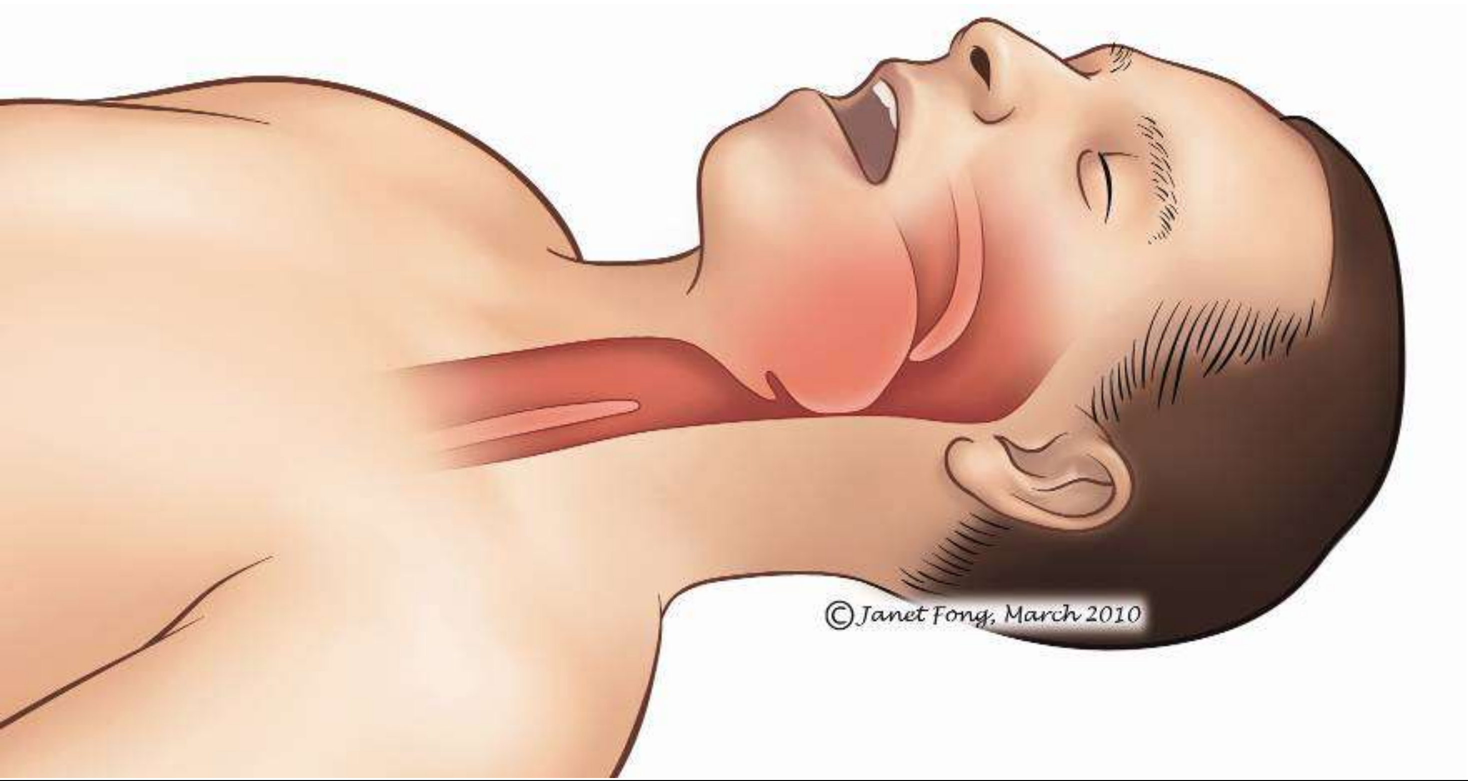
- You are called to see a patient who is unconscious and making a loud snoring noise
- O_2 2 l/min via nasal prongs
- SpO_2 98%

Case

- Does this patient require any airway intervention?

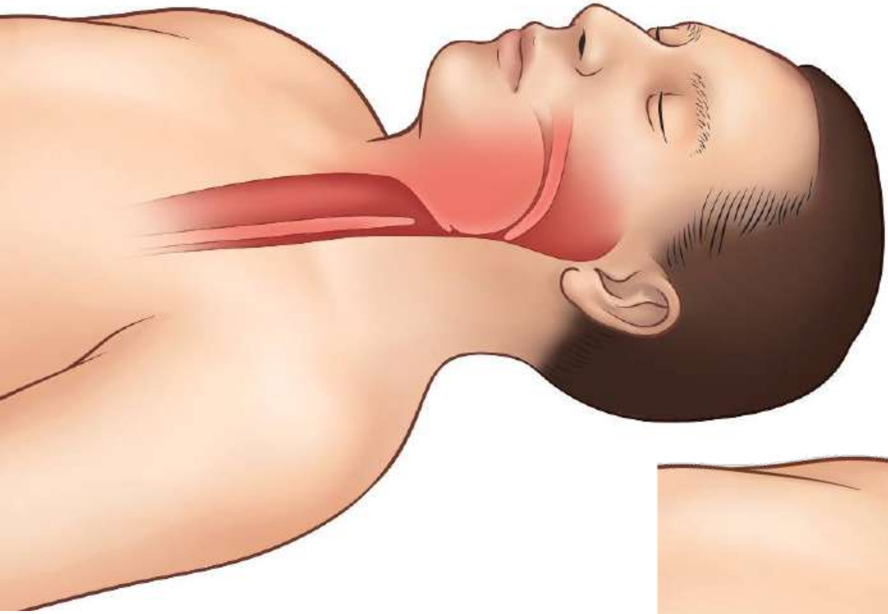
Airway obstruction

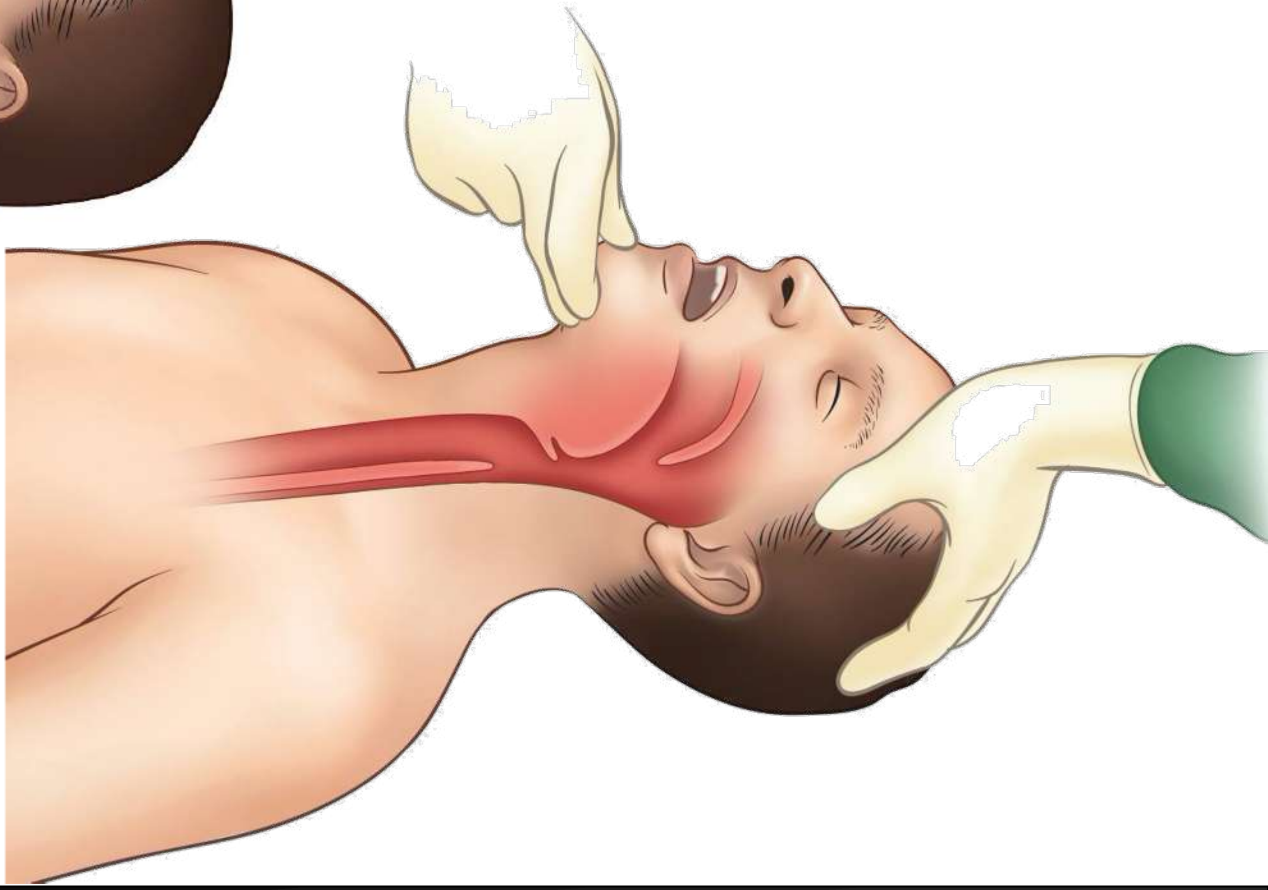
- Normal SpO_2 does not exclude obstruction

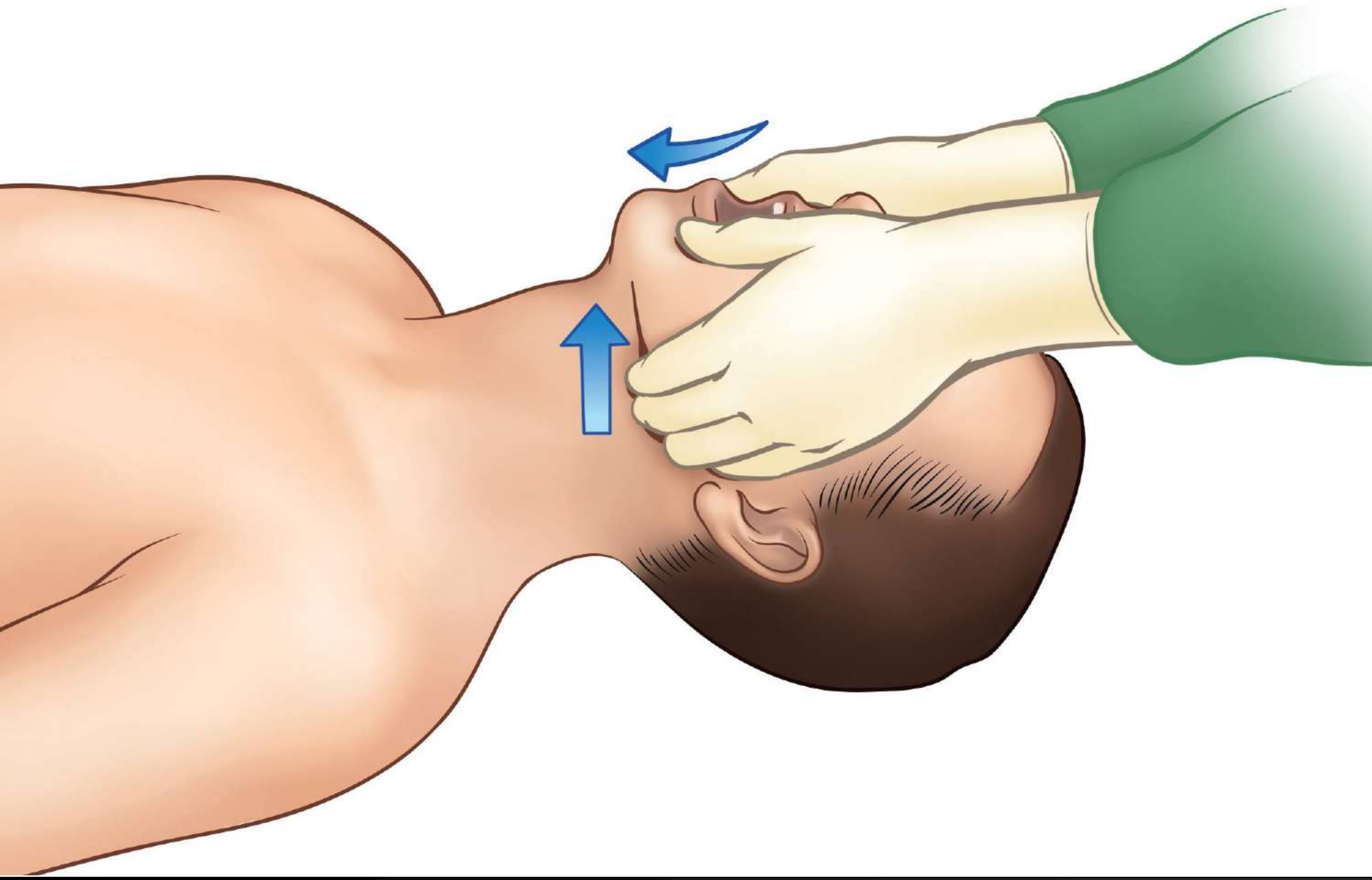


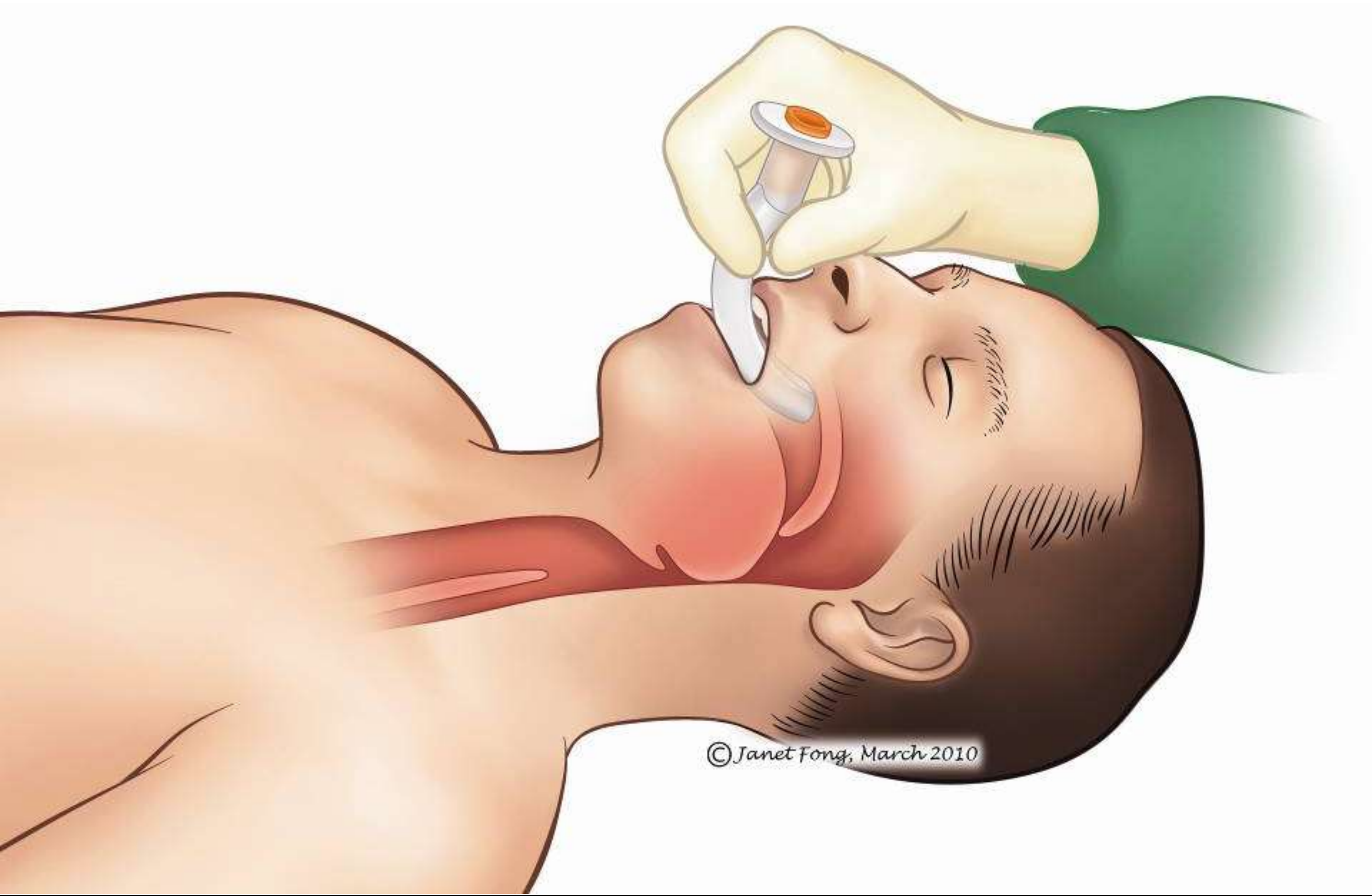
Airway management

- Can be extremely difficult, even for an expert
- Call for help early

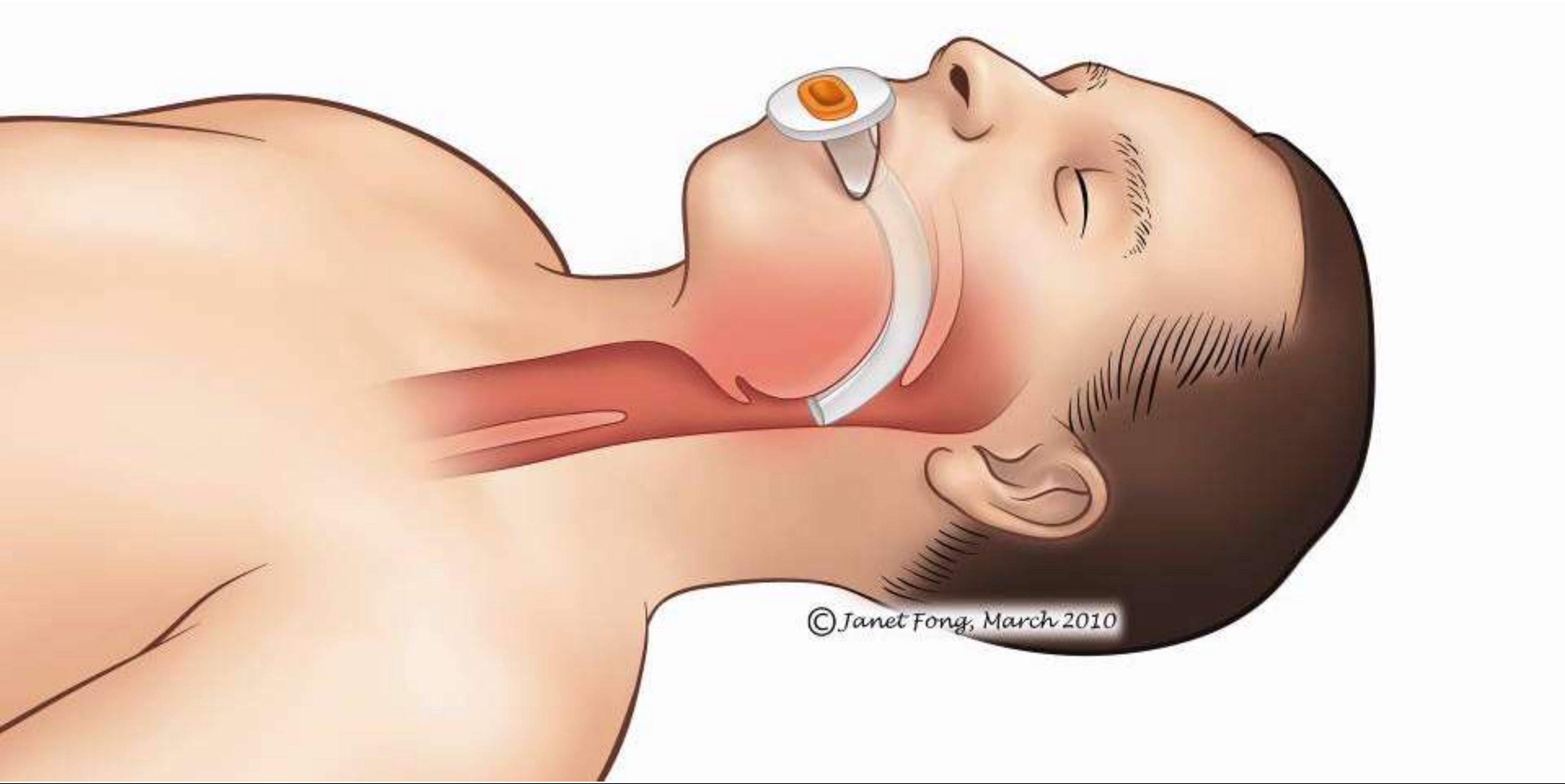








BASIC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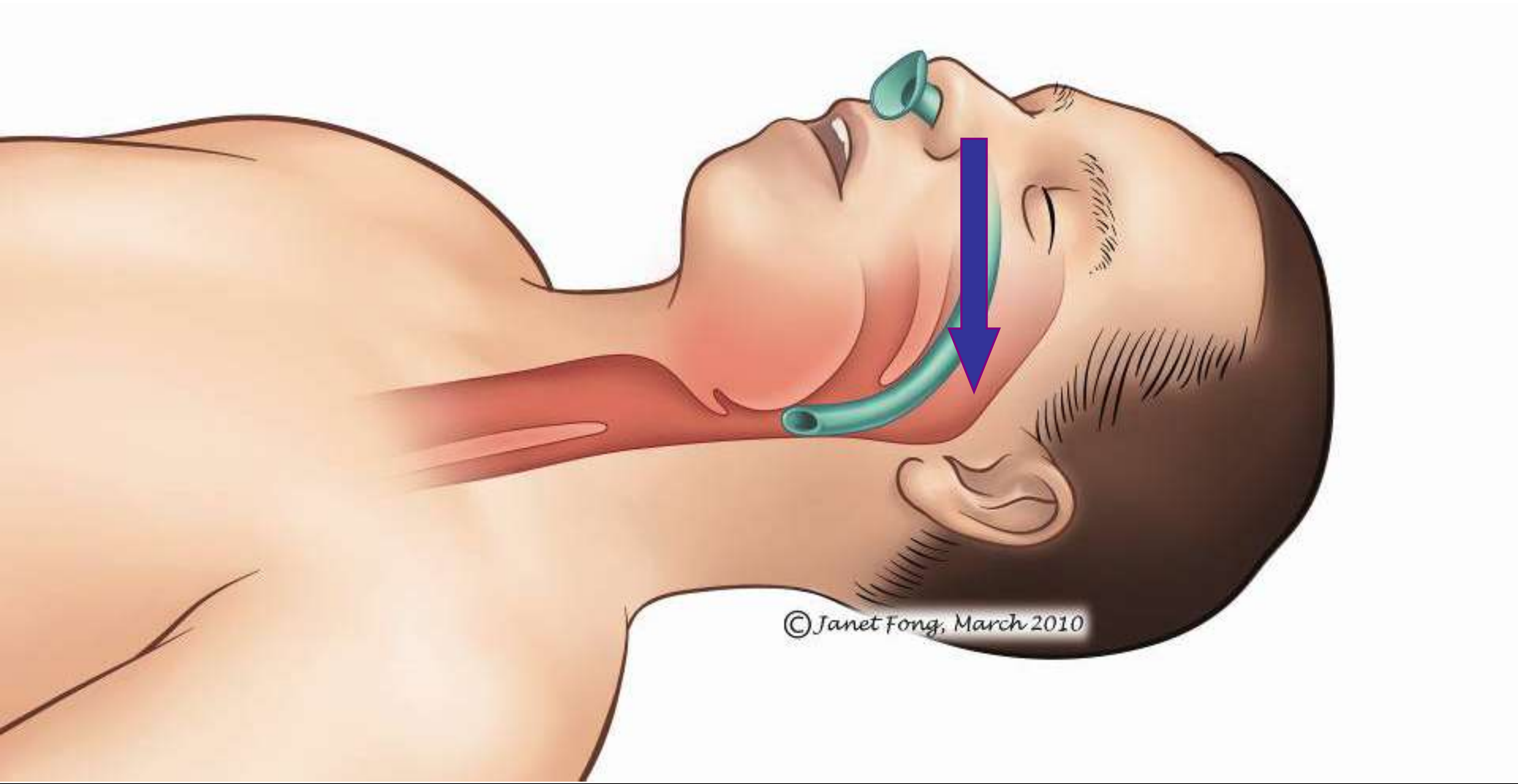
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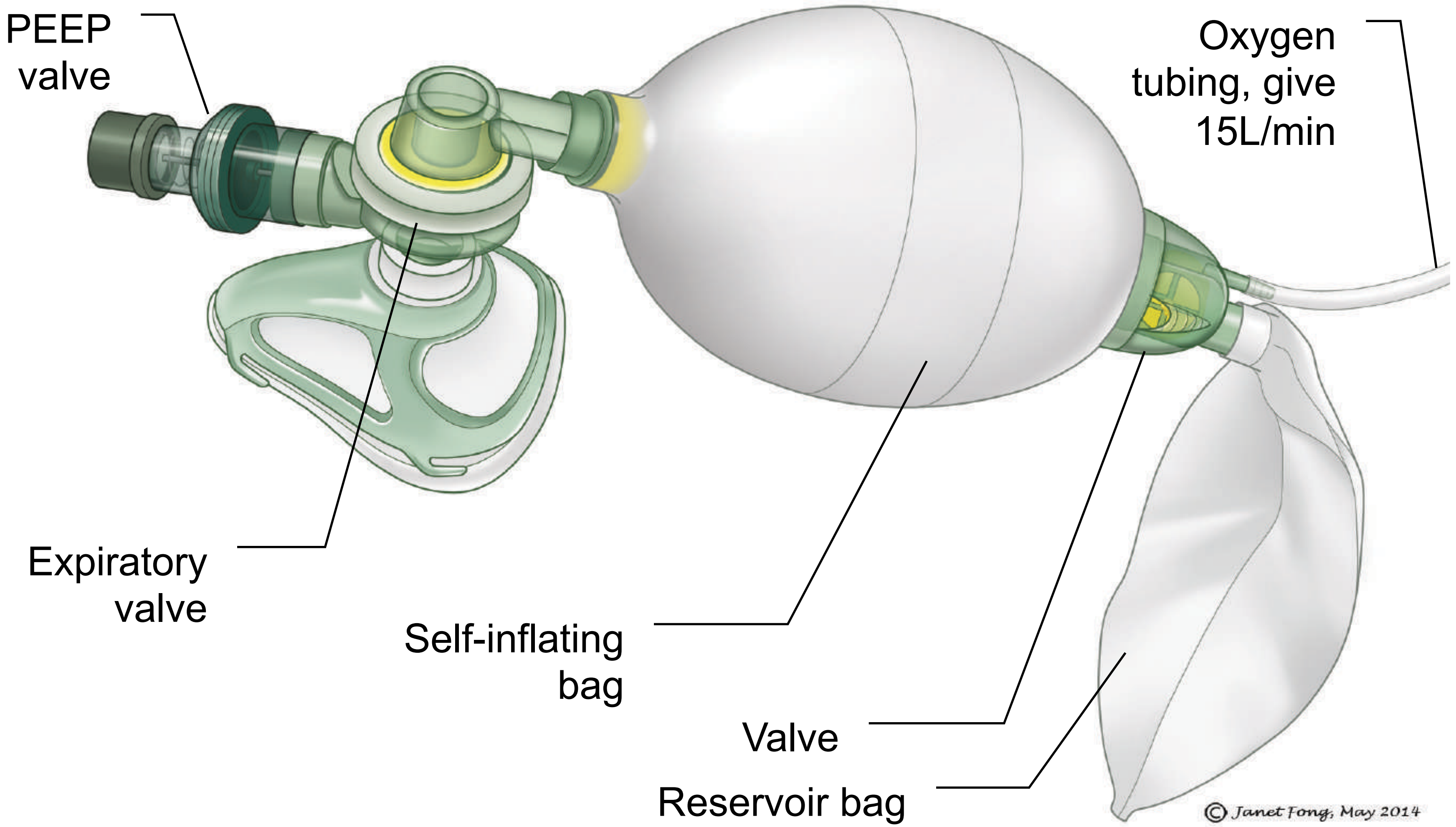
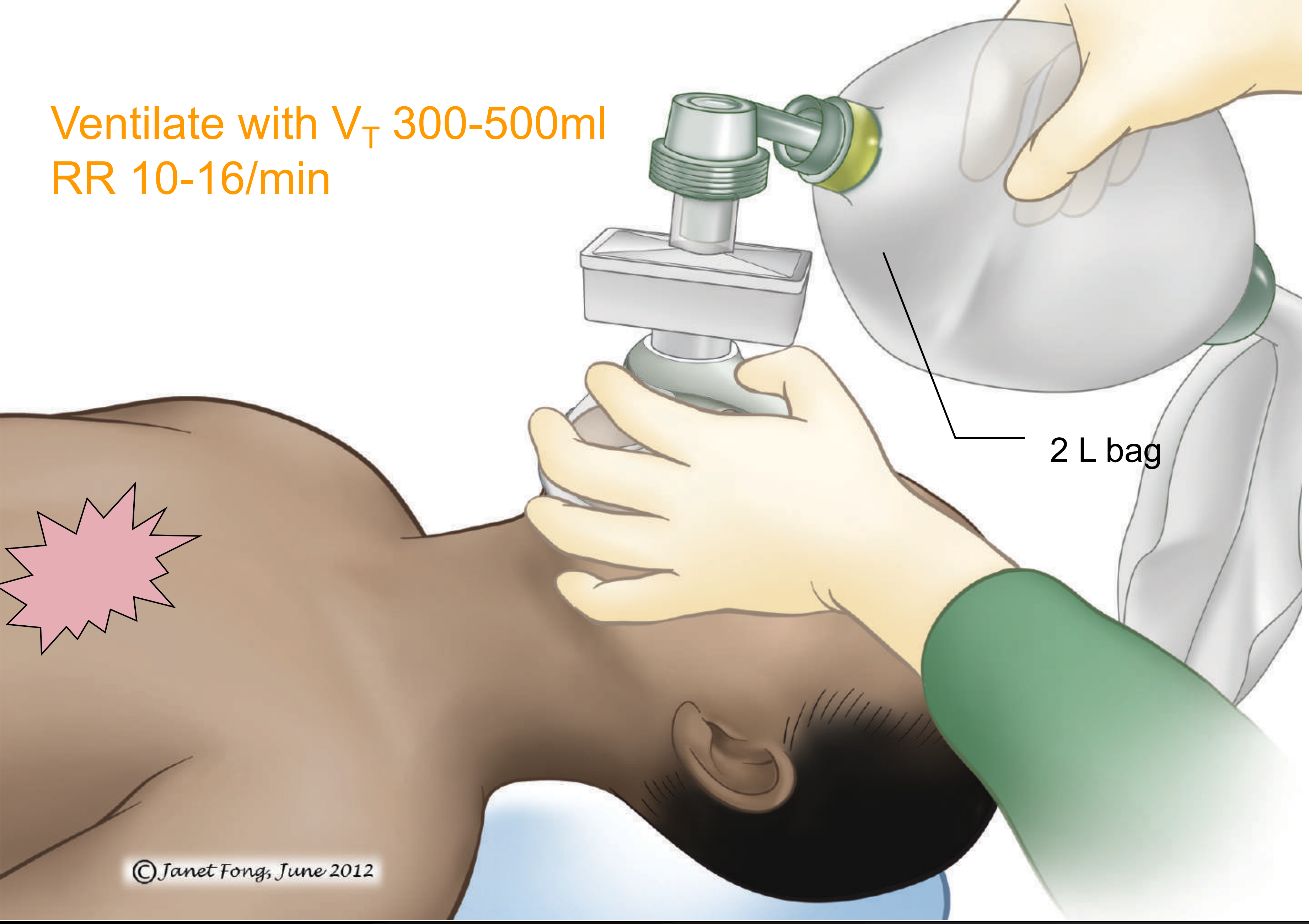
Case

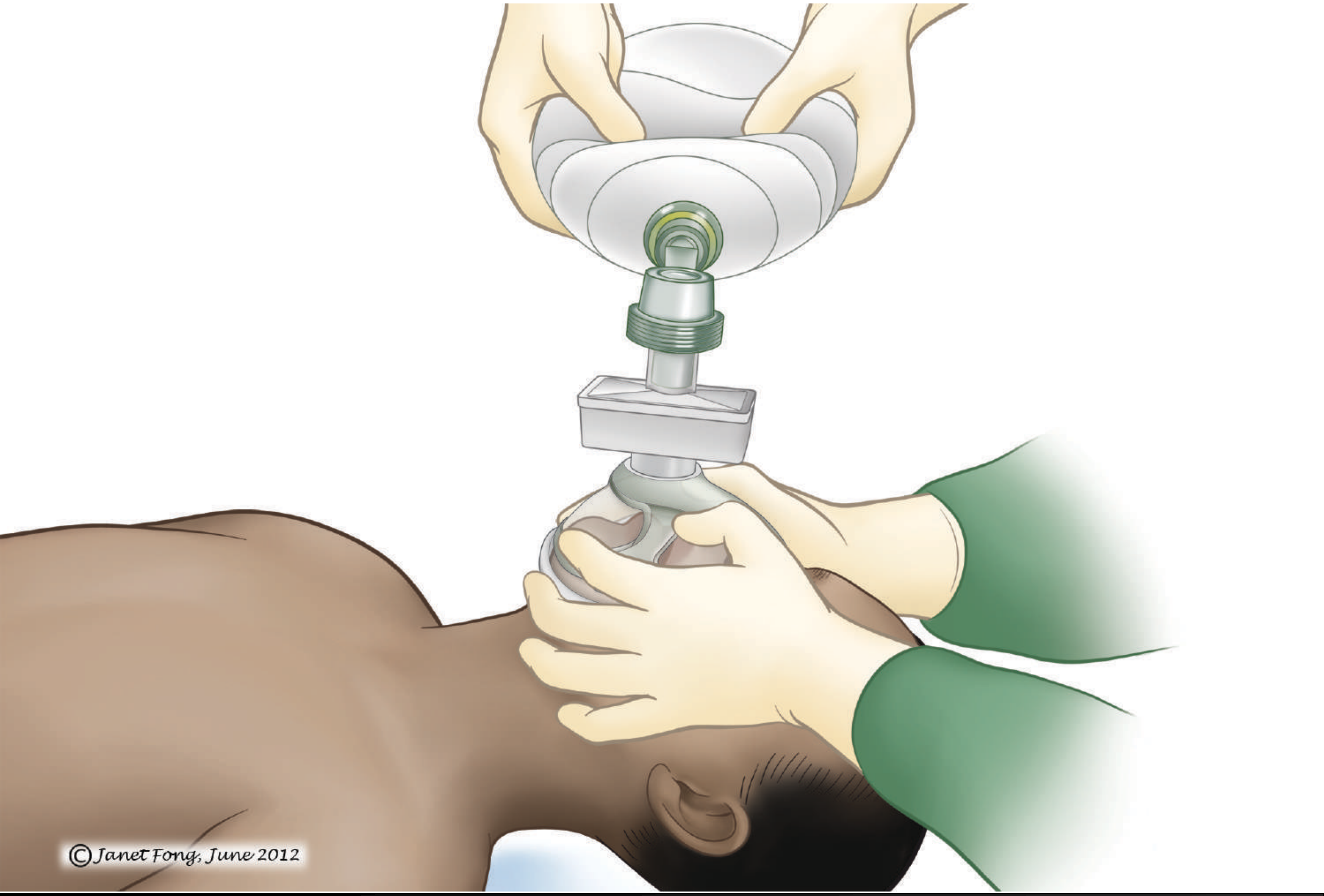
- Airway patency restored
  - Look, listen, feel
- Respiratory rate low

Breathing, ventilation



Ventilate with V_T 300-500ml
RR 10-16/min

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Case

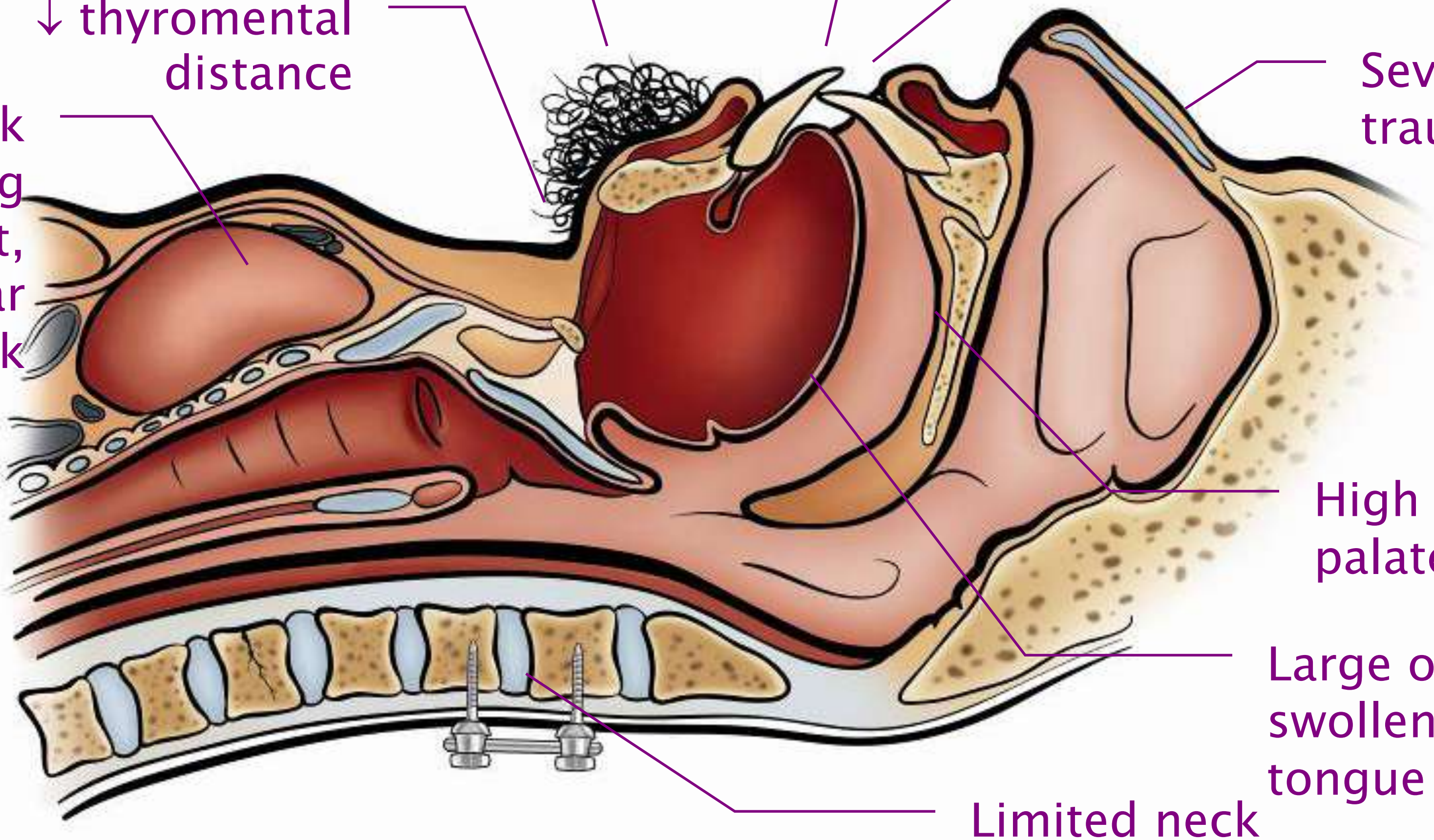
- What next?
- Intubate?

Indications for intubation

- Hypoxemic respiratory failure
- Hypercapnic respiratory failure
- Patient unlikely to be able to maintain an unobstructed airway
- Airway protection
- Airway suction

Timing

- Indication
  - Severe hypoxia?
  - Airway protection?
- Difficulty
- Your skill


Beard may hide receding chin, makes bag-mask ventilation difficult
↓ thyromental distance
Neck swelling or short, muscular neck
Anterior bite, protruding teeth
Small mouth
Severe facial trauma
High curved palate
Large or swollen tongue
Limited neck movement

Obstructing lesion in oropharynx or larynx

- High risk of failure to intubate, failure to ventilate
- Do NOT attempt rapid sequence induction

Anticipated difficult airway

- Call for help immediately while applying basic airway techniques
- High flow oxygen via bag-valve-mask resuscitator
- Make preparations for advanced airway techniques
- Wait for help
- If patient develops complete airway obstruction or cardiorespiratory arrest is imminent, attempt an advanced airway technique
  - Choice of technique is dependent on the situation and your skill in performing the technique

Case

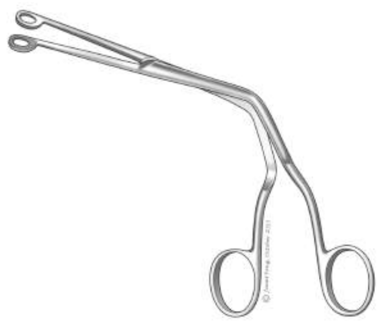
- Decide to go ahead with intubation
- (Inform patient)

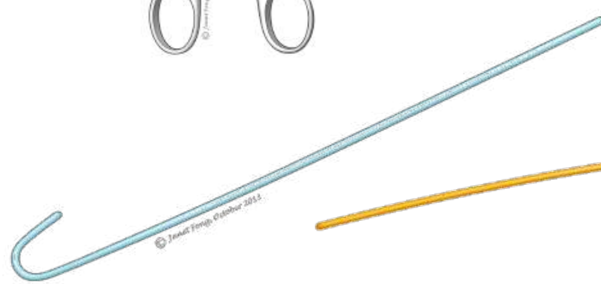
Prepare

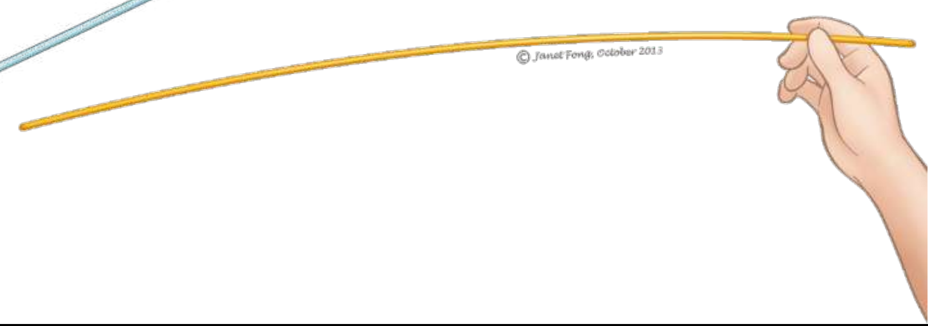
- Equipment
  - Intubation equipment
  - Monitoring
  - Suction
  - Bag-mask resuscitator
  - Artificial airway
- Does the equipment work?

Intubation equipment

- Laryngoscopes
  - Blades
- ETTs
- Forceps
- Stylet
- Syringe



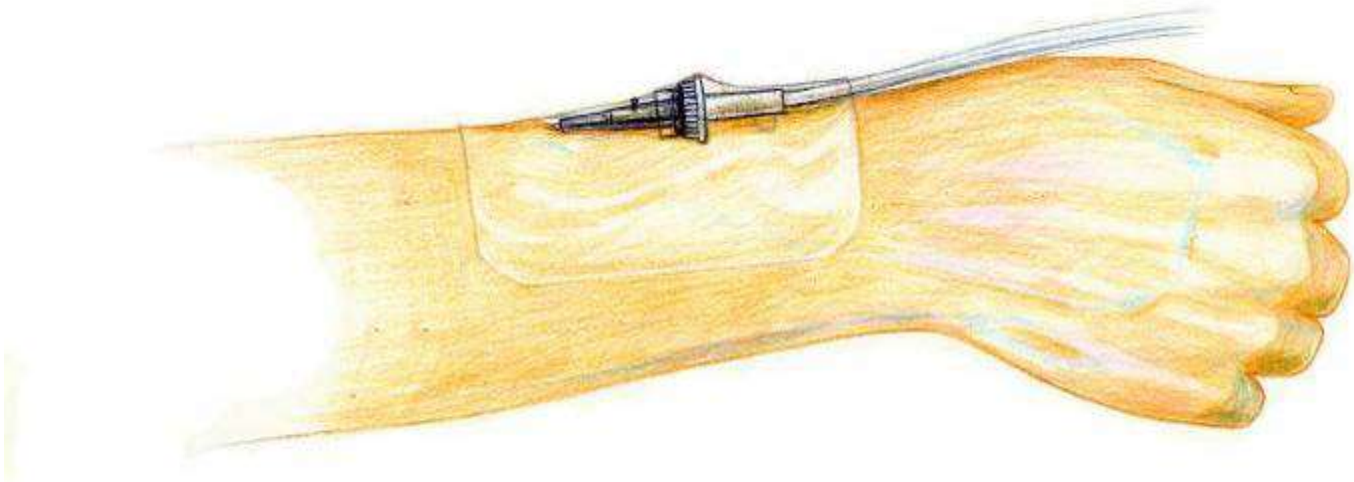




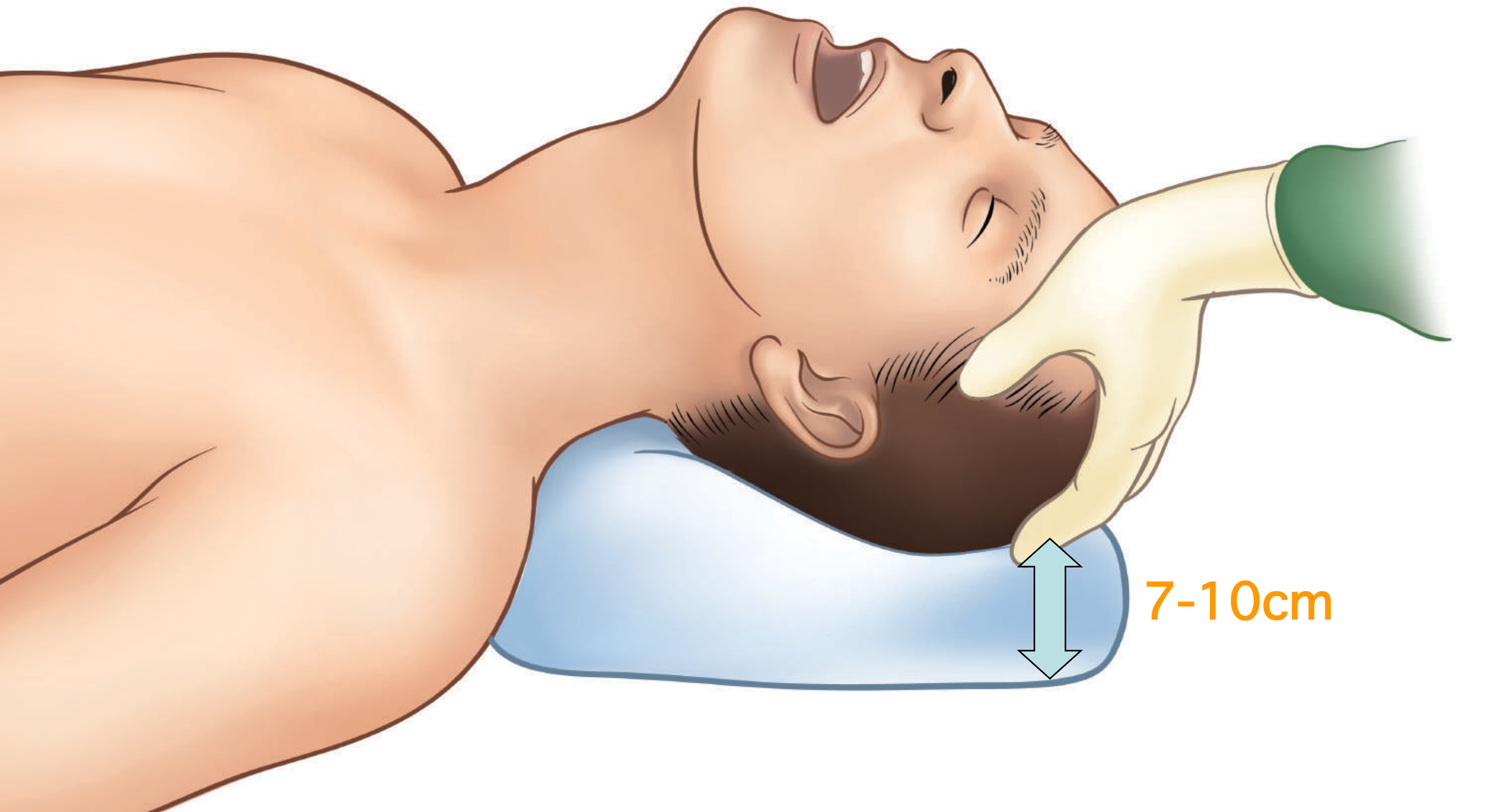
Laryngoscope

Drugs

- Induction agent
- Muscle relaxant
- Oxygen
- Sedative
- Resuscitation drugs
- Fluids



Preparation

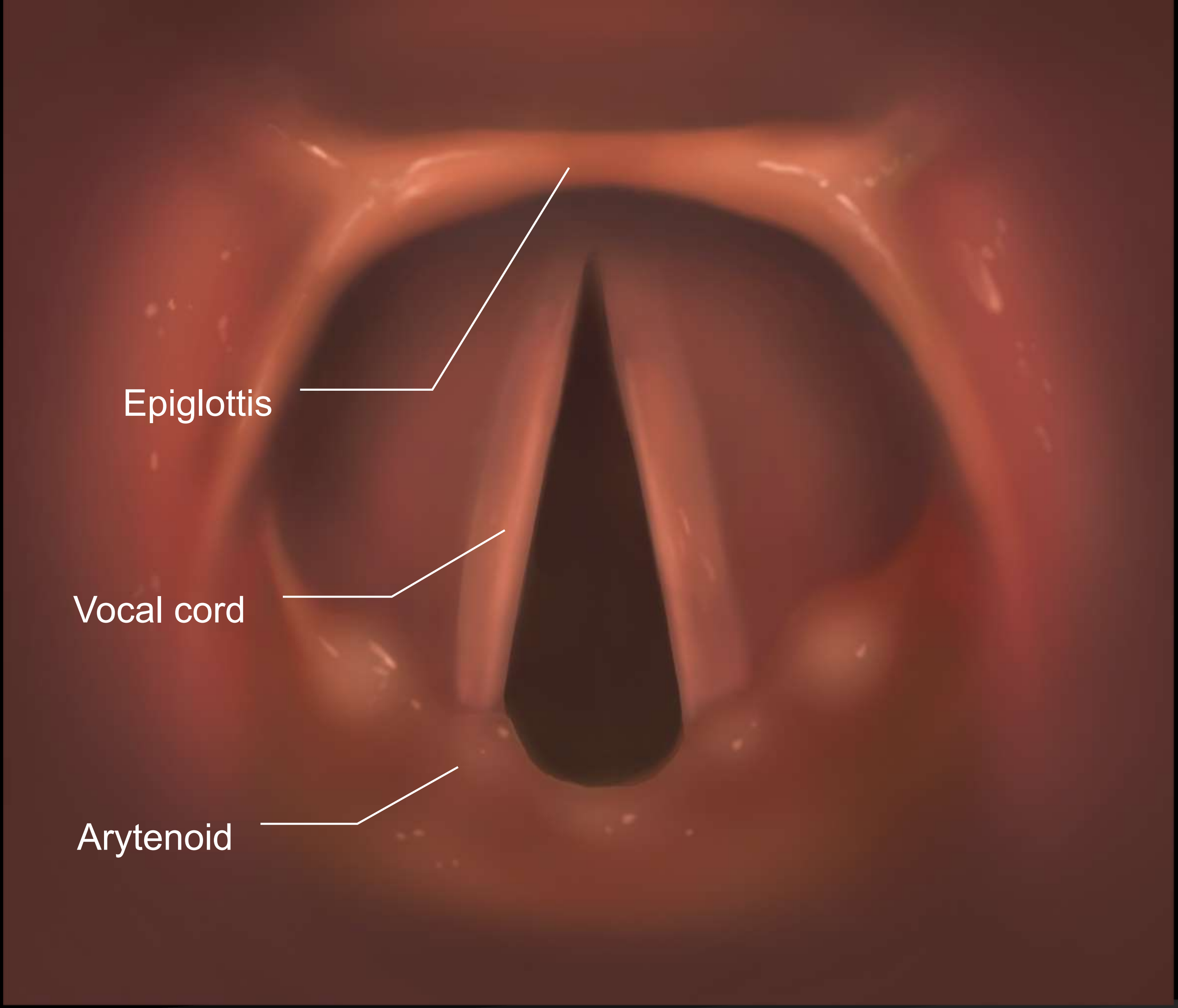


People

- Clear roles
  - Drug administration
    - What
    - How much
    - When
  - Equipment
  - Cricoid pressure
    - No other role

Hypnosis and Muscle Relaxants

- Only if required
- Must be confident of being able to maintain and control the airway
- Be careful of suxamethonium side-effects, Rocuronium is an alternative, but is long acting

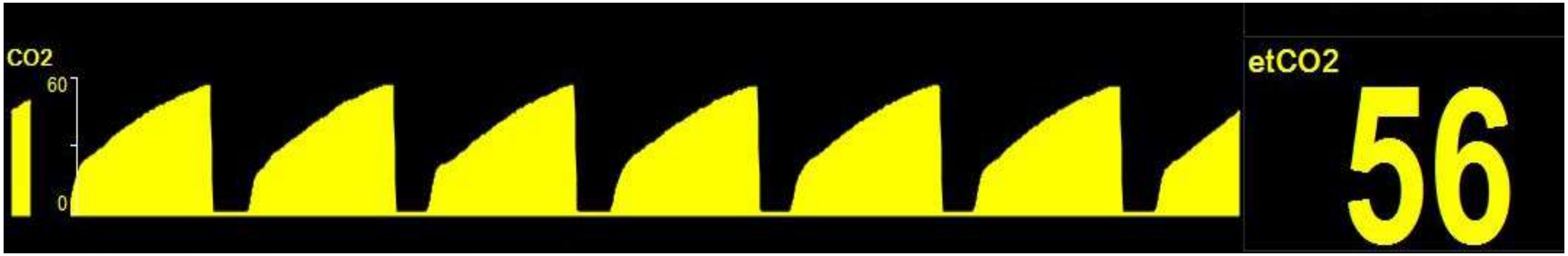

Epiglottis
Vocal cord
Arytenoid

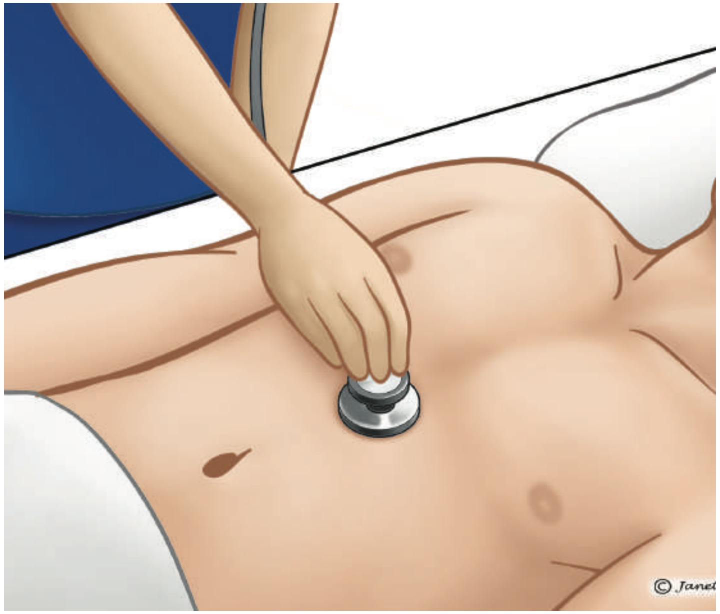
Rapid Sequence Intubation

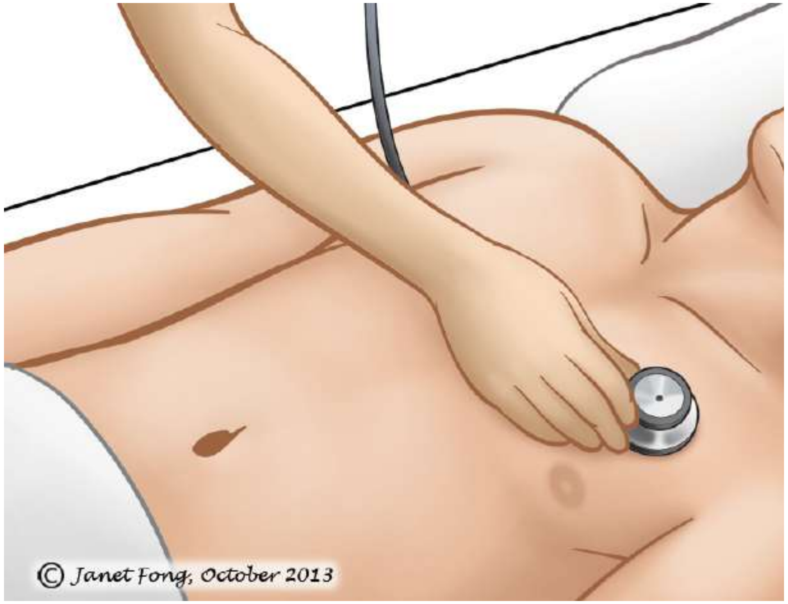
- Pre-oxygenate
- Cricoid pressure
- Pre-determined boluses of drugs
- Avoid ventilation if possible*
- Intubate immediately
- Release cricoid pressure when tube position confirmed

Cricoid pressure

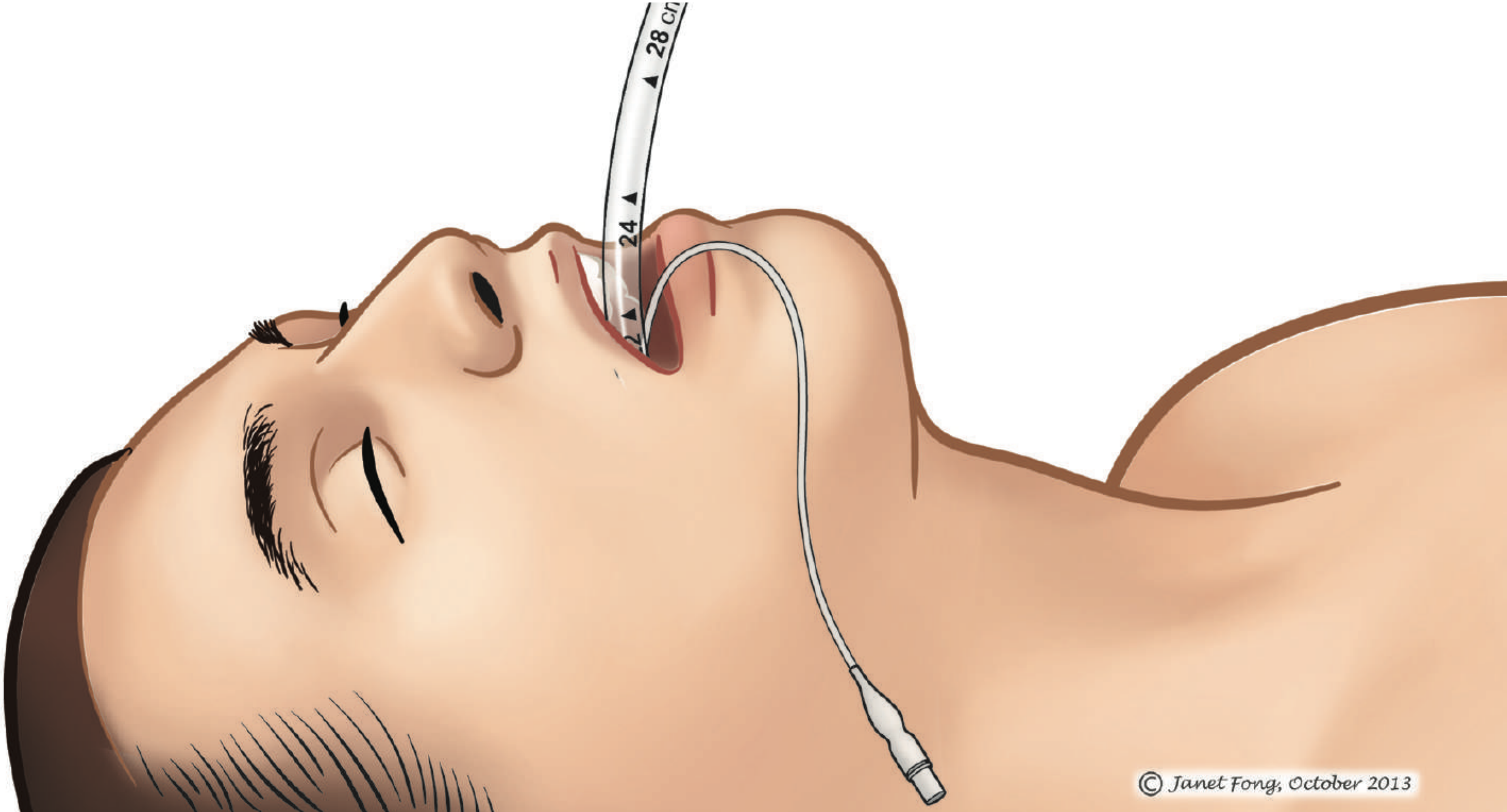
Confirm tube position



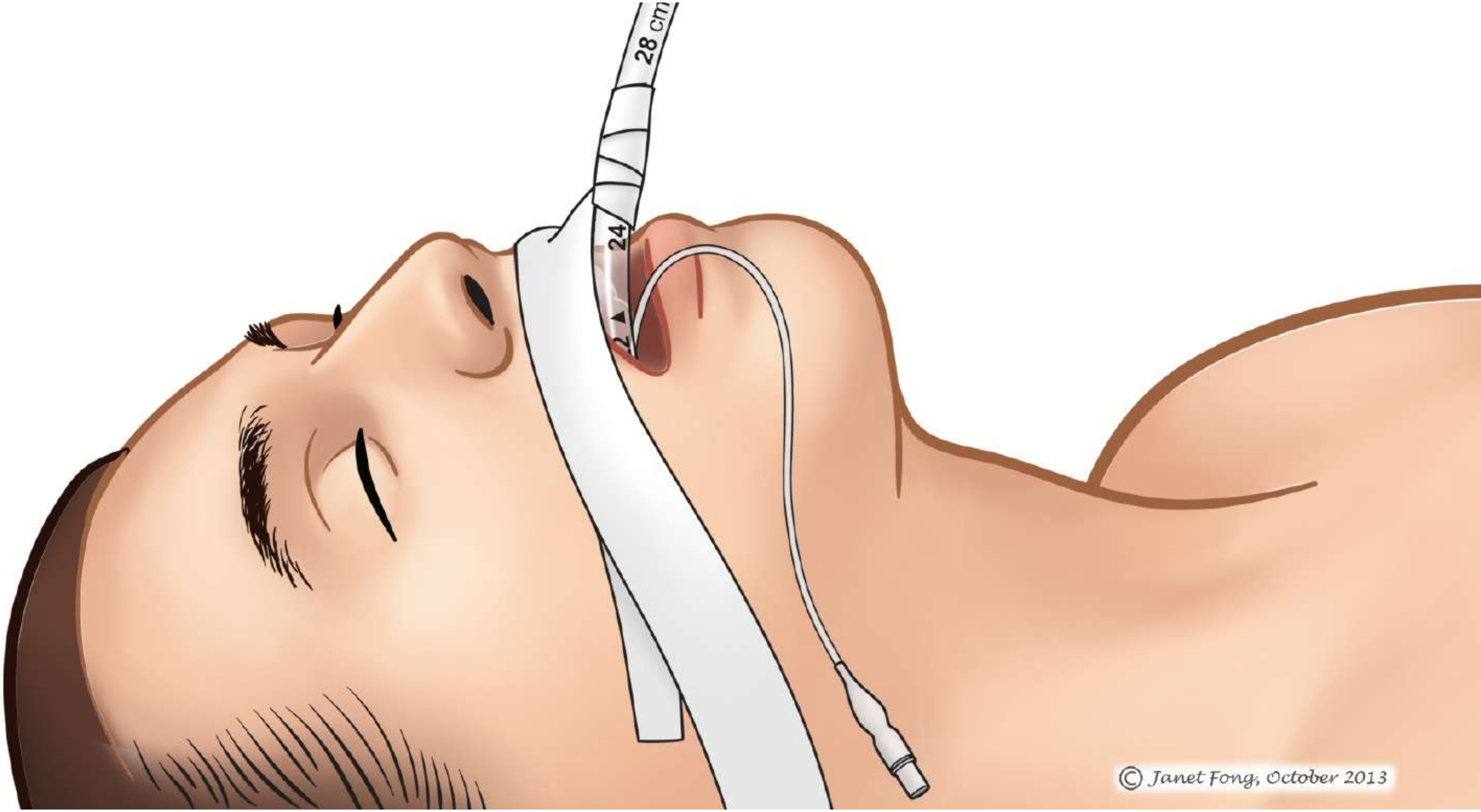




Correct depth

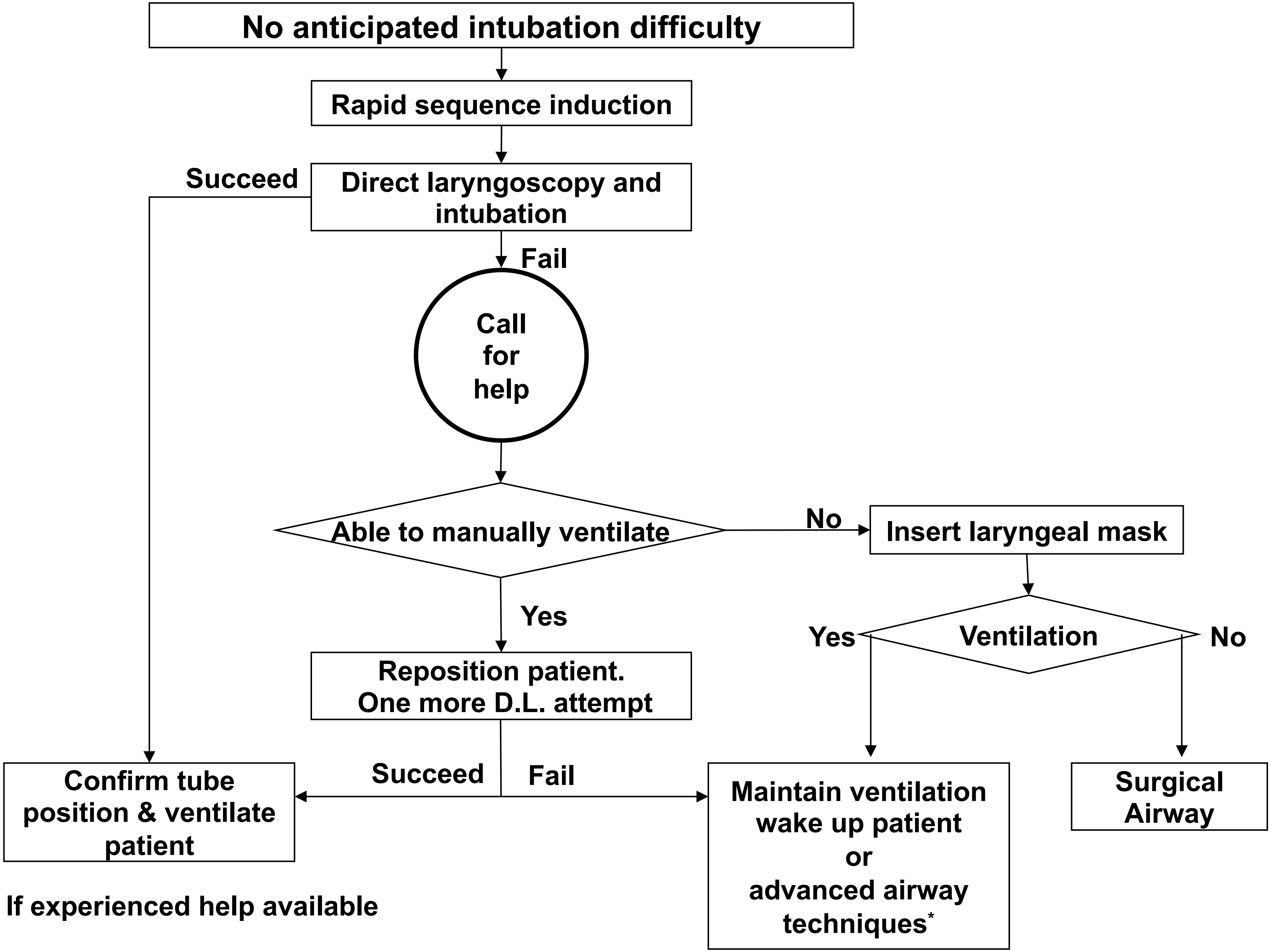


Secure the tube

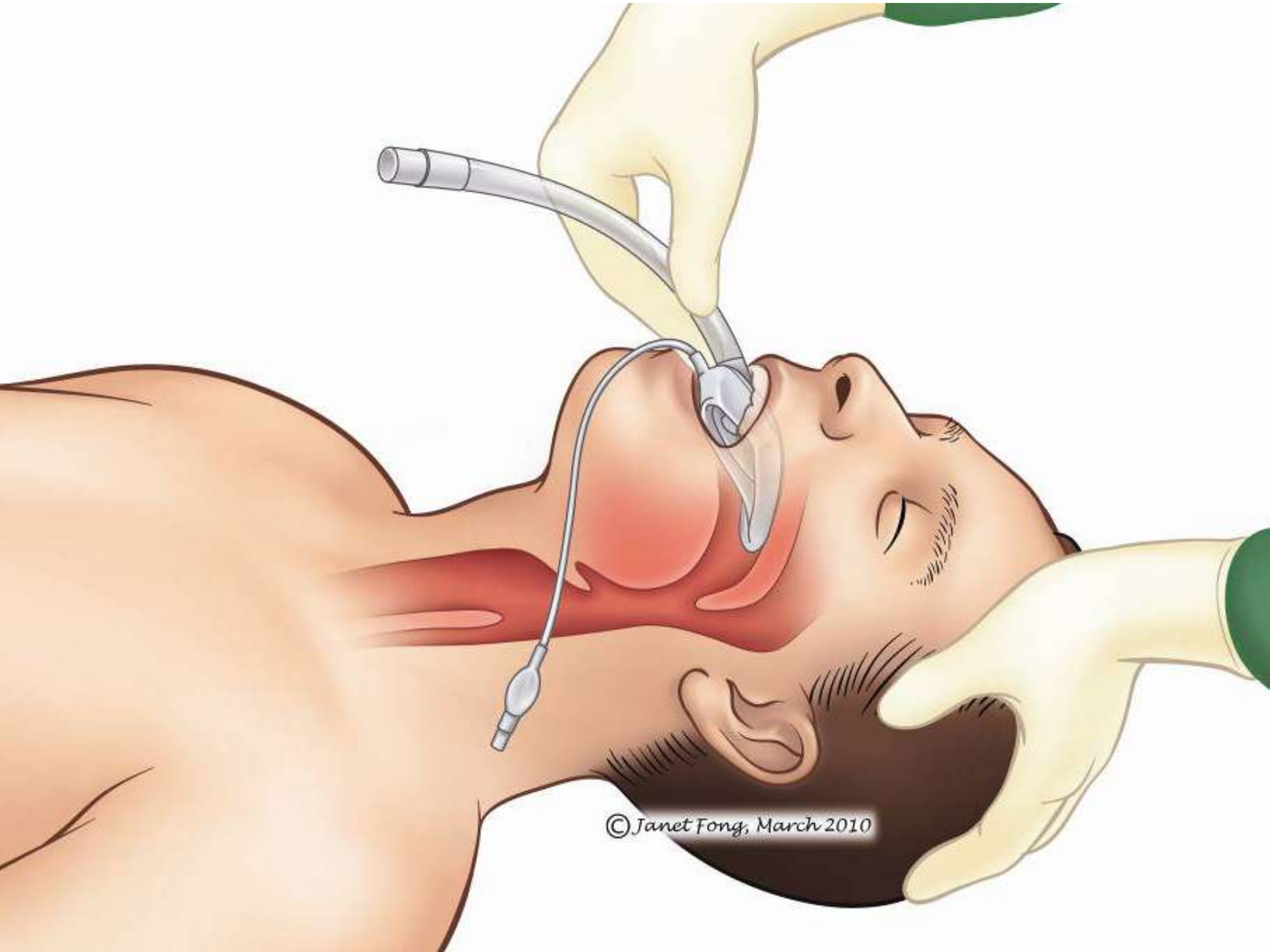


Failed Intubation

- Risk of failure about 1-5%
- Consequences of hypoxia potentially catastrophic
- Have a plan



* If experienced help available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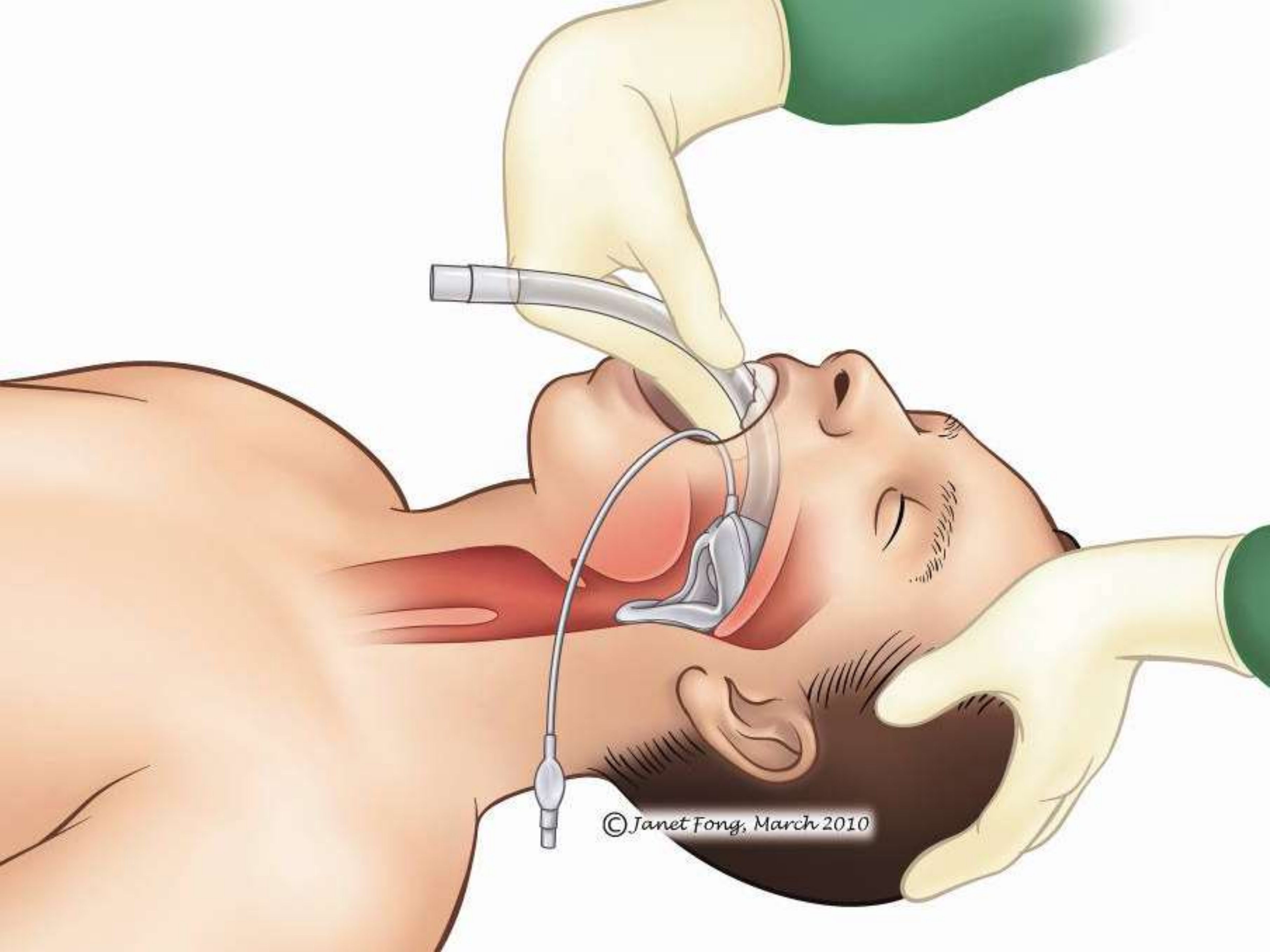
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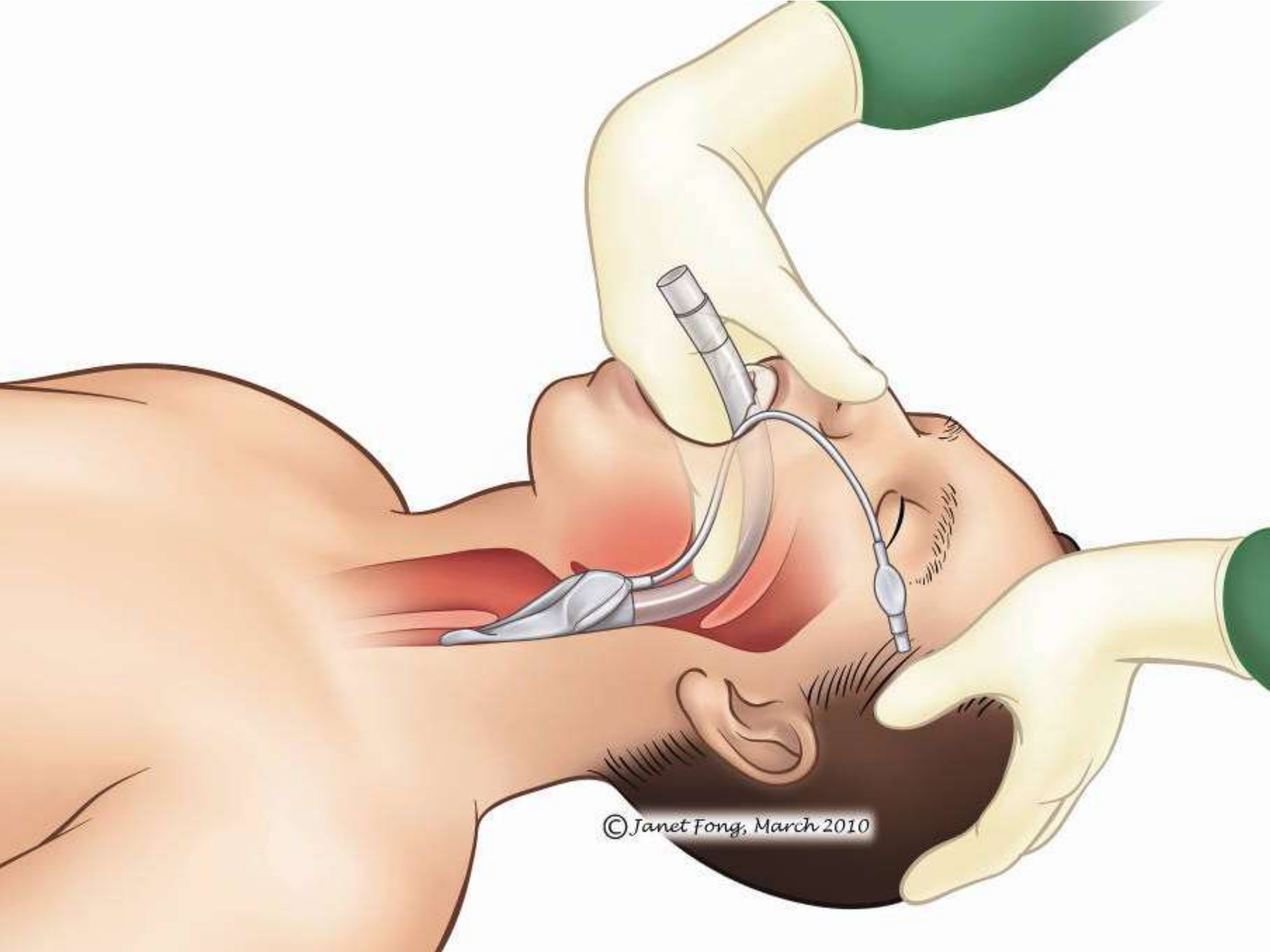
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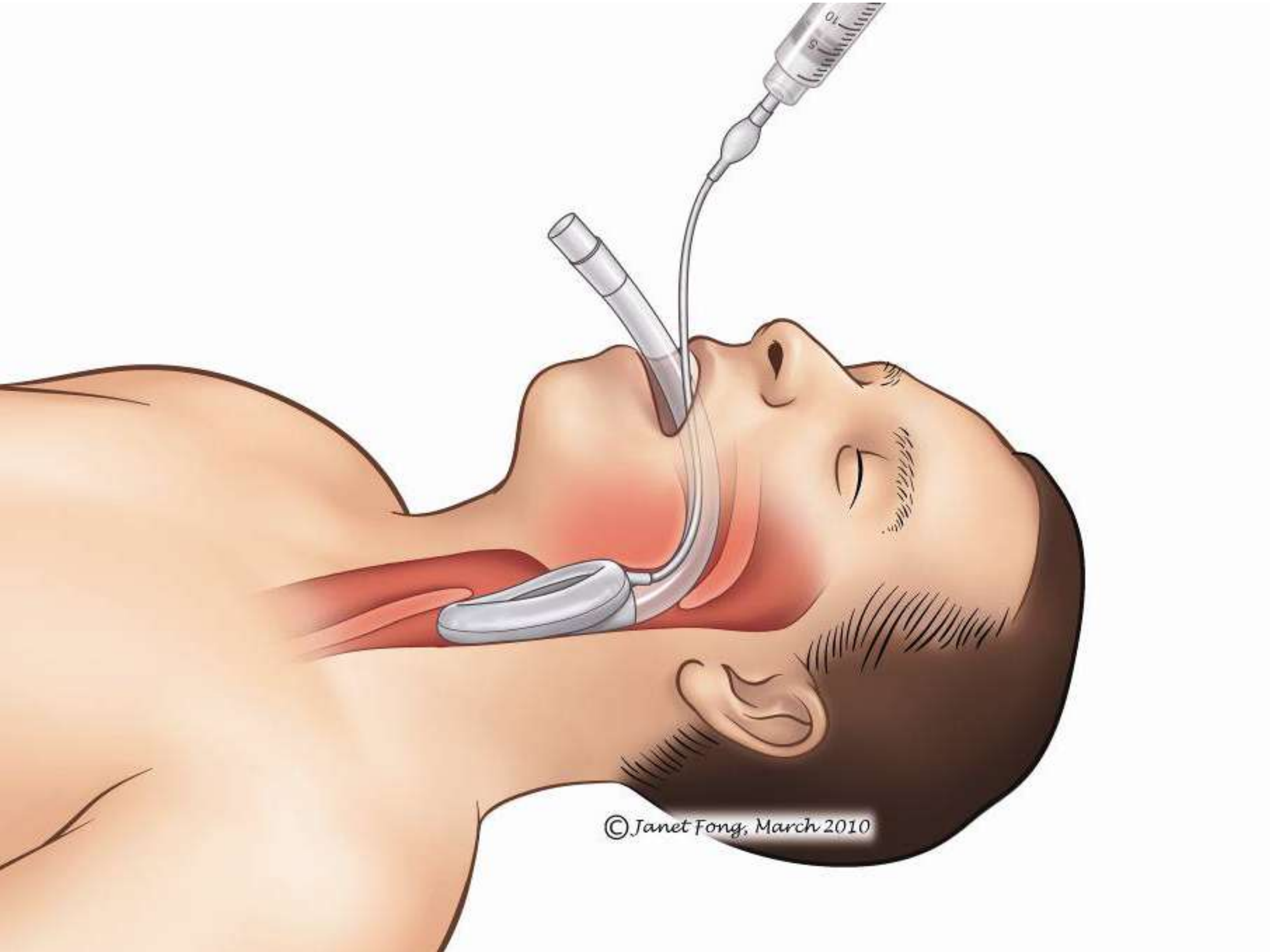
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Summary

- Maintaining airway is a life saving skill
- Timing of intubation depends on:
  - Indication
  - Difficulty
  - YOUR skill
- Appropriate preparation of equipment, drugs, personnel, patient

Any questions?

Arterial blood gases

Continuing development of BASIC is supported by an unrestricted educational grant from

MAQUET Dräger medical
A Dräger and Siemens Company

Fall 1

64-jährige Patientin mit Abgeschlagenheit/Müdigkeit seit mehreren Wochen.

pH		7.31
pCO_2	kPa	2.5
	mmHg	18.7
pO_2	kPa	12.0
	mmHg	90
HCO_3^-	mmol/L	7.8
Lactat	mmol/L	13.3
Natrium	mmol/L	138
Kalium	mmol/L	3.8
Chlorid	mmol/L	112
Albumin	g/L	21

1. pH ? 7.31 HCO_3^- ? 7.8mmol/L

→ Metabolische Azidose

2. Kompensation ?

↓ pCO_2 0.16kPa pro ↓ mmol/L HCO_3^-

16.2 x 0.16kPa = 2.5kPa ← 24 - 7.8 = 16.2mmol/L

Erwartetes pCO_2:

5.2kPa - 2.5kPa = 2.7kPa 2.5kPa

→ adäquate Kompensation

→ keine zusätzliche Störung

Fall 1

64-jährige Patientin mit Abgeschlagenheit/Müdigkeit seit mehreren Wochen.

pH		7.31
pCO_2	kPa	2.5
	mmHg	18.7
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HCO_3^-	mmol/L	7.8
Lactat	mmol/L	13.3
Natrium	mmol/L	138
Kalium	mmol/L	3.8
Chlorid	mmol/L	112
Albumin	g/L	21

1. Metabolische Azidose ?

→ Berechnung der Anionenlücke

$Na^+ - (Cl^- + HCO_3^-)$

138 - (112 + 7.8) = 18.2mmol/L

Normale Anionenlücke (8-16mmol/L) ?

Korrektur: 2.5mmol/L pro 10G/L Albumin

Norm Anionenlücke$_{\text{Alb korr}}$ = 3-11mmol/L

→ Metabolische Azidose mit erhöhter Anionenlücke

Fall 1

64-jährige Patientin mit Abgeschlagenheit/Müdigkeit seit mehreren Wochen.

pH		7.31
pCO_2	kPa	2.5
	mmHg	18.7
pO_2	kPa	12.0
	mmHg	90
HCO_3^-	mmol/L	7.8
Lactat	mmol/L	13.3
Natrium	mmol/L	138
Kalium	mmol/L	3.8
Chlorid	mmol/L	112
Albumin	g/L	21

Metabolische Azidose mit positiver Anionenlücke: Differentialdiagnose ?

Methanol-Ethylenglykol

Urämie

Diabetische oder alkoholische Ketoazidose

Paraldehyd

Isoniazid

Lactatazidose

Salizylat

Fall 1

Laktatazidose: Differentialdiagnose ?

Typ A Laktatazidose Gewebshypoperfusion Supply Demand Mismatch	**Typ B Laktatazidose** Keine Gewebshypoperfusion Impaired tissue oxygen utilization	**D Laktatazidose**
▪ Vermindertes O_2 - Angebot Schock CI > 3, ScvO2 76% Hypoxämie NB, paO2 > 10kPa Schwere Anämie Hb +/- 7 CO Vergiftung nein ▪ Erhöhter O_2 - Bedarf Grand Mal Epilepsie nein Extreme Exercise nein	▪ Sepsis CRP 91, PCT 0.8, Lc 3.8 ▪ Diabetes mellitus nein ▪ Malignom ?! ▪ Thiamin Mangel ? ▪ Inborn errors of Metabolism nein ▪ HIV Infektion nein ▪ Medikamente/Toxine nein ▪ Leberversagen LDH 3828, GOT 170, GPT 34, Bili 38, INR 1.3, Q 63, FV 39	▪ Short bowel syndrome nein ▪ GI Malabsorption nein Diffus intravaskuläres B-Zell-Lymphom

Fall 2

18-jähriger Patient, bekannte CF, gelistet für LTPL, aktuell Infektexazerbation

pH		7.27
pCO_2	kPa	19
	mmHg	142
pO_2	kPa	11.6
	mmHg	87
HCO_3^-	mmol/L	66
Lactat	mmol/L	0.9
Chlorid	mmol/L	66

1. pH ? 7.27 HCO_3^- ? 66.3mmol/L

⟶ Respiratorische Azidose

2. Kompensation ?

↑ HCO_3^- 3mmol/L pro ↑ kPa pCO_2

14 x 3mmol/L = 42mmol/L ← 19kPa - 5kPa = 14kPa

Erwartetes HCO_3^-:

24mmol/L + 42mmol/L = 66mmol/L 66mmol/L

⟶ adäquate Kompensation

⟶ keine zusätzliche Störung

Fall 2

Intubation ja/nein?

pH		7.269
pCO_2	kPa	19
	mmHg	142
pO_2	kPa	11.6
	mmHg	87
HCO_3^-	mmol/L	66
Lactat	mmol/L	0.9
Chlorid	mmol/L	66

pH		7.135
pCO_2	kPa	28
	mmHg	210
pO_2	kPa	17
	mmHg	127
HCO_3^-	mmol/L	68
Lactat	mmol/L	0.8
Chlorid	mmol/L	65

Fall 2

Geschwindigkeit der CO_2- Senkung?

pH		7.135
pCO_2	kPa	28
	mmHg	210
pO_2	kPa	17
	mmHg	127
HCO_3^-	mmol/L	68
Lactat	mmol/L	0.8
Chlorid	mmol/L	65

pH		7.61
pCO_2	kPa	7.2
	mmHg	54
pO_2	kPa	7.2
	mmHg	54
HCO_3^-	mmol/L	56
Lactat	mmol/L	2.0
Chlorid	mmol/L	74

+ 2 Tage

Posthyperkapnische Alkalose

Fall 3

51-jähriger Patient mit respiratorischer Insuffizienz. Weitere Anamnese unklar.

pH		7.32
pCO_2	kPa	9.3
	mmHg	70
pO_2	kPa	10
	mmHg	75
HCO_3^-	mmol/L	35

1. pH ? 7.32 HCO_3^- ? 35mmol/L

→ Respiratorische Azidose → Akut ? → Chronisch ?

2. Akute Kompensation ?

↑ HCO_3^- 0.8mmol/L pro ↑ kPa $paCO_2$

4.3 x 0.8mmol/L = 3.4mmol/L

9.3kPa - 5kPa = 4.3kPa

Erwartetes HCO_3^-:

24mmol/L + 3.4mmol/L = 27.4mmol/L 35mmol/L

→ inadäquate Kompensation

→ zusätzliche Störung: metabolische Alkalose

Fall 3

51-jähriger Patient mit respiratorischer Insuffizienz. Weitere Anamnese unklar.

pH		7.32
pCO_2	kPa	9.3
	mmHg	70
pO_2	kPa	10
	mmHg	75
HCO_3^-	mmol/L	35

1. pH ? 7.3 HCO_3^- ? 35mmol/L
2

→ Respiratorische Azidose → Akut ? → Chronisch ?

2. Chronische Kompensation ?

↑ HCO_3^- 3mmol/L pro ↑ kPa $paCO_2$

4.3 x 3mmol/L = 12.9mmol/L

9.3kPa - 5kPa = 4.3kPa

Erwartetes HCO_3^-:

24mmol/L + 12.9mmol/L = 36.9mmol/L 35mmol/L

→ adäquate Kompensation

→ keine zusätzliche Störung

Fall 3

51-jähriger Patient mit respiratorischer Insuffizienz. Weitere Anamnese unklar.

pH		7.32
pCO_2	kPa	9.3
	mmHg	70
pO_2	kPa	10
	mmHg	75
HCO_3^-	mmol/L	35

Respiratorische Azidose

Akute Störung → Akute respiratorische Azidose + metabolische Alkalose (bspweise Erbrechen)

Chronische Störung → Chronisch respiratorische Azidose (bspweise COPD)

Anamnese ergänzen!

Fall 4

18-jährige Patientin, sucht wegen Schwäche die Notfallstation auf.

pH		7.51
pCO_2	kPa	8.9
	mmHg	66.7
pO_2	kPa	7.2
	mmHg	54
HCO_3^-	mmol/L	38
Na	mmol/L	147
K	mmol/L	2.1
Cl	mmol/L	83
Albumin	g/L	34

1. pH ? 7.51 HCO_3^- ? 38mmol/L

→ Metabolische Alkalose

2. Kompensation ?

↑ pCO_2 0.08kPa pro ↑ mmol/L HCO_3^-

14 x 0.08kPa = 1.12kPa ← 38mmol/L - 24mmol/L = 14mmol/L

Erwartetes pCO_2:

5kPa + 1.12kPa = 6.12kPa 8.9

→ inadäquate Kompensation

→ zusätzliche Störung: respiratorische Azidose

BASIC

Ursache der metabolischen Alkalose - respiratorischen Azidose?

Hypochloräme Alkalose

↓

Harn Chlorid

< 20mmol/L	> 20mmol/L
↓	↓
Chlorid sensitive Alkalose	Chlorid insensitive Alkalose

Chlorid sensitive Alkalose:

- Gastrointestinaler Verlust Erbrechen, Verlust via MS
- Diuretika-Gabe
- Erhohlung nach Hyperkapnie
- Chloridverlust durch Diarrhoe
- CF (Schweissverlust)

Chlorid insensitive Alkalose:

- Diuretika
- Hoher Blutdruck
  Prim. Hyperaldosteronismus, Cushing, Ectop ACTH Prod, Mineralocoricoid-ähnlich Substanzen, Liddle Syndrom
- Tiefer Blutdruck
  Bartter Syndrom, Gitelman Syndrom
- Schwere Kaliumdepletion

Fall 4

18-jährige Patientin, sucht wegen Schwäche die Notfallstation auf.

pH		7.51
pCO_2	kPa	8.9
	mmHg	66.7
pO_2	kPa	7.2
	mmHg	54
HCO_3^-	mmol/L	38
Na	mmol/L	147
K	mmol/L	2.1
Cl	mmol/L	83
Albumin	g/L	34
U_{Cl}	mmol/L	8
U_{Na}	mmol/L	14

1. Ursache der metabolischen Alkalose ?

Induziertes Erbrechen bei Bulimie

- Metabolische Alkalose mit tiefem U_{Cl}
- Zeichen der Volumendepletion
  - Hypernatriämie, tiefes U_{Na}
- Zeichen der Mangelernährung
  - Hypoalbuminämie, schwere Hypokaliämie

2. Ursache der respiratorischen Azidose ?

- Alveoläre Hypoventilation bei muskulärer Schwäche bei schwerer Hypokaliämie

Fall 5

33-jährige Patientin,
bekanntes Sjögren-Syndr,
Muskelschwäche

pH		7.15
pCO_2	kPa	4.5
	mmHg	34
HCO_3^-	mmol/L	12
Na	mmol/L	134
K	mmol/L	1.5
Cl	mmol/L	112
pH_{Urin}		6.5
U_{Na}	mmol/L	44
U_K	mmol/L	10
U_{Cl}	mmol/L	35

1. pH ? 7.15 HCO_3^- ? 12mmol/L

→ Metabolische Azidose

2. Kompensation ?

↓ $paCO_2$ 0.16kPa pro ↓ mmol/L HCO_3^-

12 x 0.16kPa = 1.92kPa ← 24mmol/L - 12mmol/L = 12mmol/L

Erwartetes $paCO_2$:

5kPa - 1.92kPa = 3.08kPa 4.5

→ inadäquate Kompensation

→ zusätzlich Störung: respiratorische Azidose

BASIC

Ursache der metabolische Azidose - respiratorischen Azidose?

Fall 5

33-jährige Patientin,
bekanntes Sjögren-Syndr,
Muskelschwäche

pH		7.15
pCO_2	kPa	4.5
	mmHg	34
HCO_3^-	mmol/L	12
Na	mmol/L	134
K	mmol/L	1.5
Cl	mmol/L	112
pH_{Urin}		6.5
U_{Na}	mmol/L	44
U_K	mmol/L	10
U_{Cl}	mmol/L	35

1. Ursache der metabolischen Azidose ?

⟶ Berechnung der Anionenlücke

$Na^+ - (Cl^- + HCO_3^-)$

134 - (112 + 12) = 10mmol/L

⟶ Metabolische Azidose mit normaler Anionenlücke

Urin-Anionenlücke (UAG)

$(U_{Na} + U_K) - U_{cl}$: Norm 40 ± 20mmol/L

↓ Extrarenale Ursache	↓ Renale Ursache
UAG wird negativ	UAG bleibt positiv

(44 + 10) - 35 = 19mmol/L

Fall 5

33-jährige Patientin, bekanntes Sjögren-Syndr, Muskelschwäche

pH		7.15
pCO_2	kPa	4.5
	mmHg	34
HCO_3^-	mmol/L	12
Na	mmol/L	134
K	mmol/L	1.5
Cl	mmol/L	112
pH_{Urin}		6.5
U_{Na}	mmol/L	44
U_K	mmol/L	10
U_{Cl}	mmol/L	35

1. Ursache der metabolischen Azidose mit normaler Anionenlücke und positiver Urin-Anionenlücke?

Metabolische Azidose - normale Anionenlücke	
Urin AG negativ	**Urin AG positiv**
Extrarenale Ursache	**Renale Ursache (RTA)**
• **GI** (Diarrhoe, externe Drainage von Pankreassaft, Galle)	**P_K erniedrigt/normal** •RTA Typ I (U-pH > 5.5) •RTA Typ II (U-pH < 5.5)
• **Urinary diversion**	**P_K erhöht** •RTA Typ IV
• **Hyperalimentation**	

→ RTA Typ I im Rahmen des Sjögren - Syndroms

Fall 5

33-jährige Patientin,
bekanntes Sjögren-Syndr,
Muskelschwäche

pH		7.15
pCO_2	kPa	4.5
	mmHg	34
HCO_3^-	mmol/L	12
Na	mmol/L	134
K	mmol/L	1.5
Cl	mmol/L	112
pH_{Urin}		6.5
U_{Na}	mmol/L	44
U_K	mmol/L	10
U_{Cl}	mmol/L	35

2. Ursache der respiratorischen Azidose ?

→ Atemmuskellähmung bei schwerer Hypokaliämie

Arrhythmias

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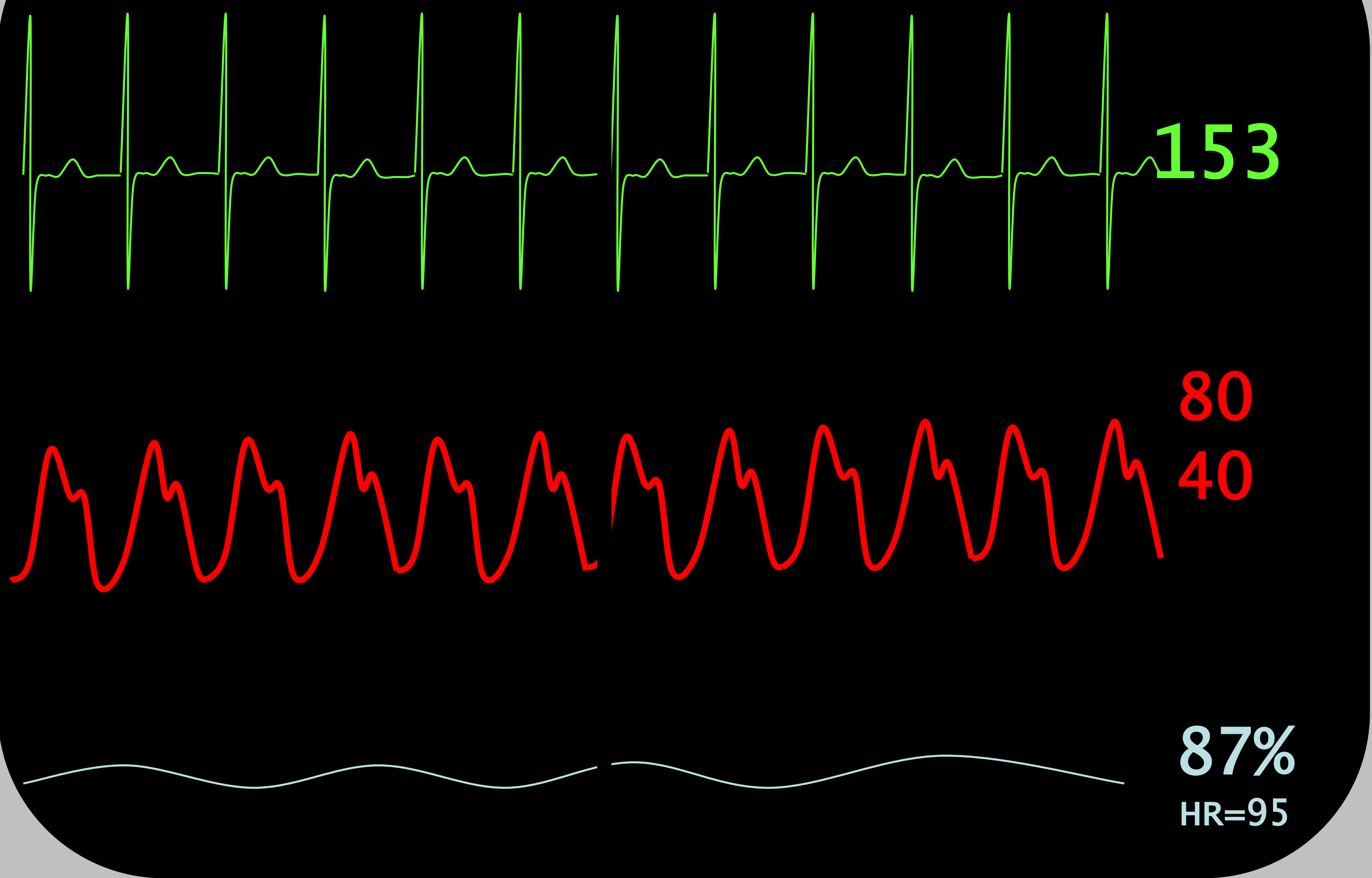
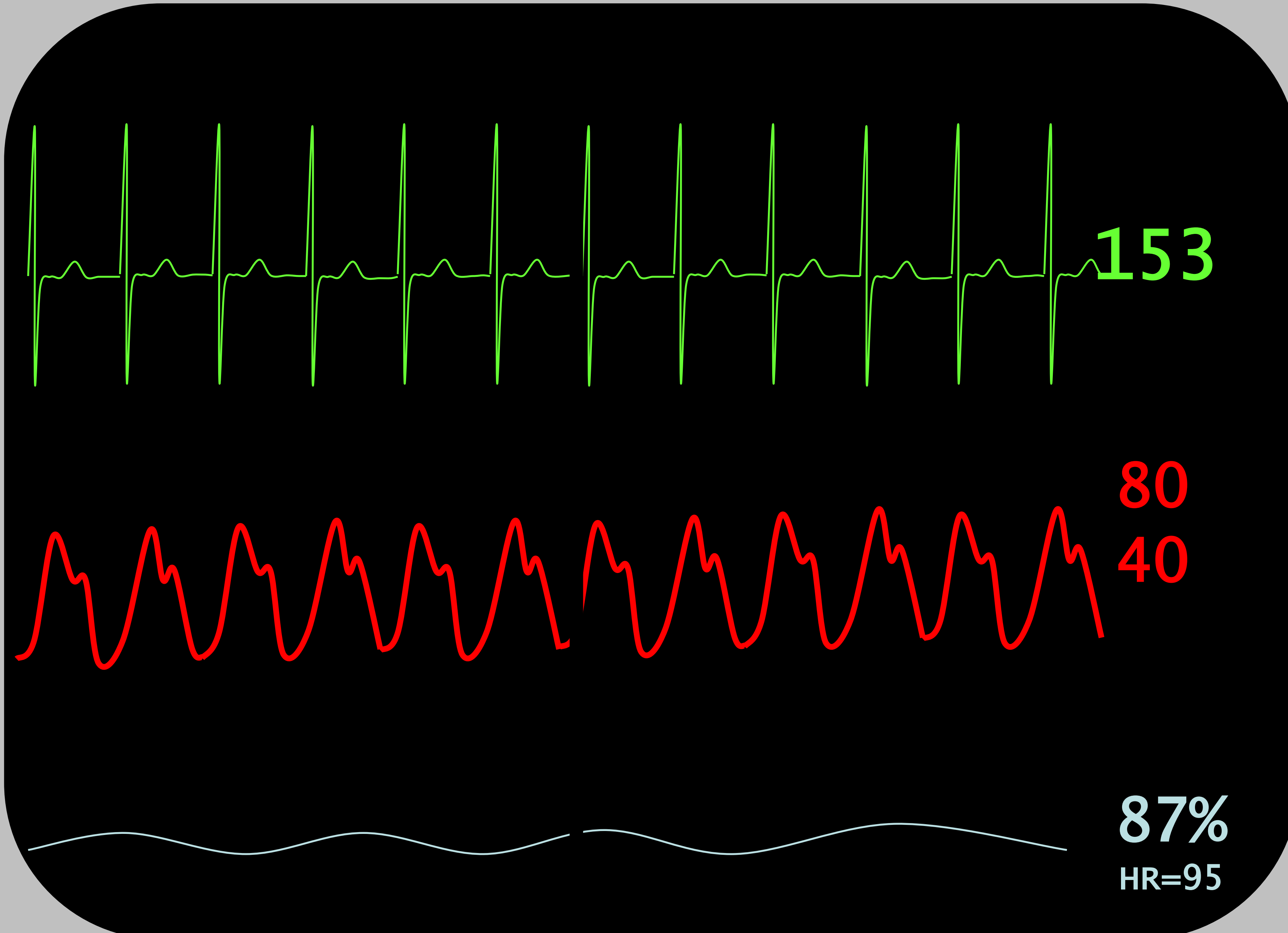
Case A

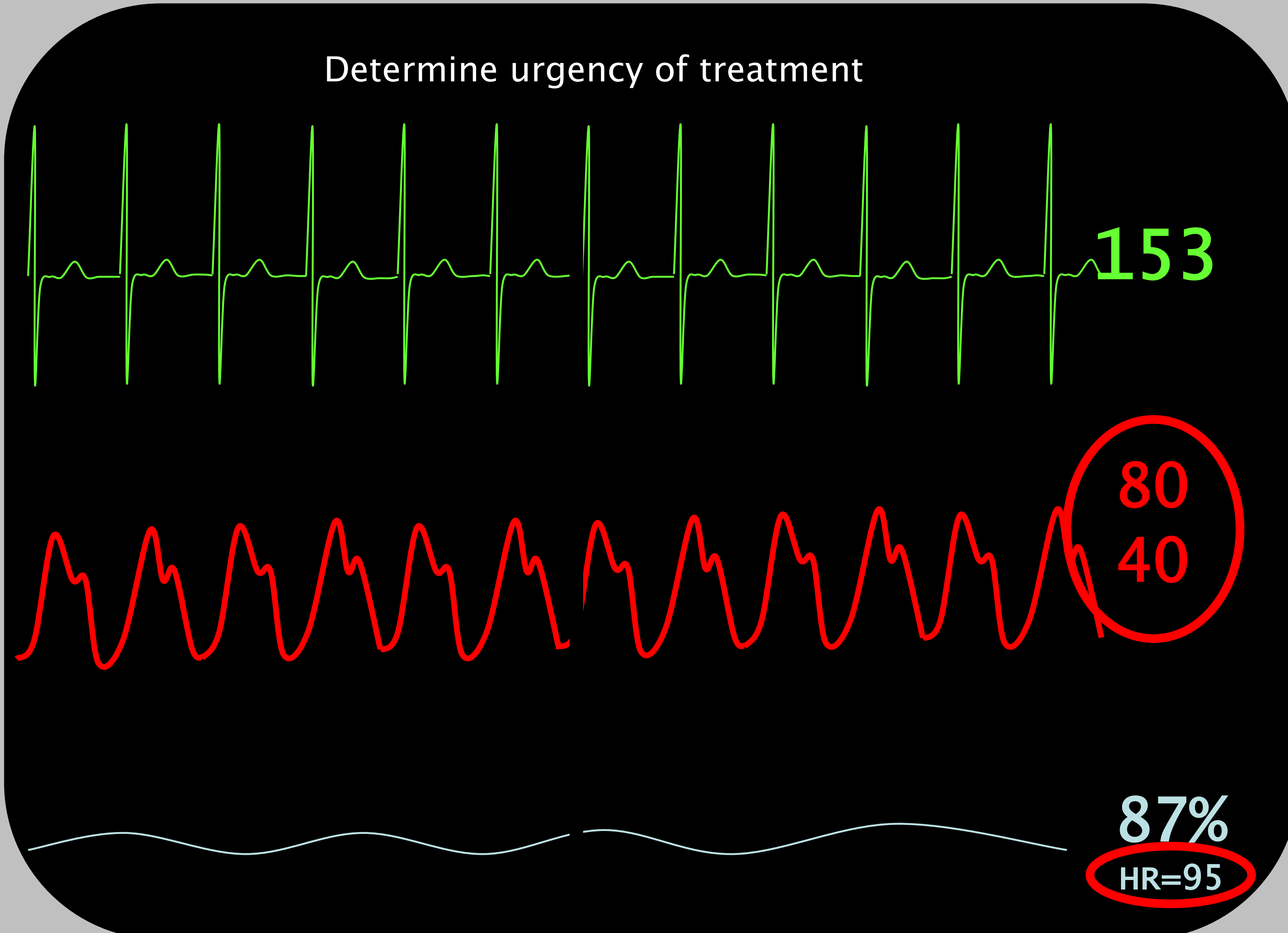
- 32 year old man
- Admitted with pneumonia
- Sudden onset of tachycardia
- Management?

Decide how much rhythm contributes to clinical deterioration




153
80
40
87%
HR=95


153
80
40
87%
HR=95

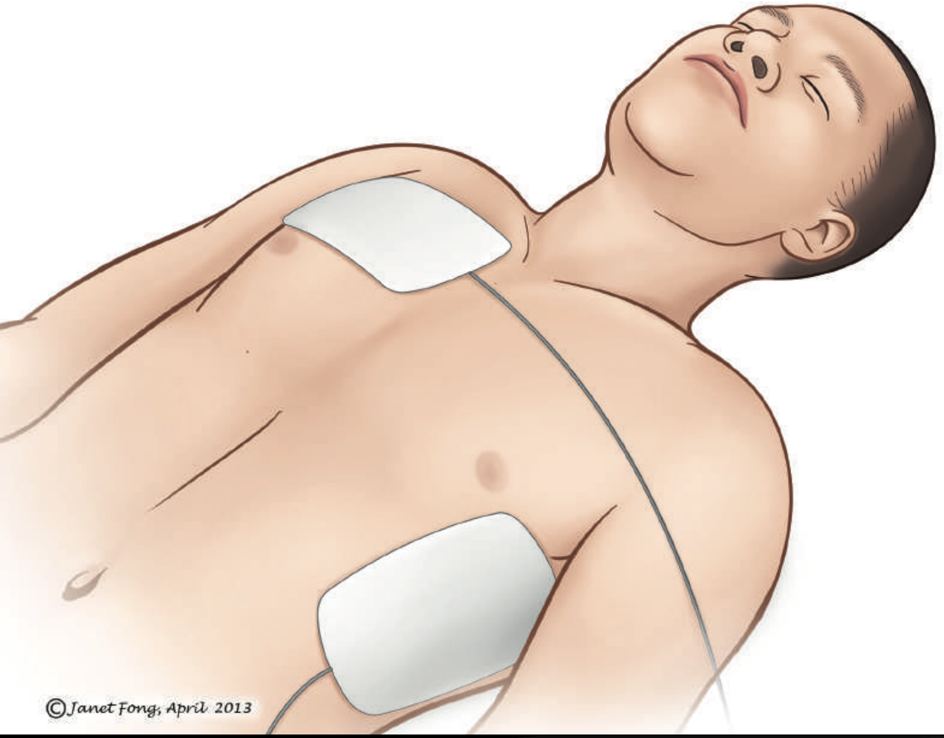

Determine urgency of treatment
153
80
40
87%
HR=95

DC cardioversion

- Narrow complex tachyarrhythmia
  - Start with:

PSVT and atrial flutter
- 50-100 J (biphasic)

AF
- 120-200J (biphasic)

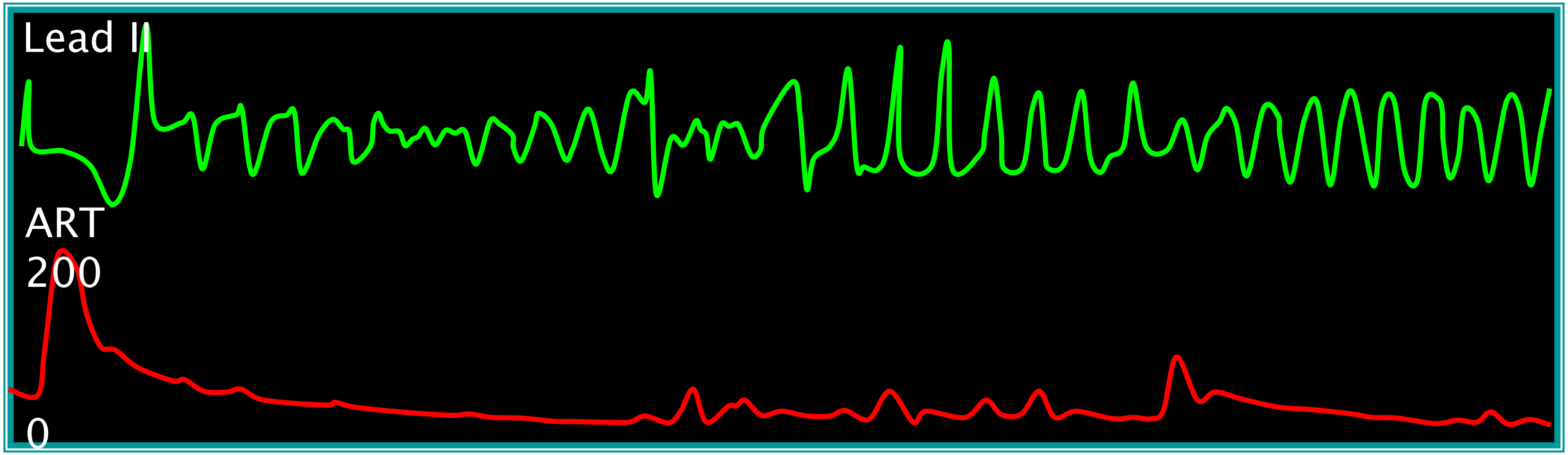


Treatment

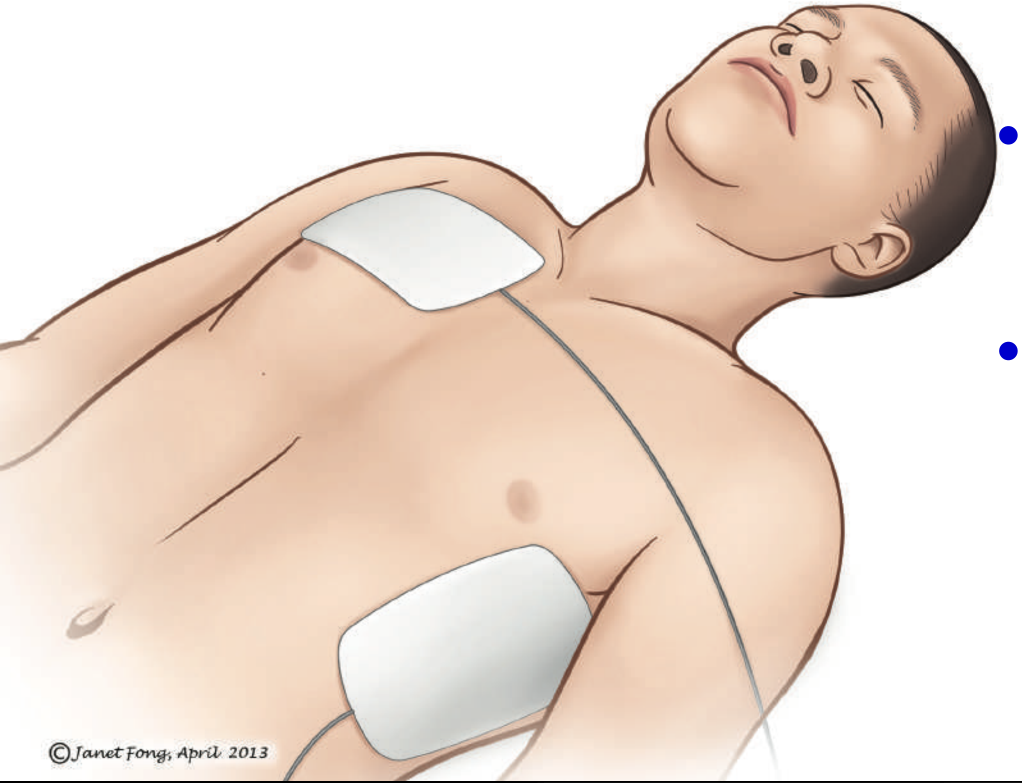
- Treat underlying cause
- Correct precipitating abnormalities eg hypokalaemia

Case B

- 45 yr old woman
- Sudden collapse at home, followed by convulsion
- GCS 7/15, intubated for airway protection
- Following CT develops this arrhythmia:



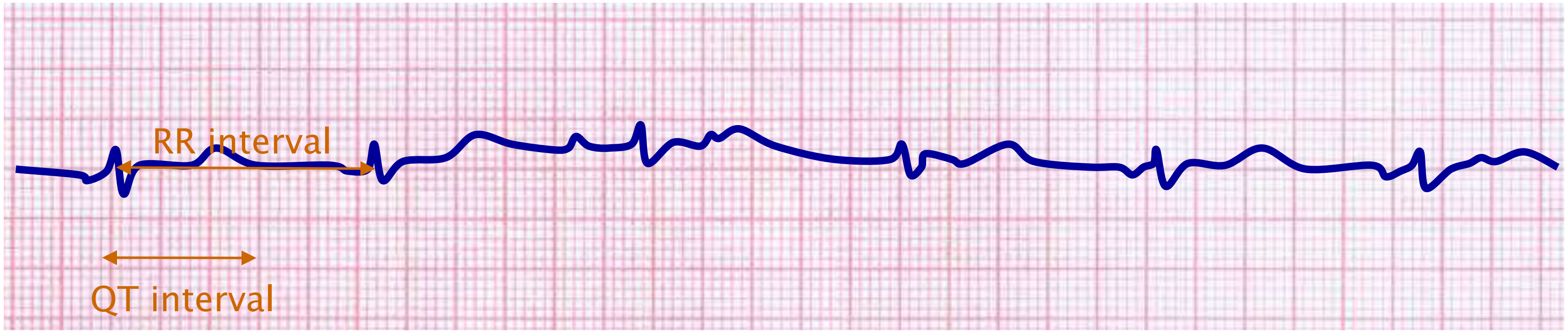
Treatment



- Defibrillation
  - 150-200J (biphasic)
- Treat as for VF
- NOT cardioversion

Management

- Check QTc
  - >460 msec: torsades de pointes
  - <460 msec: ischaemic in origin until proved otherwise



$$QT_c = \frac{\text{QT interval}}{\sqrt{\text{RR interval}}}$$

Causes of long QTc

- Congenital
- Acquired
  - Electrolyte abnormalities: ↓K,↓Mg
  - Hypothermia
  - Drugs
    - Class I and III anti-arrhythmics
    - Antimicrobials (erythromycin, ketoconazole)
    - Tricyclics
  - Intracranial bleeding

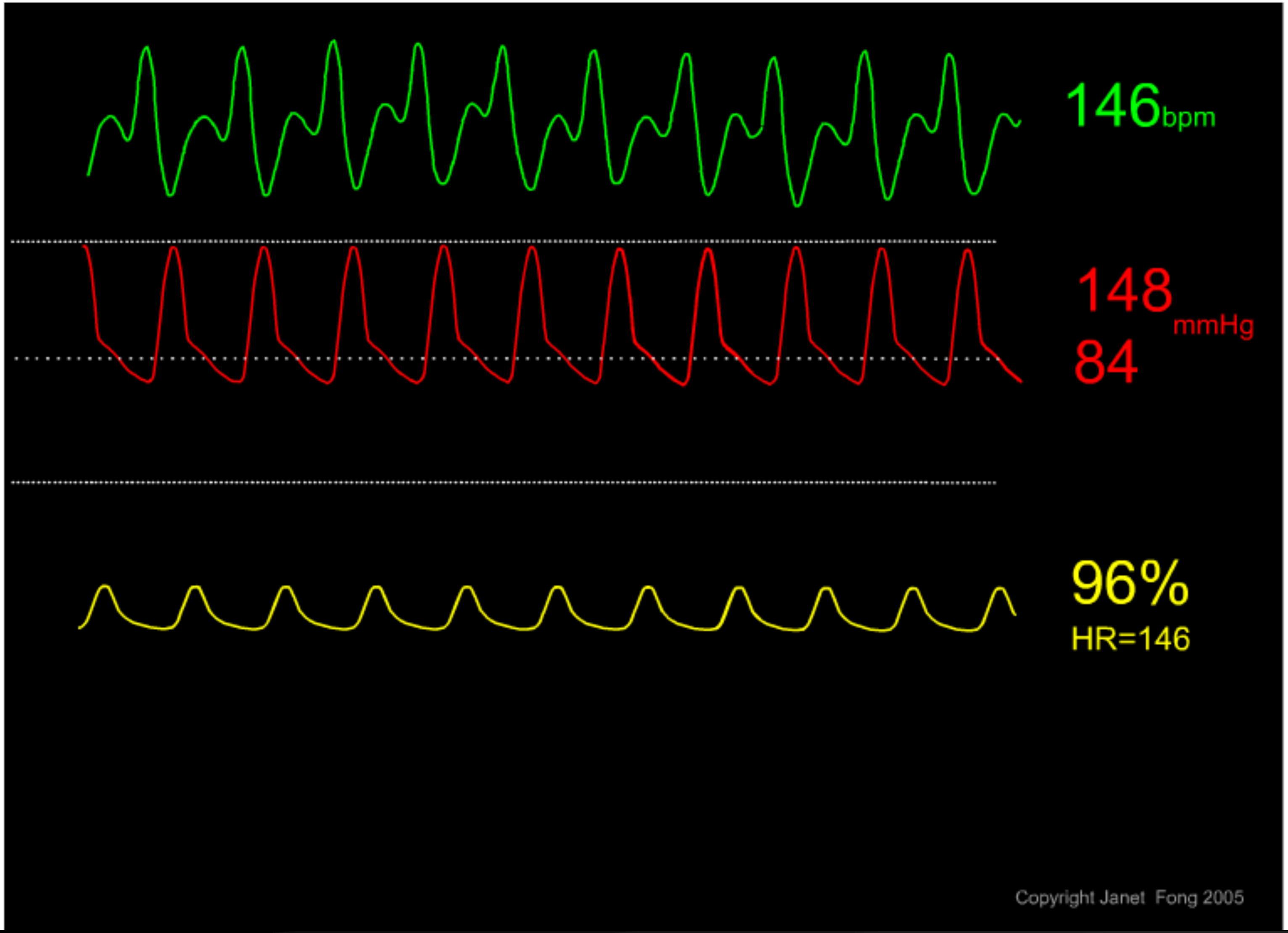
Management of torsades

- Withdraw precipitating agent
- IV magnesium 5-10 mmol over 15 minutes irrespective of serum level
- Rapid ventricular pacing

Polymorphic VT & MI

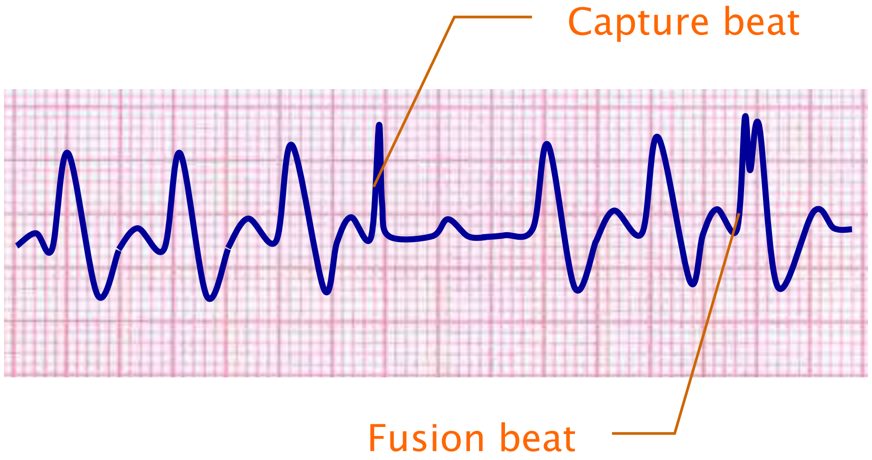
- Consider withdrawal of catecholamines (if tolerated)
- Anti-angina therapy, especially ß blocker, revascularization or IABP
- IV amiodarone or lignocaine if above measures contraindicated

Case C



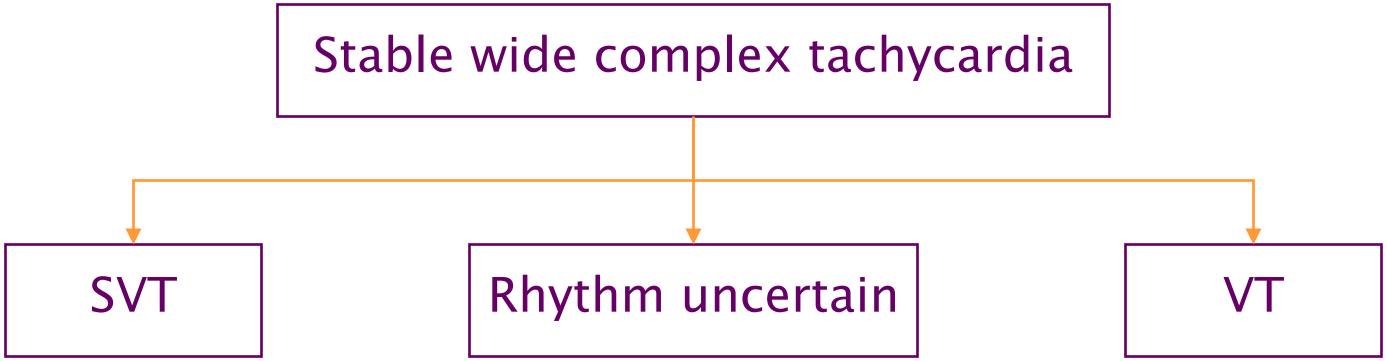
VT or SVT?

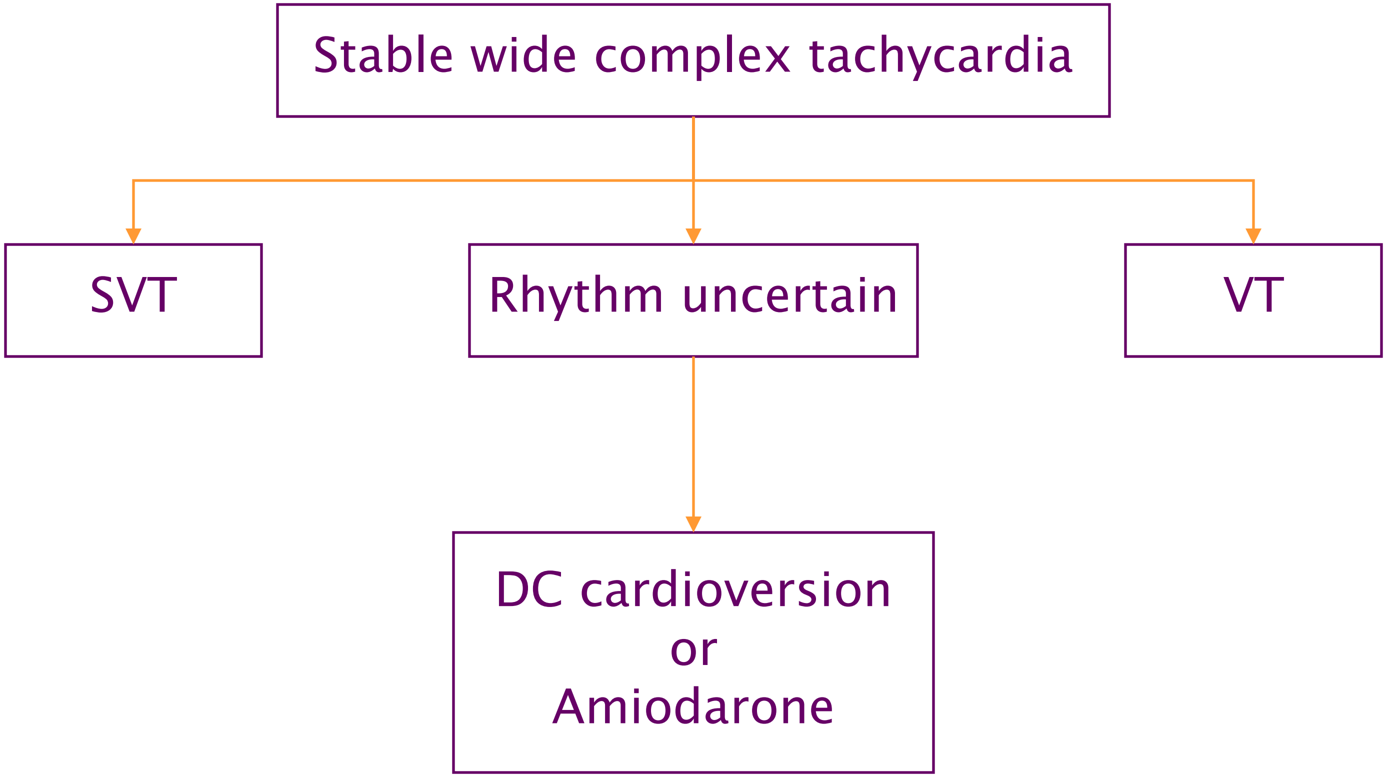
- VT
  - History
    - IHD
    - Structural heart disease
  - ECG criteria
    - AV dissociation on ECG
      - Fusion beats
      - Capture beats
      - Other criteria too complicated

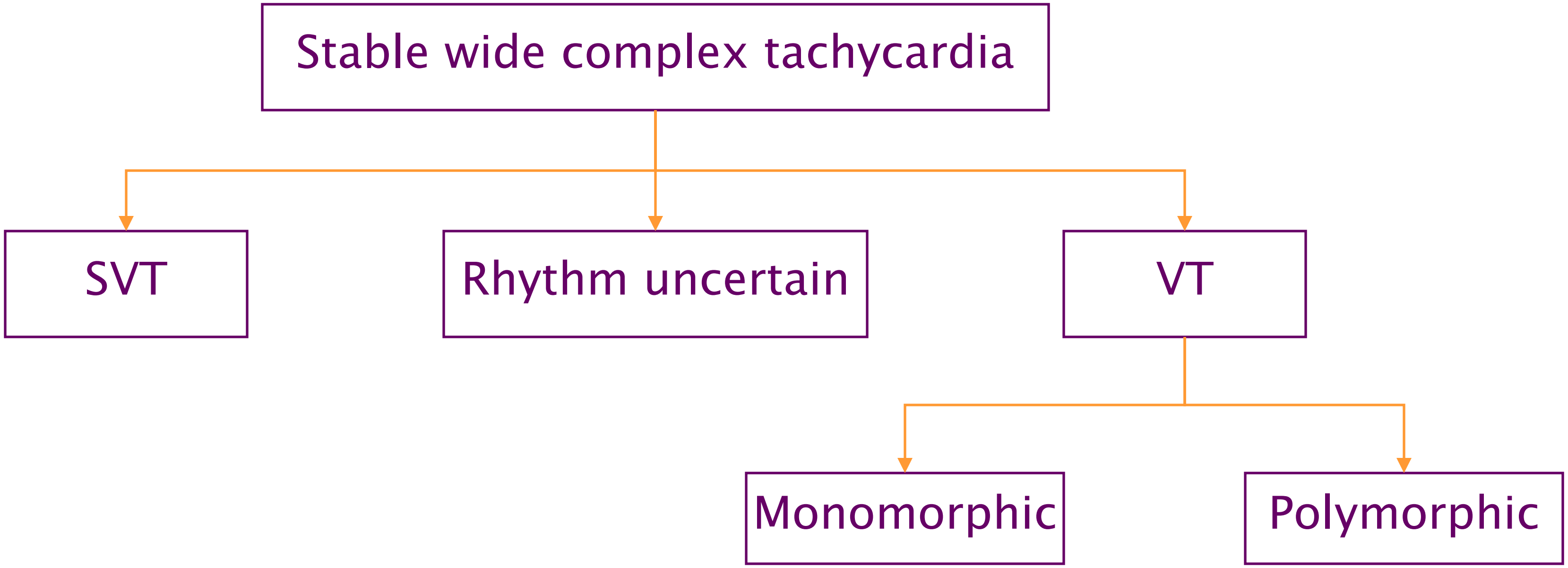


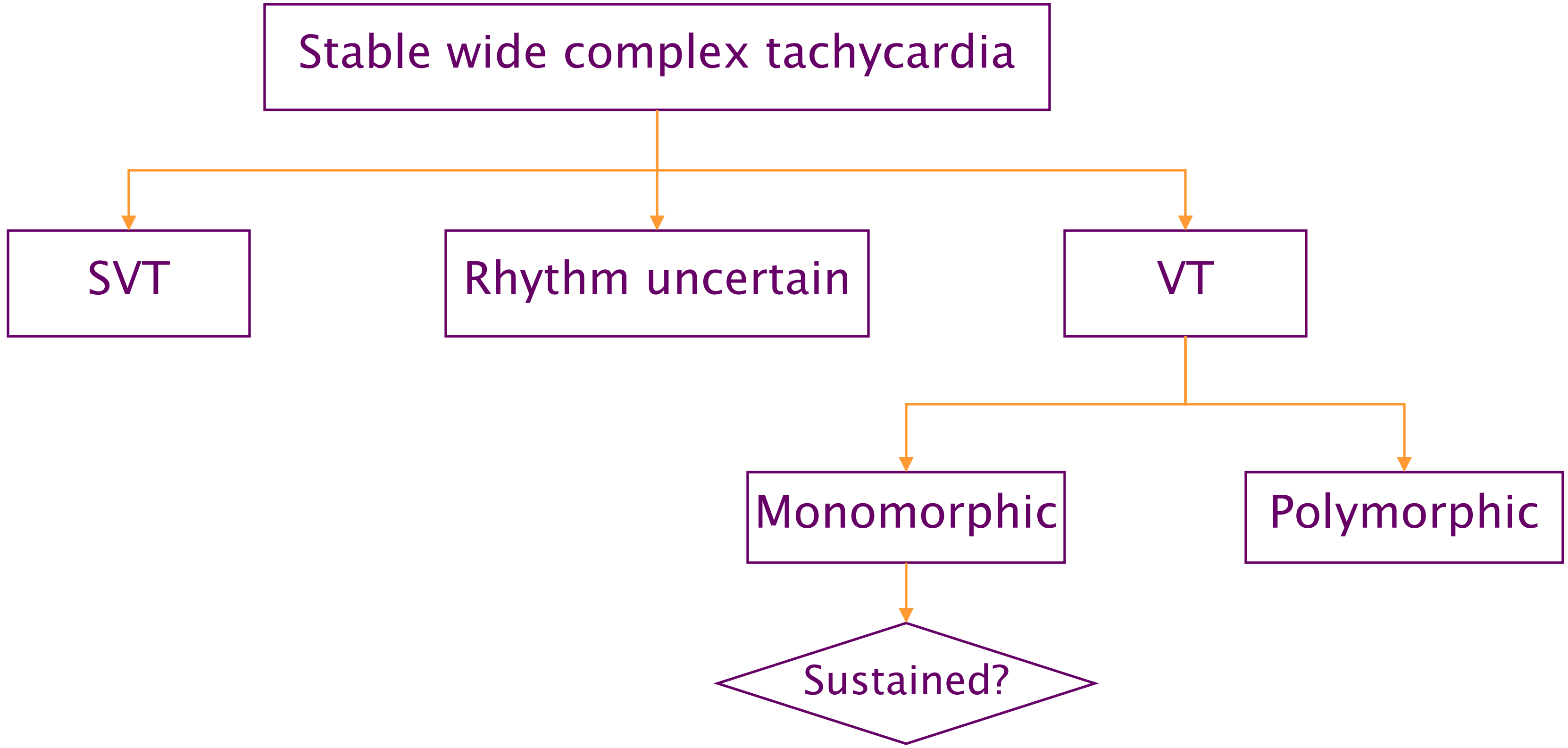
VT or SVT?

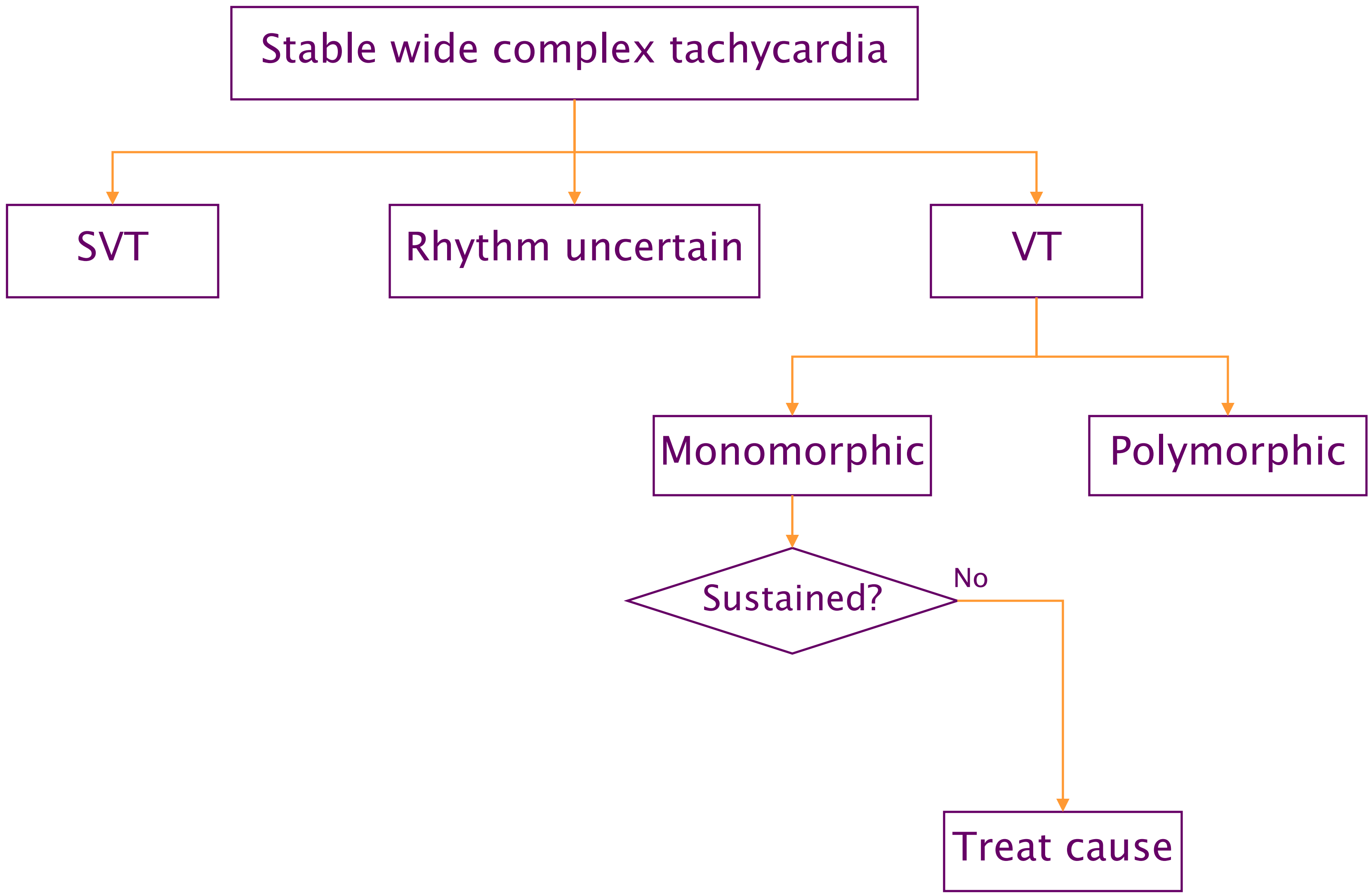
- SVT
  - History
    - Previous aberrant rhythms
    - Accessory pathways
    - BBB
    - Rate dependent BBB
  - ± slowed/abolished by carotid sinus massage

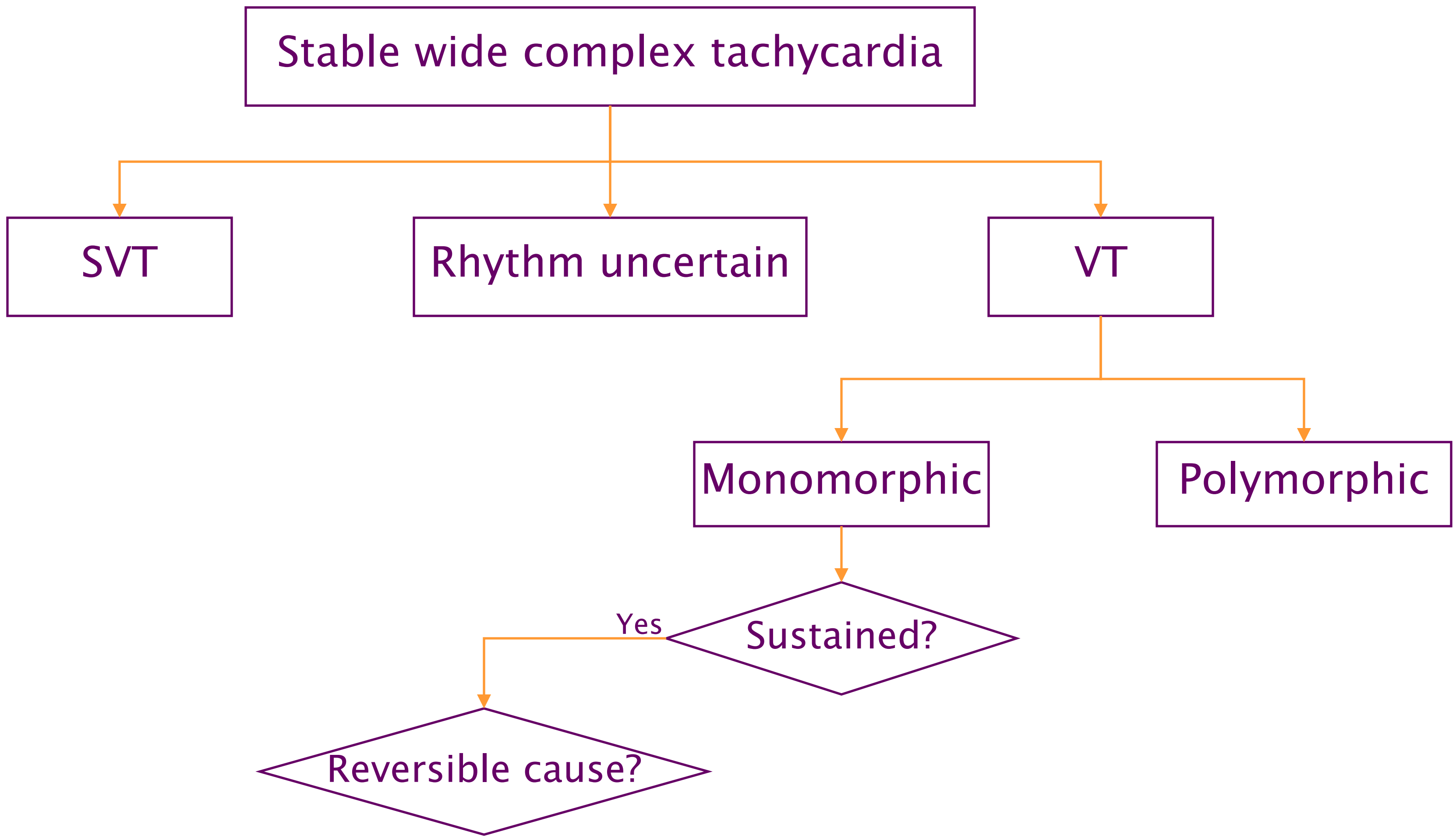

Stable wide complex tachycardia
SVT
Rhythm uncertain
VT

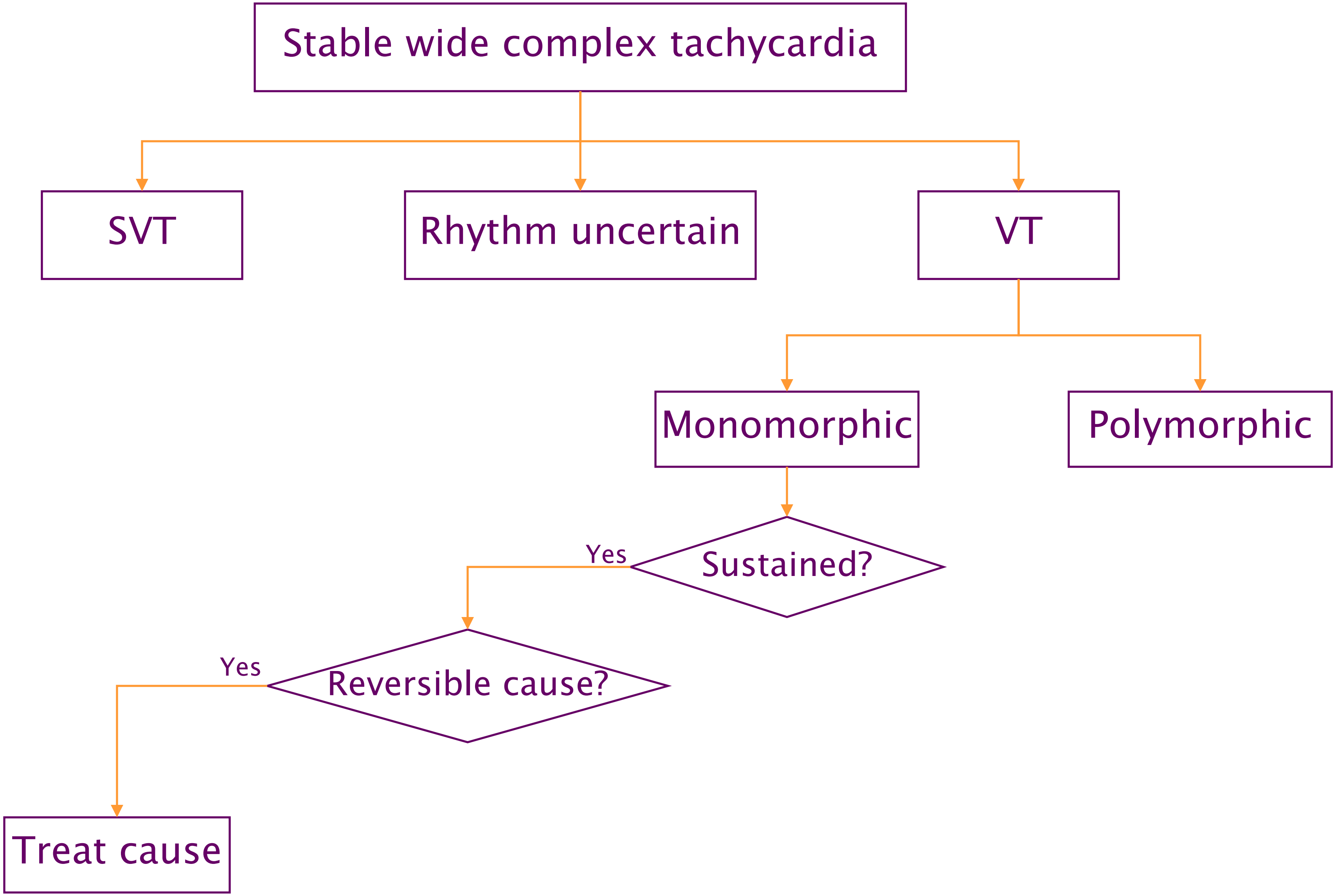

Stable wide complex tachycardia
SVT
Rhythm uncertain
VT
DC cardioversion
or
Amiodarone

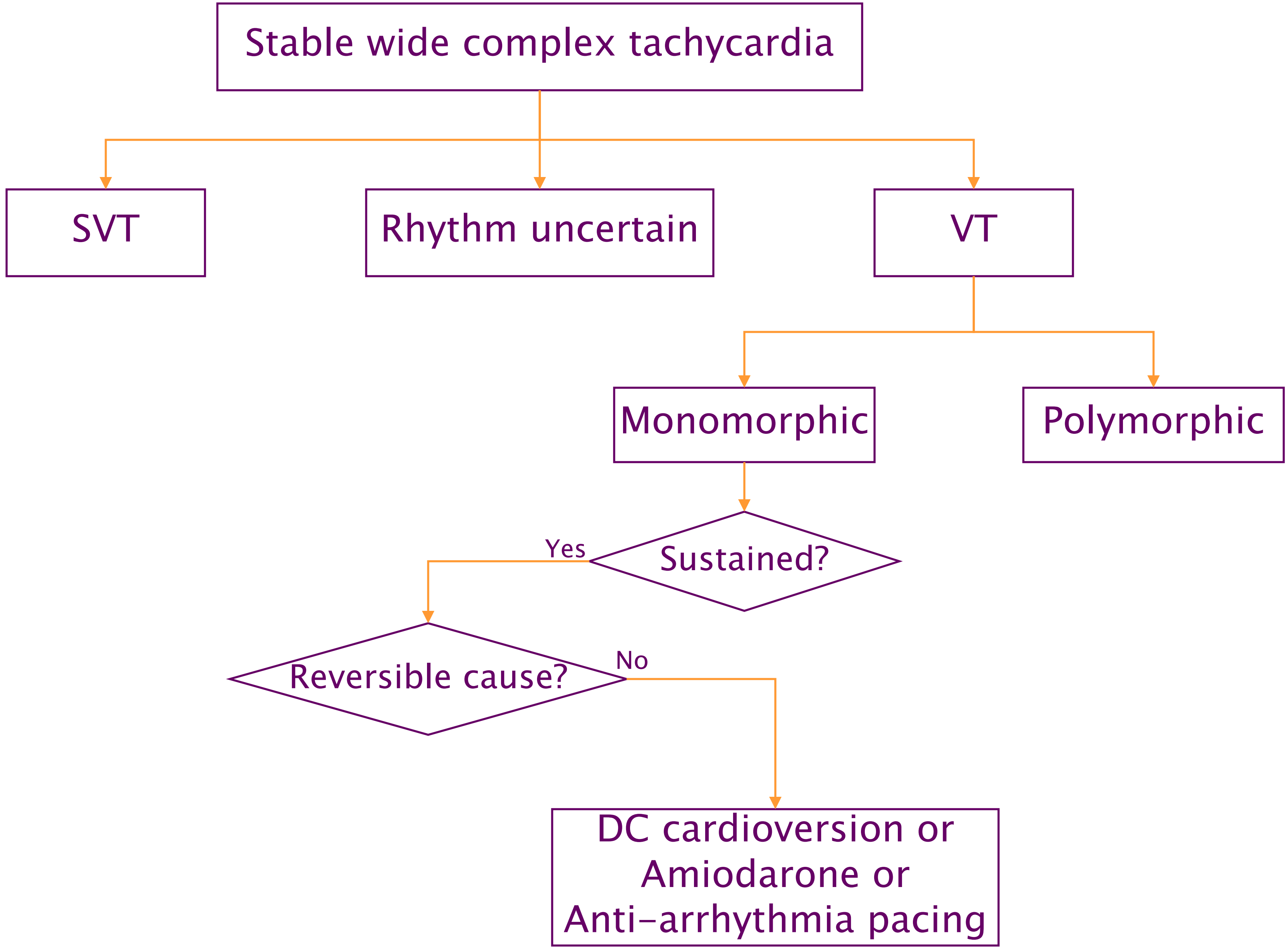

Stable wide complex tachycardia
SVT
Rhythm uncertain
VT
Monomorphic
Polymorphic


Stable wide complex tachycardia
SVT
Rhythm uncertain
VT
Monomorphic
Polymorphic
Sustained?

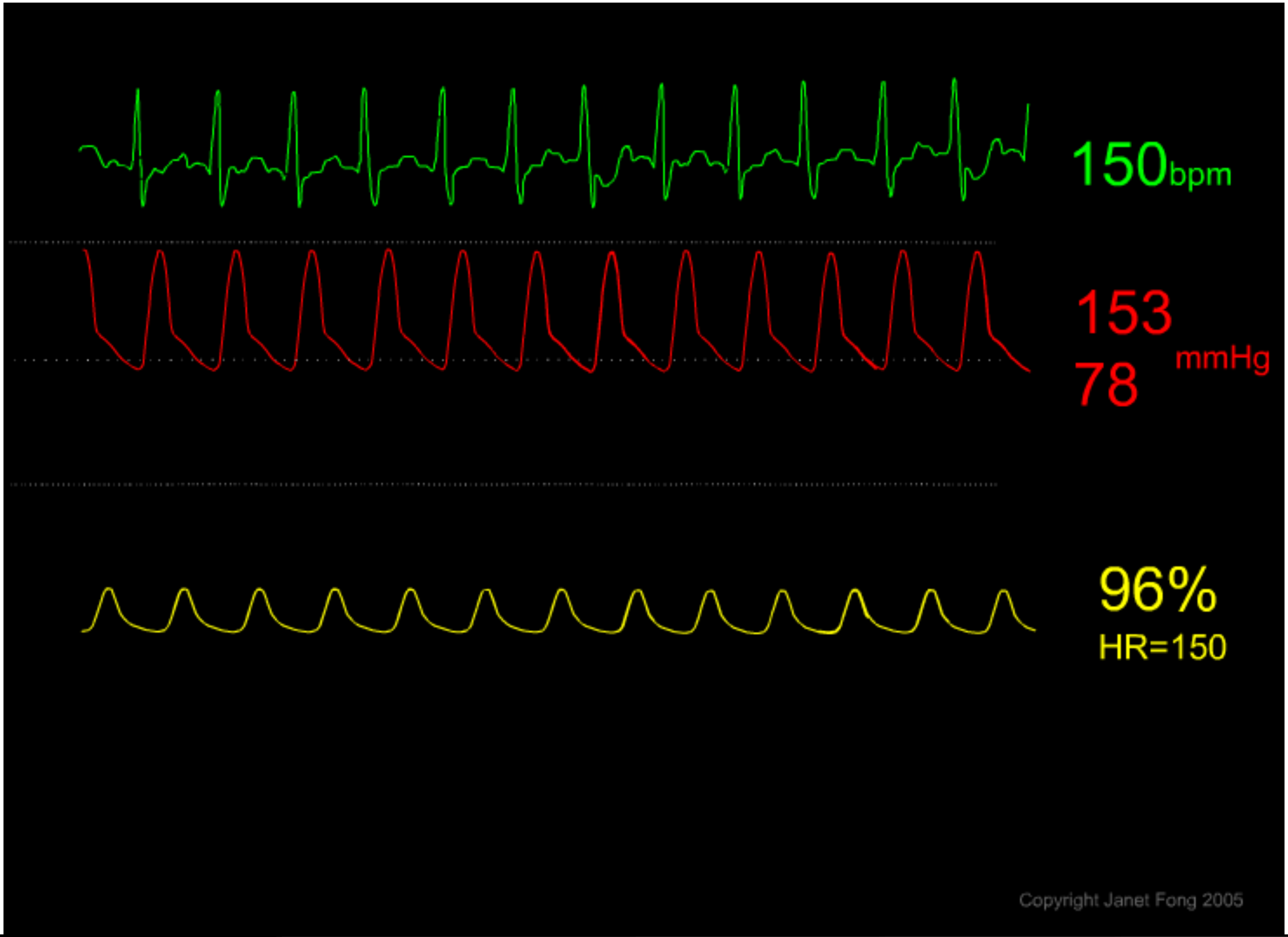

Stable wide complex tachycardia
SVT
Rhythm uncertain
VT
Monomorphic
Polymorphic
Sustained?
No
Treat cause

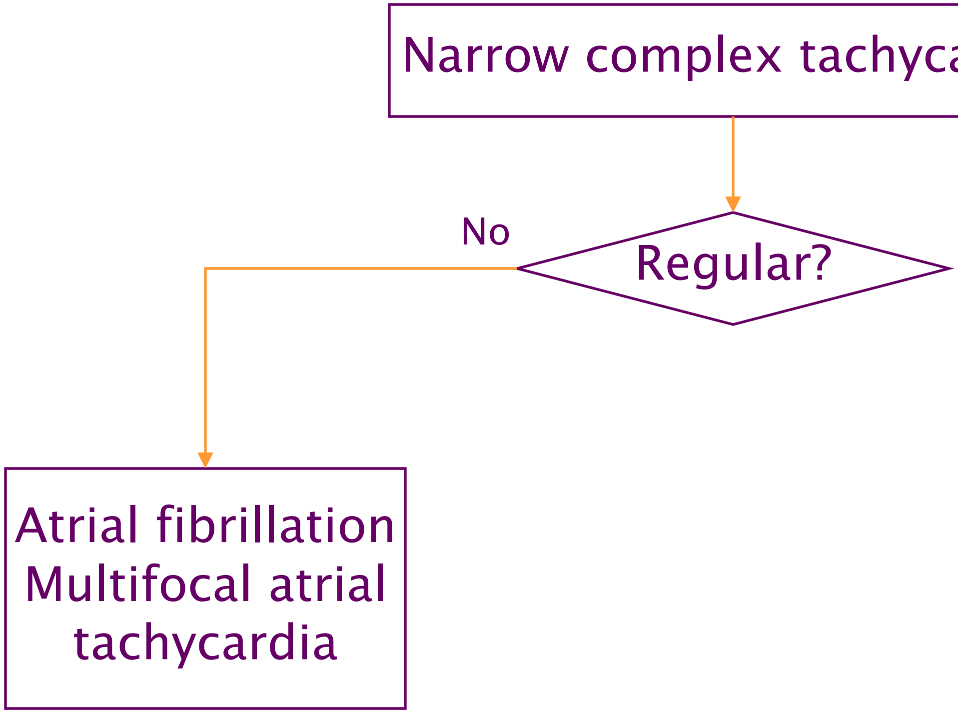

Stable wide complex tachycardia
SVT
Rhythm uncertain
VT
Monomorphic
Polymorphic
Sustained?
Yes
Reversible cause?

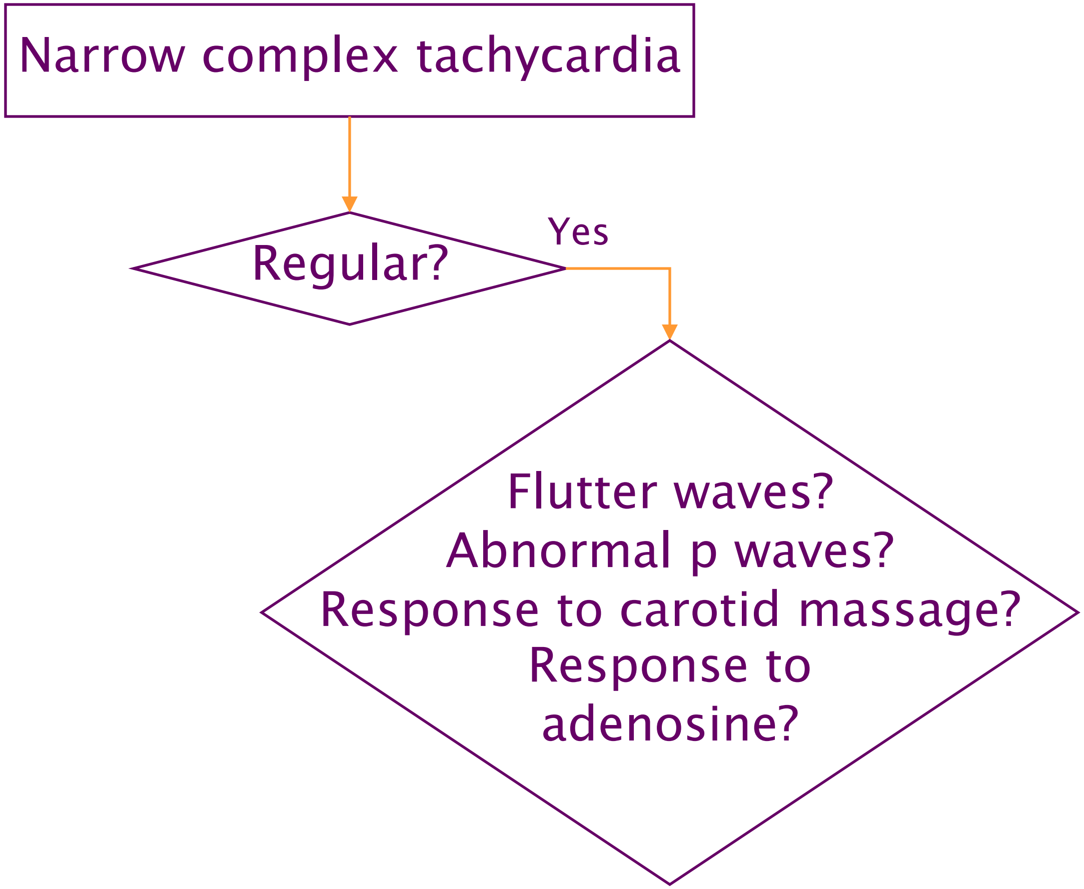

Stable wide complex tachycardia
SVT
Rhythm uncertain
VT
Monomorphic
Polymorphic
Sustained?
Yes
Reversible cause?
Yes
Treat cause


Stable wide complex tachycardia
SVT
Rhythm uncertain
VT
Monomorphic
Polymorphic
Sustained?
Yes
Reversible cause?
No
DC cardioversion or
Amiodarone or
Anti-arrhythmia pacing

Case D




Narrow complex tachycardia
Regular?
No
Atrial fibrillation
Multifocal atrial tachycardia

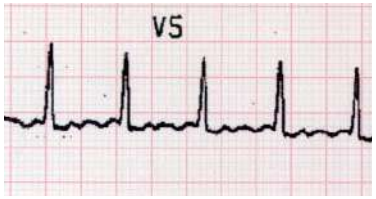

Narrow complex tachycardia
Regular?
Yes
Flutter waves?
Abnormal p waves?
Response to carotid massage?
Response to
adenosine?

Narrow complex tachycardia

Regular?

Yes

Flutter waves?
Abnormal p waves?
Response to carotid massage?
Response to adenosine?



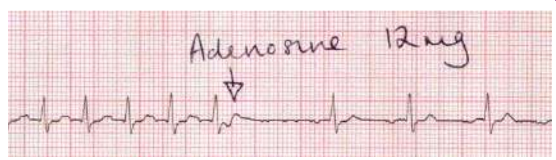
Atrial flutter

Narrow complex tachycardia

Regular?

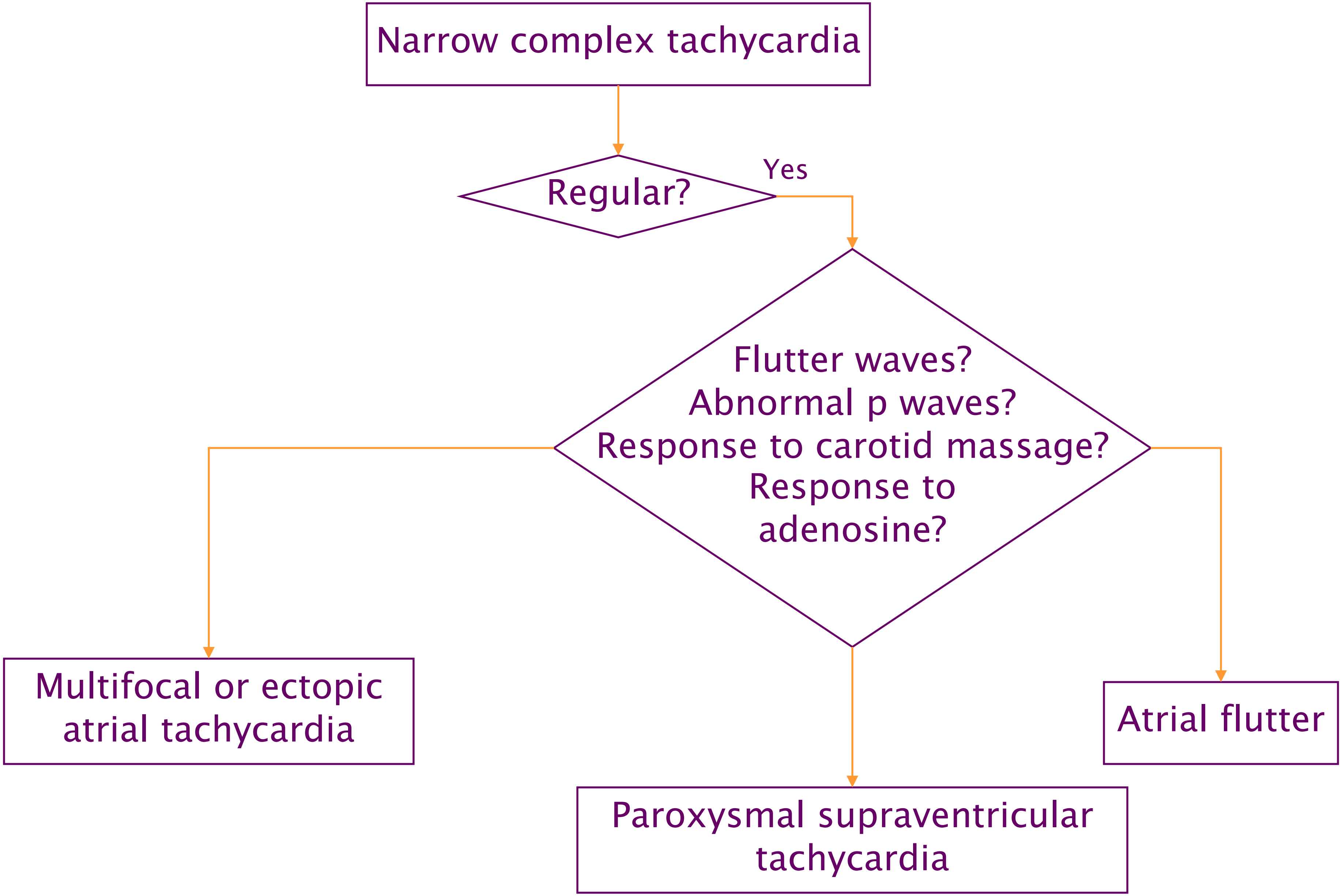
Yes

Flutter waves?
Abnormal p waves?
Response to carotid massage?
Response to adenosine?



Atrial flutter

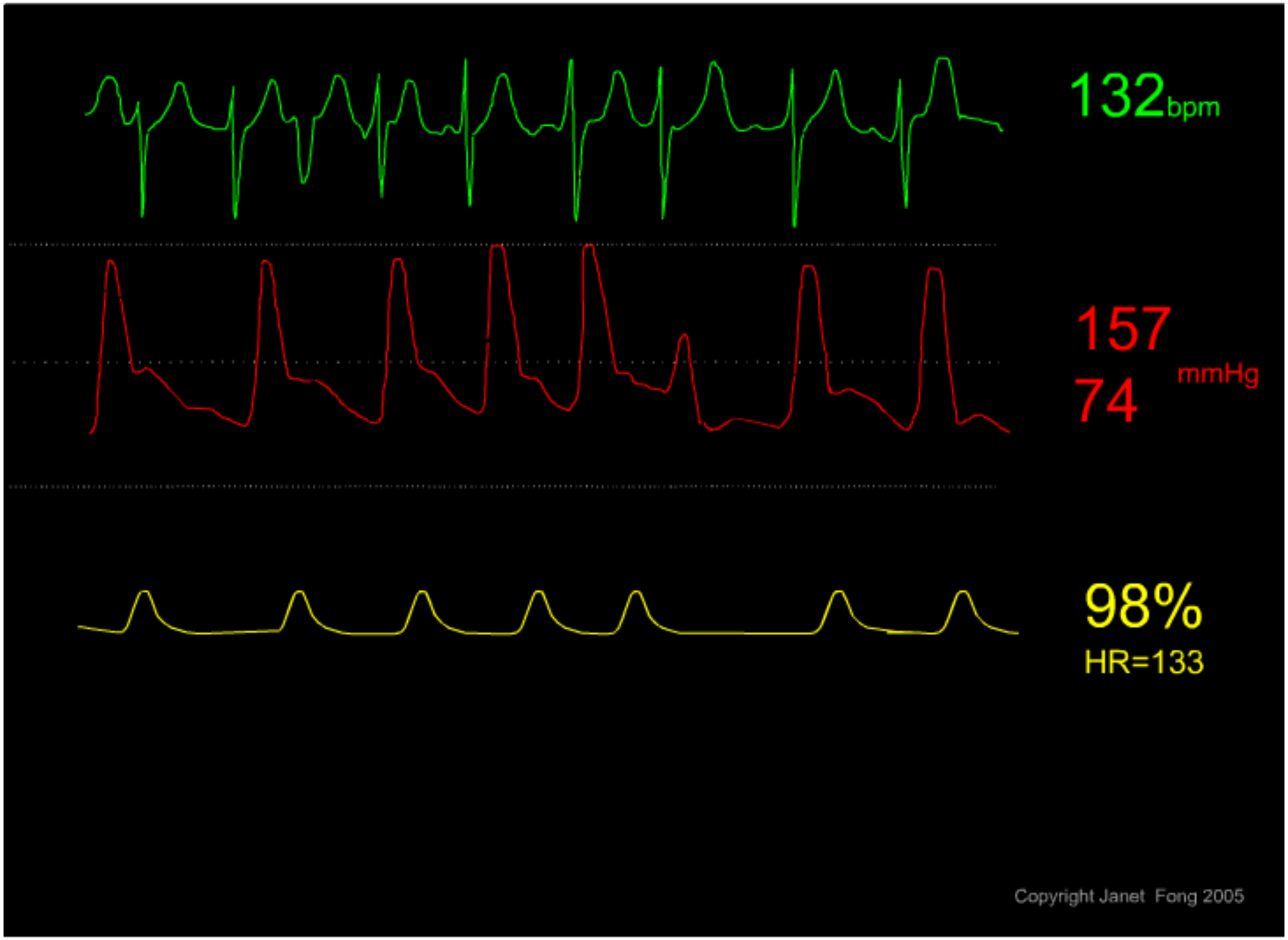
Paroxysmal supraventricular tachycardia


Narrow complex tachycardia
Regular?
Yes
Flutter waves?
Abnormal p waves?
Response to carotid massage?
Response to adenosine?
Multifocal or ectopic atrial tachycardia
Paroxysmal supraventricular tachycardia
Atrial flutter

Treatment of PSVT

- Good LV function
  - Ca channel blocker
    - Verapamil
    - Diltiazem
  - ß blocker
  - Amiodarone
- Poor LV function
  - Amiodarone
  - Diltiazem

Case E



Irregular rhythm

- Atrial fibrillation
- Atrial ectopics
- Multifocal atrial tachycardia

Treatment of AF

- Aims
  - Chronic
    - Rate control
  - Acute
    - Restoration of sinus rhythm
    - Rate control
  - Paroxysmal
    - Restoration of sinus rhythm
    - Secondary prevention
  - Prevention of complications

Pharmacological treatment

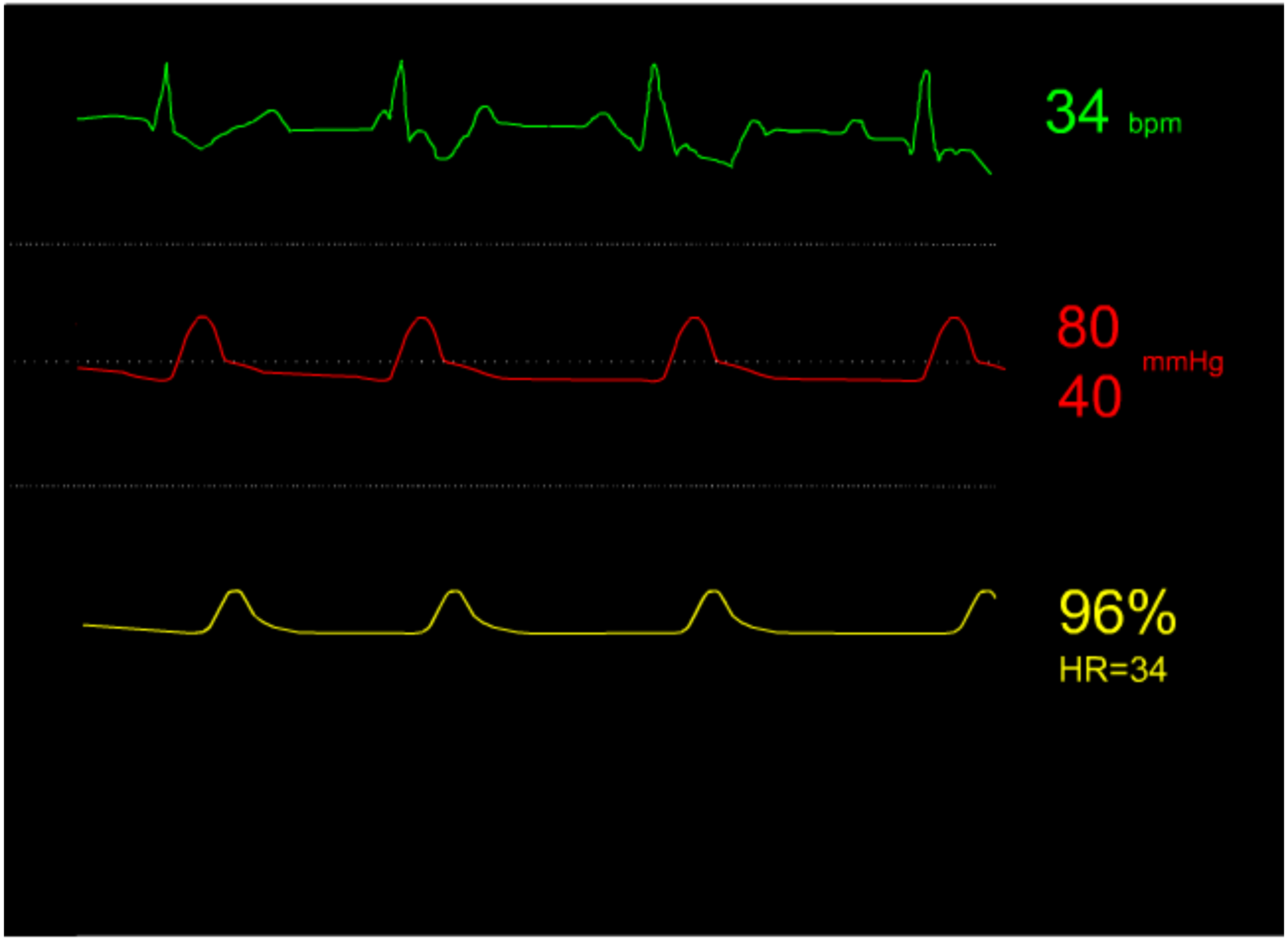
- Rate control only
  - β blockers
  - Diltiazem, verapamil
  - Digoxin (heart failure)
- Rate control & pharmacological cardioversion
  - Amiodarone
  - Procainamide
  - Ibutilide

Prevention of complications

- AF >48h
  - Consider anti-coagulation to prevent systemic embolization
  - Use agents that may convert rhythm with extreme caution unless patient anticoagulated
  - Anticoagulation for 3 weeks prior to cardioversion

Case F

- 78 year old man with dizziness



Look for cause

- Myocardial infarction
- Drugs
- Electrolytes (K, Mg, Ca)
- Hypothyroidism
- Hypothermia
- Sepsis
- Endocarditis
- Vagal-mediated in ICU
  - Intubation, suctioning, ↑ICP, urination, defaecation, vomiting, retching

Summary

- Tachycardias
  - Is the arrhythmia pathological?
  - Is the patient shocked?
    - (Yes ⇒ cardioversion)

  - Treat/correct precipitants
  - Control or abort arrhythmia
  - Prevent complications

Summary

- Bradycardias
  - Is the patient shocked?
    - Yes ⇒ atropine ± epinephrine
  - What is the arrhythmia?
  - Atropine then isoproterenol/epinephrine or pacing

Any questions?

Assessment of the seriously ill

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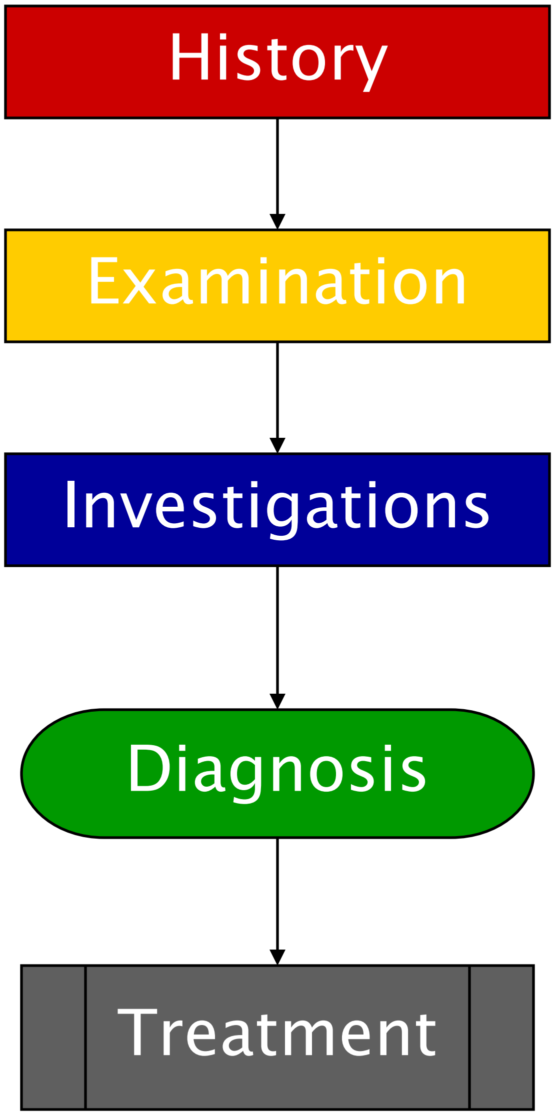
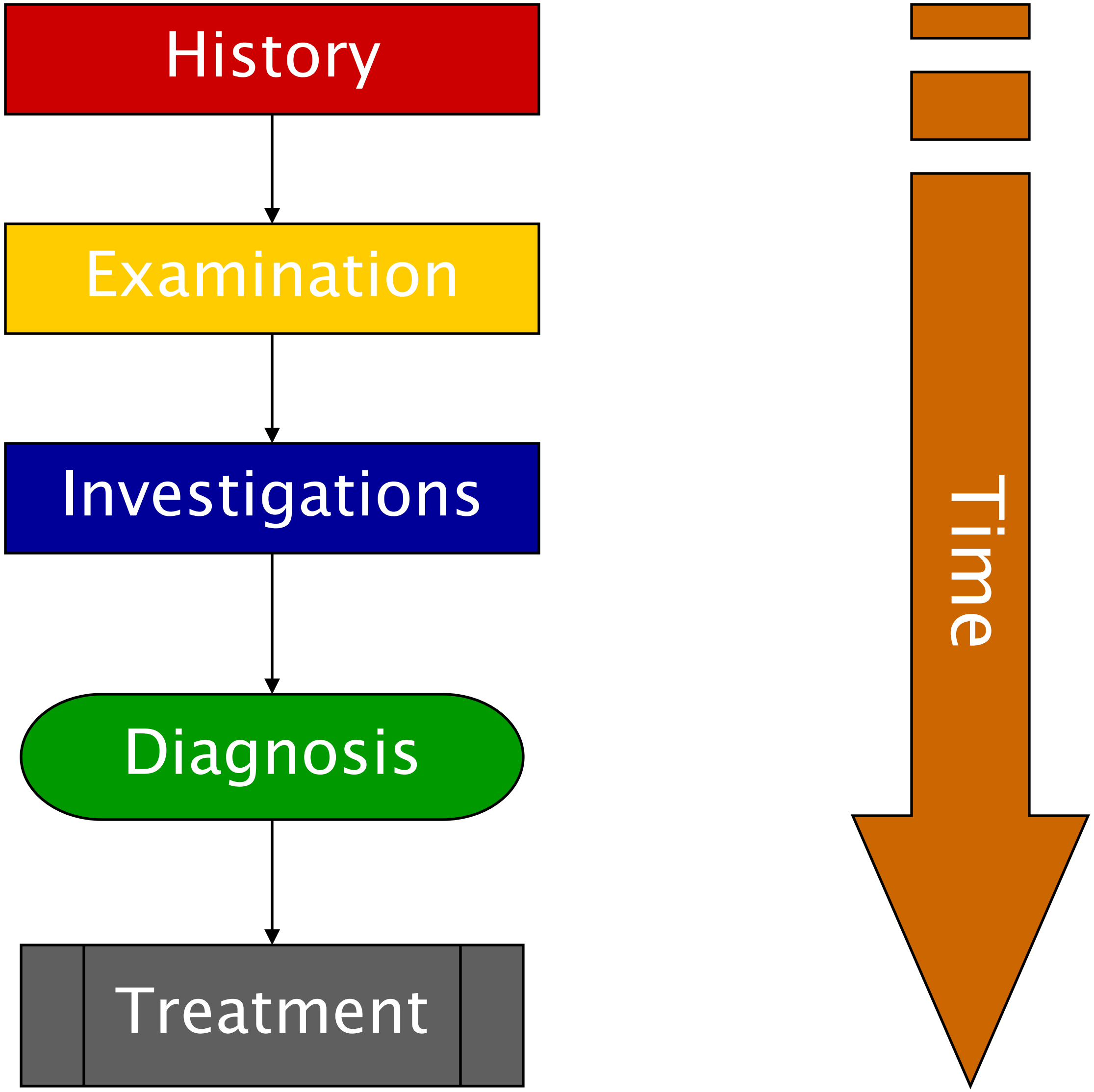
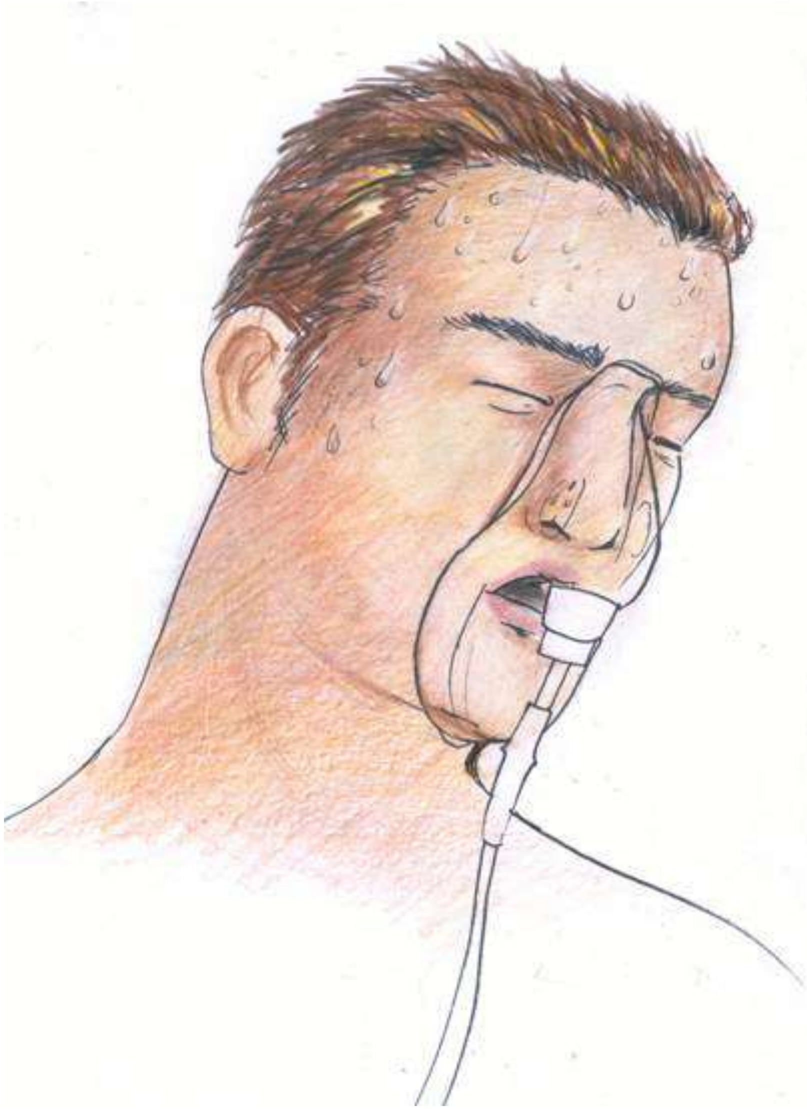
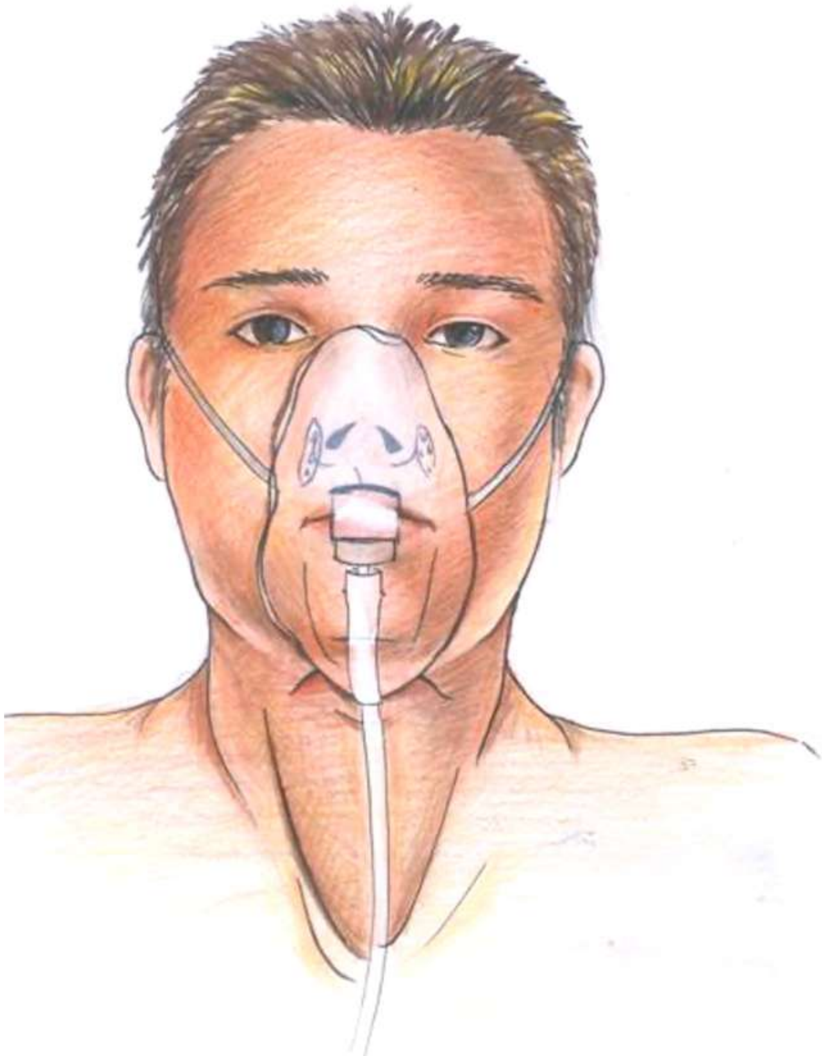
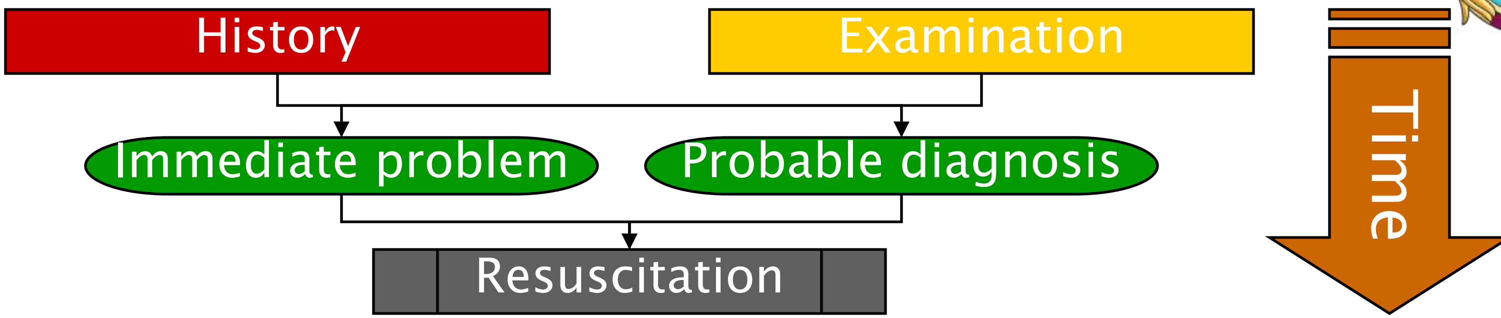

History
Examination
Investigations
Diagnosis
Treatment

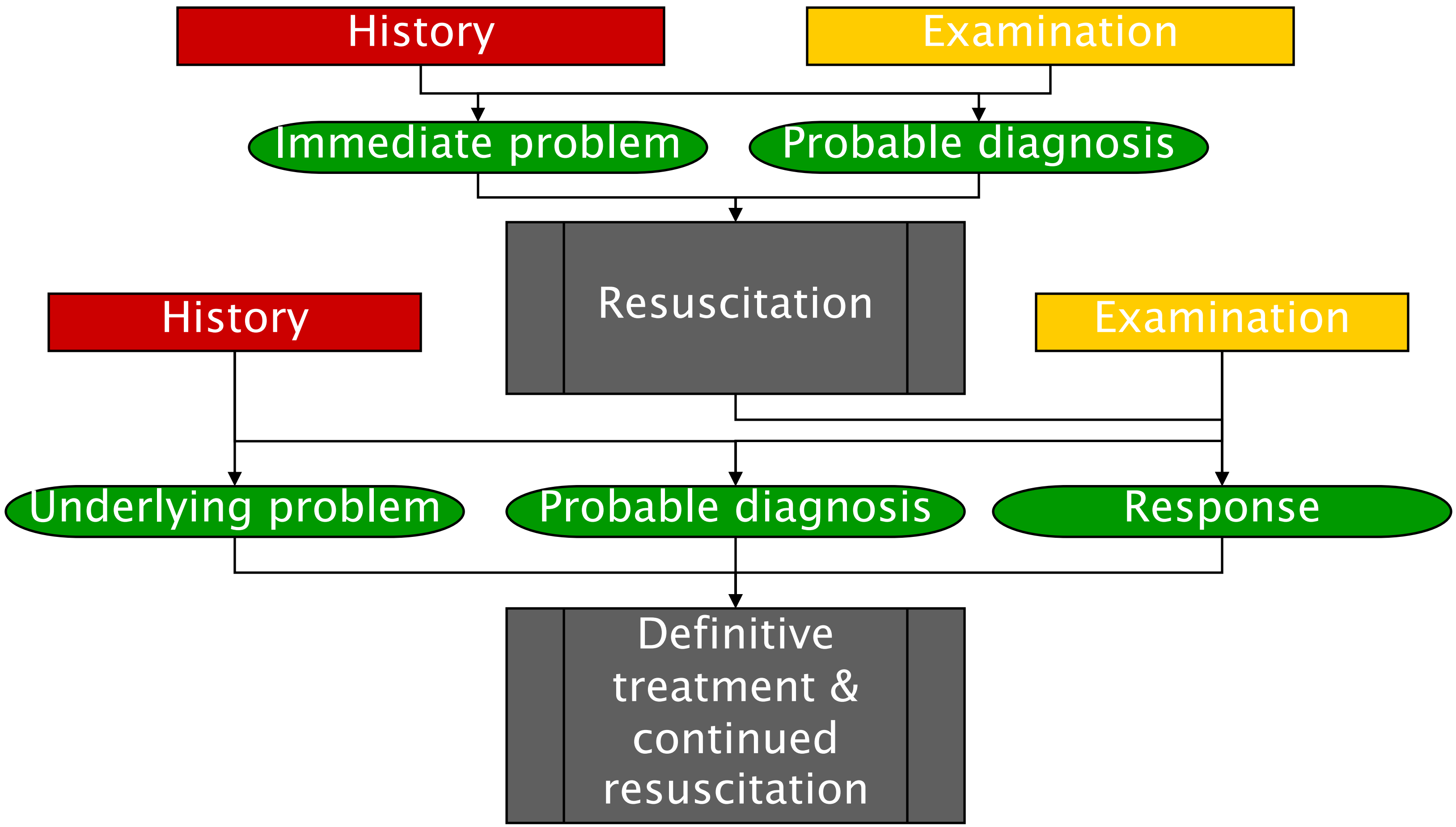

History
Examination
Investigations
Diagnosis
Treatment
Time

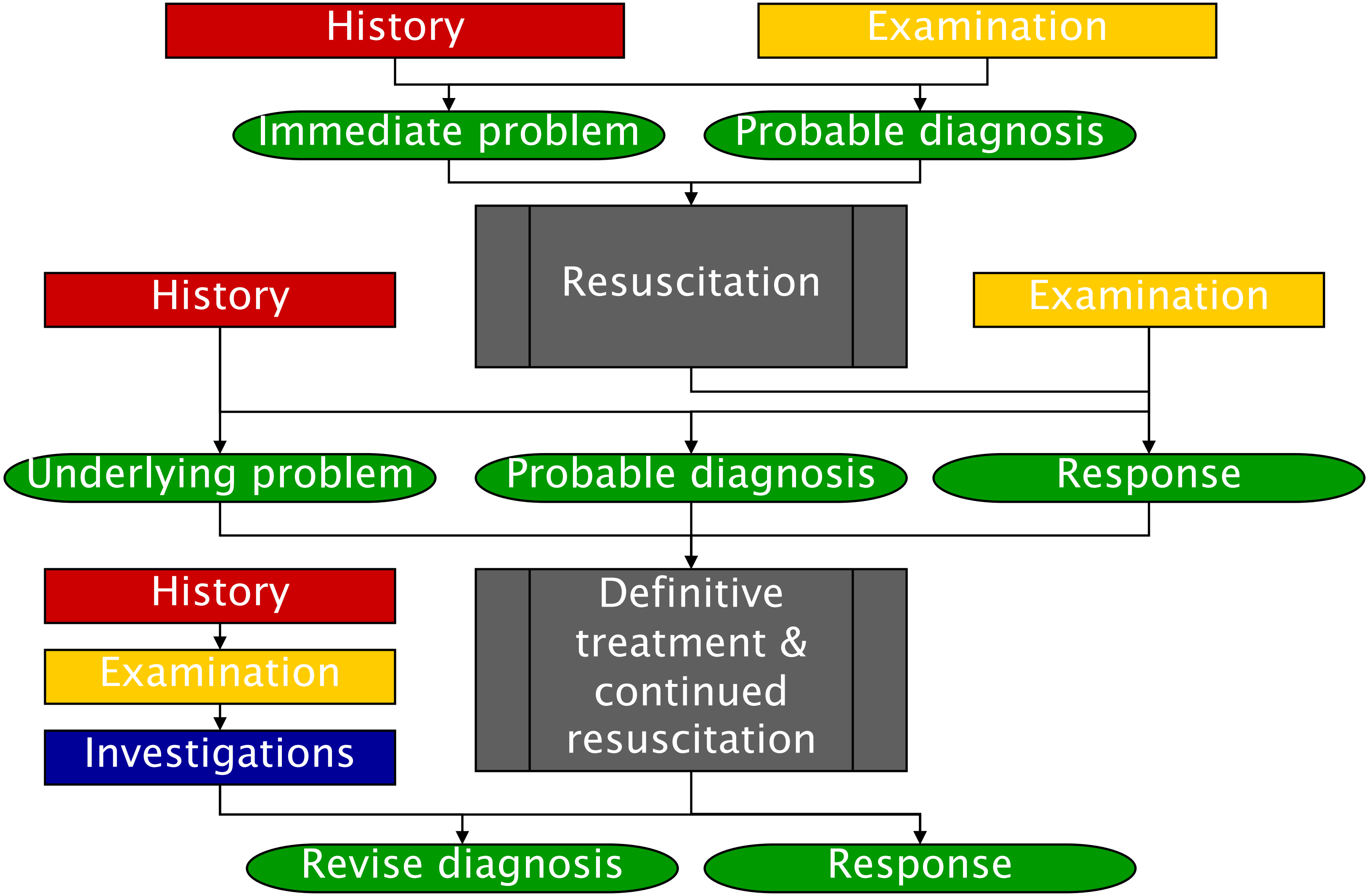
How much time?

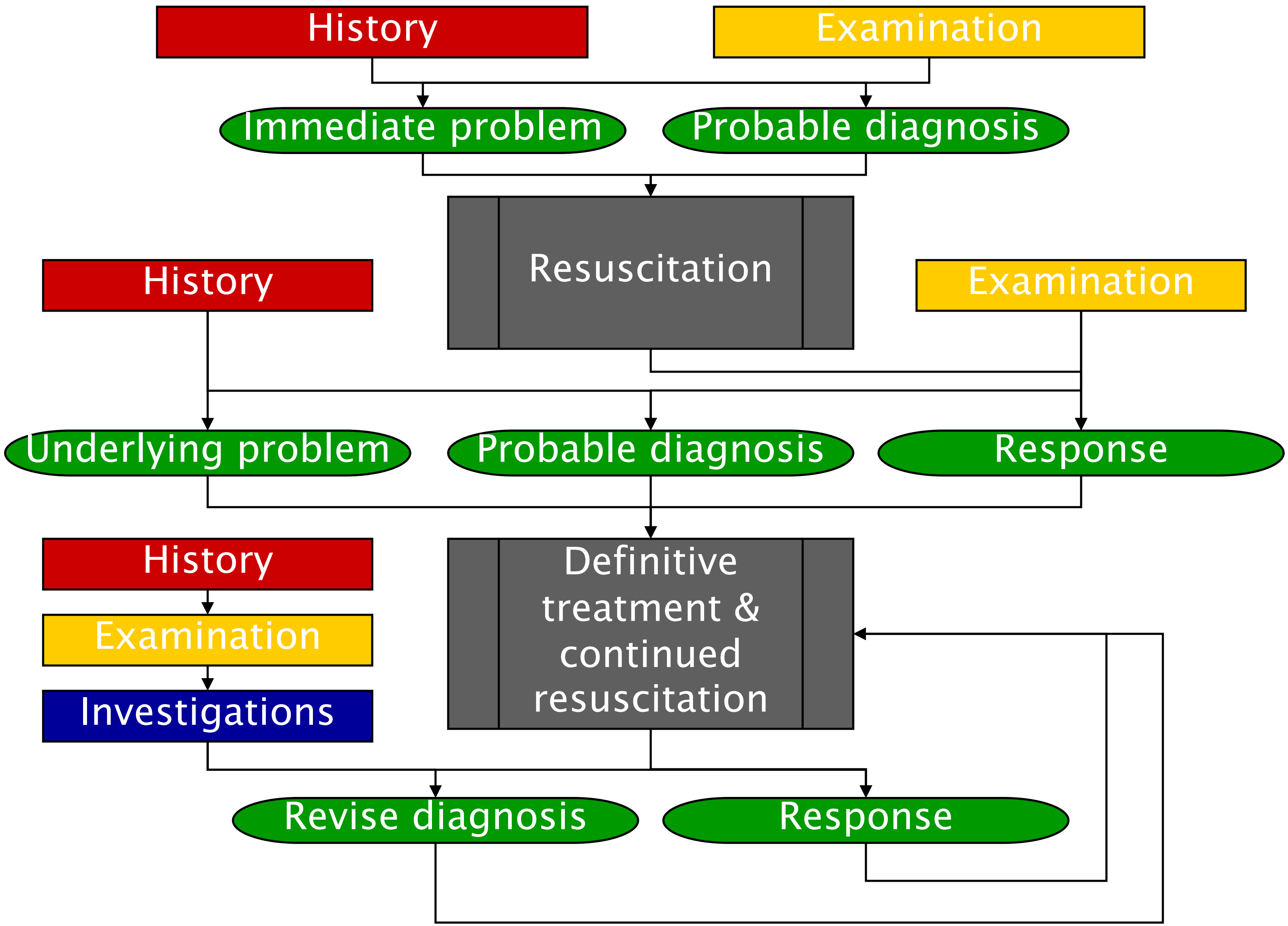





History
Examination
Immediate problem
Probable diagnosis
Resuscitation
Time


History
Examination
Immediate problem
Probable diagnosis
Resuscitation
History
Examination
Underlying problem
Probable diagnosis
Response
Definitive treatment & continued resuscitation


History
Examination
Immediate problem
Probable diagnosis
Resuscitation
History
Examination
Underlying problem
Probable diagnosis
Response
History
Examination
Investigations
Definitive treatment & continued resuscitation
Revise diagnosis
Response


History
Examination
Immediate problem
Probable diagnosis
Resuscitation
History
Examination
Underlying problem
Probable diagnosis
Response
History
Examination
Investigations
Definitive treatment & continued resuscitation
Revise diagnosis
Response

Shock forms

Loss of circulating volume
↓
Decrease in venous return

Loss of contractility- major arrhythmia
↓
Failure of the pump function

Obstruction

(pulmonary embolism, tension pneumothorax, cardiac tamponade)

Loss of vascular tone
↓
Maldistribution of blood flow

History

- Patient
- Relatives
- Medical/paramedical staff
- Notes & charts

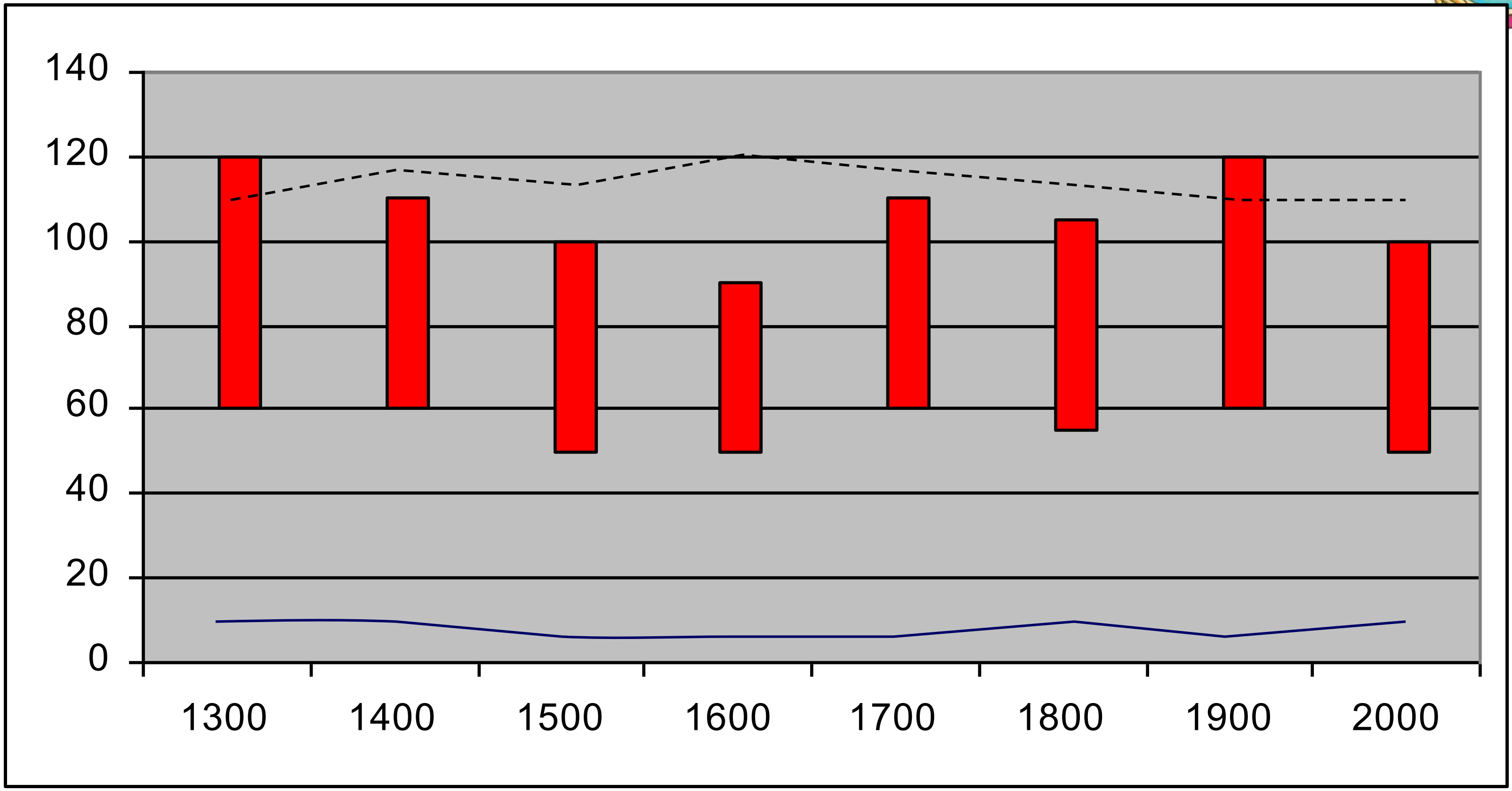
History

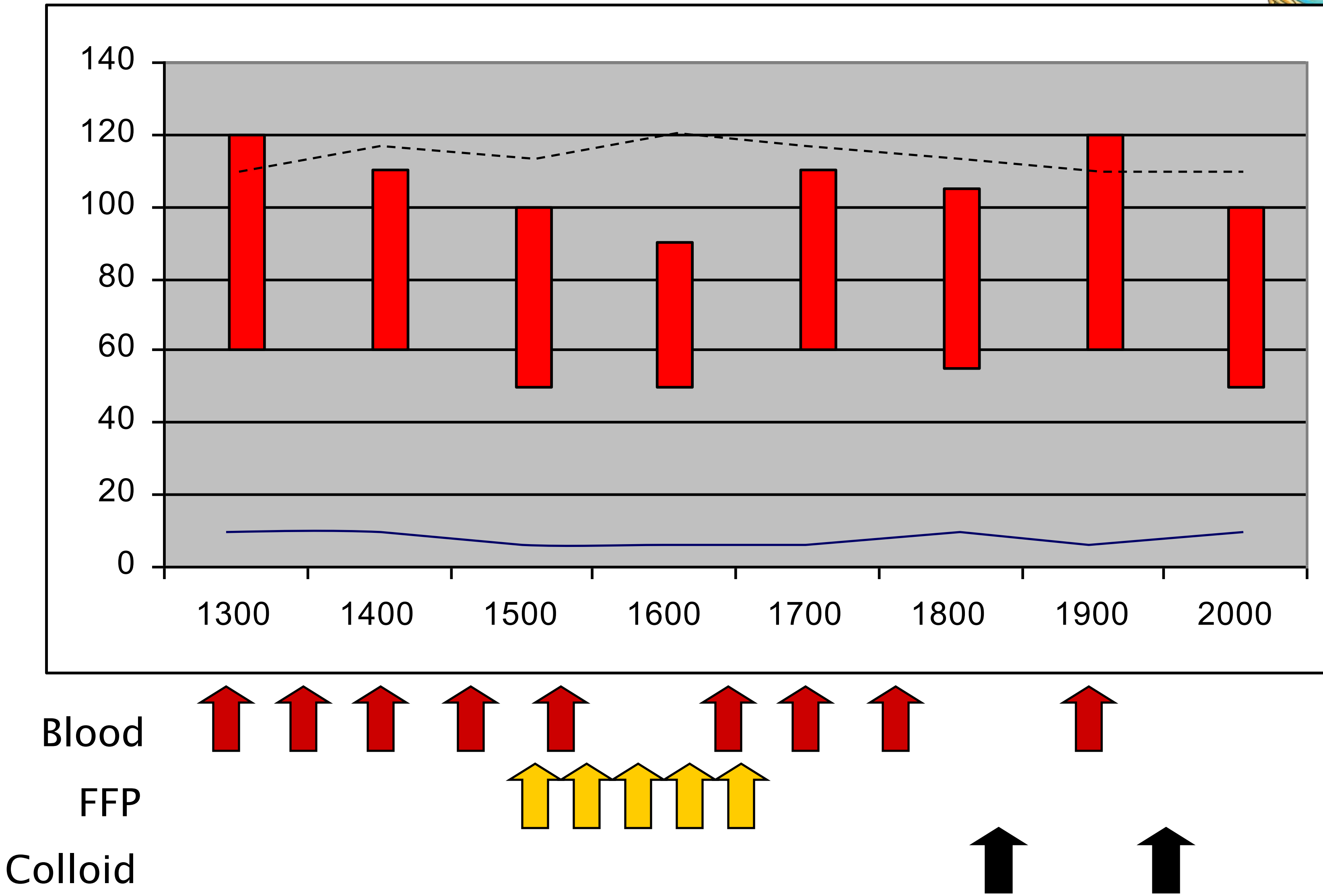
- Immediate problem
- Physiological reserve
  - Exercise tolerance
  - Previous major illnesses

History

- Immediate problem
- Physiological reserve
  - Exercise tolerance
  - Previous major illnesses
- Treatment
  - Definitive
  - Supportive

Support


140
120
100
80
60
40
20
0
1300
1400
1500
1600
1700
1800
1900
2000



140
120
100
80
60
40
20
0
1300
1400
1500
1600
1700
1800
1900
2000
Blood
FFP
Colloid

History

- Later
  - Full history

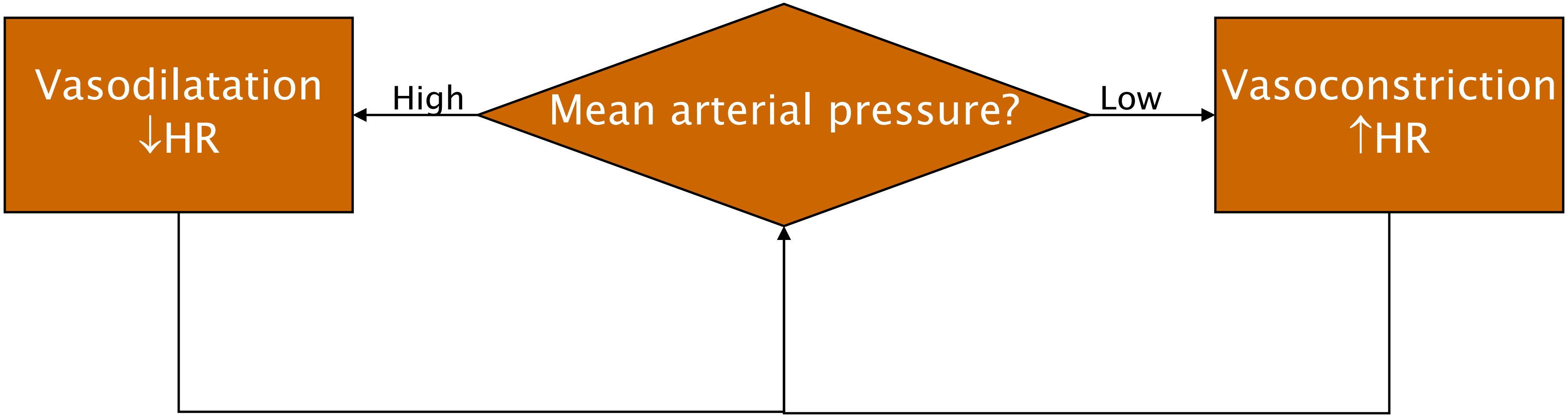
Examination

- Initial aim:
  - Rapidly determine appropriate resuscitation
  - Assess severity

  ∴ Limited examination
- Subsequent full examination

Difficult to assess

Homeostasis



- Assess compensatory response
- Failure of compensation

  ⇒ Very severe disease

Investigations

- Depend on clinical presentation
- Useful “routine” investigations
  - Glucose
  - Electrolytes: Na, K, Ca, Mg, PO_4
  - Renal function tests
  - Liver function tests
  - CRP, PCT
  - TSH
  - LDH
  - Cardiac enzymes, (pro) BNP
  - Complete blood count
  - Clotting (including D-Dimer)

  - ABG (arterial and central venous)
  - ECG, CXR, CT scan, bedside echo
  - Pregnant?

Airway

- Key points
  - Stridor may be absent in airway obstruction, particularly in severe cases
  - Normal oxygen saturation does not exclude compromised airway
  - Hypercarbia and ↓ consciousness ⇒ compensatory mechanisms exhausted
  - ↓HR in patient with airway obstruction ⇒ impending cardiorespiratory arrest
  - Assess sympathetic response

Breathing

- Key points
  - Marked tachypnoea good marker of severely ill patient
  - Pulse oximetry useful, BGA useful
    - Significant desaturation late feature of inadequate ventilation
  - Tachypnoea in absence of respiratory failure may be due to metabolic acidosis or sepsis
  - Low respiratory rate may indicate impending respiratory arrest
  - Assess sympathetic response

Breathing

- Worry if
  - RR > 30/min (or < 8/min)
  - unable to speak 1/2 sentence without pausing
  - agitated, confused or comatose
  - cyanosed or $SpO_2 < 90\%$
  - deteriorating despite therapy

Circulation

- Key points
  - Hypotension late feature of shock
  - Assess tissue perfusion
    - Conscious level
    - Peripheries
    - Urine output
    - Acidosis
  - Assess cause of shock
    - HR, JVP, peripheries

Hypotension

- lowest acceptable BP depends on usual BP for each patient
- treat all non-pregnant, non-anaesthetised adults with systolic BP < 90 mm Hg as seriously ill
- a few will have no other signs of shock, but still need to be treated with great caution

Conscious state

- Key points
  - ↓ consciousness in absence of neurological disease ⇒ severe systemic disease

Report the critically ill patient

- Key points
  - think about the situation and your patient before you start to report
  - know your patient !
  - know your questions
  - use a standardized way to report your patient

(I) S B A R

S	**Situation**	**Who (you) – Who (the patient), what, why, when, how**
B	**Background**	**Previous illness, Risk factors, Medication**
A	**Acutal Situation**	**Breathing, Airway** **Spontaneus, settings on the ventilator, BGA results**
		Circulation **Rhythm, Blood pressure** **vasoactive drugs, inotropes** **hemodynamic monitoring** **BGA results**
		Neuro **Level of consciousness** **Sedatives**
		Renal and hepatic function
		Monitoring (arterial line, central venous catheter…)
R	**Recommendation**	**We would…** **We ask you…**

Summary

- Altered conscious state
- Hypotension
- Tachycardia
- Tachypnoea
- Cyanosis/hypoxia
- Oliguria
- Acidosis

- Learn how to report your patient

Any questions?

Basic haemodynamic monitoring

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Definition

Basic Hemodynamic Monitoring

- Continuous documentation of heart rate, blood pressure and indirect markers of tissue perfusion such as skin perfusion, urine output, arterial and ventral venous oxygenation and lactate

Aim of hemodynamic monitoring

- Early identification and correction of cardiovascular instability by the continuous assessment of heart rate, filling status, blood flow and pressure.

Case presentation

- 45 years-old male is admitted to the emergency department after complaining about abdominal pain for 24 hours and confusion since a few hours.
- On admission BP was 110/50 mmHg, HR 123 b/min, RR 32 b/min and SpO_2 95%.
- Clinical examination revealed cold extremities and a painful abdomen with signs of cholecystitis

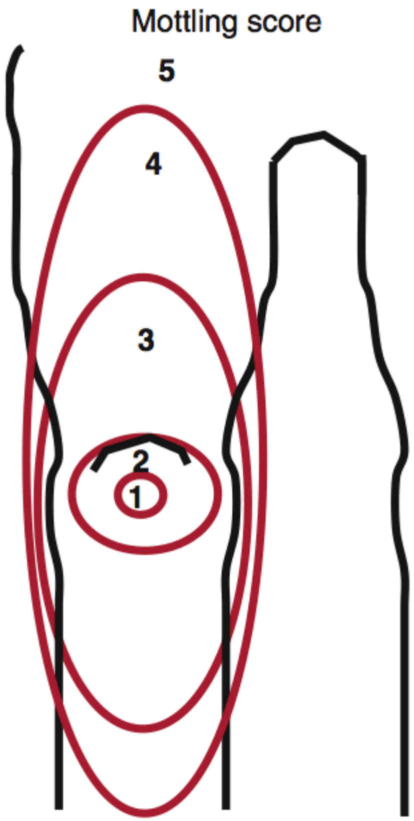
Clinical assessment

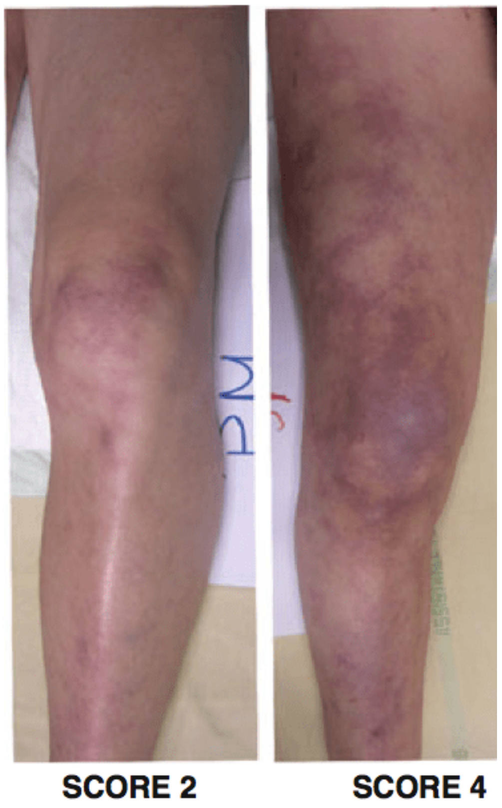
- Systemic circulation
  - BP 110/50 mmHg, HR 123 b/min
    → Compensatory sympathetic stimulation
    - Tachycardia
    - Sweating
    - Vasoconstriction

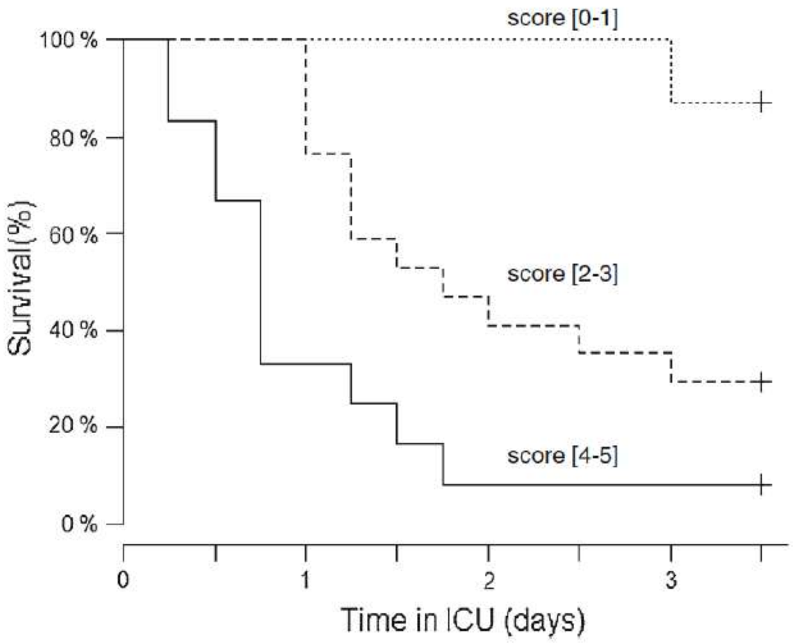
- End-organ function
  - Mental state (GCS 12)
  - Tachypnoe (RR 32 b/min)
  - Lactate 3.2 mmol/L

Clinical examination

– Mottling score







Ait-Oufella H. et al ICM 2011 37:801

Clinical examination

– Capillary refill time

- Assess peripherally and centrally
- Press for 10-15 sec. and then release
- Normal values
  - ✓ Finger: < 2 sec.
  - ✓ Patella: < 5 sec.

Case presentation

- Arterial blood sampling was performed:
  - CRP 40 mg/l, PCT 24 µg/l, Creatinine 143 µg/l, Urea 10.4 mmol/l. GOT 89 U/L, GPT 58 U/L, Alk Phos 230 U/L, P-amylasis 45 U/L.
  - Hb 155 g/l, Hk 47%, Lc 1.2 G/L Tc 86 G/L, Quick 45%
  - Venous blood gas shows pH 7.39, HCO^{3-} 16 mmol/L, BE -5.2, lactate 3.2 mmol/L

Open questions

- What's the problem?
- Does my patient need an ICU?
  - If yes why?

Does my patient needs an ICU?

- **YES** → Sepsis, risk of death > 40% → transfer as soon as possible to your ICU

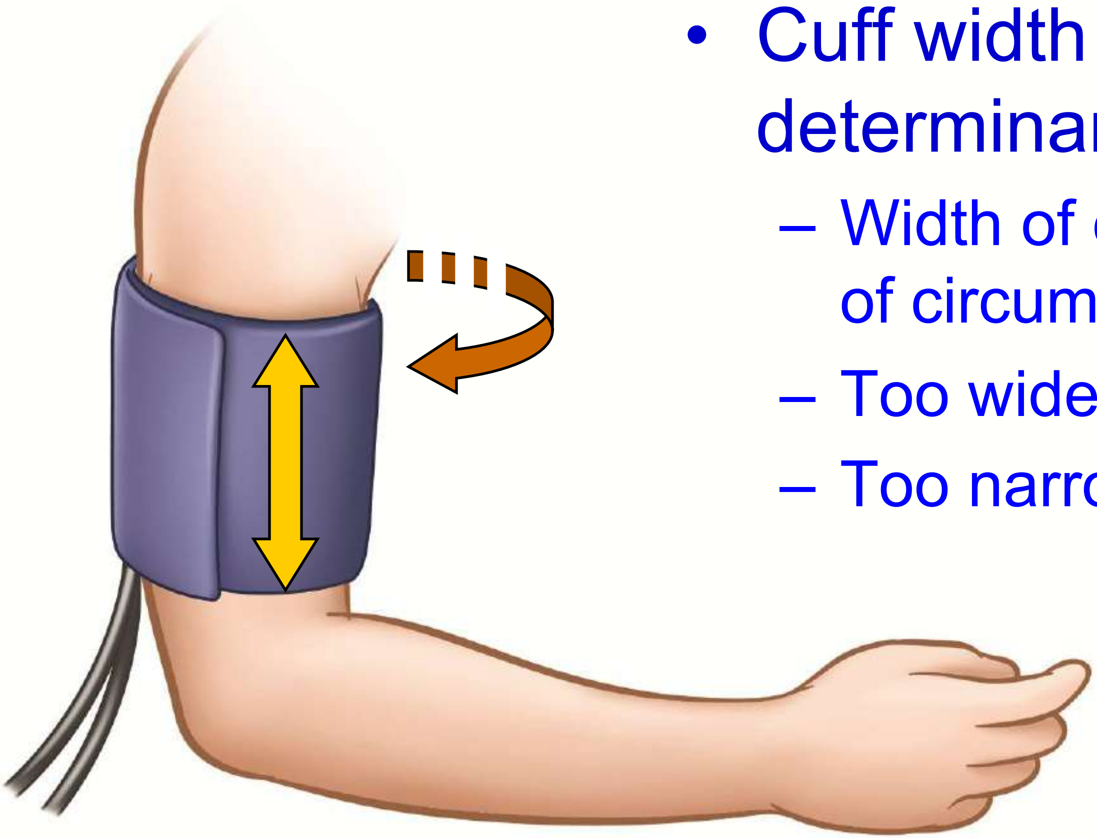
CAVE before transfer! Blood culture → AB treatment is started → Fluids

- Does my patient need a monitoring?
  - If yes, what a kind of monitoring does he need?
    - BP non-invasive vs. invasive?
    - Central venous line?
    - Urine catheter?
    - Advanced hemodynamic monitoring?

Measuring blood pressure

- Non-invasive automated devices
- Invasive measurements

Non-invasive BP



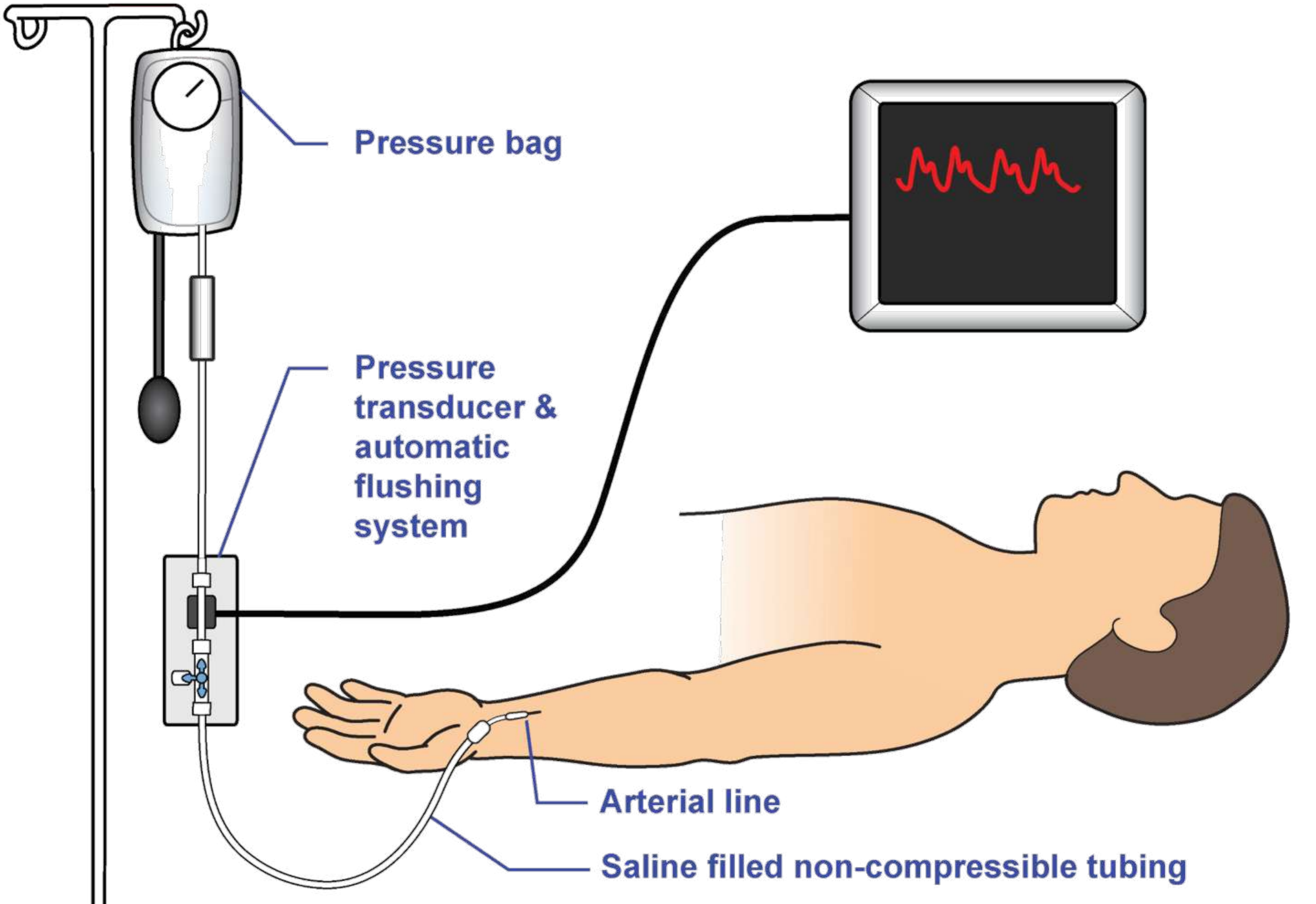
- Cuff width most important determinant of accuracy
  - Width of cuff should be ~40% of circumference of arm
  - Too wide ⇒ underestimate
  - Too narrow ⇒ overestimate

Non-invasive BP

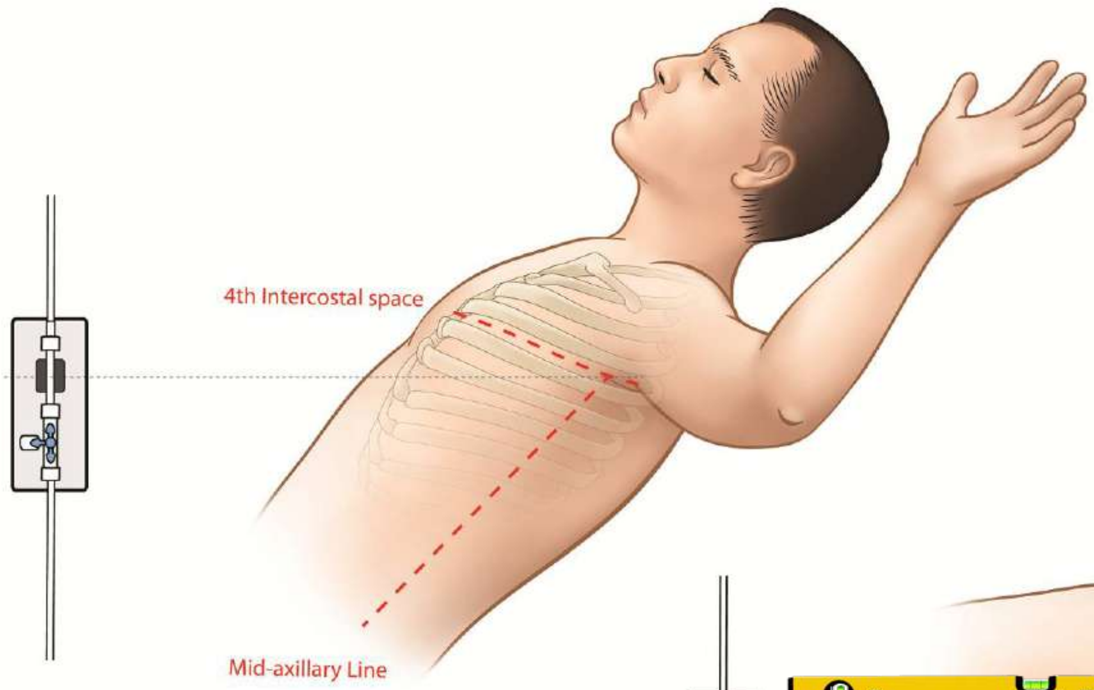
- Under-read at high pressures
- Over-read at low pressures
- Less accurate during arrhythmias

Arterial line

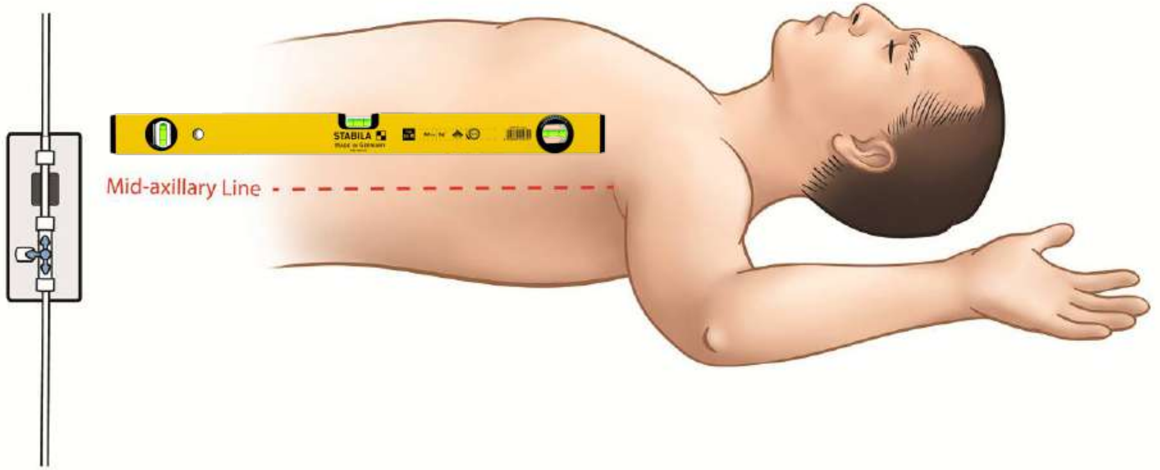
- Advantages
  - Continuous monitoring
  - Allows blood sampling
  - More accurate


Pressure bag
Pressure transducer & automatic flushing system
Arterial line
Saline filled non-compressible tubing

Transducer position



Zeroing

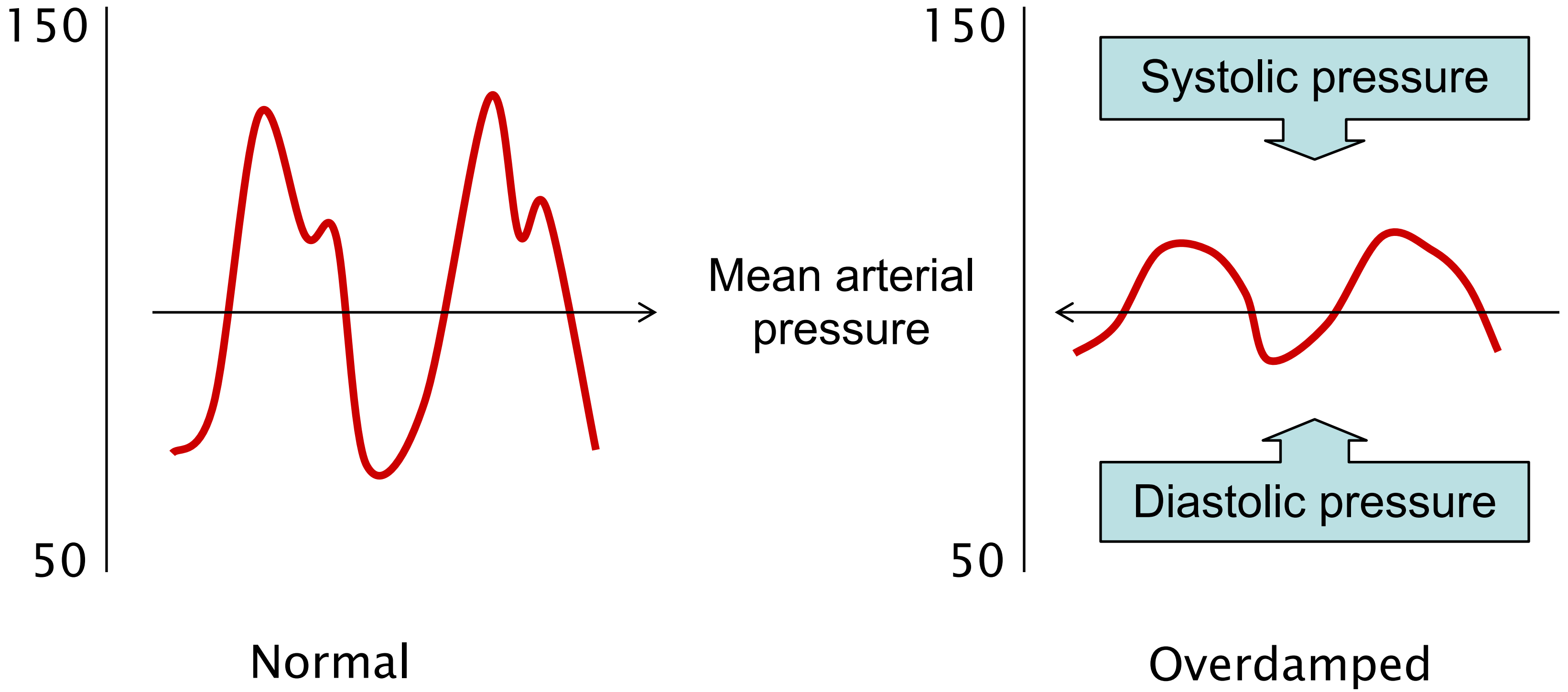


4th intercostal space
mid-axillary line

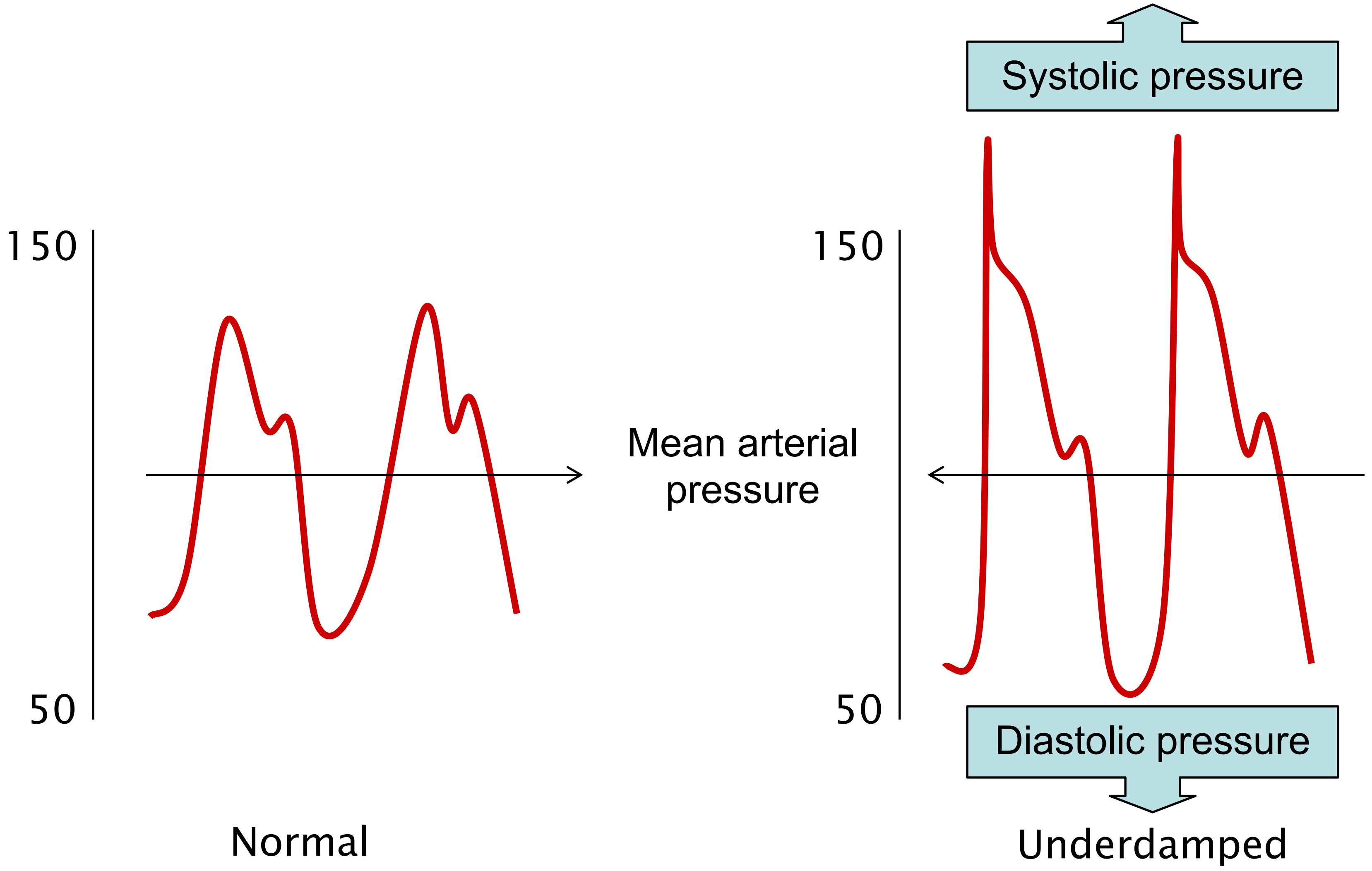
Sources of error

- Transducer position
- Zeroing
- Damping
  - Over-damping
  - Underdamping

Overdamping



Underdamped



Arterial line

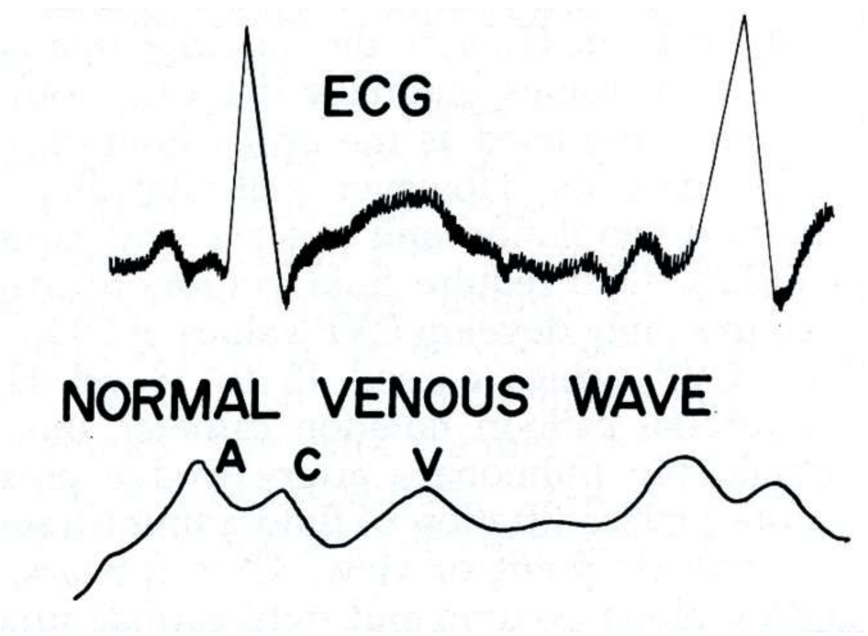
- Complications
  - Ischaemia
  - Thrombosis
  - Embolism
  - Infection
  - Haemorrhage
  - Accidental drug injection
  - Damage to artery

Which pressure?

- Systolic pressure
  - Bleeding risk, arterial wall stress
- Diastolic pressure
  - Peripheral vascular tone
  - Perfusion of left ventricle
- Mean pressure
  - Perfusion of most other organs

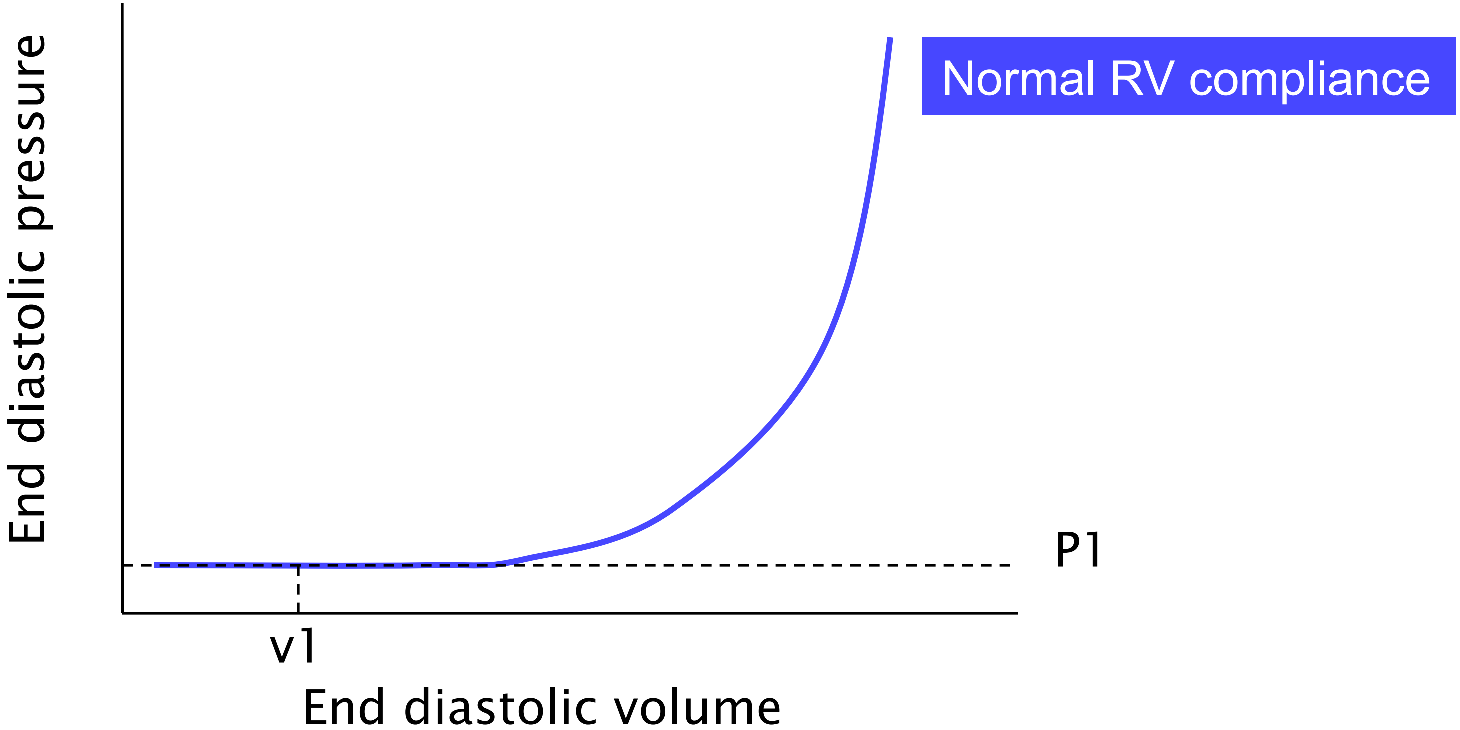
Central venous pressure

- Central venous pressure is determined by
  - right ventricular end-diastolic compliance
  - Right ventricular filling

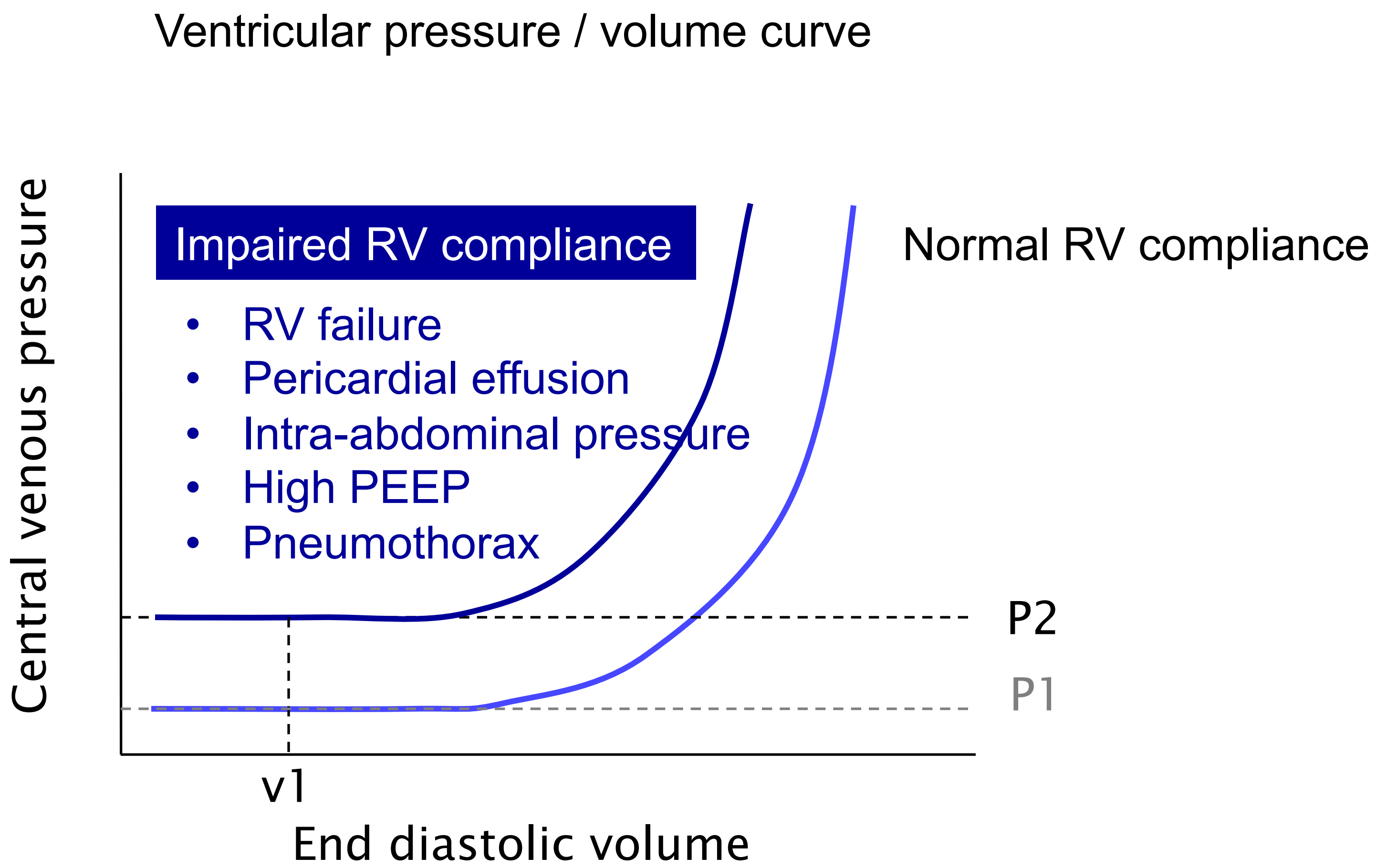


CVP interpretation

Ventricular pressure / volume curve

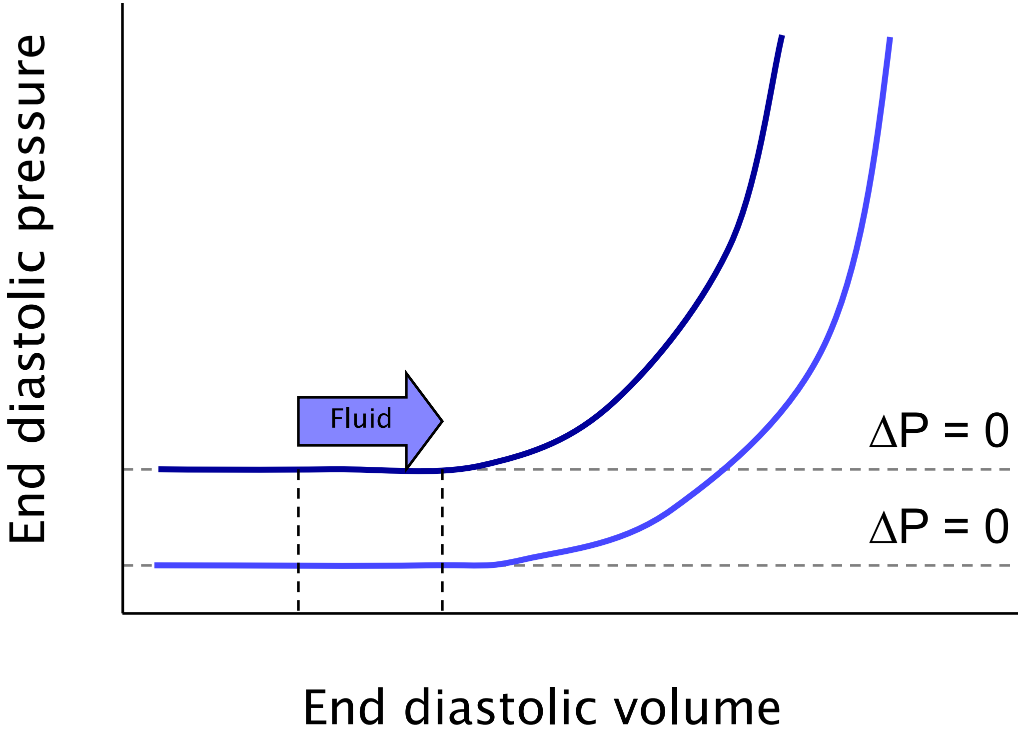


CVP interpretation



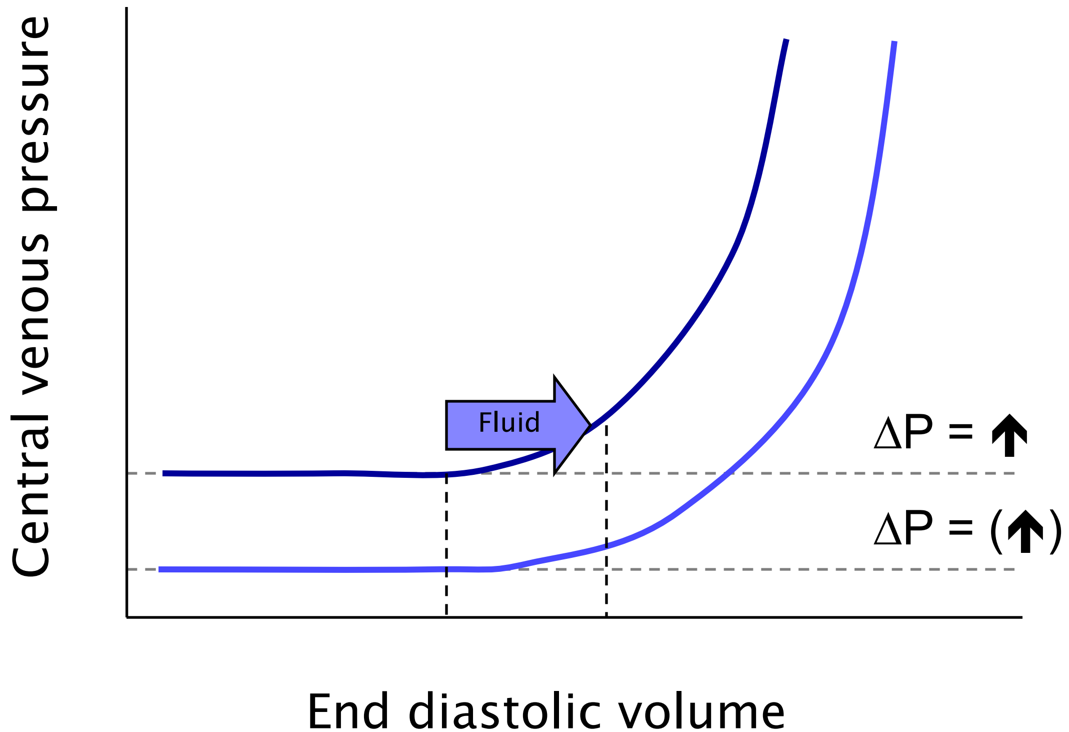
CVP interpretation

Ventricular pressure / volume curve



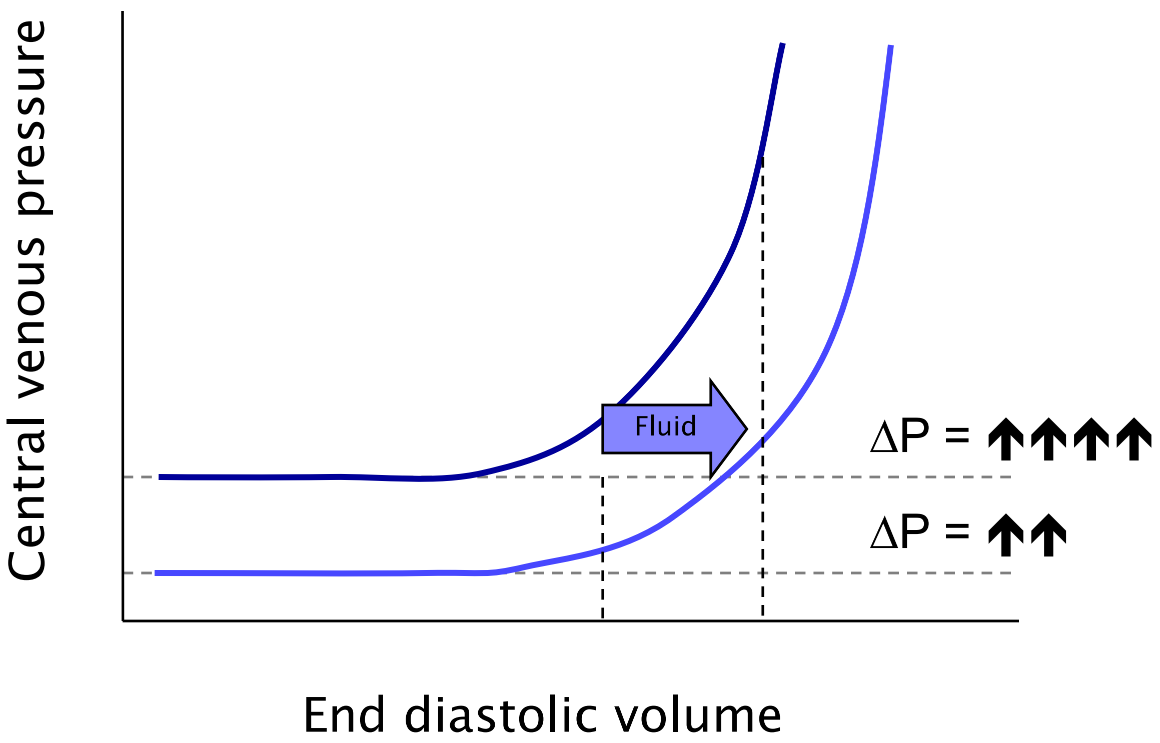
CVP interpretation

Ventricular pressure / volume curve



CVP interpretation

Ventricular pressure / volume curve

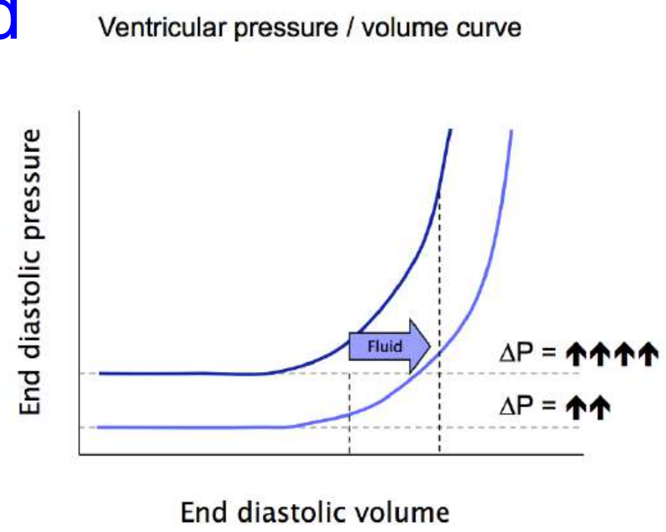


Rule of thumb

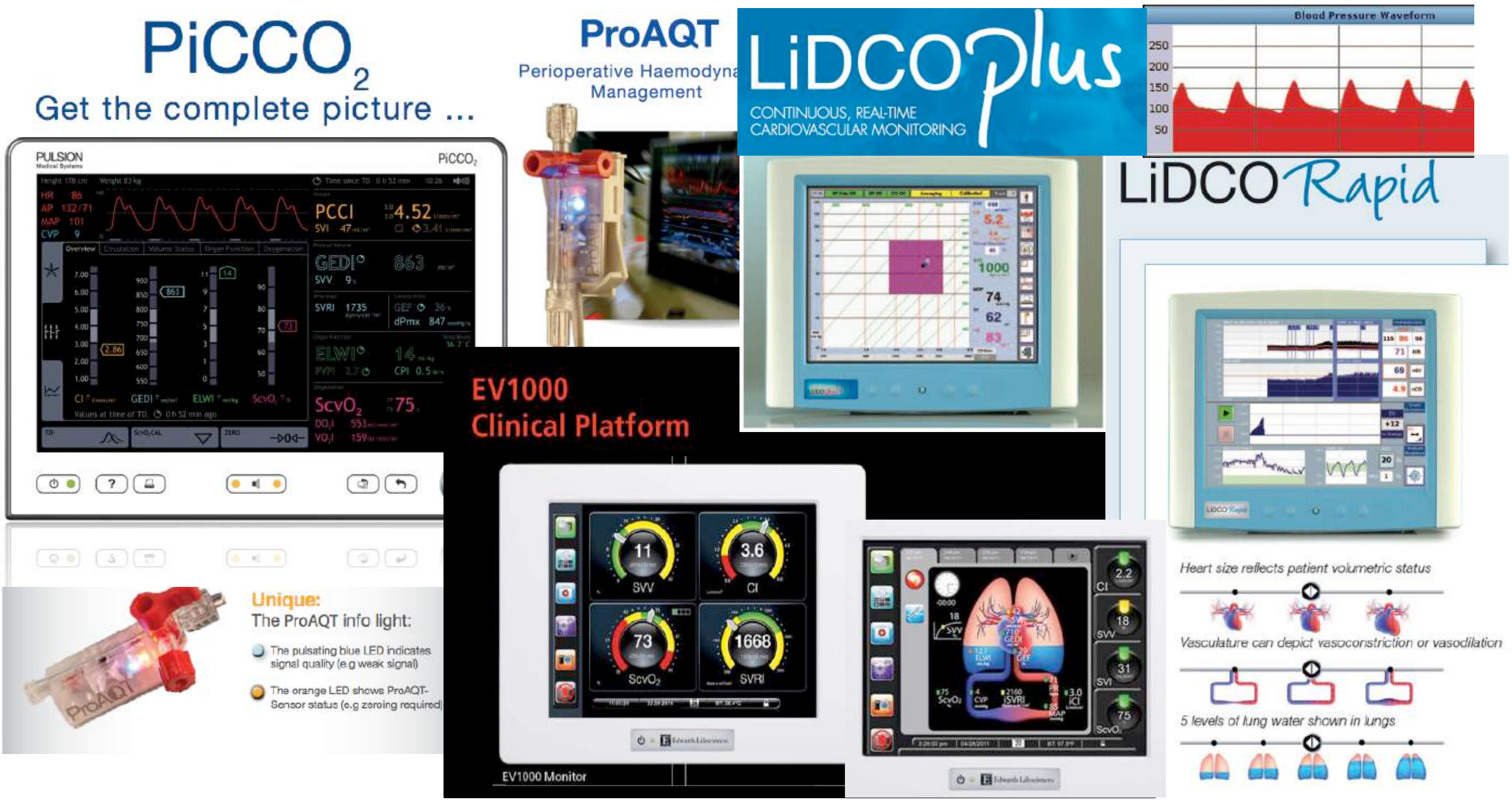
- Change in CVP measured before and 5 mins after bolus of fluid
  - 0-3 mmHg: underfilled
  - 3-5 mmHg: adequately filled
  - 5-7 mmHg: overfilled

CAVE

Poor marker of preload!



Advanced hemodynamic monitoring



Switch to other presentation

PPV Limitations

- Irregular cardiac rhythm (i.e. Atrial fibrillation)
  - Variable stroke volume
  - Variable pulse pressure
- Spontaneous breathing
  - Intrathoracic pressure changes difficult to predict
    - All breaths must be control breaths

PPV Limitations

- Low V_T
  - Smaller change in intrathoracic pressure ⇒ smaller variation in pulse pressure
  - If PPV <10% and $V_T \leq 6$ ml/kg PBW
    - Increase V_T to 8 ml/kg temporarily to assess PPV
- Low lung compliance
  - Stiff lung as it is the case in severe ARDS (compliance < 30 cmH_2O)

Summary

- Clinical monitoring by nurse and doctor are the most important
- Is there and what is the problem?
  - Define you working diagnosis
  - Define you treatment goals
- Which monitoring do I need?
  - Clinical monitoring
  - Basic monitoring: ECG, arterial pressure, (CV-line for the measurement of: CVP, ScvO2)
  - Advanced hemodynamic monitoring: continous pulse analysis for PPV, transpulmonary thermodilution for CO

Any questions?

Shock

Continuing development of BASIC is supported by an unrestricted educational grant from

MAQUET   Dräger medical
A Dräger and Siemens Company

Shock

- State in which there is inadequate oxygen delivery to the tissues to meet demand

$$DO_2 = CO \times CaO_2$$

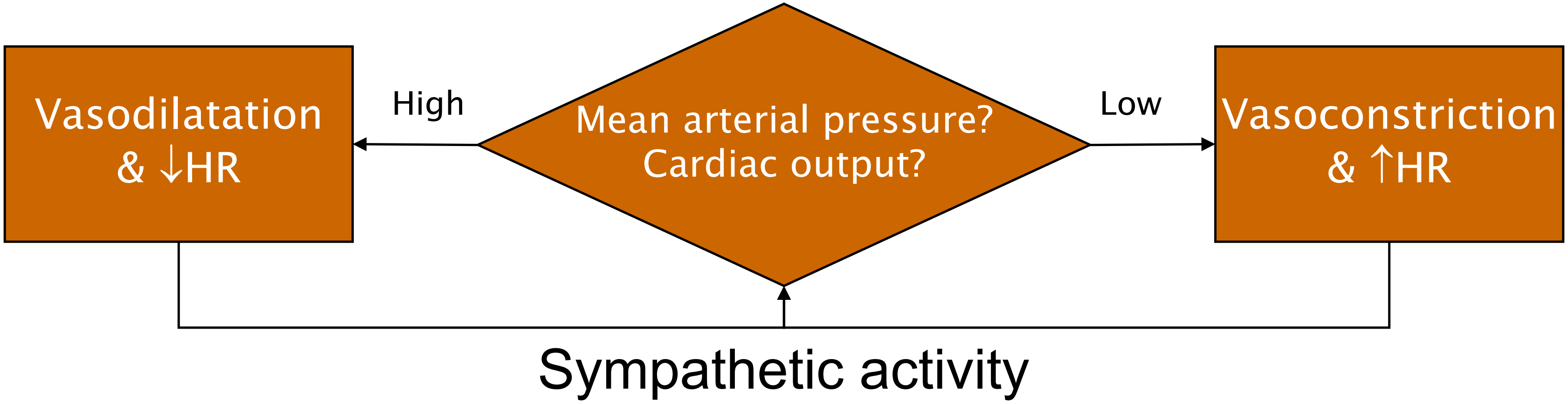
DO_2 = oxygen delivery; CO = cardiac output; CaO_2 = arterial oxygen content

Shock

- Initial assessment
  - Focused clinical examination (CNS, Heart, Lung, Skin, Abdomen)
  - Vital signs (BP, HR)
  - Oxygenation status (SpO_2 / FiO_2)
  - Metabolic status (venous or arterial ABG)

CAVE: Hypotension need not be present

Hypotension does not need to be present!



Hypotension = Failure of compensation

⇒ Very severe disease

Shock: Definition

- Hypoperfusion despite adequate fluid resucitation
  - BP ≤ or ≥ 90
  - Tachycardia
  - Tachypnoea (low $PaCO_2$)
  - Mottled skin
  - Centralization
  - Lactic acidosis
  - Oligo-anuria

Case A

- Our clinical case: 45 years old male
  - BP 70/50/35 mmHg
  - SaO_2 95% $ScvO_2$ 57%
  - Mottled skin
  - $PaCO_2$ 2.7 kPa
  - Lactate 6.[illegible] mmol/l
  - pH 7.25
  - Urine output = 10 ml/h

Mean BP = 50 mmHg
What does is mean?

Global haemodynamic relationships

$$MAP = CO \times TPR$$

Global haemodynamic relationships

Peripheral perfusion

$$MAP = CO \times TPR$$

Global haemodynamic relationships

Peripheral perfusion

$$MAP = CO \times TPR$$

$$HR \times SV$$

Global haemodynamic relationships

$$MAP = CO \times TPR$$

$$HR \times SV$$

Fluid respon-sivness

Preload Afterload Contractility

Case A

- Differential diagnosis
  - Septic shock (distributive)
  - Cardiogenic shock
  - Obstructive shock due to pulmonary embolus
  - Hypovolemic shock

Clinical presentation of shock

	HR	JVP or CVP	Peripheries
Cardiac	↑ or ↓↓ or ↑↑	↑	Cold
Hypovolaemic	↑	↓	Cold
Distributive	↑	↓-N	Warm
Obstructive*	↑	↑↑	Cold

* Obstructive shock due to cardiac tamponade, tension pneumothorax or massive PE

Case A

- Differential diagnosis
  - Septic shock (distributive)
  - Cardiogenic shock
  - Obstructive shock due to pulmonary embolus
  - Hypovolemic shock
- **What next?**

Case A

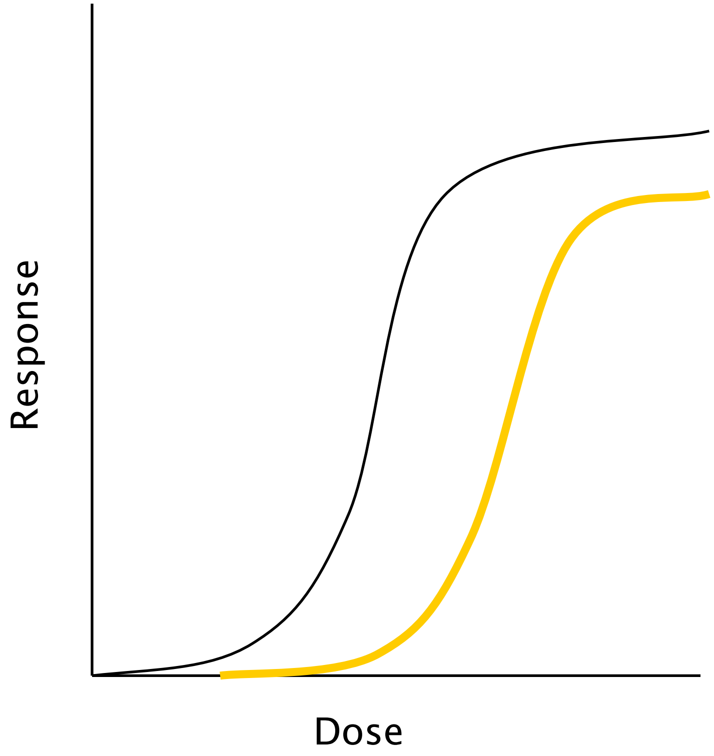
- Start fluid infusion
- Give oxygen
  – Oxygenation status unclear at the moment
- Investigations
  – ECG
  – Echocardiography
  – Biochemistry, complete blood count

Immediate resuscitation of the shocked patient

- Immediately life threatening hypotension
  - Norepinephrine boluses
  - Vasopressor infusion
  - Fluids

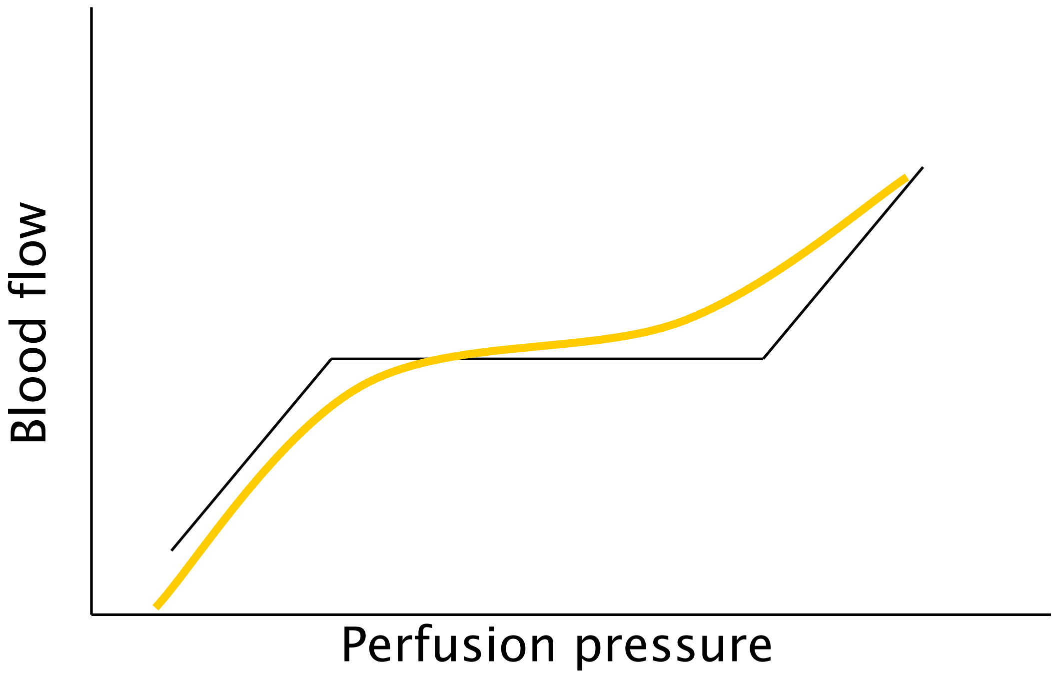
How much?

- Altered dose-response curve in critically ill, particularly sepsis
  - Excessive vasodilation
  - Metabolic acidosis



Blood Pressure

- Autoregulation of organ perfusion pressure

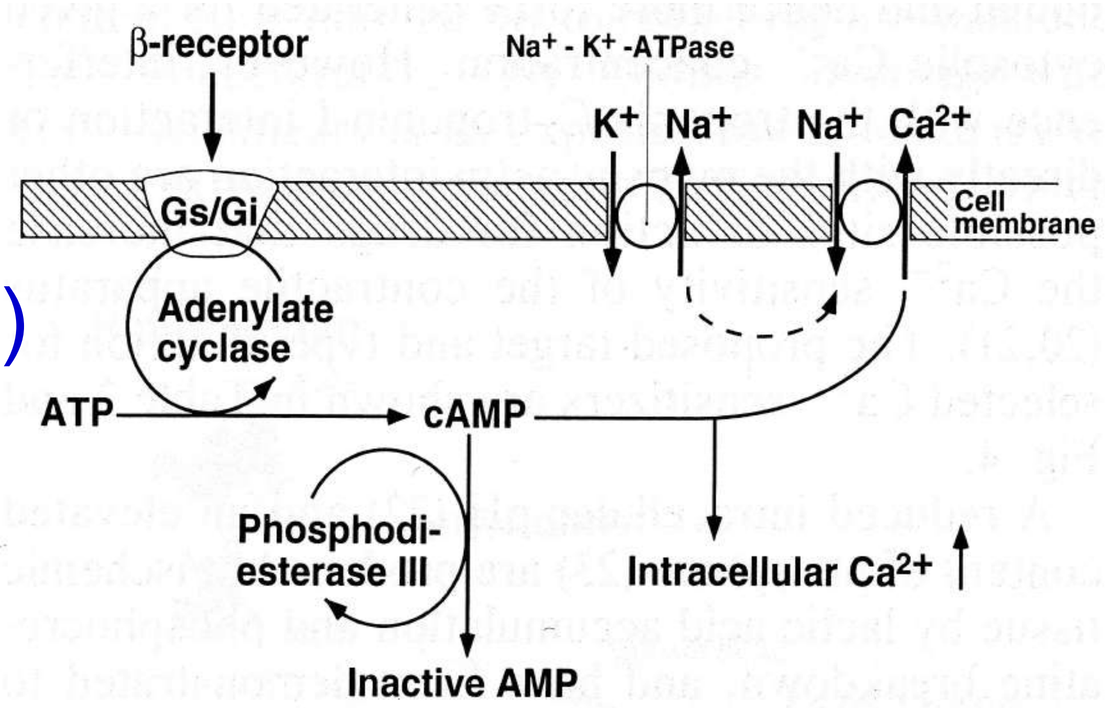


What if fluids are not enough?

- Inadequate cardiac output (SV * HR)
  - Inotrope
- Inadequate blood pressure
  - Vasopressor

Dobutamine

- Inotrope (β_1)
- Vasodilator (β_2)
- Positive chronotrope (β_1)



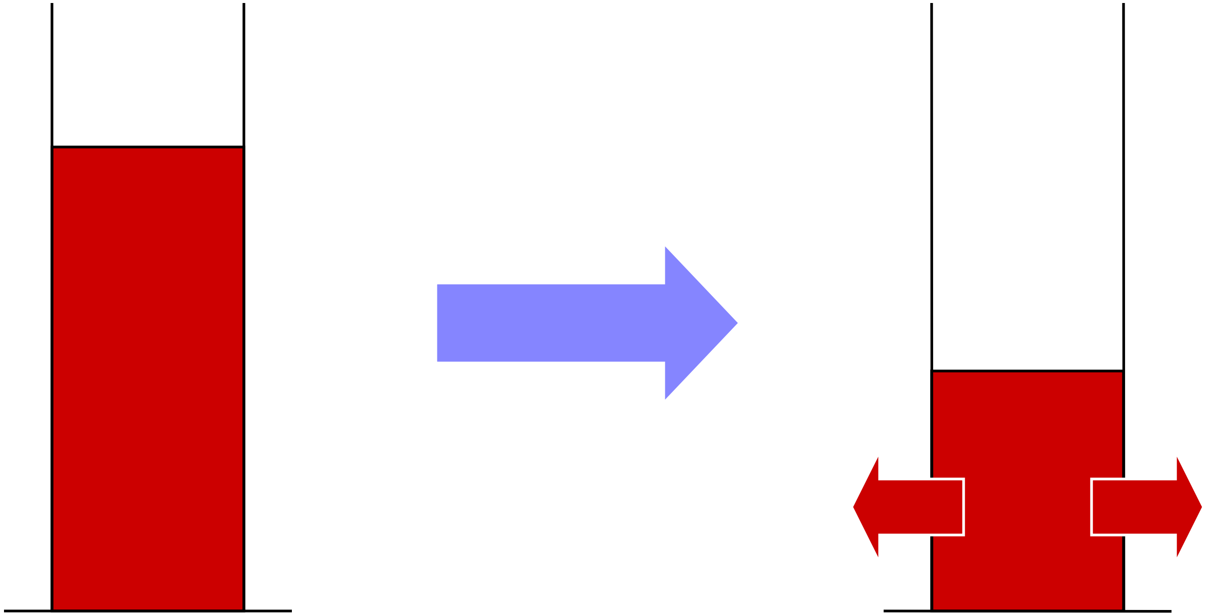
Norepinephrine

- Vasopressor (α)
- Increases cardiac output in hypotensive patients
  - Increases venous return to the heart
  - Increases coronary perfusion and hence cardiac function

Mechanisms of Shock

- Excessive loss of fluids
- Sepsis
- Cardiac failure
- Obstruction to blood flow

Hypovolaemic

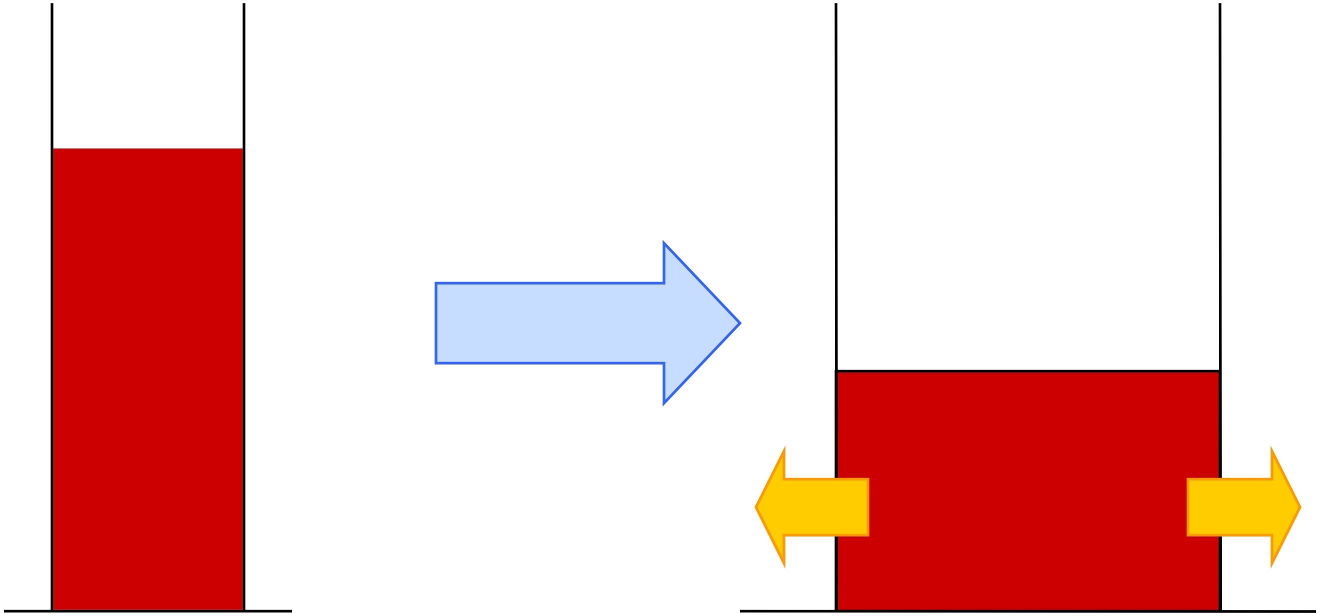


Hypovolaemic: Treatment

- Fluid resuscitation
  – Crystalloids, colloids
  – Blood products
- Vasopressors
  – Life threatening hypotension
  – Distributive shock phase
- Treat underlying cause

Septic

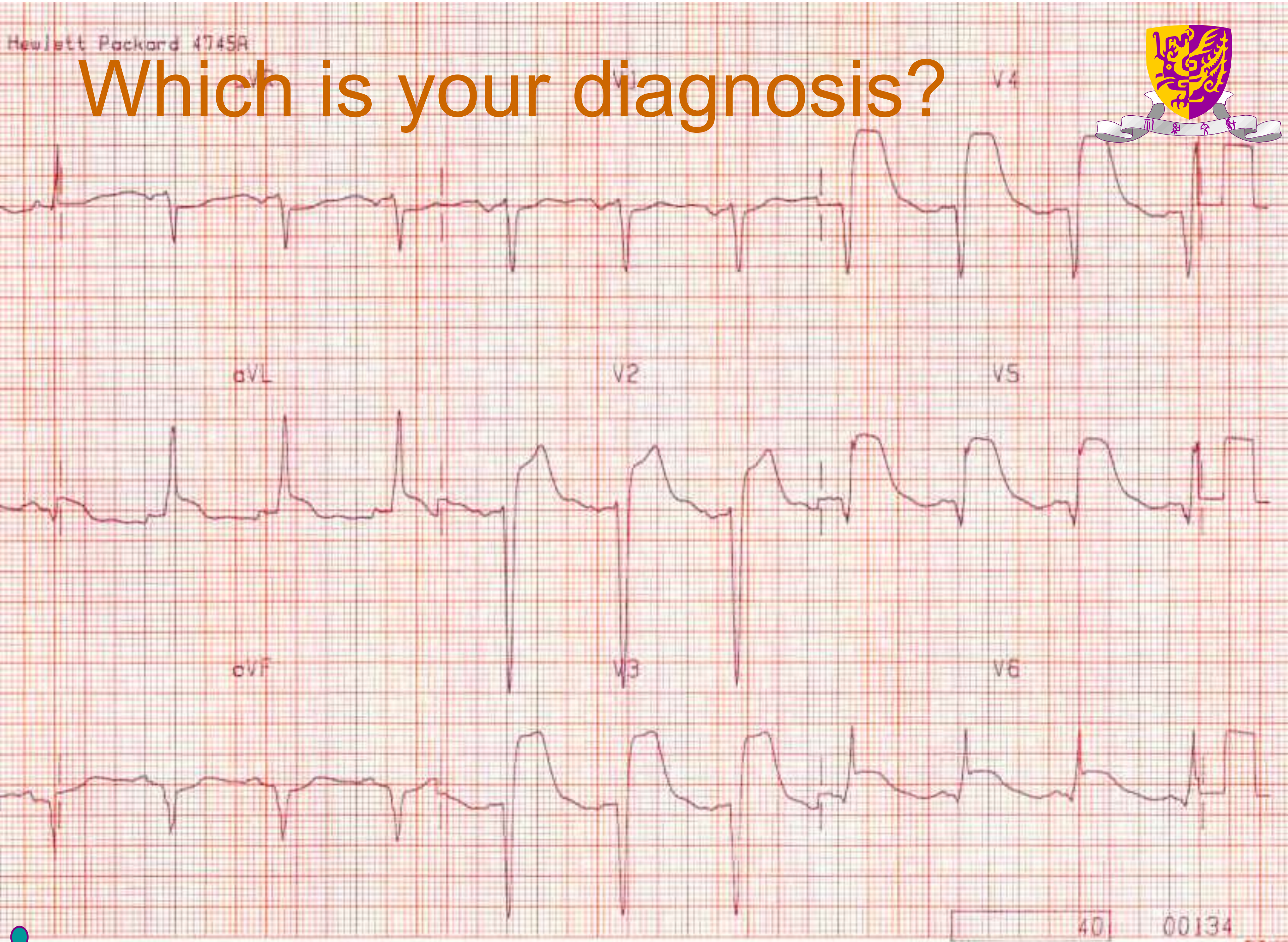
- Pathophysiology
  - ✓ Vasodilatation
  - ✓ Capillary leak
  - ✓ Cardiomyopathy



Septic: Treatment

- Norepinephrine or epinephrine (or dopamine)
- Dobutamine
  - Tissue hypoperfusion despite normotension
- Blood cultures
- Antibiotics (within 1 hours of diagnosis!)
- Source control

Which is your diagnosis?



Cardiogenic

- Coronary perfusion to LV dependent on diastolic blood pressure
- LV function dependent on coronary perfusion
- Tachycardia decreases duration of diastole, hence stroke volume

Cardiogenic

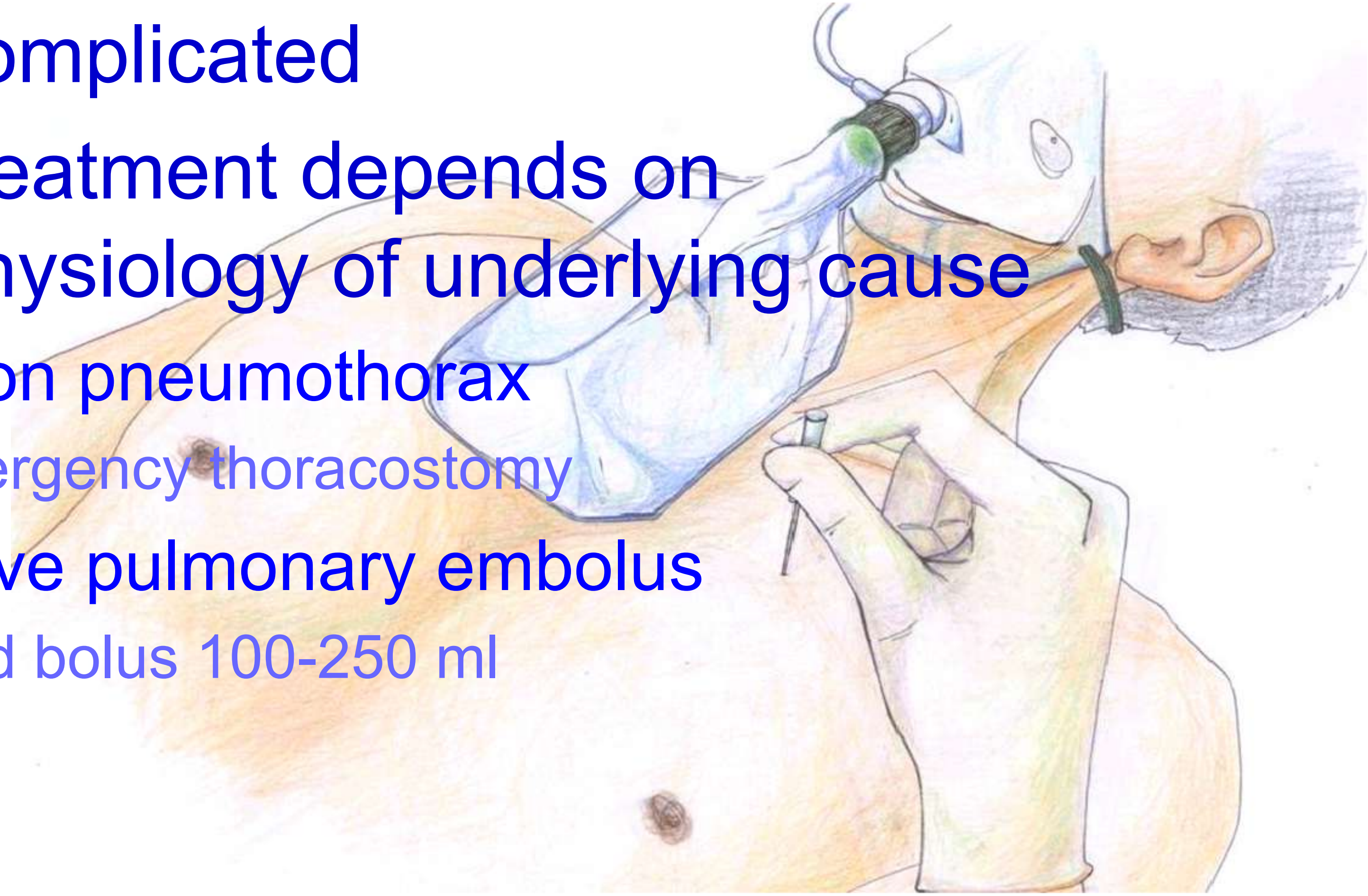
- Early referral
  - Revascularization
  - Inotropic drugs
  - LV assist devices
- Treat cause

Cardiogenic: Treatment

- Aims:
  - Adequate diastolic pressure without tachycardia
  - Increase cardiac output
- Normotensive patient with poor peripheral perfusion
  - Dobutamine infusion
- Hypotensive patient
  - Dobutamine infusion
  - Norepinephrine infusion

Obstructive

- More complicated
- Initial treatment depends on pathophysiology of underlying cause
  - Tension pneumothorax
    - Emergency thoracostomy
  - Massive pulmonary embolus
    - Fluid bolus 100-250 ml



Obstructive: Treatment

- Complex, call for help
- Early relief of obstruction
- Dobutamine-induced vasodilatation

  $\Rightarrow$ severe hypotension due to relatively fixed cardiac output
- Norepinephrine probably drug of choice for initial management

Summary

- Cardiovascular assessment
  - Assess tissue perfusion
    - Vital signs
    - Conscious state
    - Temperature of limbs
    - Skin perfusion
    - Urine output
    - pH
    - Lactate

Summary

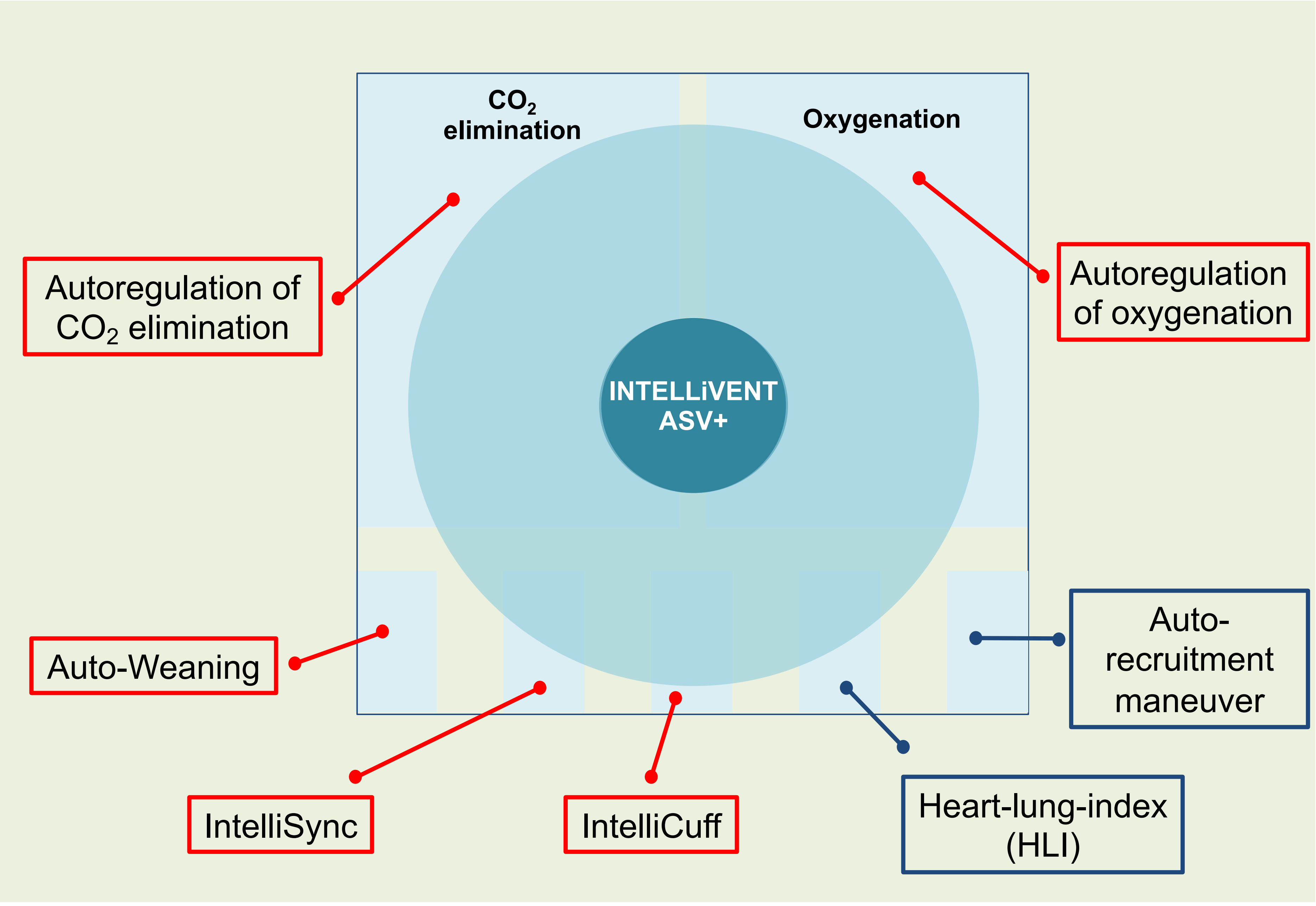
- Resuscitation
  - Fluid
  - Vasopressor to restore BP
  - Inotrope to increase cardiac output
  - Titrate against patient response

- Treat underlying cause

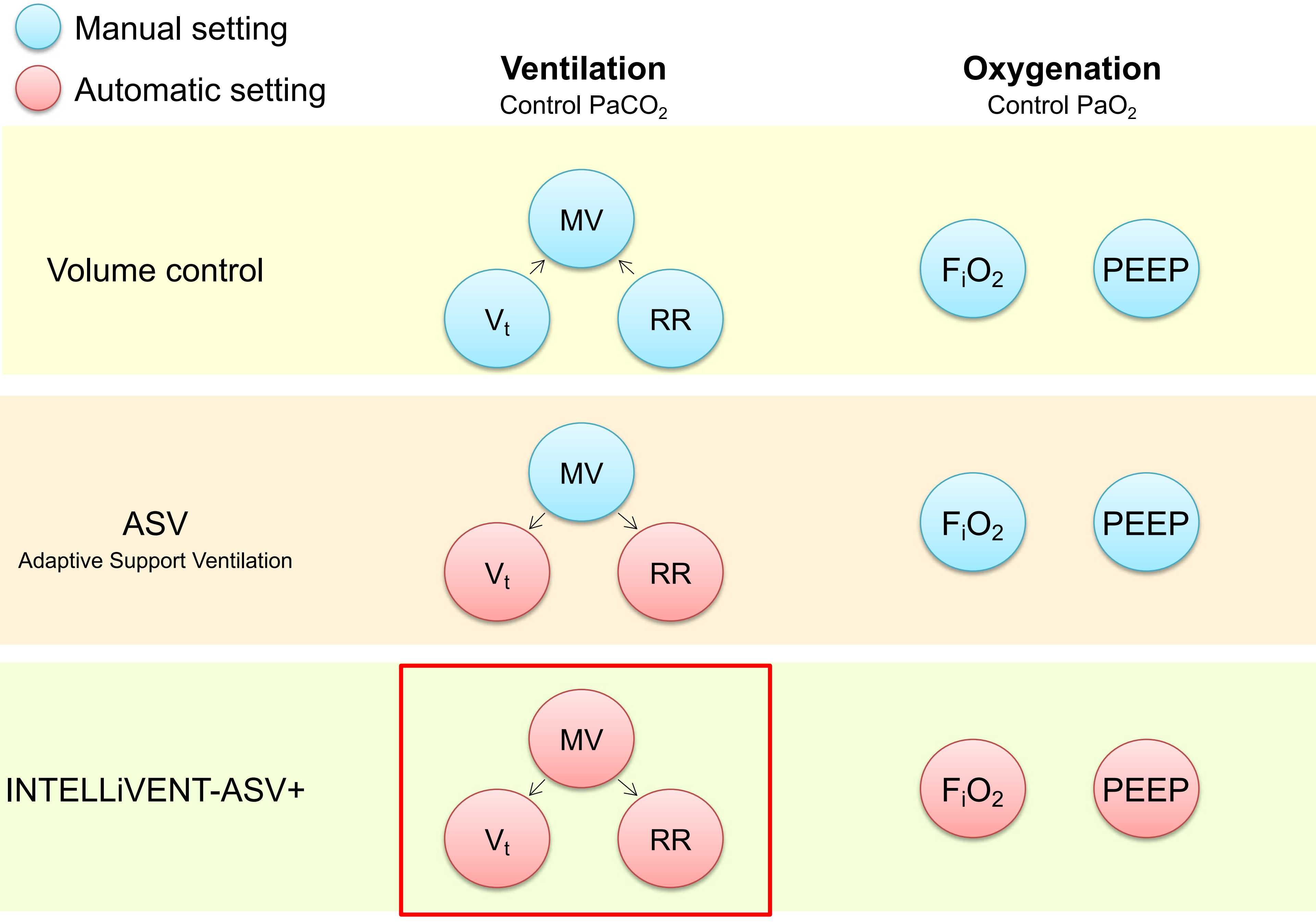
Any questions?

Einführung Beatmung

INTELLiVENT-ASV+



ASV und INTELLiVENT-ASV+



INTELLiVENT-ASV+

Ventilation
Control $PaCO_2$

INTELLiVENT-ASV+

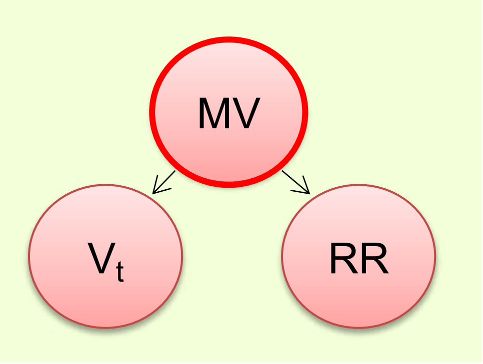
Druckkontrollierte Beatmungsform

→ Druckunterstützte Beatmungsform

Ein vordefiniertes MV wird appliziert....

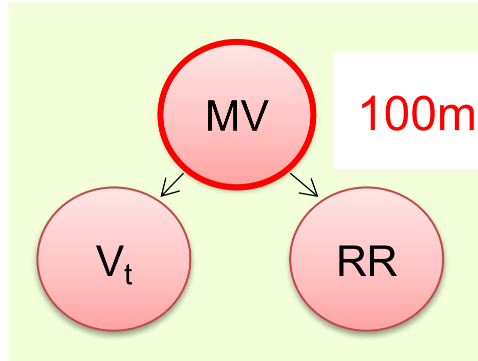
...unter Einhaltung von Lungenschutzparametern....

...eine Spontanatmung ist jederzeit möglich.



Ventilation
Control $PaCO_2$

INTELLiVENT-ASV+



100ml/kg KG = 100% MV

Ideales Körpergewicht		Normoventilation (ml/min)
Frauen: Körpergrösse (cm) - 100 - 10% Männer: Körpergrösse (cm) - 100 - 5%	→	100ml/kg KG
180cm grosse Frau Ideales Körpergewicht: 180cm-100-0.1x(180cm-100cm) =72kg	→	100%Minutenvolumen: 100ml x 72kg = 7200ml/min = 7.2L/min

INTELLiVENT-ASV+

Ventilation
Control $PaCO_2$

Otis A. Mechanics of Breathing in Man. J Appl Physiol 1950

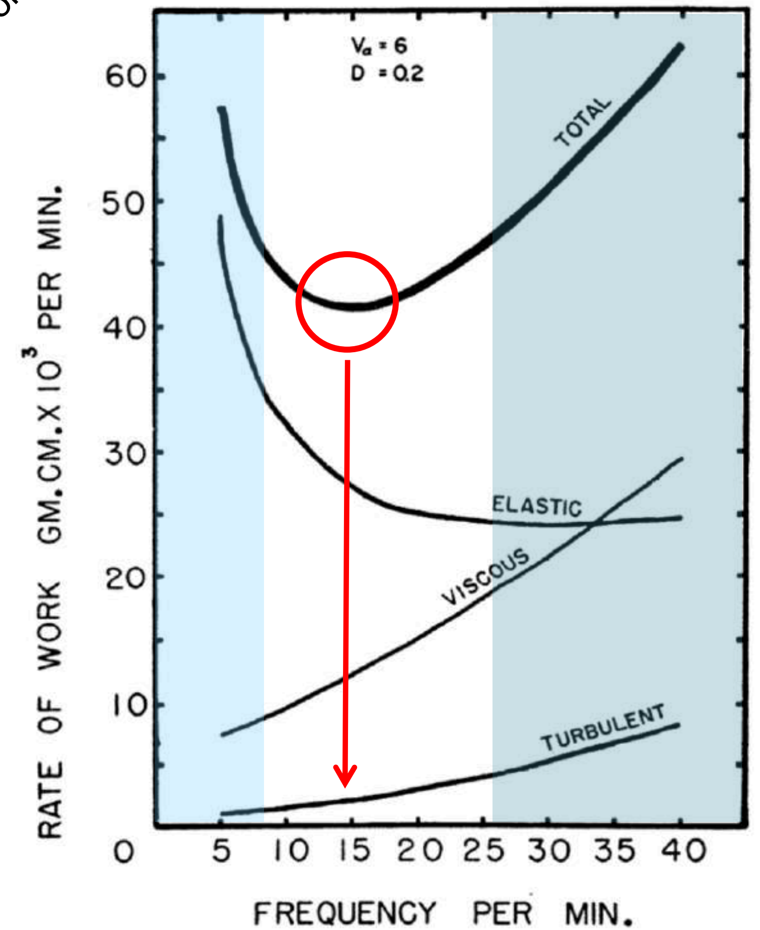
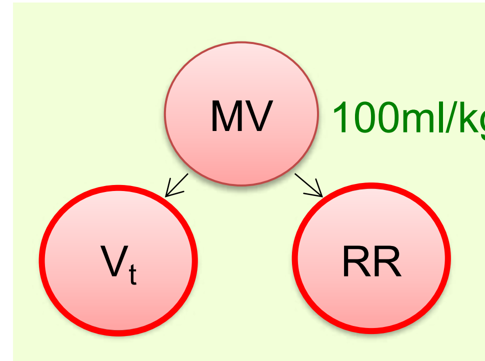


Fig. 7. Relationship of elastic, viscous, turbulent, and total work of breathing/min. to frequency of breathing when alveolar ventilation is 6 l/min., and dead space is 200 cc. Curves calculated according to *equation 13.*



100ml/kg KG = 100% MV

? ?

50x144ml ? ? 10x720ml

7.2L/min

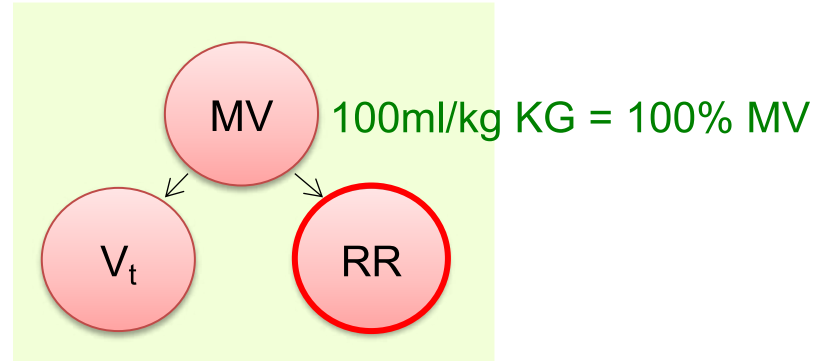
20x360ml ? ? 3x2400ml

Frequency too low: much elastic work is required to produce the tidal volume

Frequency too high: much work is uselessly done in ventilating the dead space with each breath

Ventilation
Control $PaCO_2$

INTELLiVENT-ASV+



$$f_{target} = \frac{\sqrt{1 + 2a \times RCe \times (MV - VD) / VD} - 1}{a \times RCe}$$

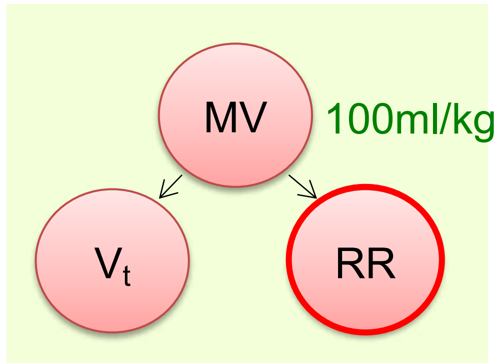
Otis Formel

f_{target} Ziel-AF für minimal Work of Breathing
a Faktor, welcher vom Flowmuster abhängt
MV Minutenvolumen
VD Totraumvolumen
RCe expiratorische Zeitkonstante

+ Mead Formel → f_{target} aus beiden Formeln wird gemittelt

Ventilation
Control $PaCO_2$

INTELLiVENT-ASV+



What‘s the idea behind?

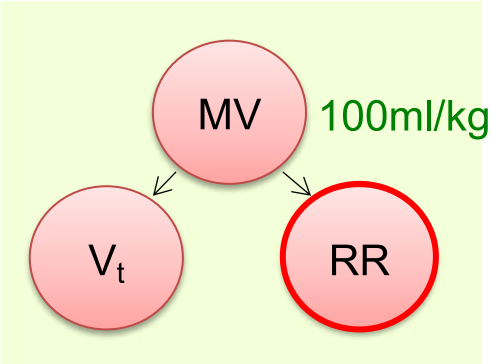
Mead und Otis-Formel

Spontan atmender Patient:
bei welcher Atemfrequenz ist die Atemarbeit am geringsten?

Kontrolliert beatmeter Patient:
bei welcher Atemfrequenz kann das vordefinierte Atemminutenvolumen mit dem geringstmöglichen Inspirationsdruck appliziert werden?

Ventilation
Control $PaCO_2$

INTELLiVENT-ASV+



100ml/kg KG = 100% MV

RCe (exspiratorische Zeitkonstante)

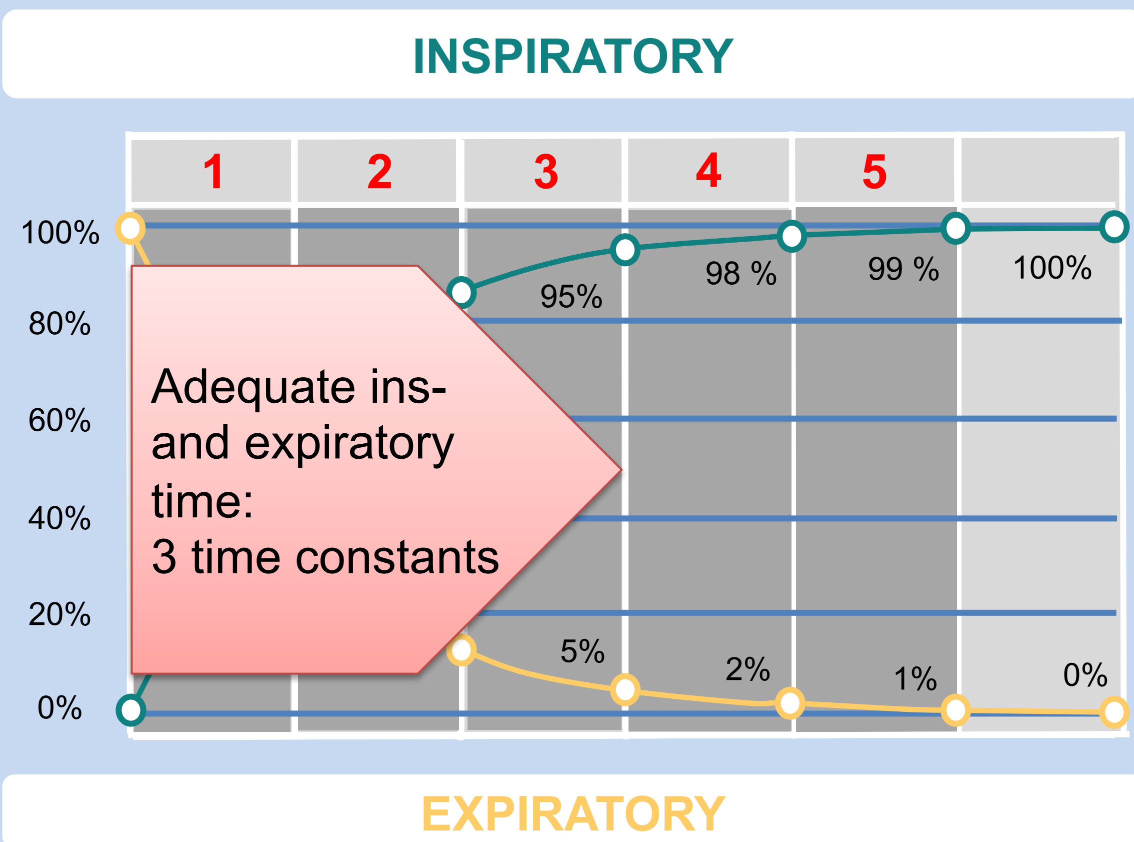
Die exspiratorische Zeitkonstante (RC) ist ein Mass für die Zeit, welche es für die Entleerung der Lunge braucht.

Sie ist ein Mass für die Compliance und die Resistance der Lunge als auch der Atemwege.

RESPIRATORY PHYSIOLOGY

TIME CONSTANT

Expressed in second. It does NOT directly indicate the length of time required to complete a flow course.



Time constant RC = Resistance (R) X Complicance (C)

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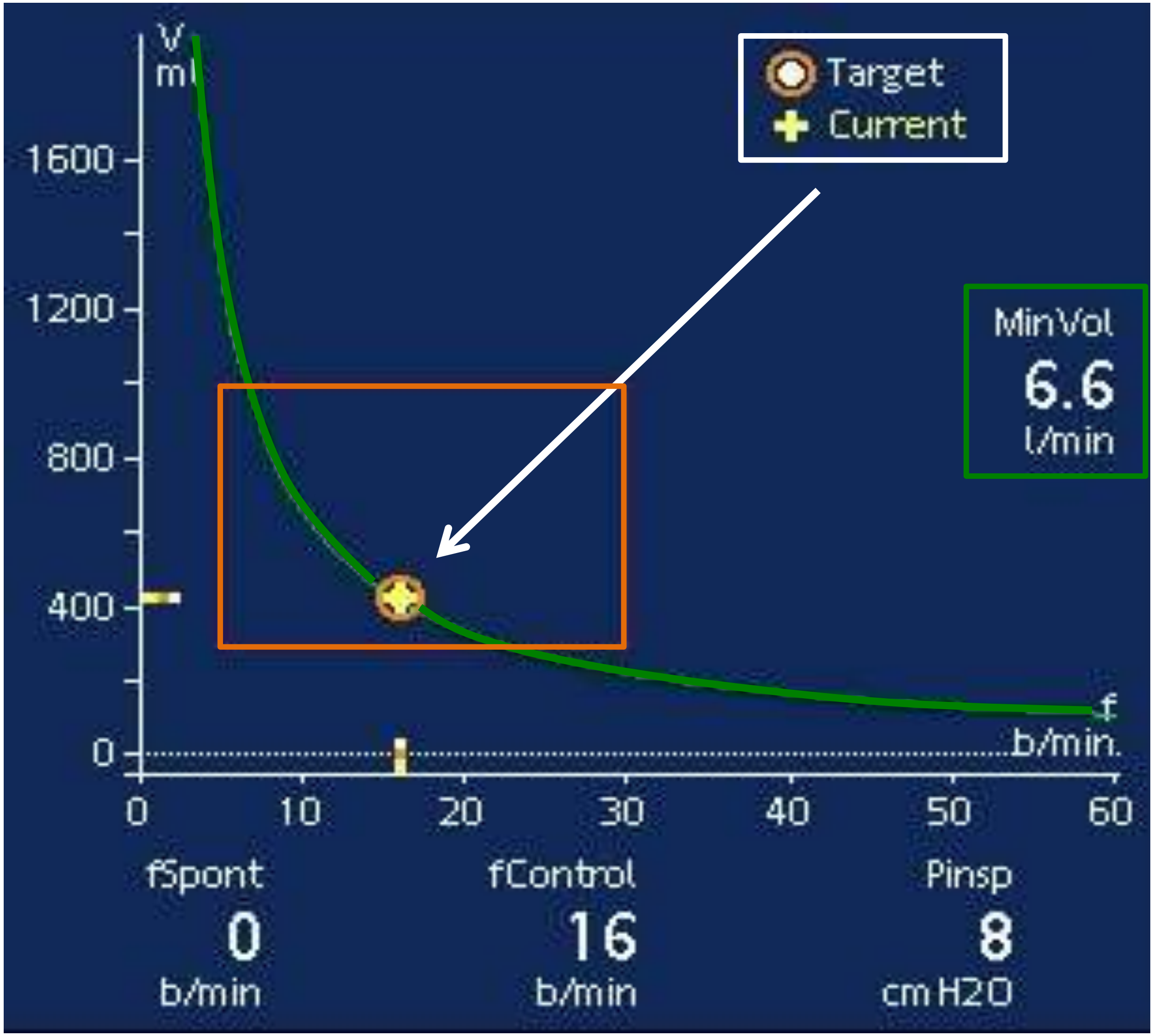
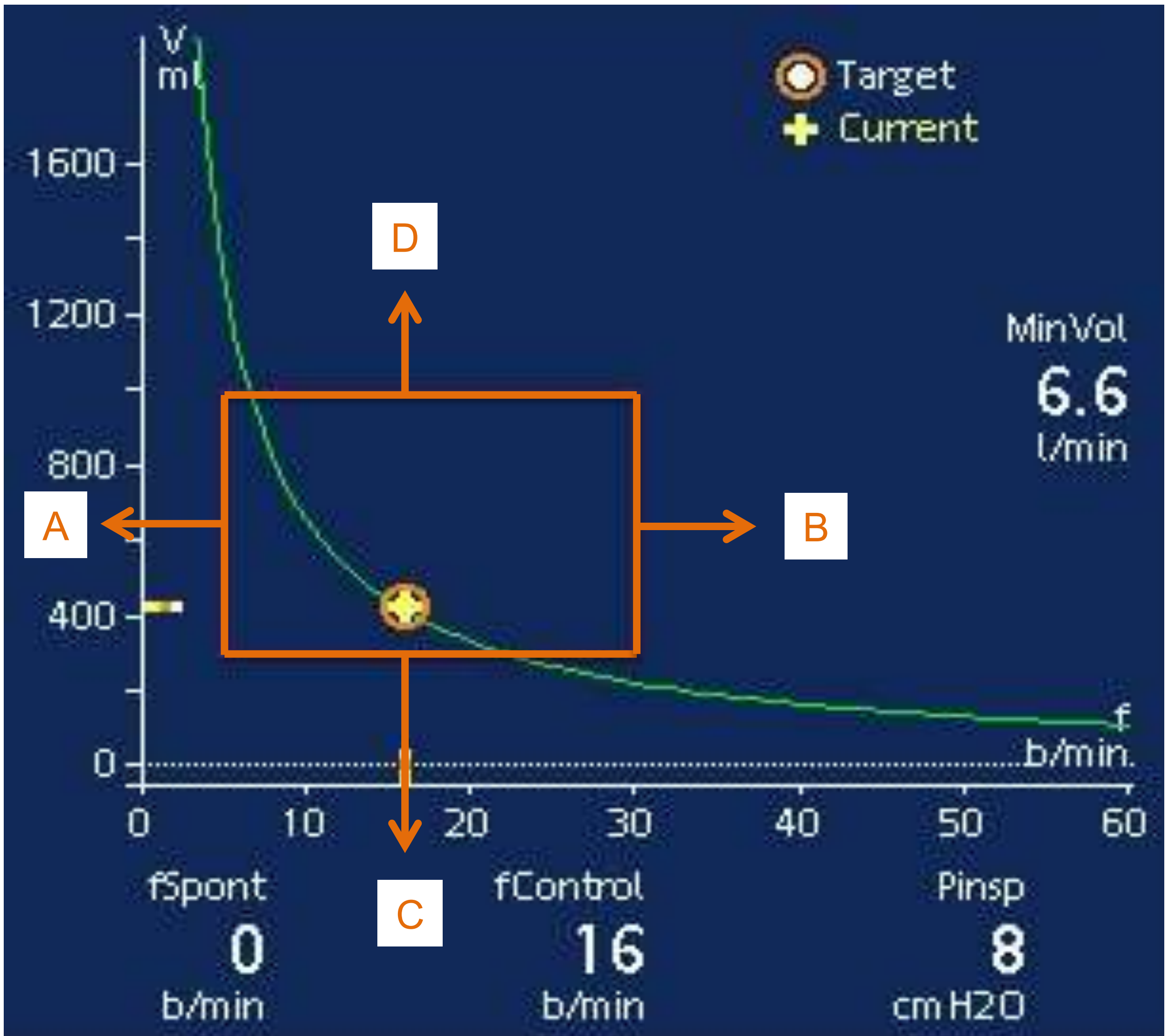
ASV Screen



Ventilation
Control $PaCO_2$

INTELLiVENT-ASV+

ASV Screen



<u>Lungenschutzregeln</u>

A Schutz vor Apnoe

B Vermeiden von dynamischer Hyper-inflation und Breath Stacking

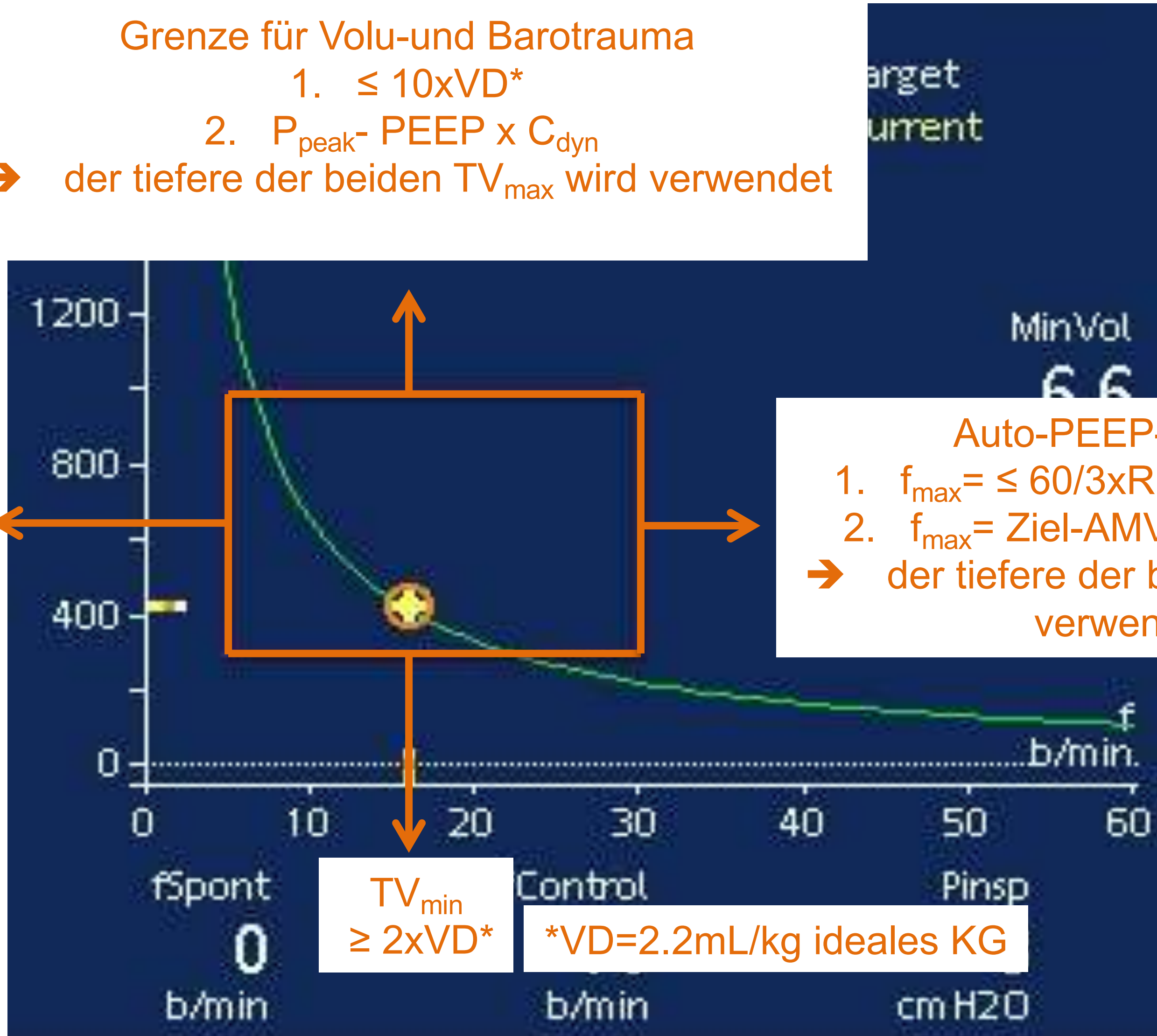
C Vermeiden von niederiger alveolarer Beatmung

D Vermeiden von zu hohen Tidalvolumina und Drücken

Ventilation
Control $PaCO_2$

INTELLiVENT-ASV+

ASV Screen Lungenschutzregeln



Grenze für Volu-und Barotrauma

1. ≤ 10xVD*
2. P_{peak}- PEEP x C_{dyn}

➔ der tiefere der beiden TV_{max} wird verwendet

Apnoe-Grenze
AF ≥ 5

Auto-PEEP-Grenze

1. f_{max}= ≤ 60/3xRC_{exp} = 20/Rc_{exp}
2. f_{max}= Ziel-AMV/minimales Vt

➔ der tiefere der beiden fmax wird verwendet

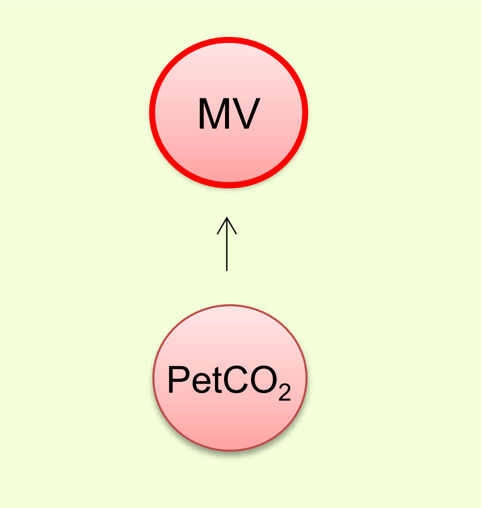
TV_{min}
≥ 2xVD*

*VD=2.2mL/kg ideales KG

Ventilation
Control $PaCO_2$

INTELLiVENT-ASV+

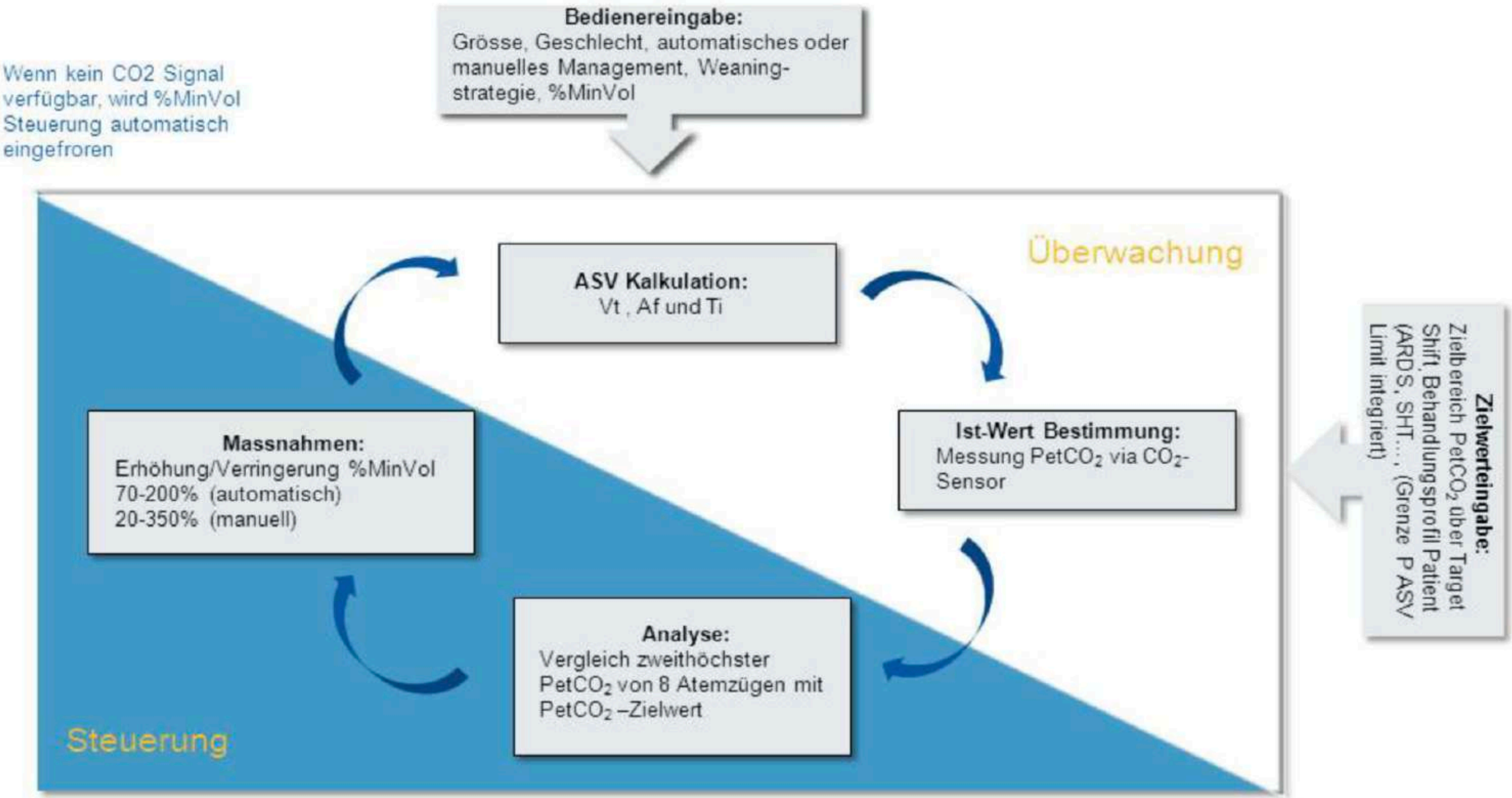
Minute Volume Adjustment in passive patients



Ventilation
Control $PaCO_2$

INTELLiVENT-ASV+

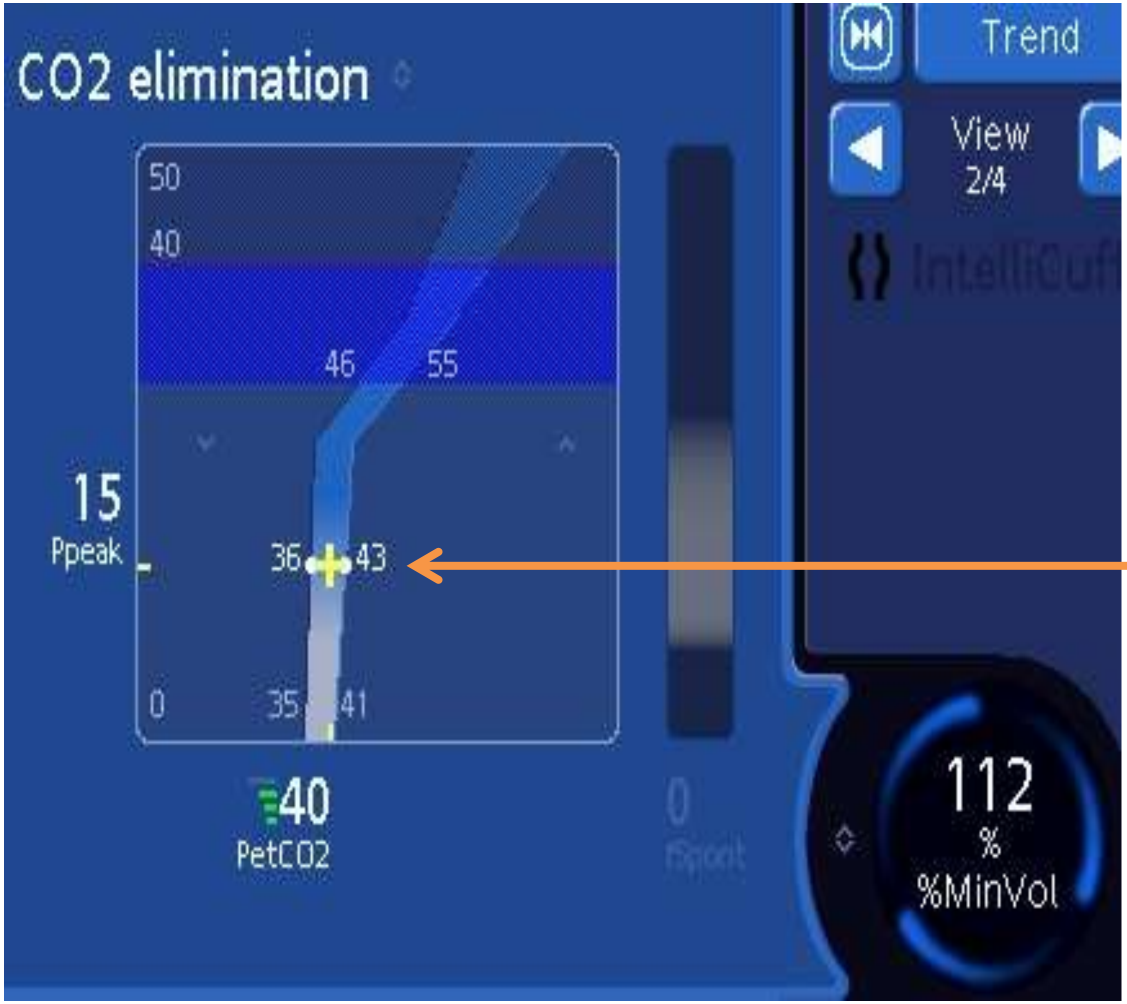
Minute Volume Adjustment in passive patients



Ventilation
Control $PaCO_2$

INTELLiVENT-ASV+

Minute Volume Adjustment in passive patients



$P_{ET}CO_2$ in target range:
Treatment kept constant

➡ Fine %MV adjustment to target the middle of range

Ventilation
Control $PaCO_2$

INTELLiVENT-ASV+

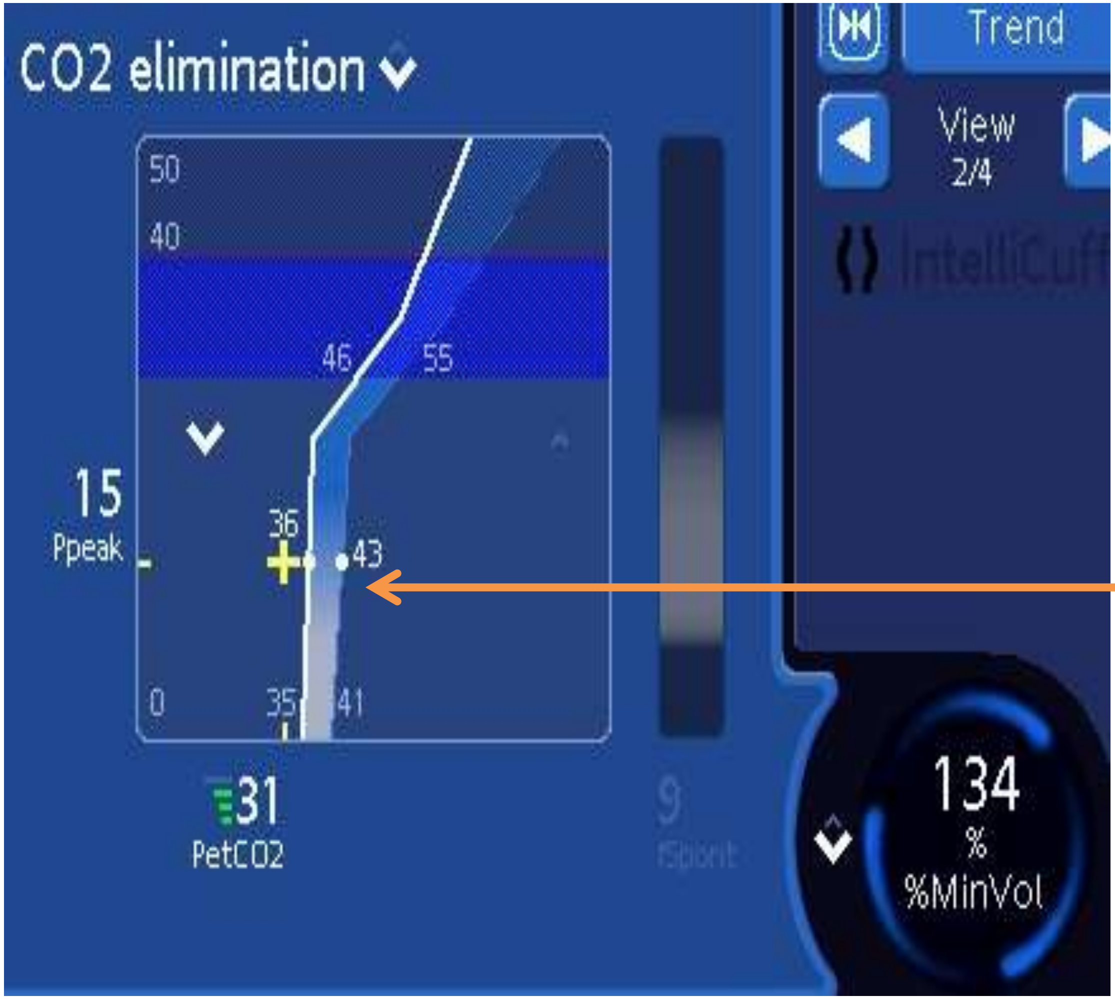
Minute Volume Adjustment in passive patients

$P_{ET}CO_2$ above target range:
Increasing target MV

Breath by breath %MV increase of 1% per breath

INTELLiVENT-ASV+

Minute Volume Adjustment in passive patients

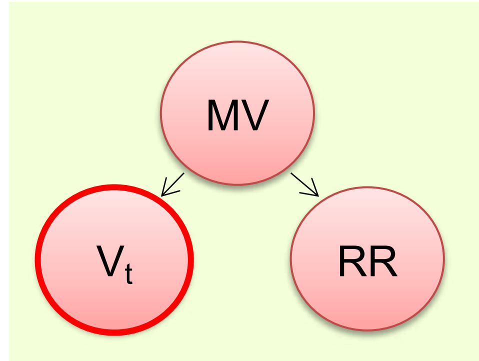


$P_{ET}CO_2$ below target range:
Decreasing target MV

Breath by breath %MV decrease of 1% per breath

Ventilation
Control $PaCO_2$

INTELLiVENT-ASV+

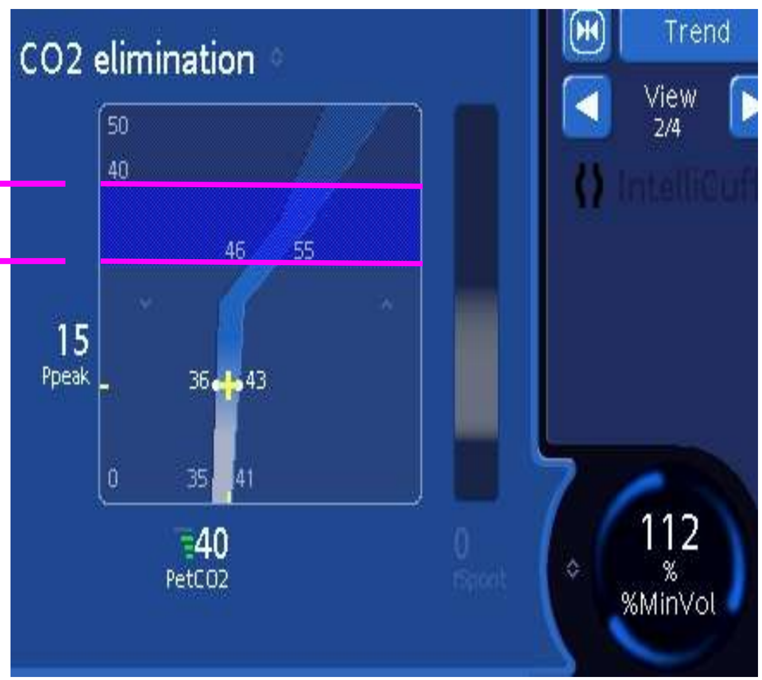


Is there a pressure limit to generate a predefined tidal volume ?

P_{ASV} limit

Hochdruck-Alarm

P_{ASV} limit

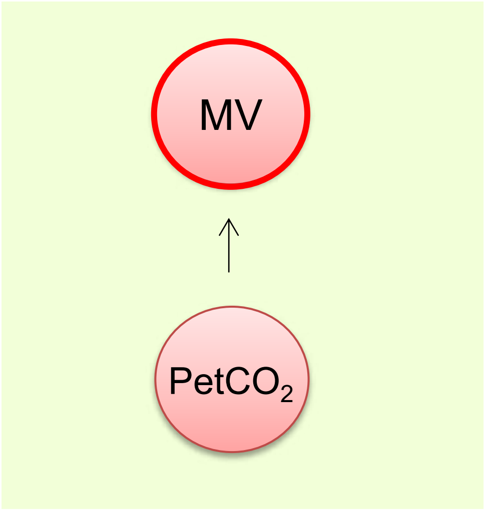


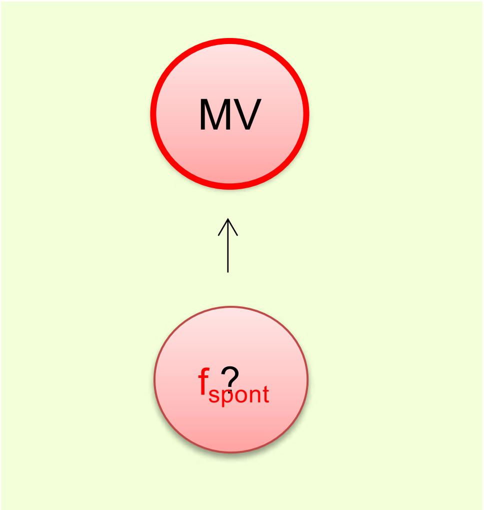
Ventilation
Control $PaCO_2$

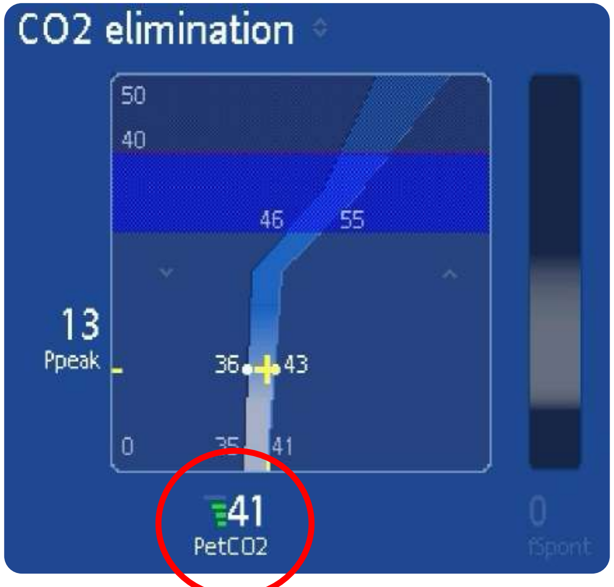
INTELLiVENT-ASV+

Minute Volume Adjustment in active patients

Passive patient → Active patient





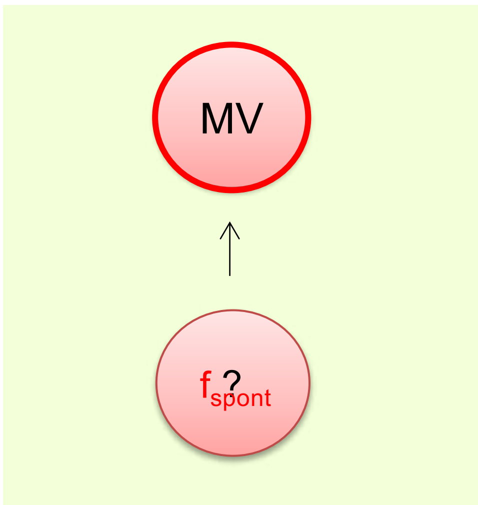


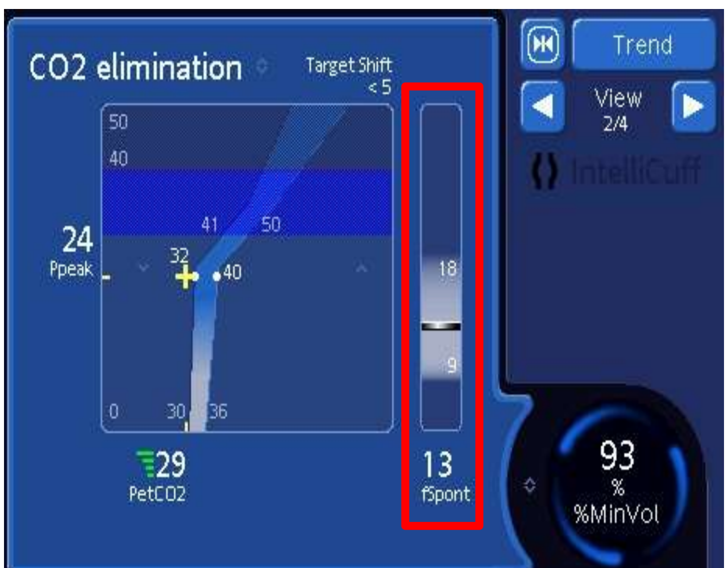
Ventilation
Control $PaCO_2$

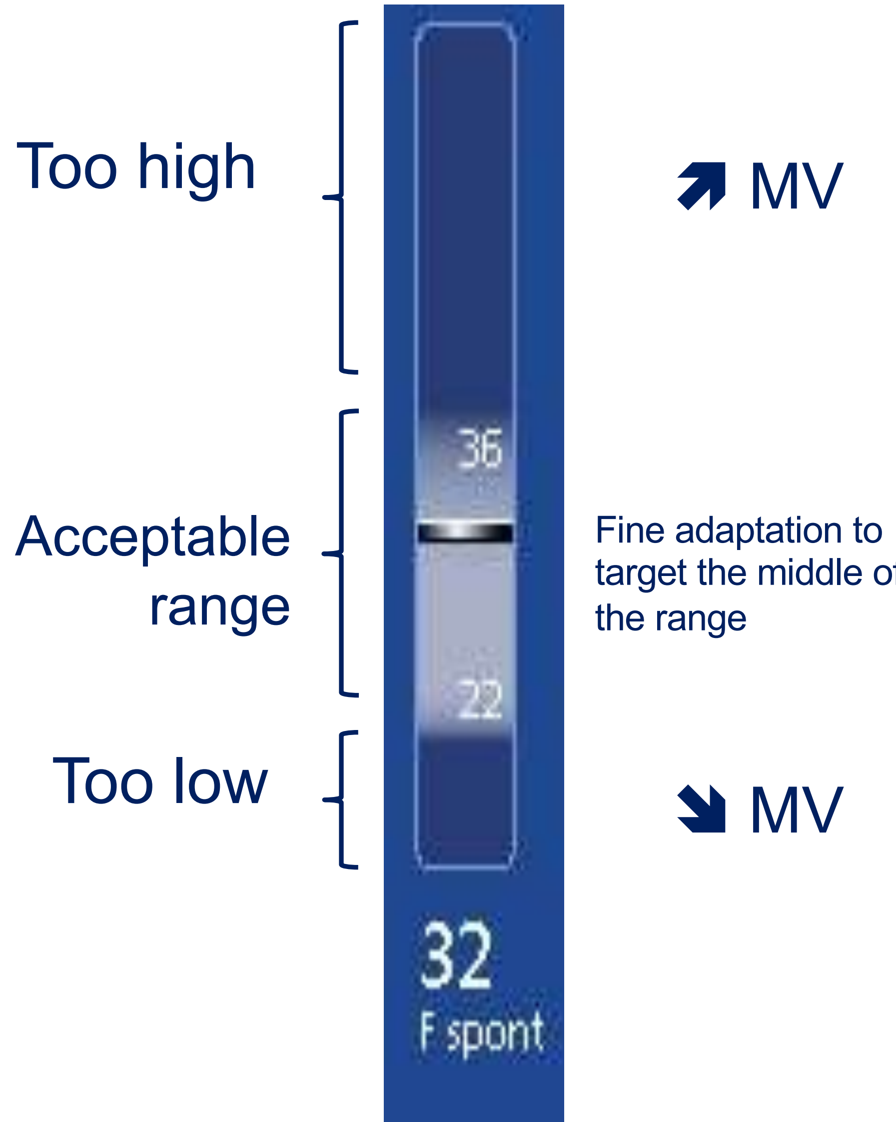
INTELLiVENT-ASV+

Minute Volume Adjustment in active patients

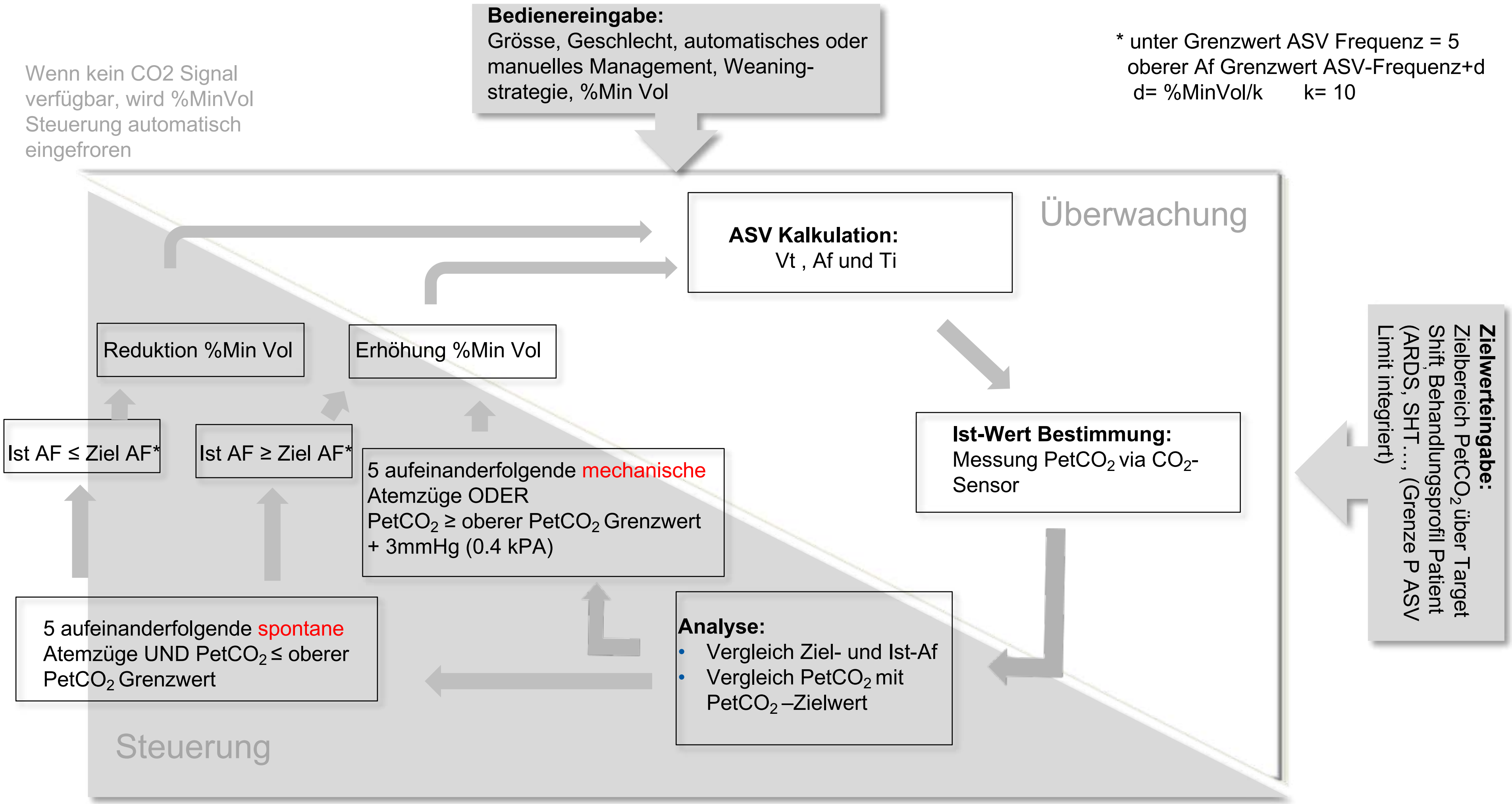
Active patient



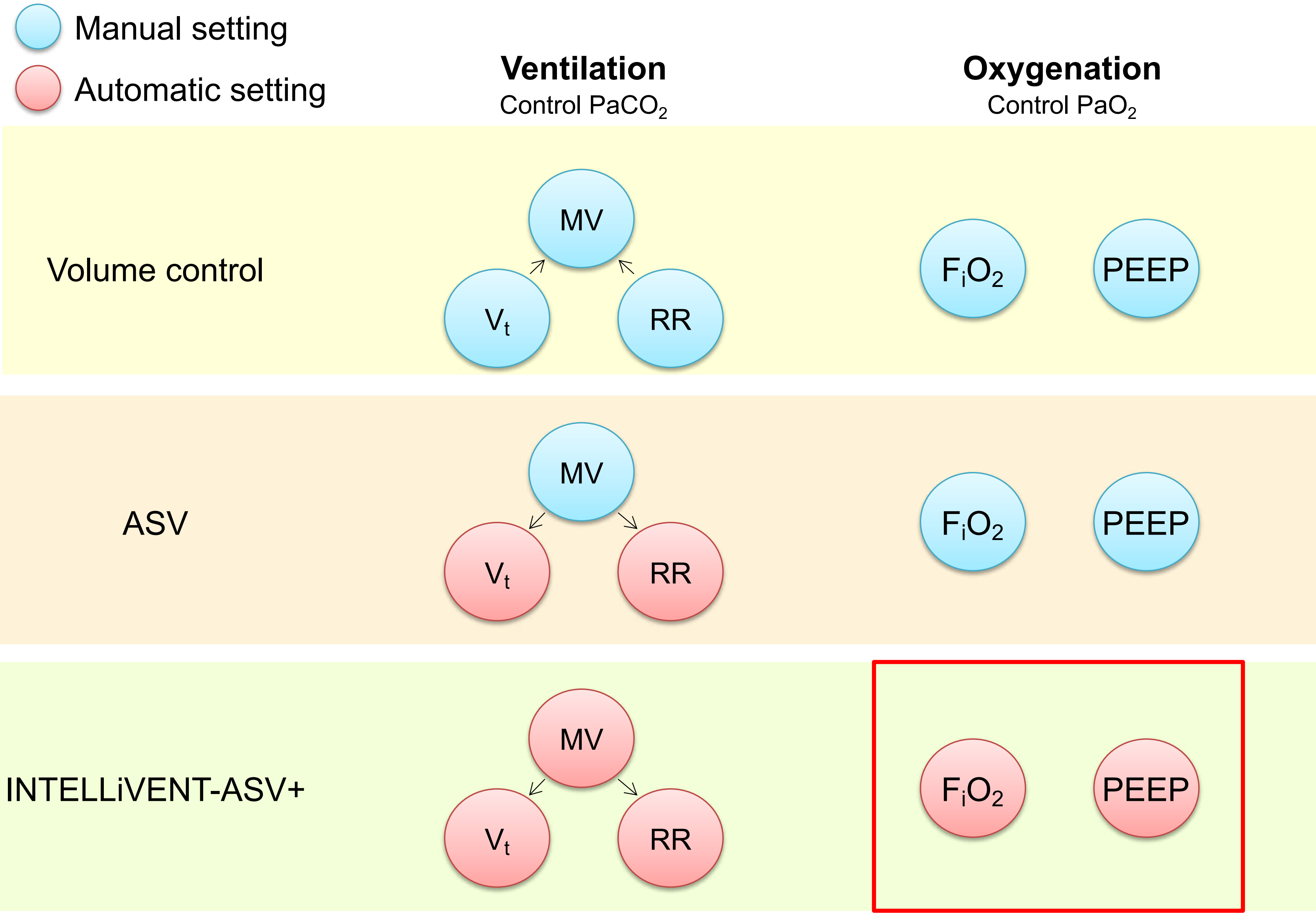




INTELLiVENT-ASV: Ventilationsmanagement aktiver Patient



ASV und INTELLiVENT-ASV+

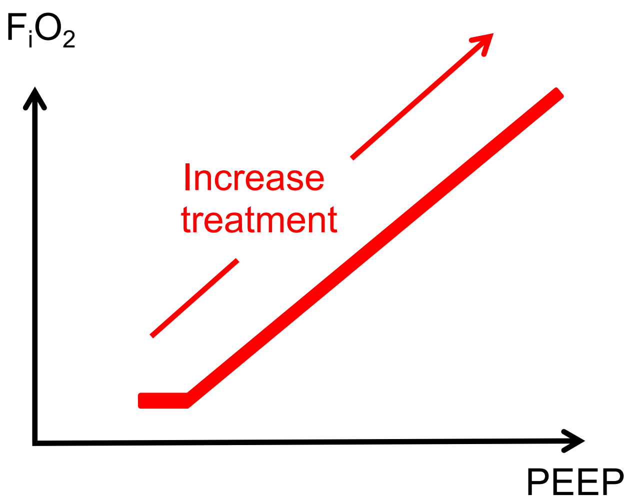


Oxygenation
Control PaO_2

INTELLiVENT-ASV+

Oxygenation
Control PaO_2

INTELLiVENT-ASV+



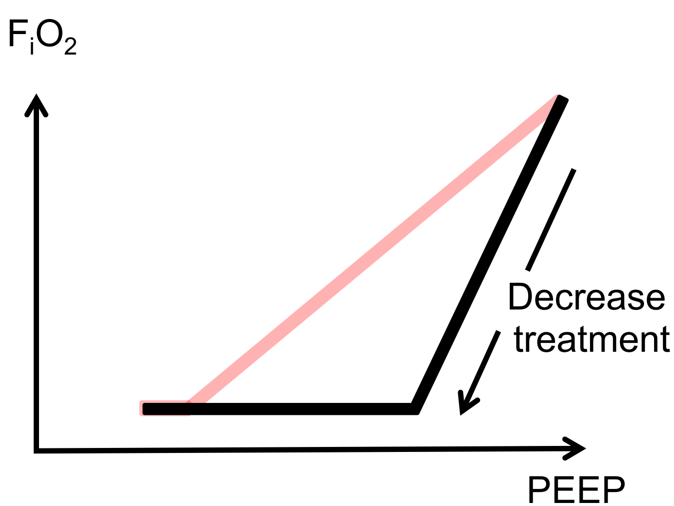
Increase treatment

- F_iO_2: 10% alle 30sec
- PEEP: 1mbar alle 6min

Higher PEEP/lower FiO2

FiO_2	0.3	0.3	0.3	0.3	0.3	0.4	0.4	0.5
PEEP	5	8	10	12	14	14	16	16

FiO_2	0.5	0.5-0.8	0.8	0.9	1.0	1.0
PEEP	18	20	22	22	22	24



Decrease treatment

- F_iO_2: 5% alle 60sec
- PEEP: 1mbar alle 6min

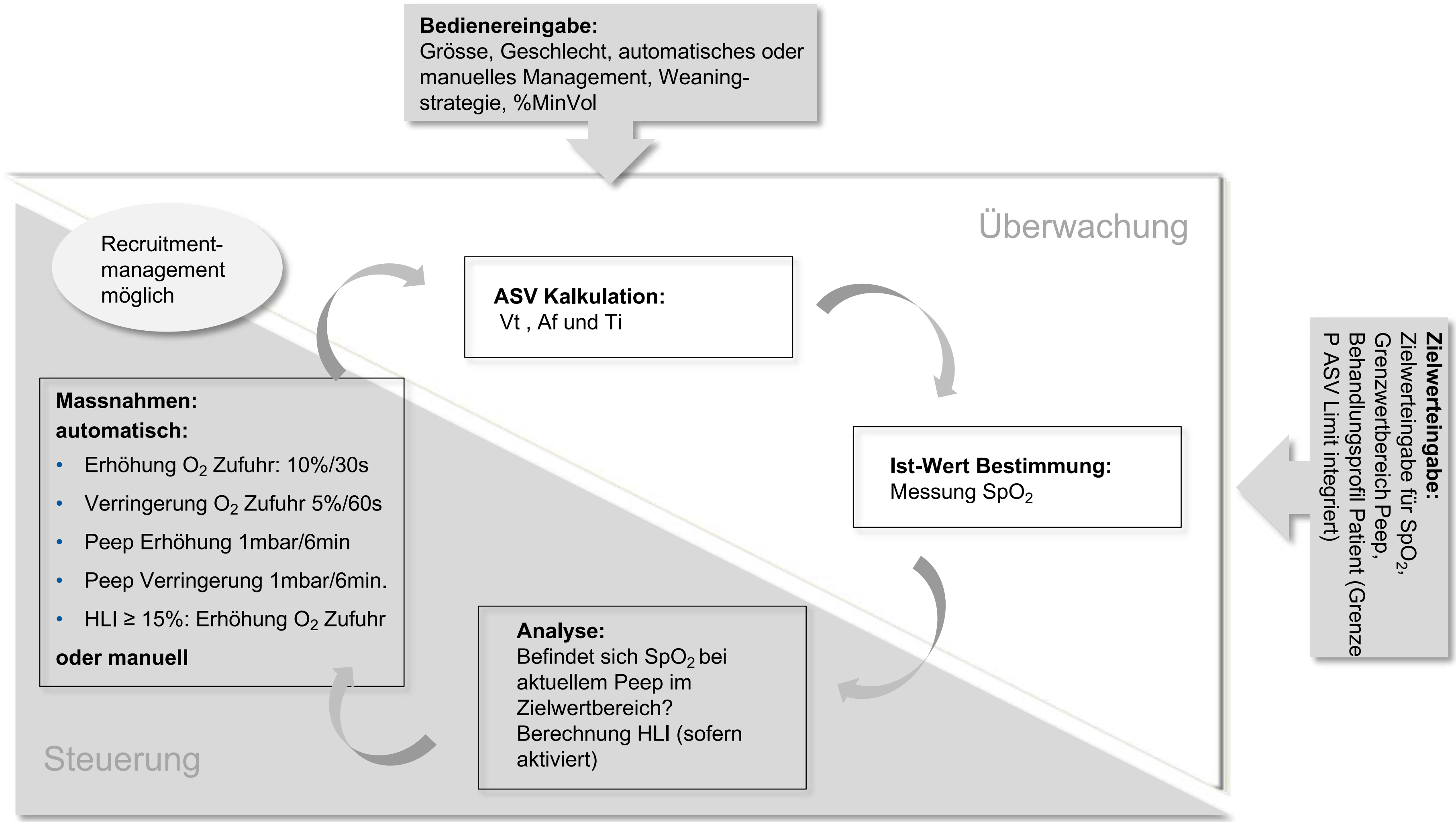
Lower PEEP/higher FiO2

FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7
PEEP	5	5	8	8	10	10	10	12

FiO_2	0.7	0.8	0.9	0.9	0.9	1.0
PEEP	14	14	14	16	18	18-24

Oxygenation
Control PaO_2

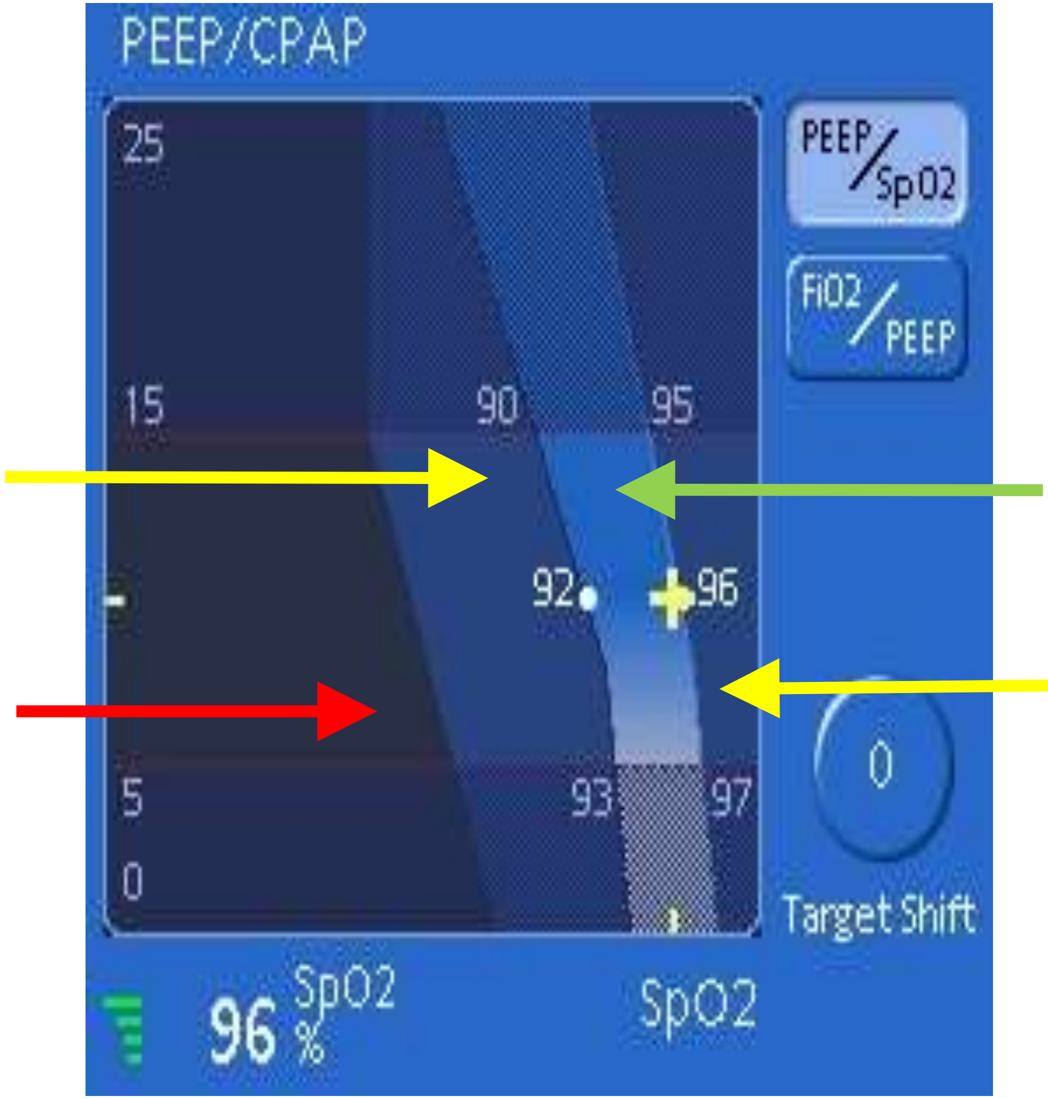
INTELLiVENT-ASV+



INTELLiVENT-ASV+

Oxygenation
Control PaO_2

Oxygenation Controller



Increase Oxygen/PEEP

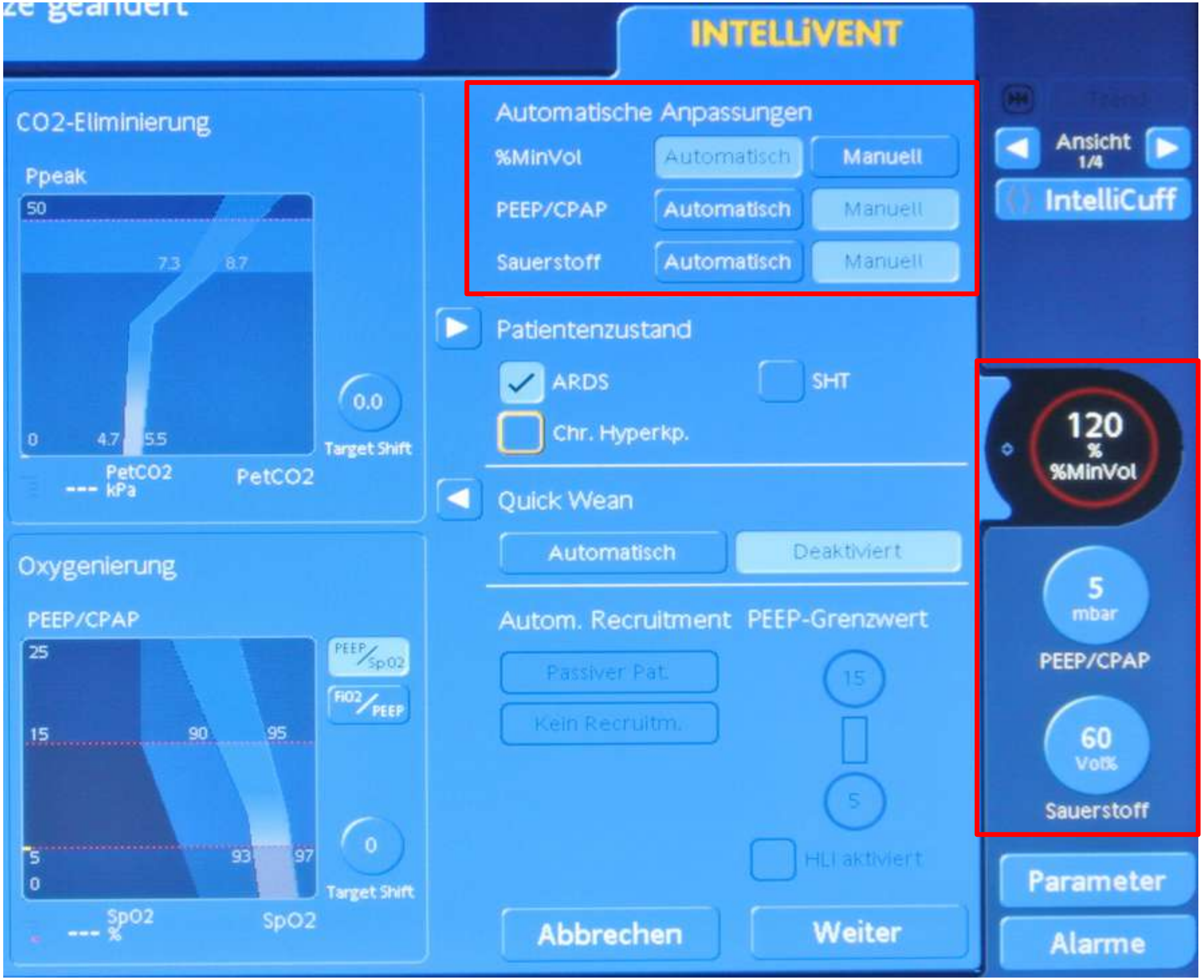
Emergency 100% Oxygen

FiO_2/PEEP maintained (fine tuning)

Decrease Oxygen/PEEP

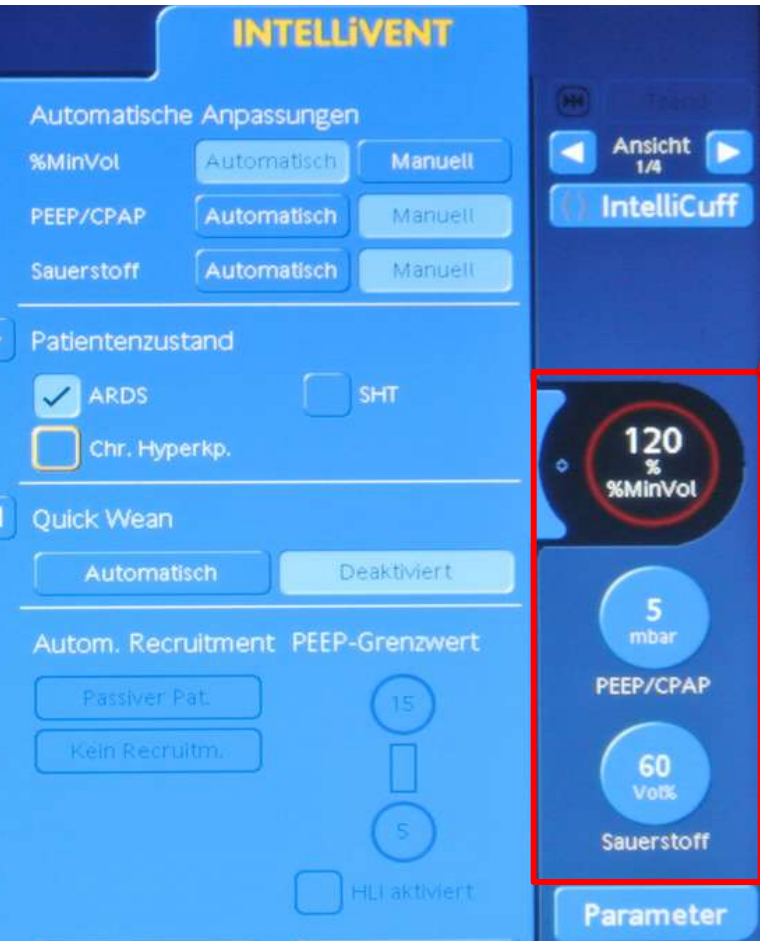
INTELLiVENT-ASV+

Controller Status



Controller Status

INTELLiVENT-ASV+



INTELLiVENT-ASV+

Patienten zustand

Patienten zustand

INTELLiVENT-ASV+

Patienten-zustand	Beatmung		Oxygenierung			Quick Wean	Autom. Recruitment	PEEP-Grenzwert Anpassung anhand HLI
	%MinVol Startwert	*Akzeptabler Bereich Spontan-atmung D=%MinVol/K*	*P ASV Limit (mbar)*	*O_2-Start-wert (%)*	*PEEP-Start-wert (mbar)*			
Normal	100	K = 10	30	60	5	deaktiviert	deaktiviert	deaktiviert
ARDS	120	K = 10	35	100	5	deaktiviert	deaktiviert	deaktiviert
Hyperkapnie	90	K = 10	25	40	manuell	deaktiviert	deaktiviert	deaktiviert
ARDS+ Hyperkapnie	110	K = 10	30	80	manuell	deaktiviert	deaktiviert	deaktiviert
Schädel-Hirn-Trauma	a)		b)	60	manuell	deaktiviert	deaktiviert	deaktiviert

a) Der Startwert wird anhand des Patientenzustands festgelegt

b) Schädel-Hirn-Trauma (SHT): 28 mbar
SHT + Hyperkapnie: 28 mbar
SHT + ARDS: 30 mbar
Übrige Kombinationen: 28 mbar

Patienten zustand

INTELLiVENT-ASV+

Normal

Patienten-zustand	Beatmung		Oxygenierung		
	%MinVol Startwert	*Akzeptabler Bereich Spontan-atmung D=%MinVol/K*	*P ASV Limit (mbar)*	*O_2-Start-wert (%)*	*PEEP-Start-wert (mbar)*
Normal	100	K = 10	30	60	5
ARDS	120	K = 10	35	100	5
Hyperkapnie	90	K = 10	25	40	manuell
ARDS+ Hyperkapnie	110	K = 10	30	80	manuell
Schädel-Hirn-Trauma	a)		b)	60	manuell

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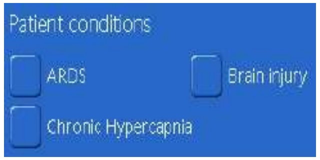

Patienten zustand

INTELLiVENT-ASV+

ARDS

Patienten-zustand	Beatmung			Oxygenierung	
	%MinVol Startwert	*Akzeptabler Bereich Spontan-atmung D=%MinVol/K*	*P ASV Limit (mbar)*	*O_2-Start-wert (%)*	*PEEP-Start-wert (mbar)*
Normal	100	K = 10	30	60	5
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SHT + Hyperkapnie: 28 mbar
SHT + ARDS: 30 mbar
Übrige Kombinationen: 28 mbar

Patienten
zustand

INTELLiVENT-ASV+

Hyperkapnie

Patienten-zustand	Beatmung		Oxygenierung		
	%MinVol Startwert	*Akzeptabler Bereich Spontan-atmung D=%MinVol/K*	*P ASV Limit (mbar)*	*O_2-Start-wert (%)*	*PEEP-Start-wert (mbar)*
Normal	100	K = 10	30	60	5
ARDS	120	K = 10	35	100	5
Hyperkapnie	90	K = 10	25	40	manuell
ARDS+ Hyperkapnie	110	K = 10	30	80	manuell
Schädel-Hirn-Trauma	a)		b)	60	manuell

a) Der Startwert wird anhand des Patientenzustands festgelegt

b) Schädel-Hirn-Trauma (SHT): 28 mbar
SHT + Hyperkapnie: 28 mbar
SHT + ARDS: 30 mbar
Übrige Kombinationen: 28 mbar

Patienten-
zustand

INTELLiVENT-ASV+

Schädelhirntrauma

Patienten-zustand	Beatmung			Oxygenierung	
	%MinVol Startwert	*Akzeptabler Bereich Spontan-atmung D=%MinVol/K*	*P ASV Limit (mbar)*	*O_2-Start-wert (%)*	*PEEP-Start-wert (mbar)*
Normal	100	K = 10	30	60	5
ARDS	120	K = 10	35	100	5
Hyperkapnie	90	K = 10	25	40	manuell
ARDS+ Hyperkapnie	110	K = 10	30	80	manuell
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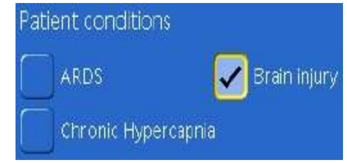
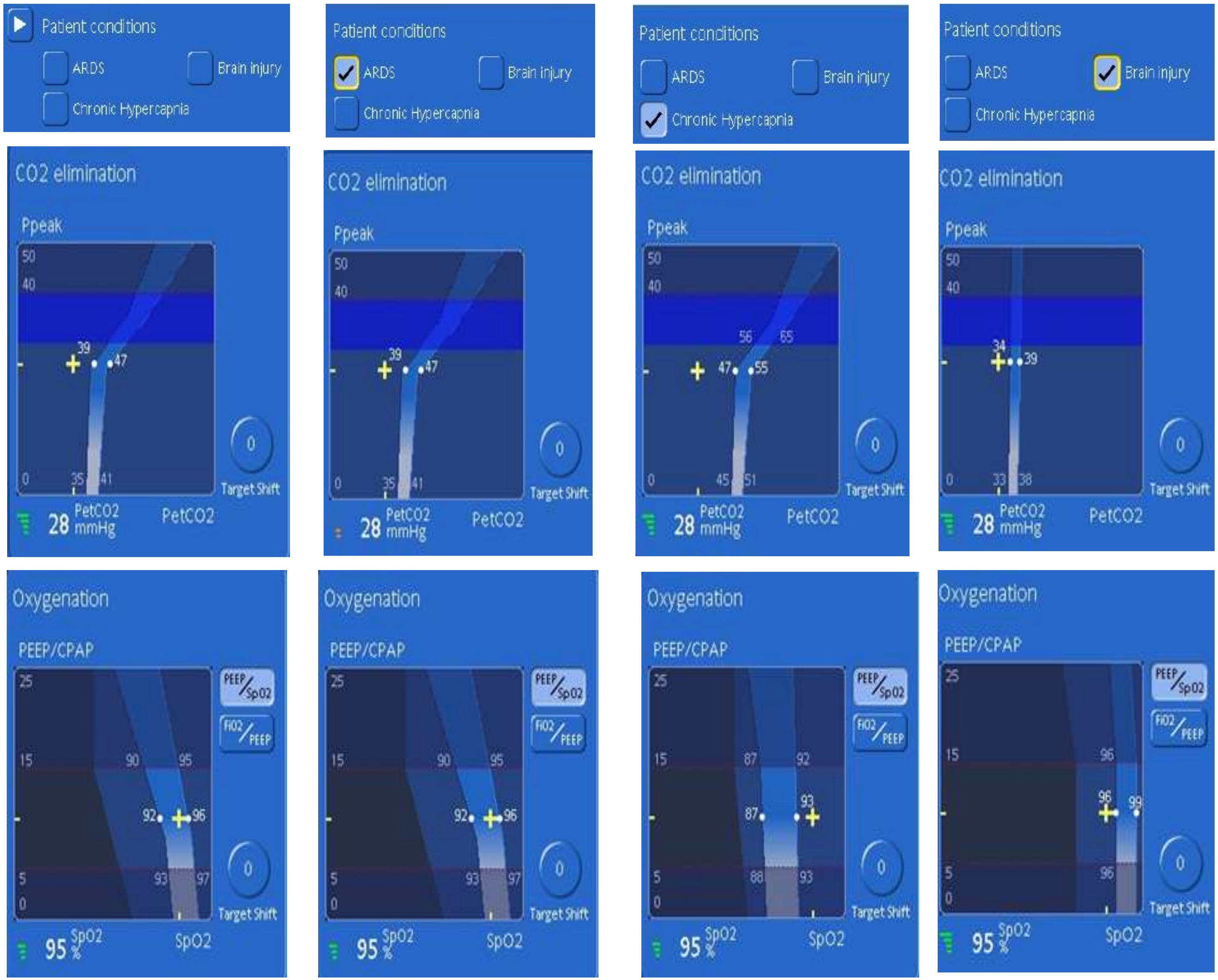


Patienten zustand

INTELLiVENT-ASV+

CO_2 und SpO_2 Targets

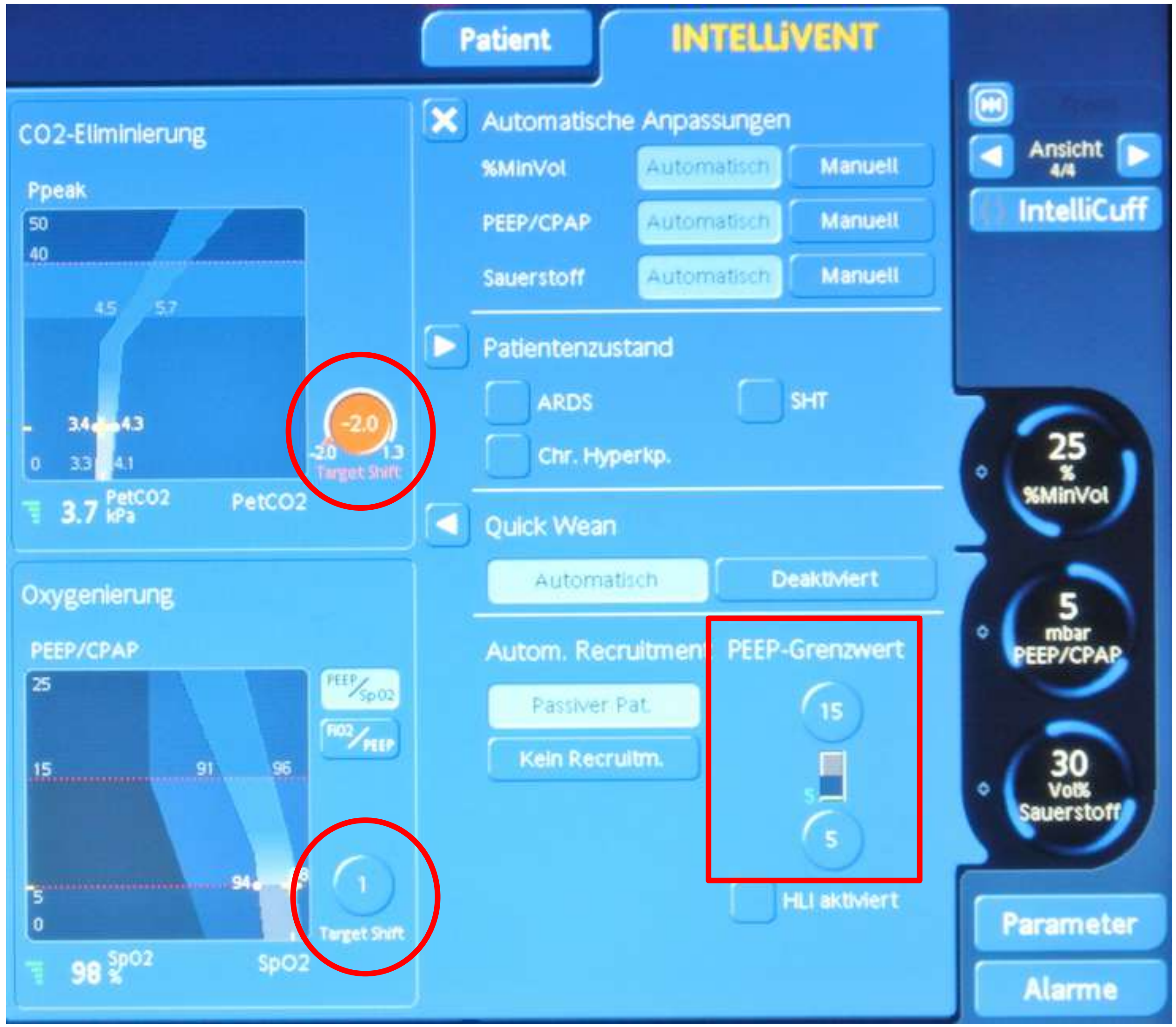


Patientenzustand

INTELLiVENT-ASV+

PEEP Grenzen und Target Shift

Patientenzustand	%MinVol [%]	PASVlimit [mbar]	FiO_2 [%]	PEEP [mbar]	PEEP-Grenzen [mbar] low	PEEP-Grenzen [mbar] high	Target Shift [mbar] CO_2	Target Shift [mbar] O_2
Normal RC_{exp} 0.5 – 0.9 s	100	30	100	5	5	12	-0.7	0
ARDS RC_{exp} < 0.5 s	140	35	100	10	10	15	-0.7	0
Chronische Hyperkapnie RC_{exp} > 0.9 s	100	35	100	8	manuell		Patientenzustand Chr. Hyperkap. 0.7	Patientenzustand Chr. Hyperkap. 0



Sensoren

INTELLiVENT-ASV+

Die notwendigen Sensoren

Ventilation

Proximal flow sensor

Airway CO2 sensor

Oxygenation

SpO2 sensors

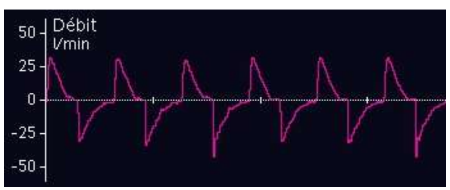
Sensoren

INTELLiVENT-ASV+

Die notwendigen Sensoren

Ventilation

Oxygenation



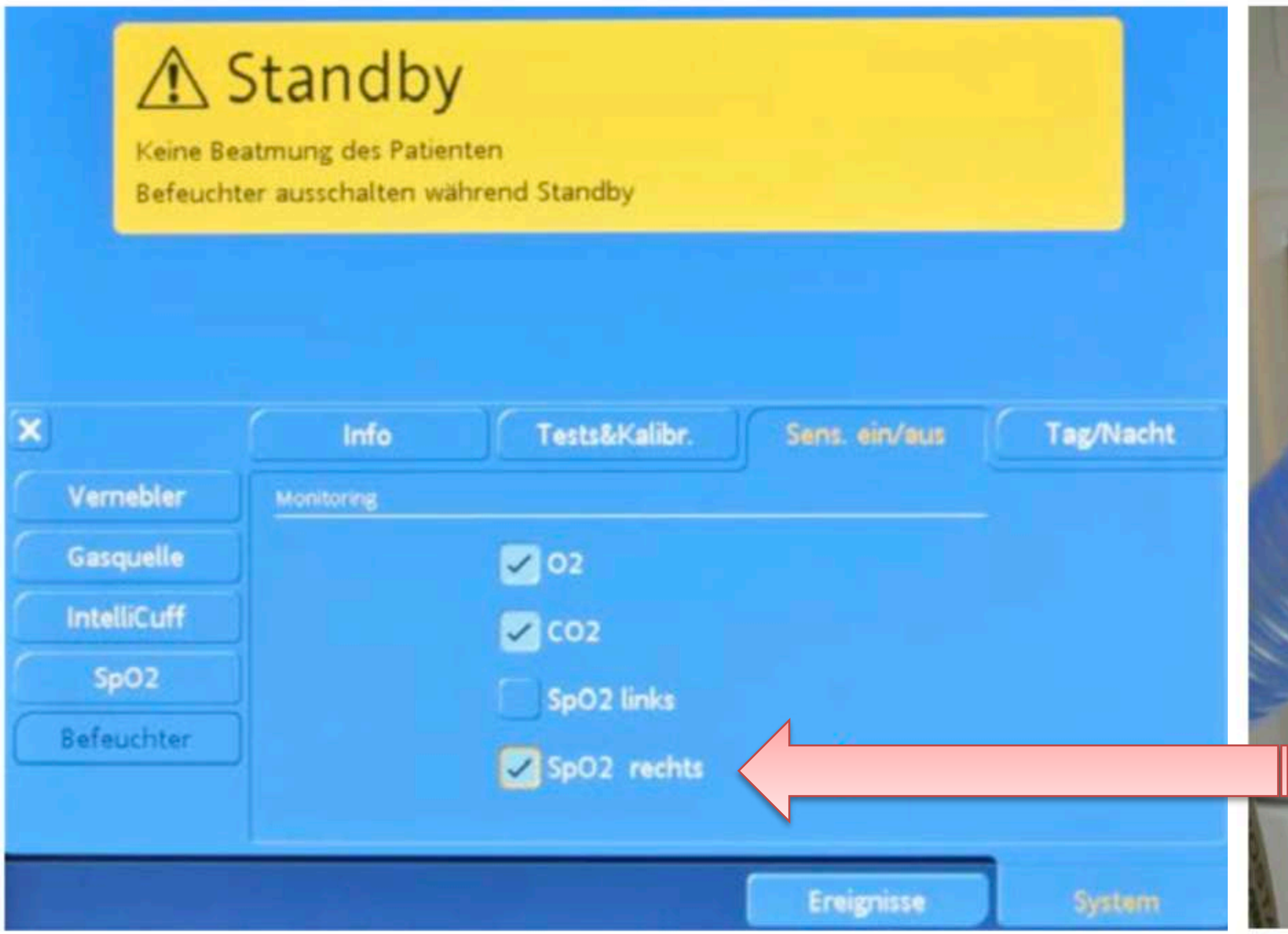
- 2^{nd} highest value of the last 8 breaths
- Quality index

- Mean on the 15 last values
- Quality index

Sensoren

INTELLiVENT-ASV+

Aktivierung der Sensoren



Basic mechanical ventilation

Continuing development of BASIC is supported by an unrestricted educational grant from

MAQUET Dräger medical
A Dräger and Siemens Company

Mechanical ventilation

- Physics
- Basic concepts
- Complications

Physics

P:	Pressure
ΔP:	Pressure Gradient
V:	Volume
$\dot{V}$:	Gas Flow
R:	Resistance
C:	Compliance
RC:	Time constant

Pressure

Force applied over a unit area

Physics

P: Pressure

ΔP: Pressure Gradient

V: Volume

$\dot{V}$: Gas Flow

R: Resistance

C: Compliance

RC: Time constant

Pressure Gradient

Refers to the difference between the pressure at area A and the pressure at area B. If both areas are connected with a tube, the pressure gradient drives the air or gas to move from the high pressure area to the low pressure area.

The gas movement is flow.

Physics

- P: Pressure
- △P: Pressure Gradient
- V: Volume
- $\dot{V}$: Gas Flow
- R: Resistance
- C: Compliance
- RC: Time constant

Volume

Gas volume is a measure of the space occupied by a quantity of gas at a given pressure.

Tidal volume and minute volume are two typical examples of gas volumes.

Gas is compressible.

Physics

P: Pressure

ΔP: Pressure Gradient

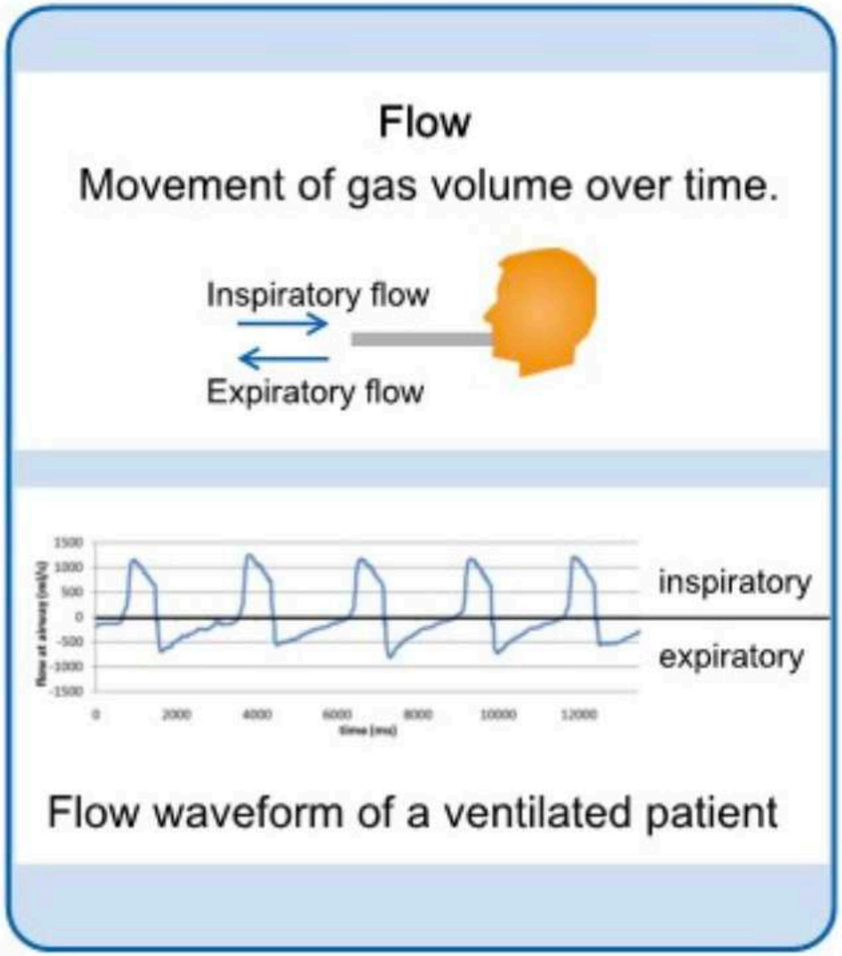
V: Volume

$\dot{V}$: Gas Flow

R: Resistance

C: Compliance

RC: Time constant



Hamilton Medical e Learning

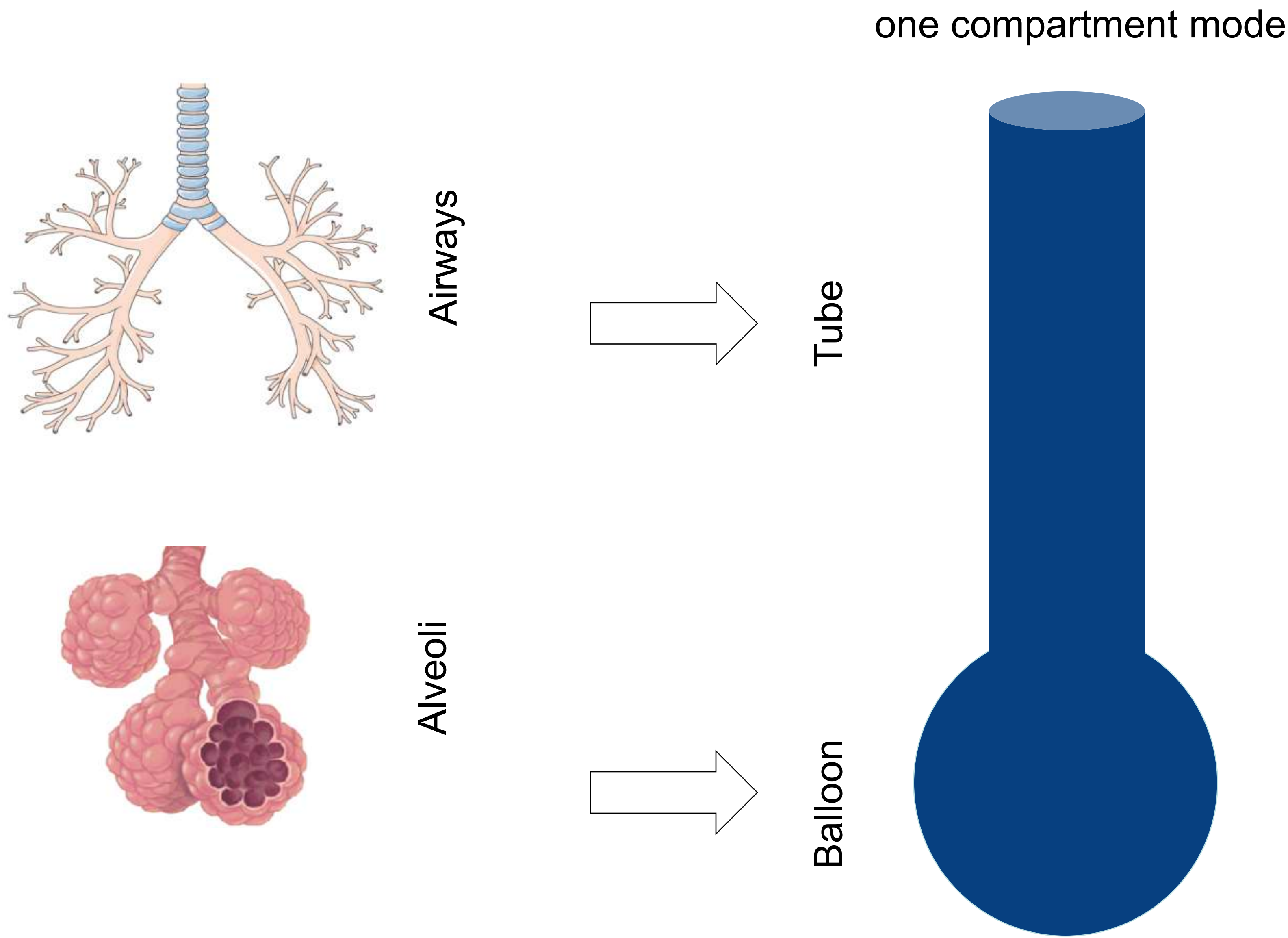
Physics

P:	Pressure
ΔP:	Pressure Gradient
V:	Volume
$\dot{V}$:	Gas Flow
R:	Resistance
C:	Compliance
RC:	Time constant

RESPIRATORY PHYSIOLOGY

Airway Lung Structure

2 parts



RESPIRATORY PHYSIOLOGY

Two main forces oppose inflation of the balloon

Impedance to flow

...which represents resistance of the airways

Impedance to volumetric expansion

...which represents compliance of the chest wall

RESPIRATORY PHYSIOLOGY

RESISTANCE

Force against gas movement

- When a gas moves through a tube, a resistance is generated
- Resistance depends on

1. Properties of the tube
   (length, internal diameter, inner surface, curvature...)
2. Properties of the passing gas
   (density, viscosity)
3. Flow

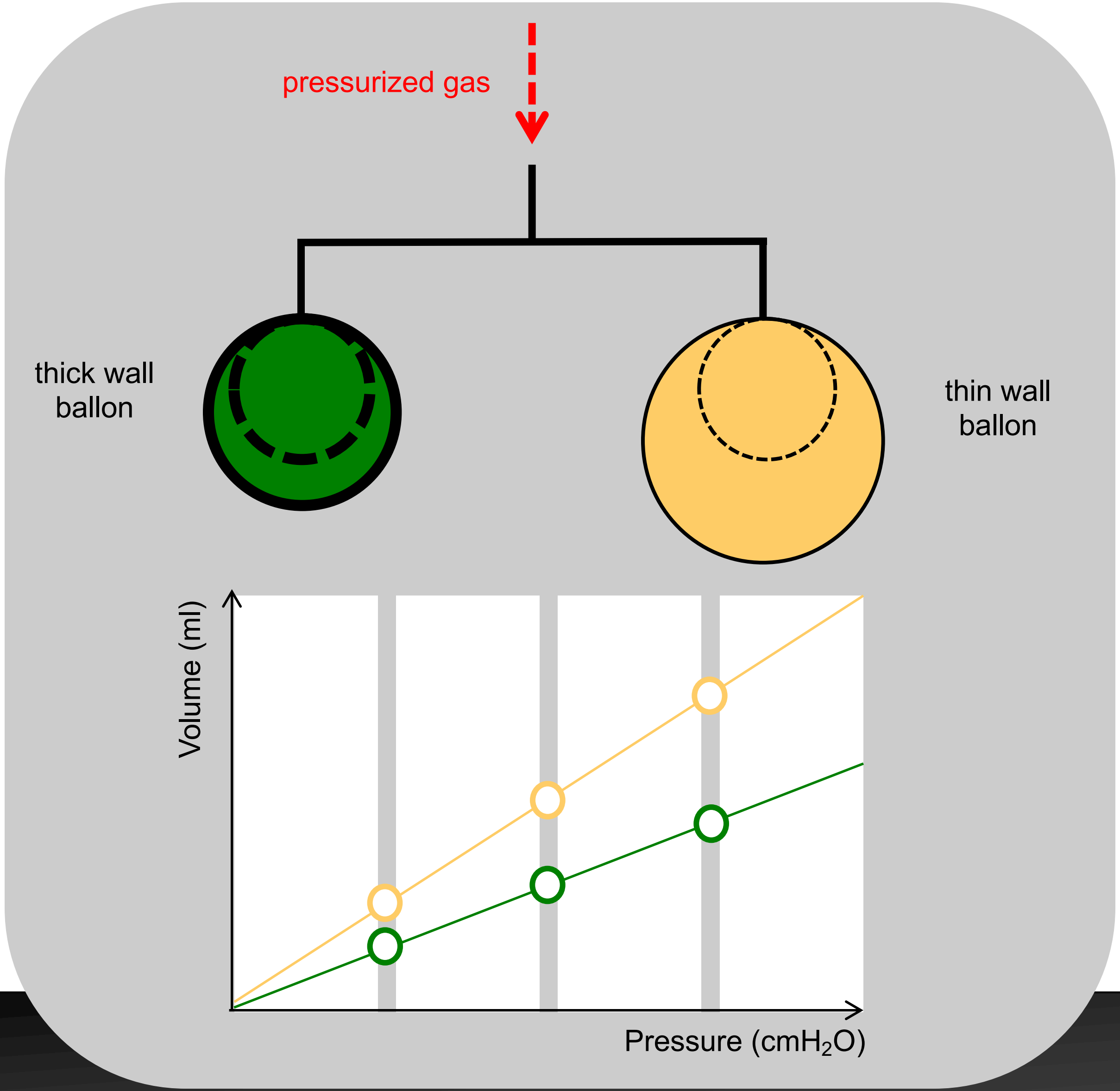
$$\text{Resistance} = \frac{\Delta\ \text{Pressure}}{\text{Flow}}$$

mbar/L/min

Ohm's law

RESPIRATORY PHYSIOLOGY

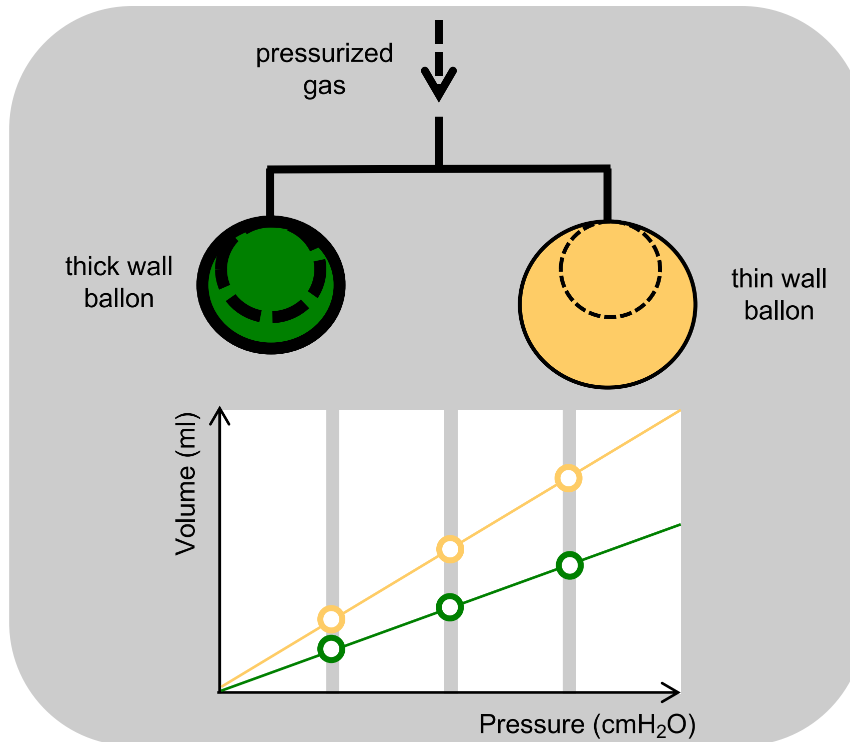
COMPLIANCE



Adapted from Hamilton Medical

RESPIRATORY PHYSIOLOGY

COMPLIANCE

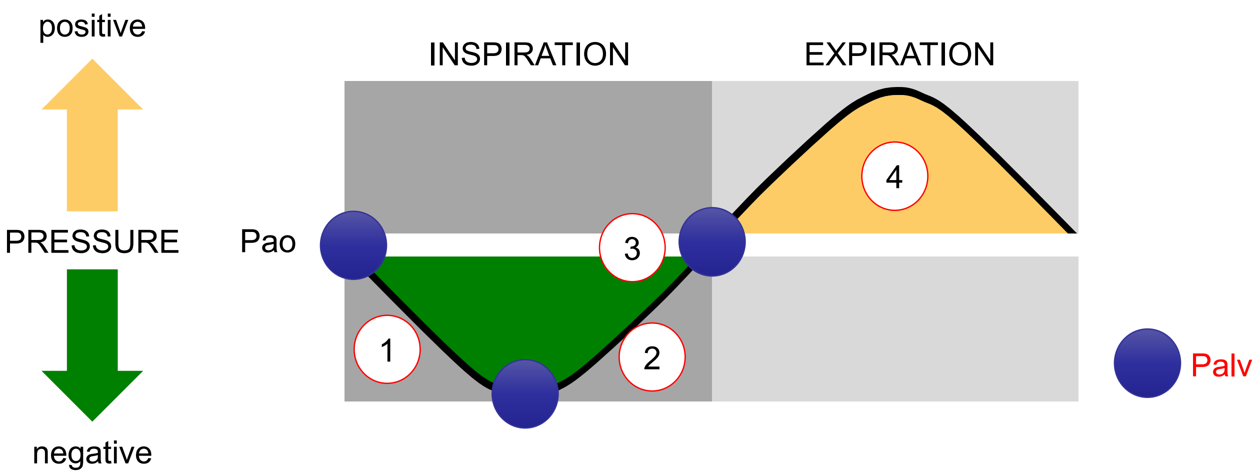


$$\text{Compliance} = \frac{\Delta\ \text{Volume}}{\Delta\ \text{Pressure}}$$ mL/cmH_2O oder L/cmH_2O

Adapted from Hamilton Medical

RESPIRATORY PHYSIOLOGY

INSPIRATION AND EXPIRATION; SPONTANEOUS BREATHING

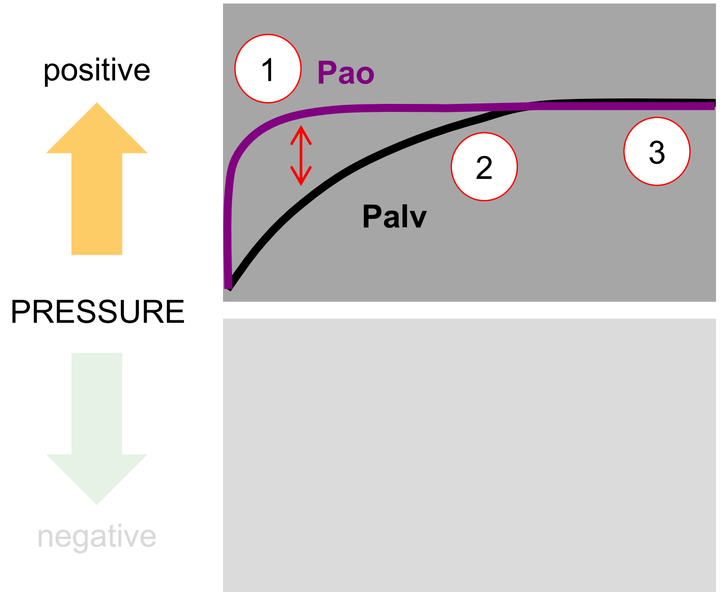


1	Downward movement of diaphragm →	alveolar pressure drops below airway opening pressure
	Following the pressure gradient →	air is sucked into the lung
2	Lungs are inflated with inspiratory flow over time →	Alveolar pressure increases gradually
3	Alveolar pressure and airway opening pressure become equal →	Flow stops
4	Relaxation of the diaphragm →	Elastic recoil force of lungs and chest wall brings the enlarged lungs bach to their resting position (FRC)

RESPIRATORY PHYSIOLOGY

INSPIRATION AND EXPIRATION; ARTIFICIAL LUNG VENTILATION

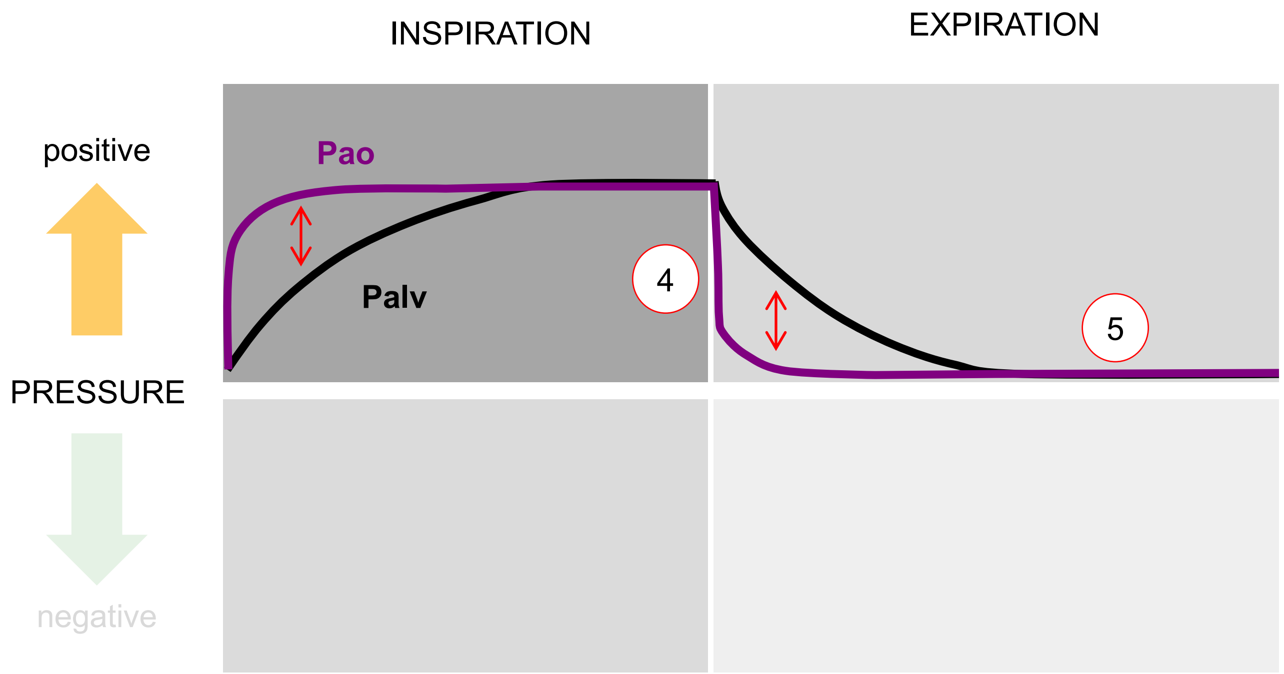
INSPIRATION



1	Ventilator raises Pao from pressure baseline to peak pressure	→	This generates a temporary gradient between Pao and Palv
		→	The gas is pushed into the lungs
2	Lungs are inflated with inspiratory flow over time	→	Alveolar pressure increases gradually
3	Alveolar pressure and airway opening pressure become equal	→	Flow stops

RESPIRATORY PHYSIOLOGY

INSPIRATION AND EXPIRATION; ARTIFICIAL LUNG VENTILATION



4 Ventilator lowers Pao from peak pressure baseline to baseline pressure → This generates a temporary gradient between Pao and Palv → The elastic recoil force of the lung-chest wall pushes the gas out of the lungs

5 Alveolar pressure and airway opening pressure become equal → Flow stops

RESPIRATORY PHYSIOLOGY

INSPIRATION AND EXPIRATION

- Flow is defined as change of volume over time
- It takes time to complete a course of volume change (inflation or deflation)
- Inflation: if time available for lung inspiration is too short, tidal volume decreases
- Deflation: if time available for lung exhalation is shorter than required, gas volume is trapped in the lungs and alveolar pressure rises

How to know or estimate objectively and individually the required time to complete a course of volume change?

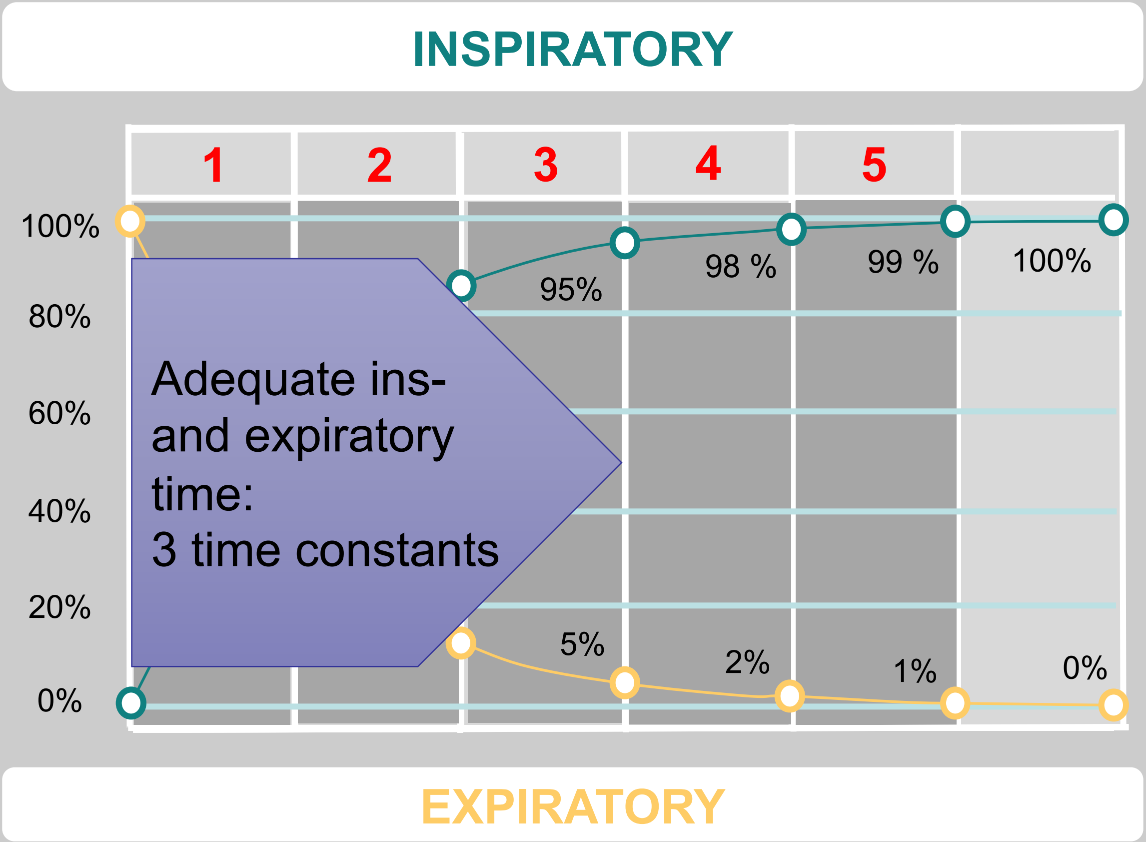
Physics

P:	Pressure
ΔP:	Pressure Gradient
V:	Volume
$\dot{V}$:	Gas Flow
R:	Resistance
C:	Compliance
RC:	Time constant

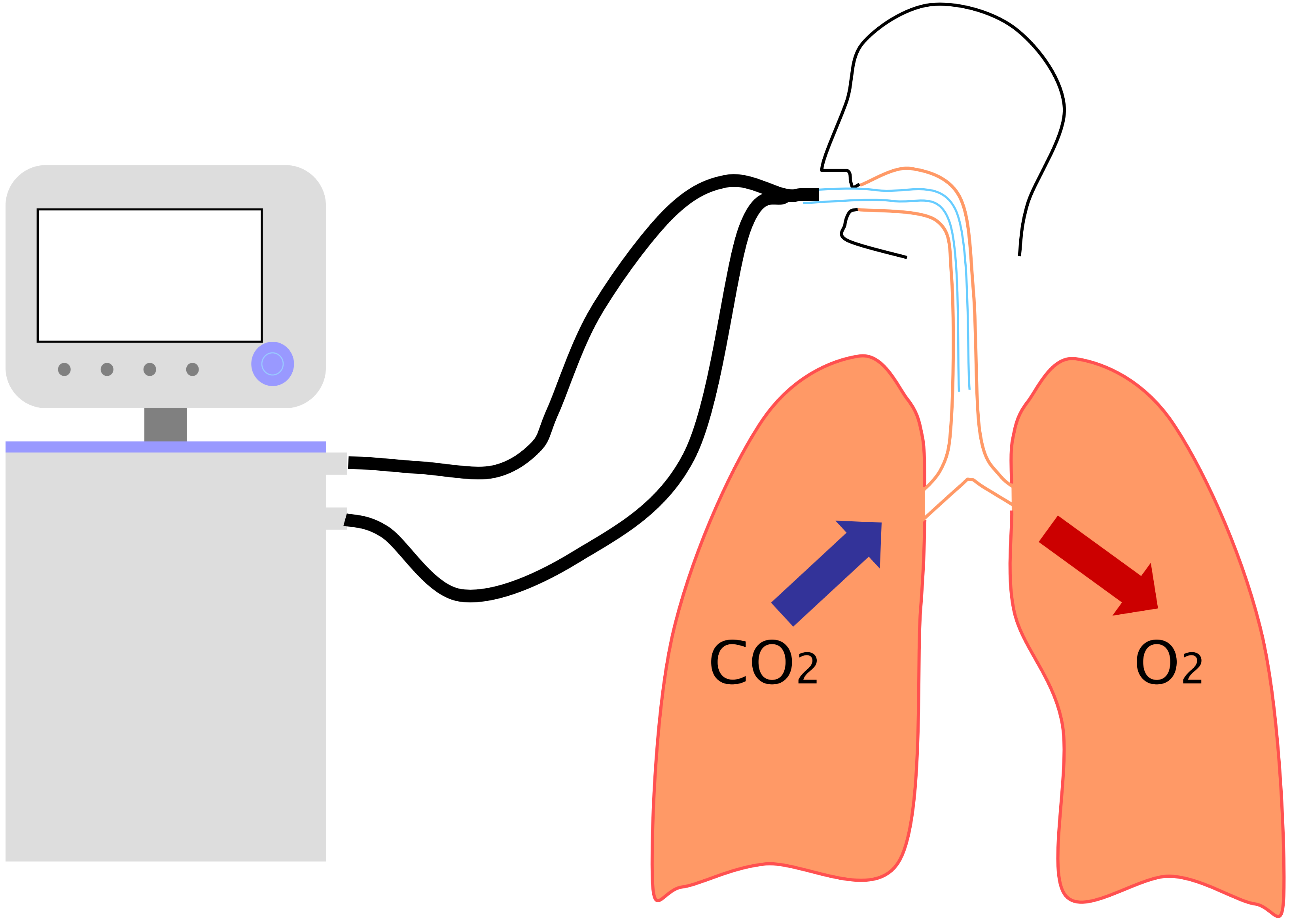
RESPIRATORY PHYSIOLOGY

TIME CONSTANT

Expressed in second



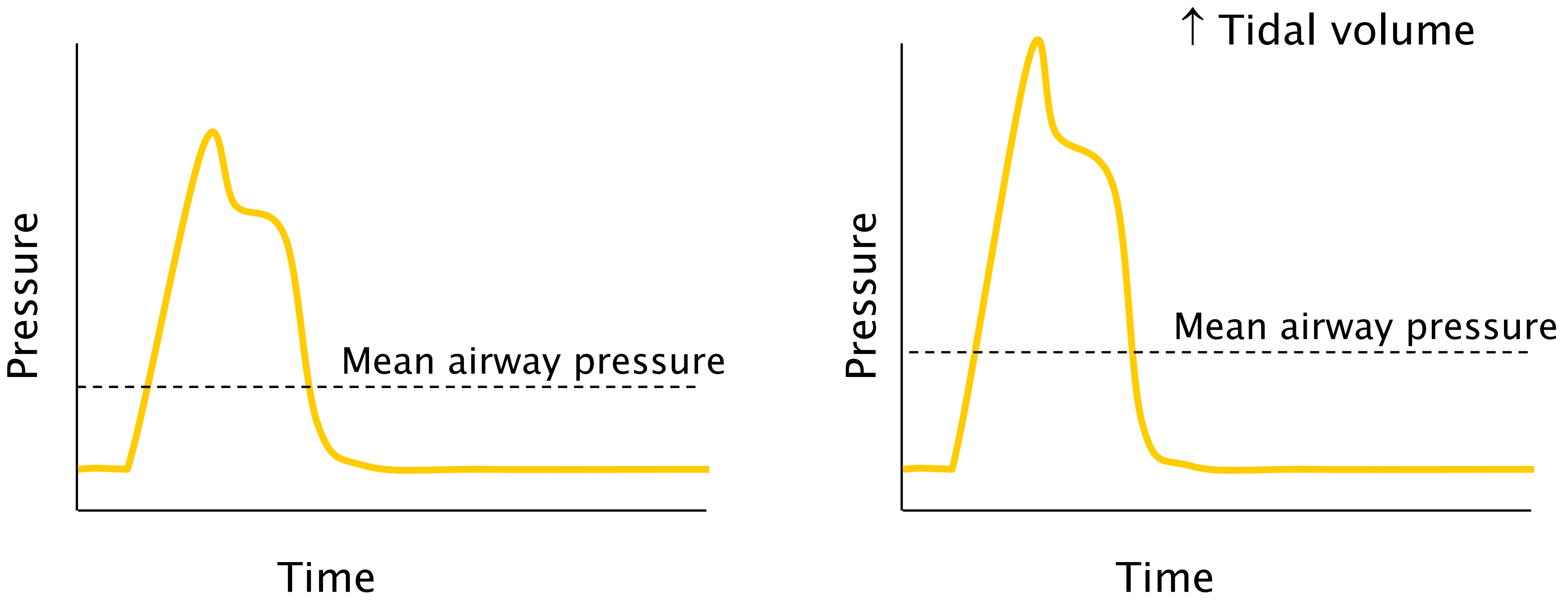
Time constant RC = Resistance (R) X Complicance (C)


CO2
O2

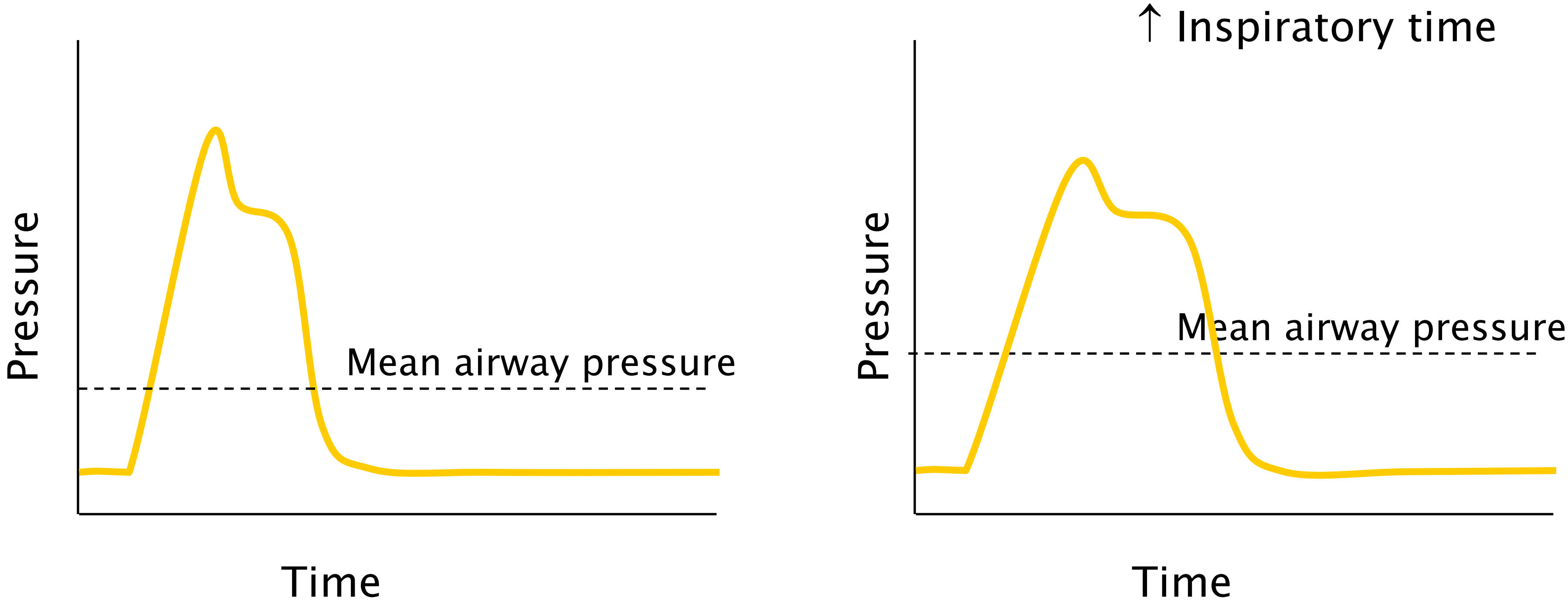
Main determinants

- Oxygen in
  - ↑ F_IO_2
  - ↑ mean alveolar pressure
  - PEEP
    - Re-open alveoli and ↓ shunt
- Carbon dioxide out
  - ↑ ventilation
    - ↑ RR
    - ↑ tidal volume

Mean airway pressure



Mean airway pressure



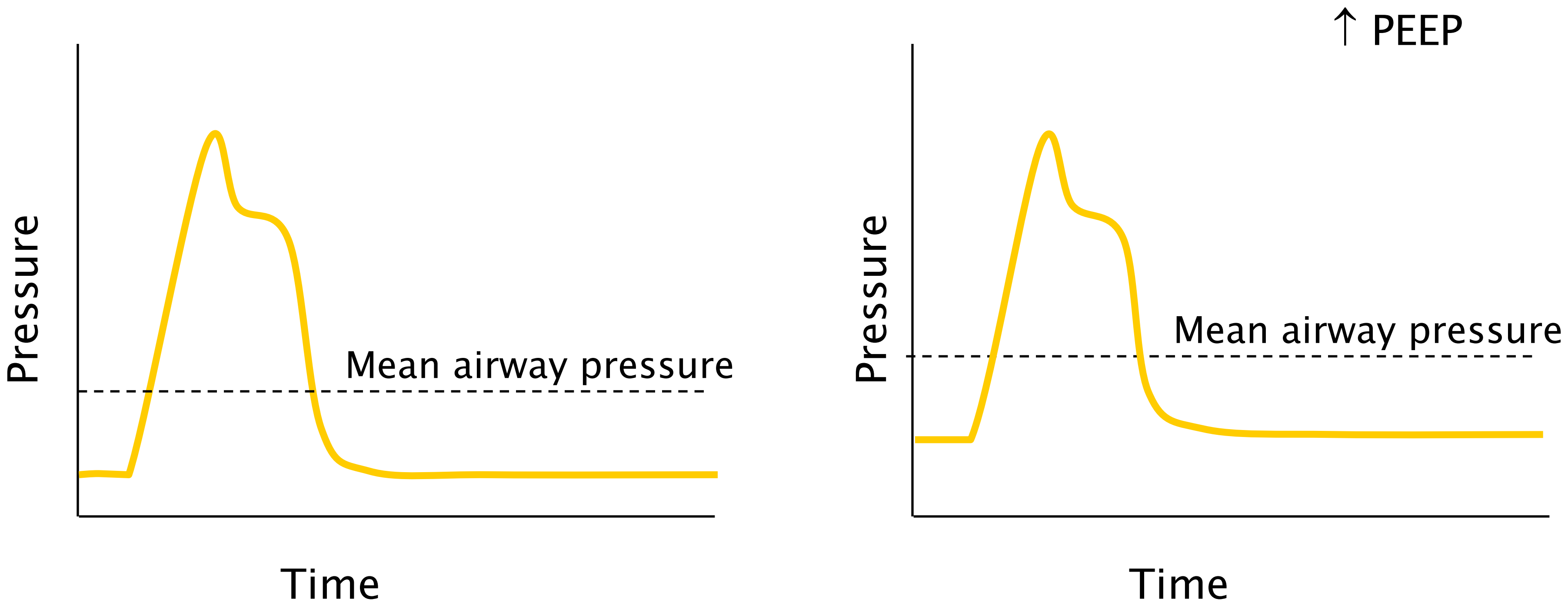
Inspiratory time

- Set as:
  - % of respiratory cycle
  - I:E ratio
  - Fixed time
  - Indirectly, by setting flow
- Expiratory time not set
  - Remaining time after inspiration before next breath

Inspiratory time

- Increased inspiratory time
  - Improved oxygenation
  - Unnatural pattern of breathing
    - Deeper sedation
  - Increased risk of gas trapping

Mean airway pressure



PEEP

- Improves oxygenation
  - ↑ mean alveolar pressure
  - ↓ shunting

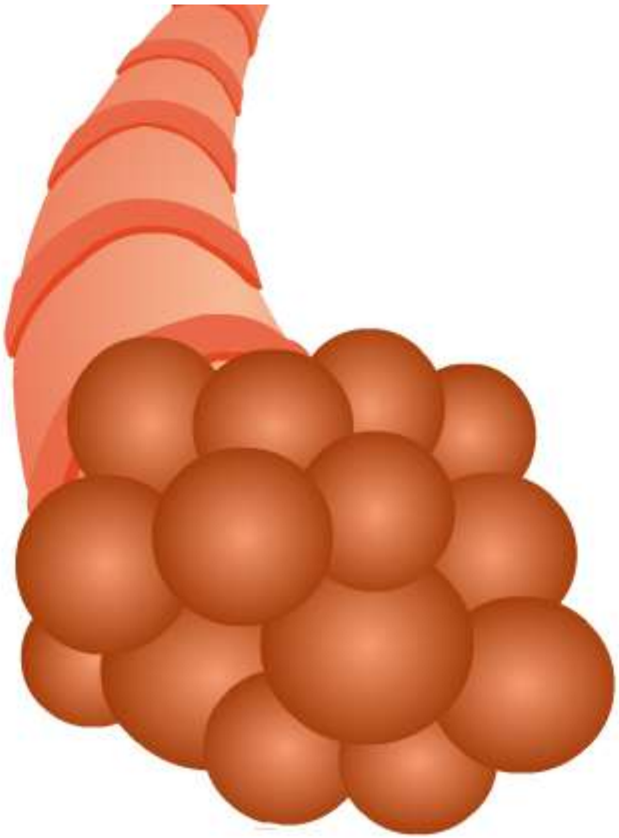
Respiratory complications

- Nosocomial pneumonia
- Ventilator-associated lung injury
- Gas trapping

Ventilator associated lung injury

- High pressures (barotrauma)
- High volumes (volutrauma)
- Shear injury

Gas trapping



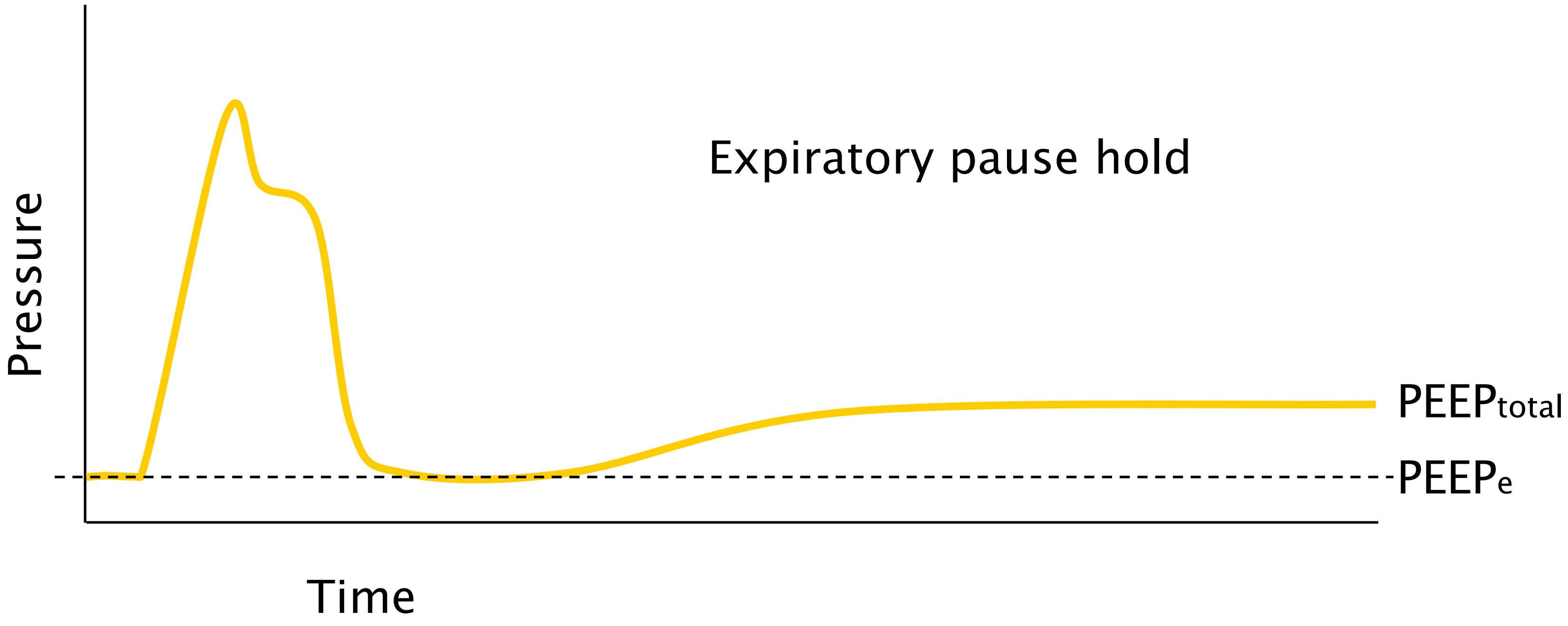
Gas trapping

- Predisposing factors:
  - Asthma or COPD
  - Long inspiratory time ($\Rightarrow$ expiratory time short)
  - High respiratory rate ($\Rightarrow$ absolute expiratory time short)
  - Large tidal volume

Gas trapping

- Effects
  - progressive hyperinflation of alveoli
  - progressive rise in end-expiratory pressure (intrinsic PEEP)

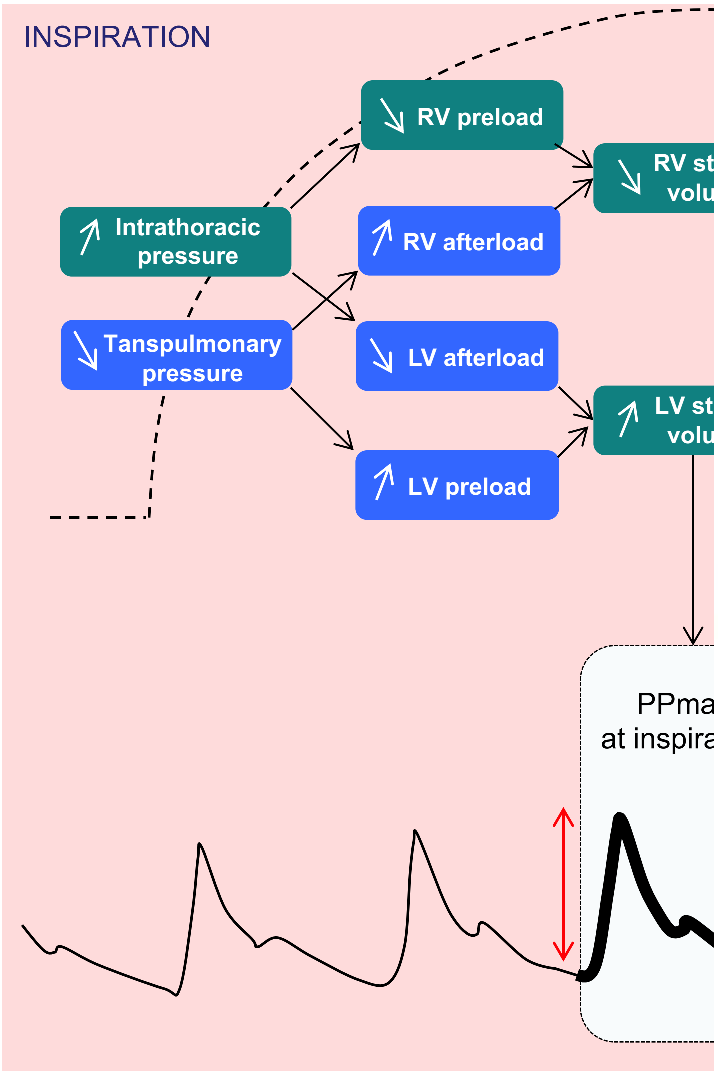
Intrinsic PEEP (PEEPi)

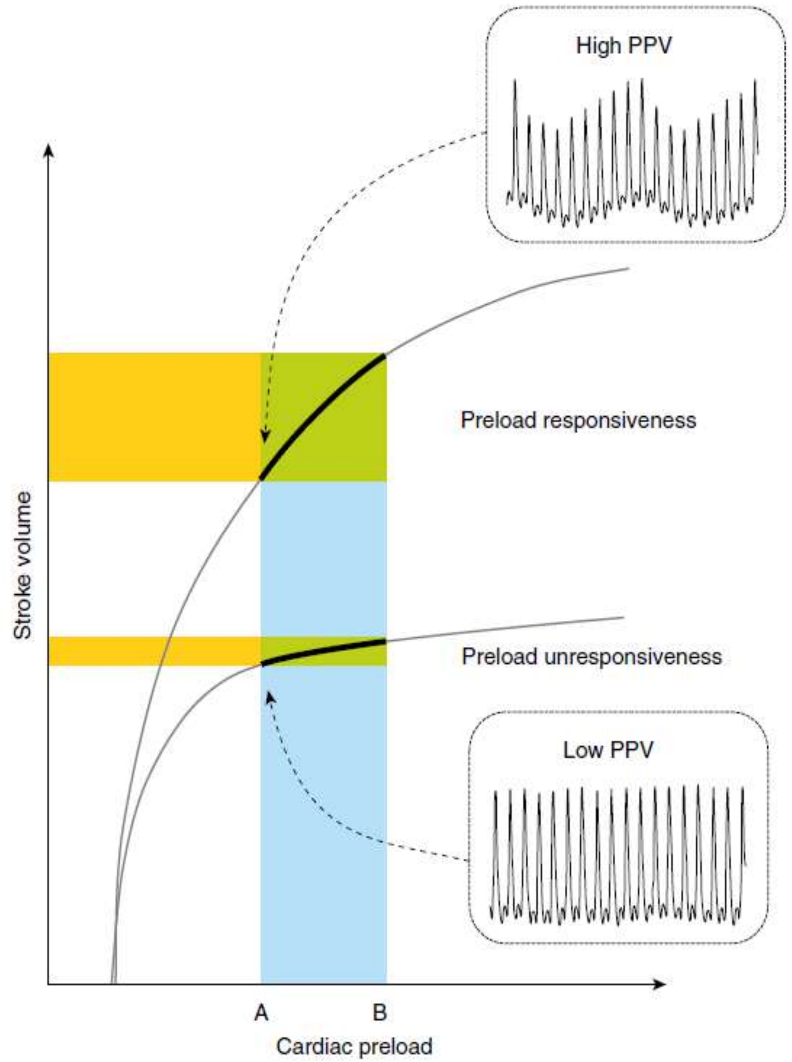


Gas trapping

- Adverse effects
  - Barotrauma
  - Cardiovascular compromise

Cardiovascular effects

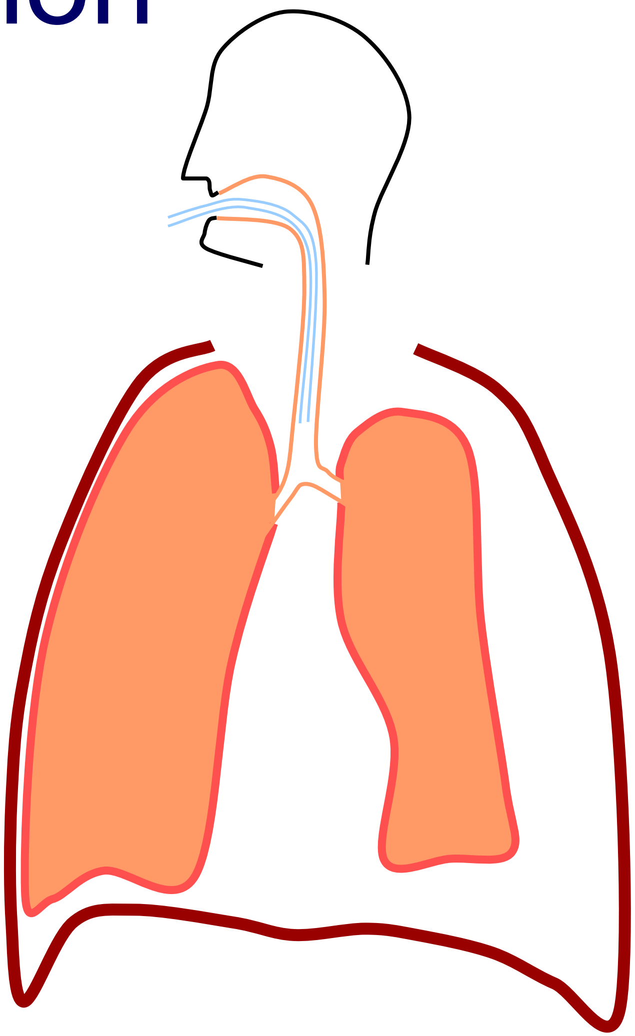




Teboul JL. Arterial pulse pressure variation with mechanical ventilation. Am J Respir Crit Care Med 2019

Hypotension

- Consider
  - Drug induced
  - Gas trapping
  - Tension pneumothorax



Summary I

- Physics: P, ΔP, $\dot{V}$, R, C, RC
- PaO_2 determined by
    - $F_I O_2$
    - Mean airway pressure (PEEP, I:E ratio)
- $PaCO_2$ determined by
    - Alveolar minute ventilation (RR, V_T)

Summary II

- Complications include
  - Hypotension
  - Ventilator-associated lung injury
  - Gas trapping

Any questions?

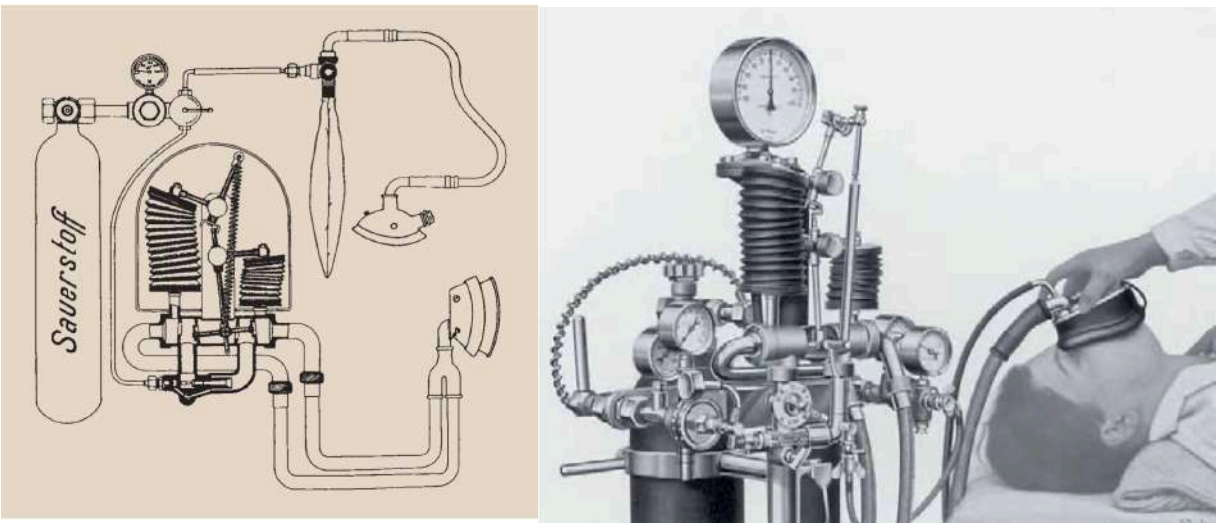
Mechanical ventilation Modes

Continuing development of BASIC is supported by an unrestricted educational grant from

MAQUET Dräger medical

A Dräger and Siemens Company

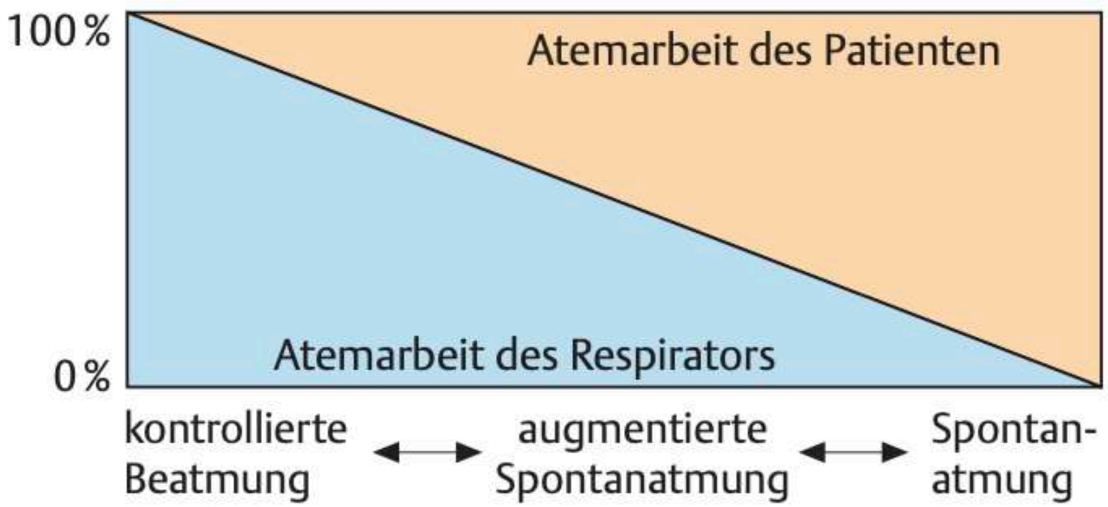
Modes



…

Pulmotor, Dräger, patented 1907

Modes



Change of pressure, volume and flow over time describes the different breathing modes

CMV

- Volume pre-set assist control
- Pressure pre-set assist control

ASB

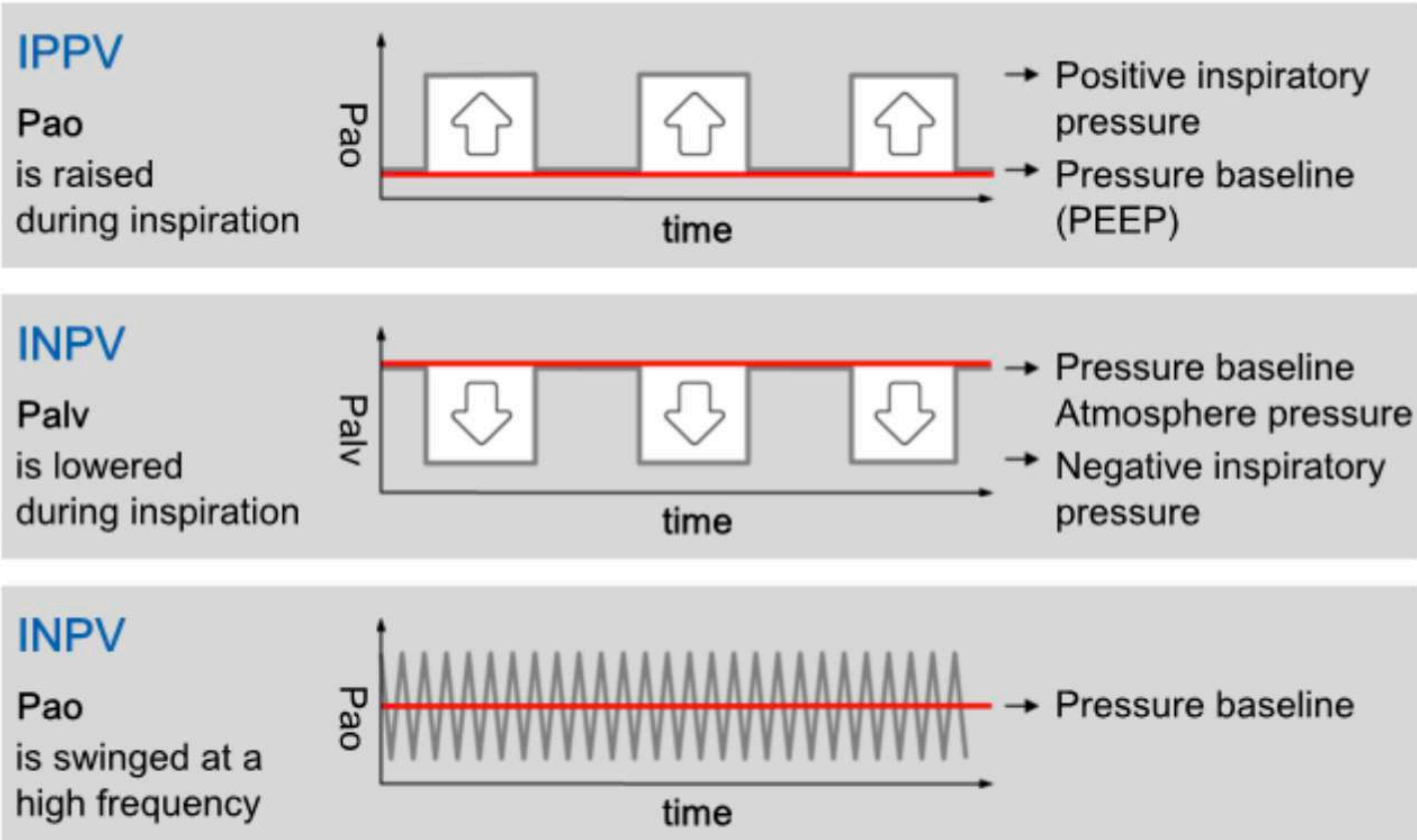
- Pressure support

Modes

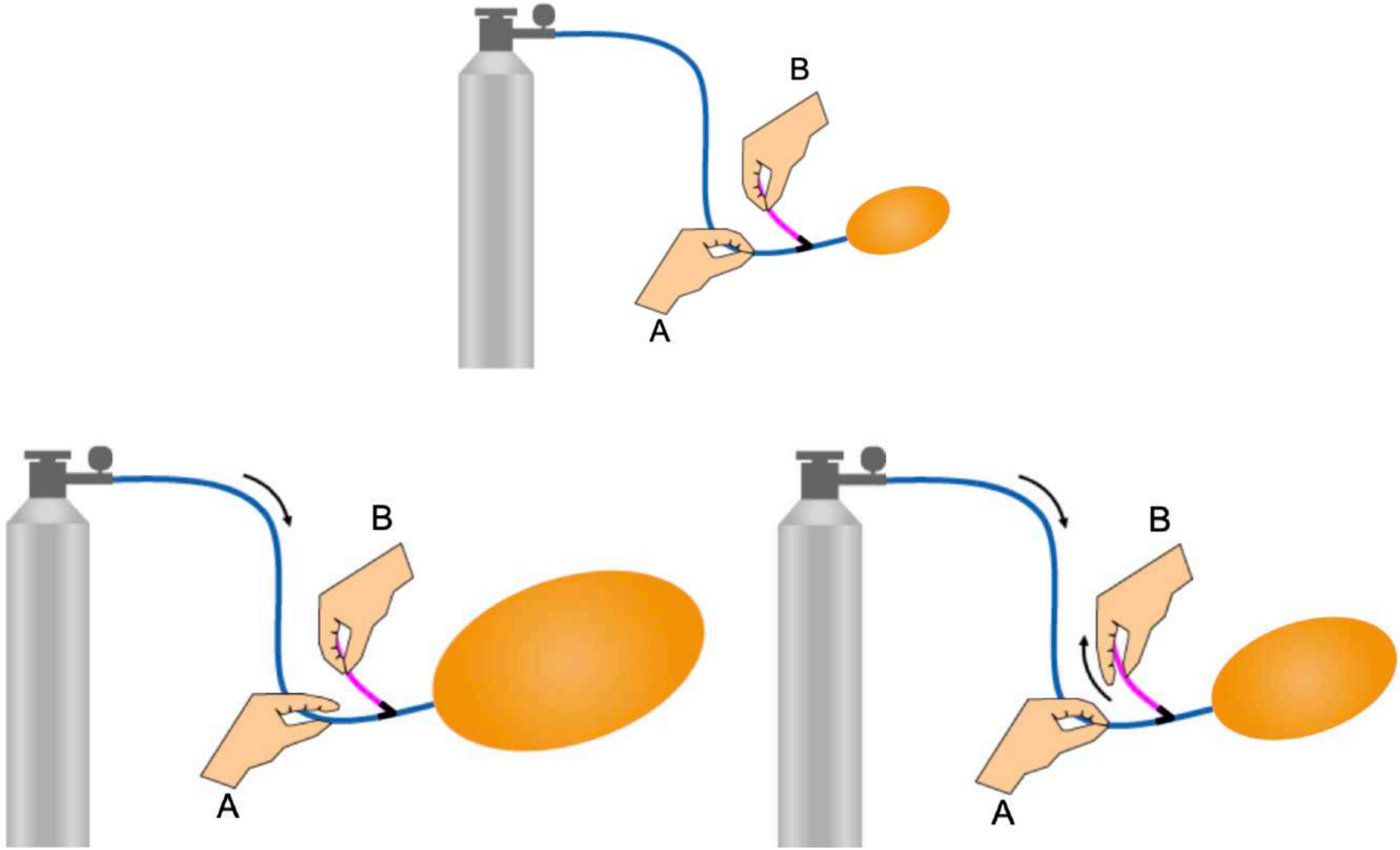
It's complicated, but easy…

Vendor	Hamilton Medical	Dräger	CareFusion	Covidien	Maquet	GE Medical	Chatburn
Ventilator	G5, S1	V500	AVEA	PB 840	SERVO-i	Carestation	---
Volume-CMV	(S)CMV	VC-AC	Volume A/C	AC-VC	Volume control*	VCV	VC-CMVs
Pressure-CMV	P-CMV	PC-AC	Pressure A/C	AC-PC	Pressure control	PCV	PC-CMVs
Adaptive-CMV	APVcmv	VC-AC + AutoFlow	PRVC A/C	AC-VC+	PRVC	PCV-VG	PC-CMVa
Volume-SIMV	SIMV	VC-SIMV	Volume SIMV	SIMV-VC	SIMV(VC) + PS	SIMV-VC	VC-IMVs,s
Pressure-SIMV	P-SIMV	PC-SIMV	Pressure SIMV	SIMV-PC	SIMV(PC) + PS	SIMV-PC	PC-IMVs,s
Adaptive-SIMV	APVsimv	VC-SIMV + AutoFlow	PRVC SIMV	SIMV-VC+	SIMV(PRVC) + PS	SIMV-PCVG	PC-IMVa,s
Pressure support	SPONT	SPN-CPAP / PS	CPAP / PSV	SPONT PSV	PS/CPAP	CPAP/PS	PC-CSVs
Volume support	VS	SPB-CPAP / VS	---	VS	VS	---	PC-CSVa
Biphasic	DuoPAP	PC BiPAP	APRV / BiPhasic	BiLevel	Bi-Vent	BiLevel	PC-IMVs,s
APRV	APRV	PC-APRV					PC-IMVs,s

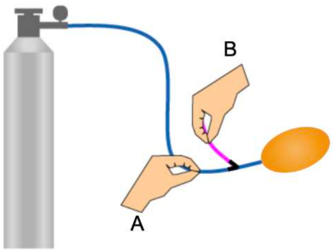
Modes

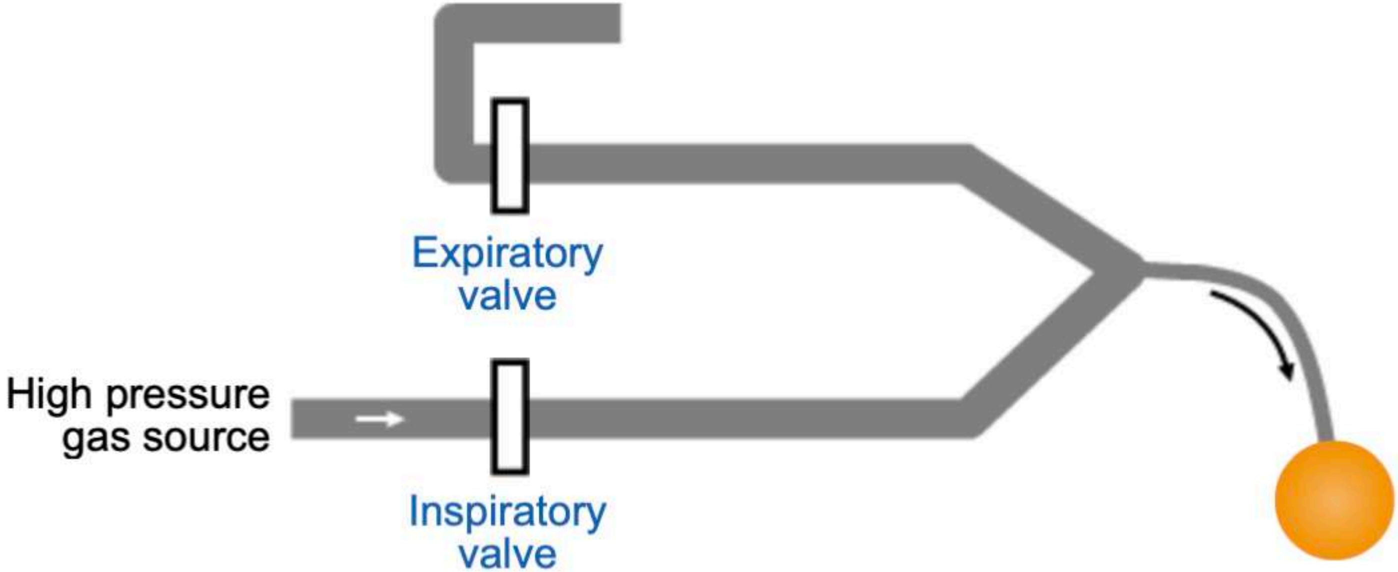

IPPV
Pao
is raised
during inspiration
Pao
time
Positive inspiratory
pressure
Pressure baseline
(PEEP)
INPV
Palv
is lowered
during inspiration
Palv
time
Pressure baseline
Atmosphere pressure
Negative inspiratory
pressure
INPV
Pao
is swinged at a
high frequency
Pao
time
Pressure baseline

Concept of a respirator

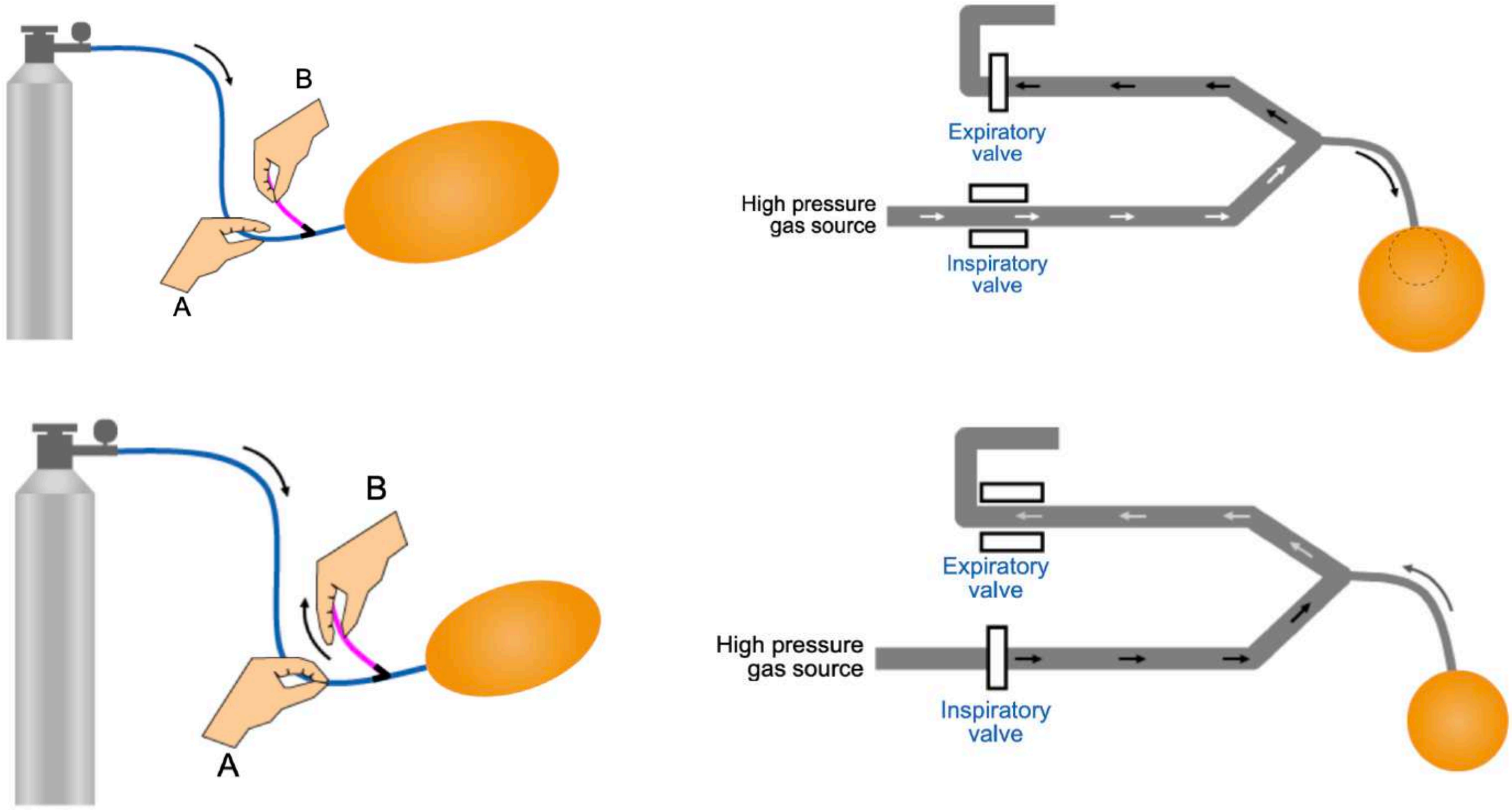


Concept of a respirator

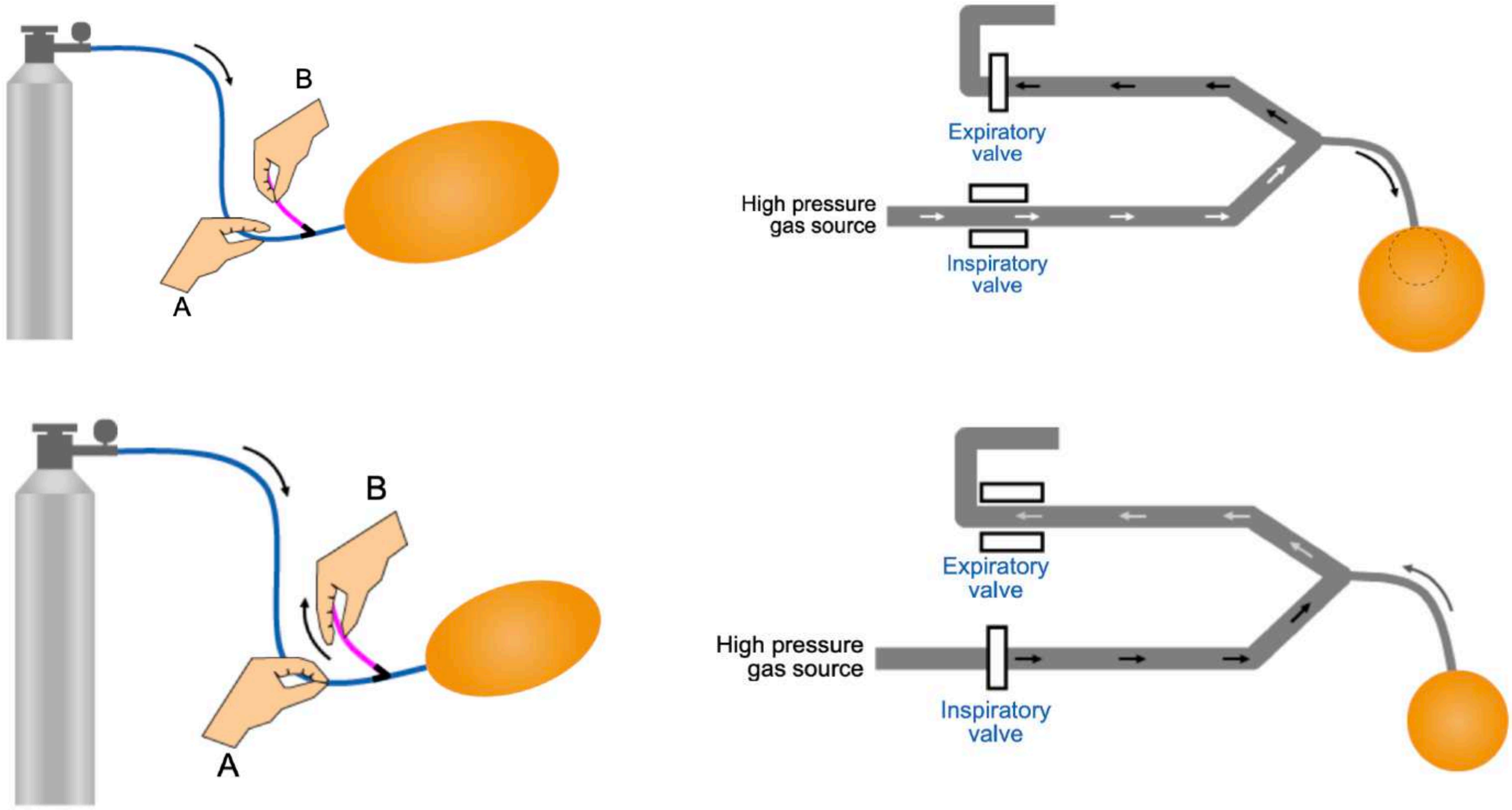




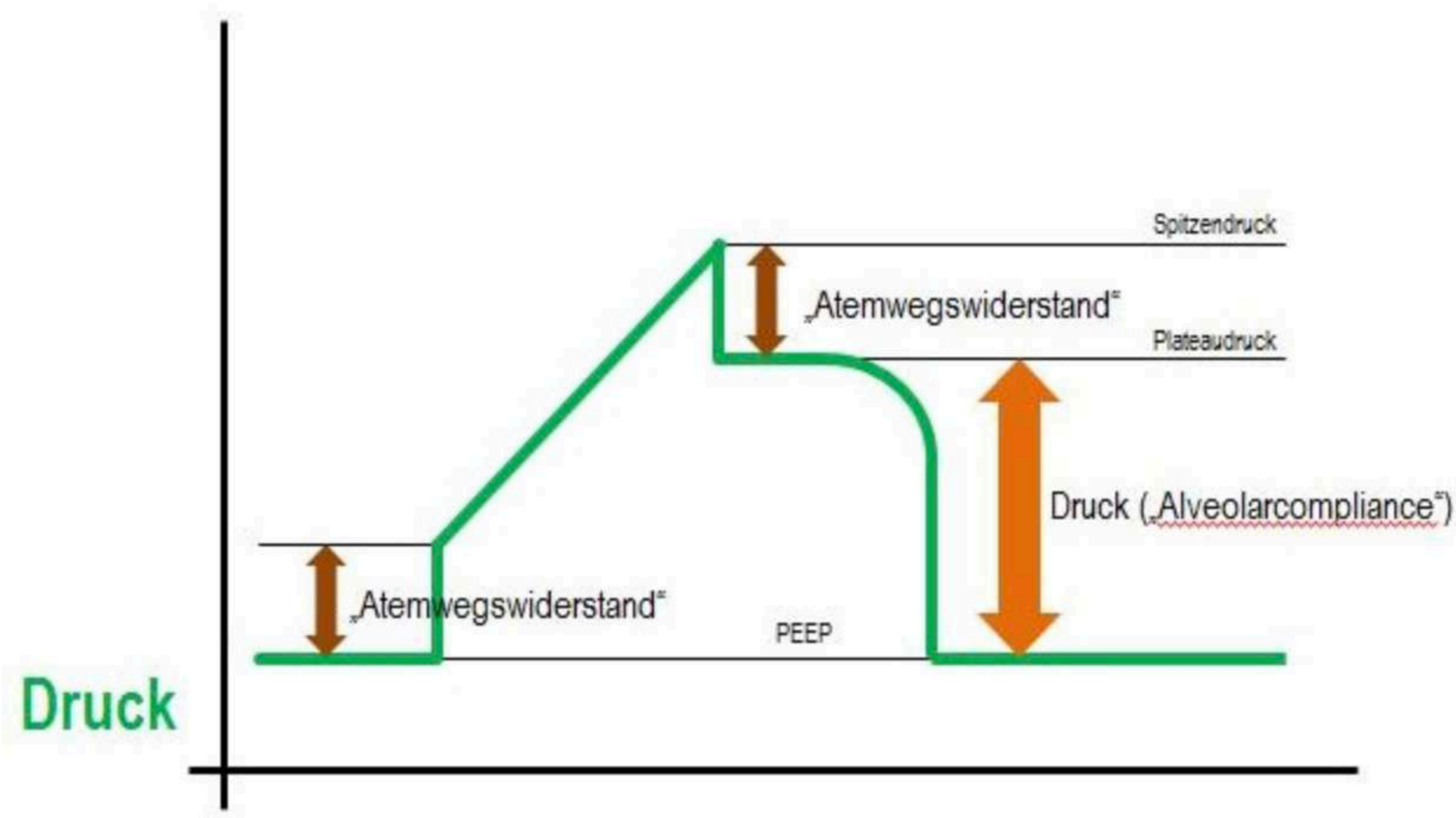
Concept of a respirator



Concept of a respirator

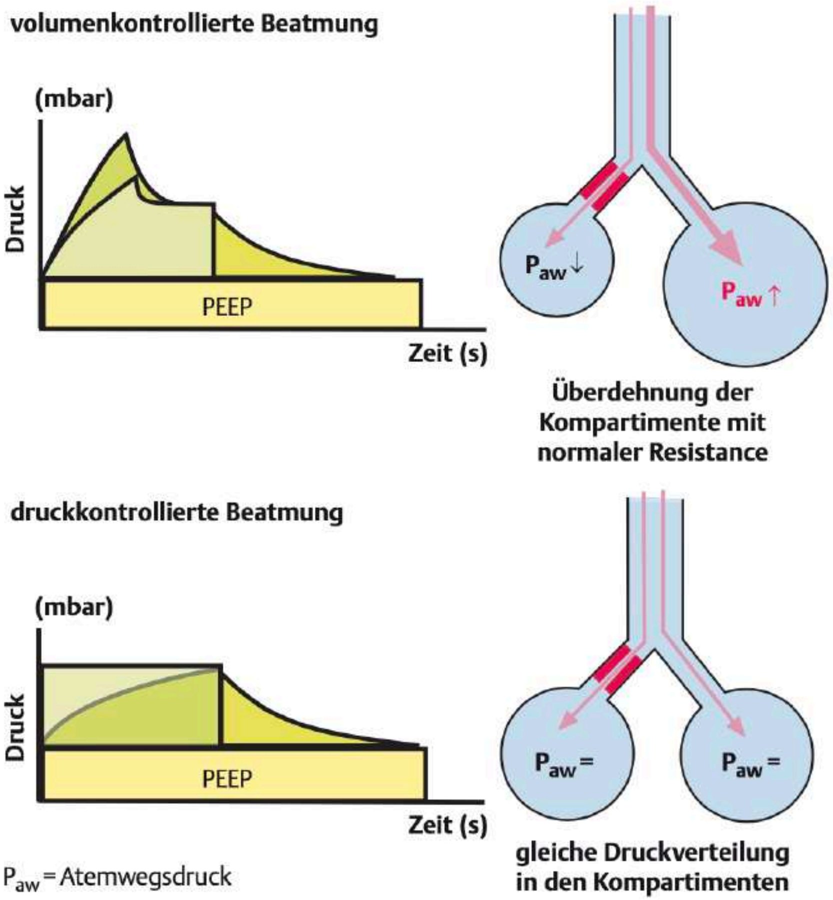


Pressure, Flow and Volume Curves

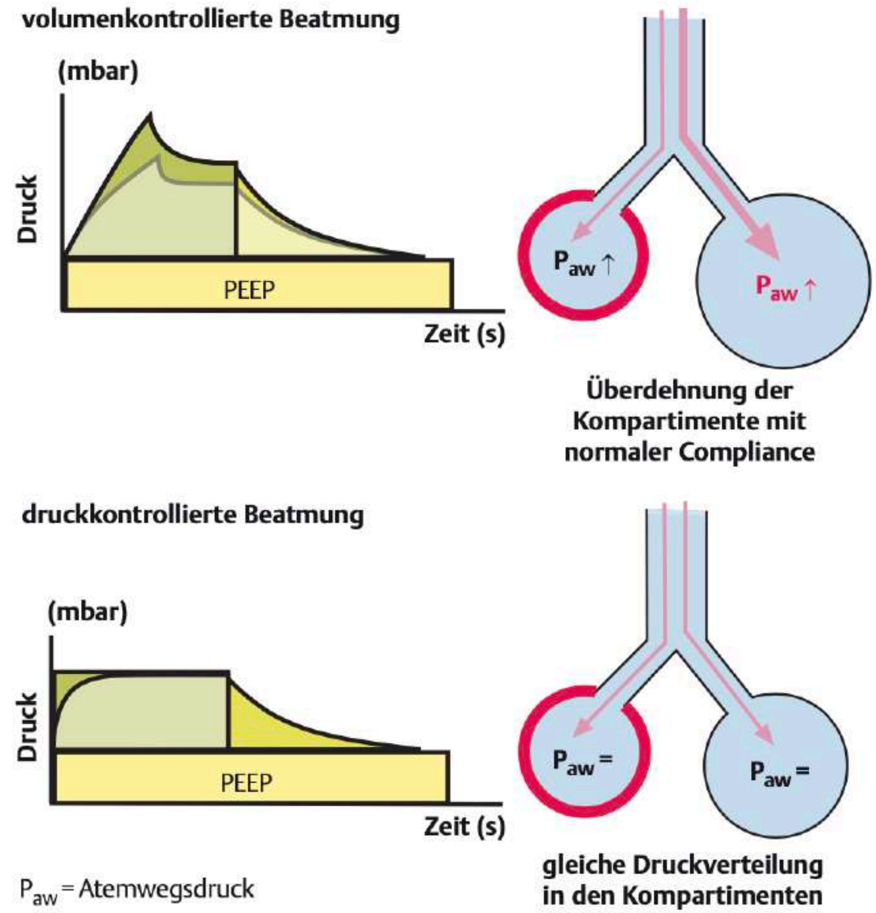


P-CMV vs V-CMV

obstructive patient

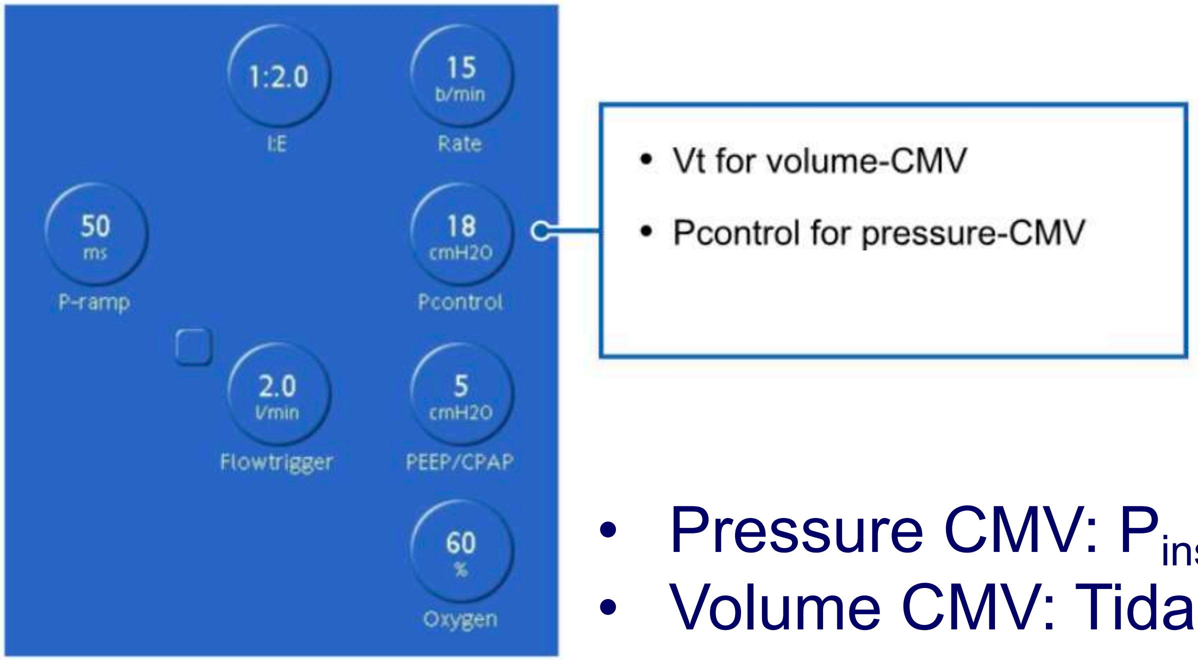


restrictive patient



Controlled mechanical ventilation (CMV)

Basic controls



- Pressure CMV: P_{insp}
- Volume CMV: Tidal volume

- Rate
- PEEP
- FiO2
- Ti or I:E

Controlled mechanical ventilation(CMV)

Basic settings

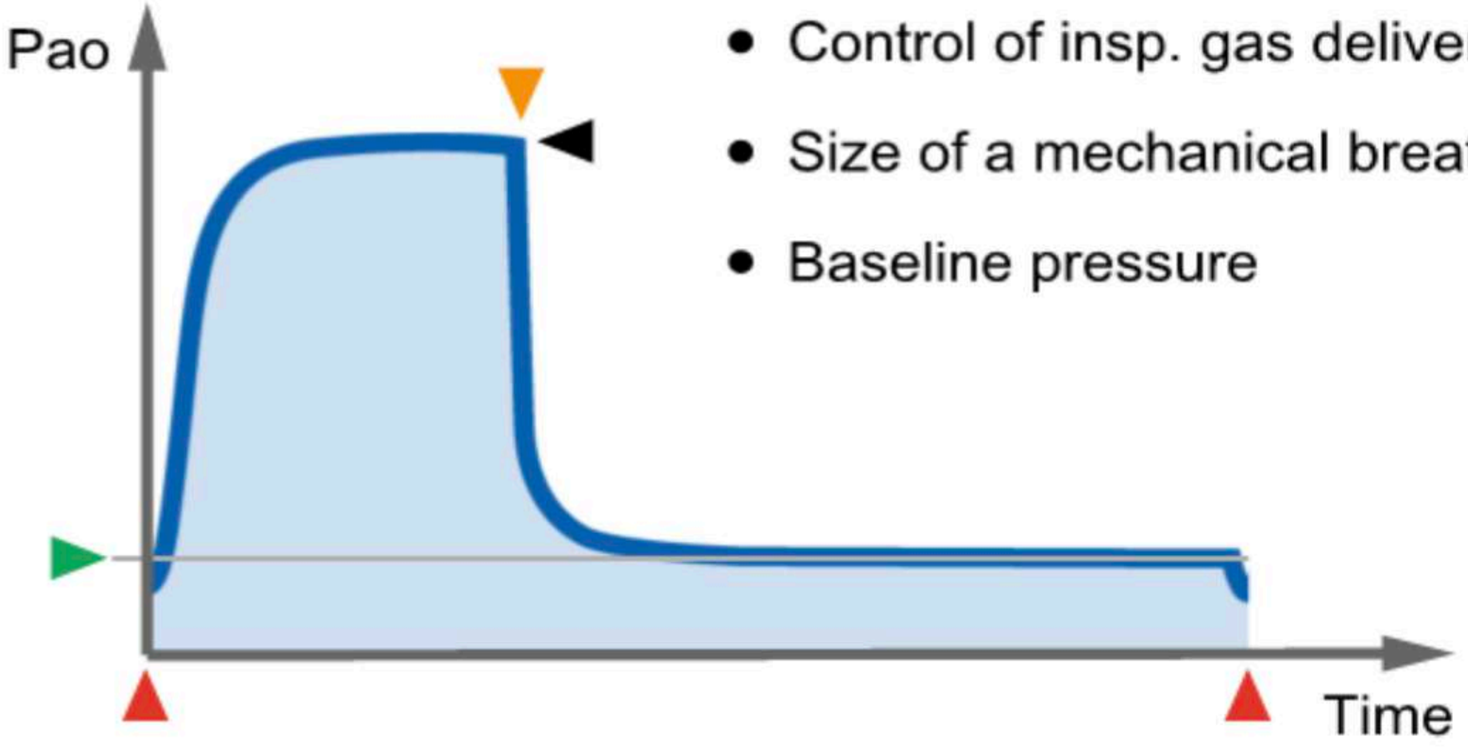
<table>
<tr><th></th><th>Rate</th><th>Tidal Volume</th><th>Phoch</th><th>I:E</th><th>PEEP</th><th>FiO2</th></tr>
<tr><td>PCV</td><td rowspan="2">10-15</td><td>6-8ml/kg*</td><td>10-12mbar über PEEP</td><td rowspan="2">1:2</td><td rowspan="2">5-8mbar</td><td rowspan="2">0.5</td></tr>
<tr><td>VCV</td><td>6-8ml/kg*</td><td>max 30mbar**</td></tr>
</table>

Goals
Normoventilation

- pH 7.35 - 7.45
- paCO2 35-45mmHg / 4.6-6kPa
- pO2 80-100mmHg / 10-13kPa

* PBW
** resp. P_{plat}-PEEP = max 15mbar = Driving pressure

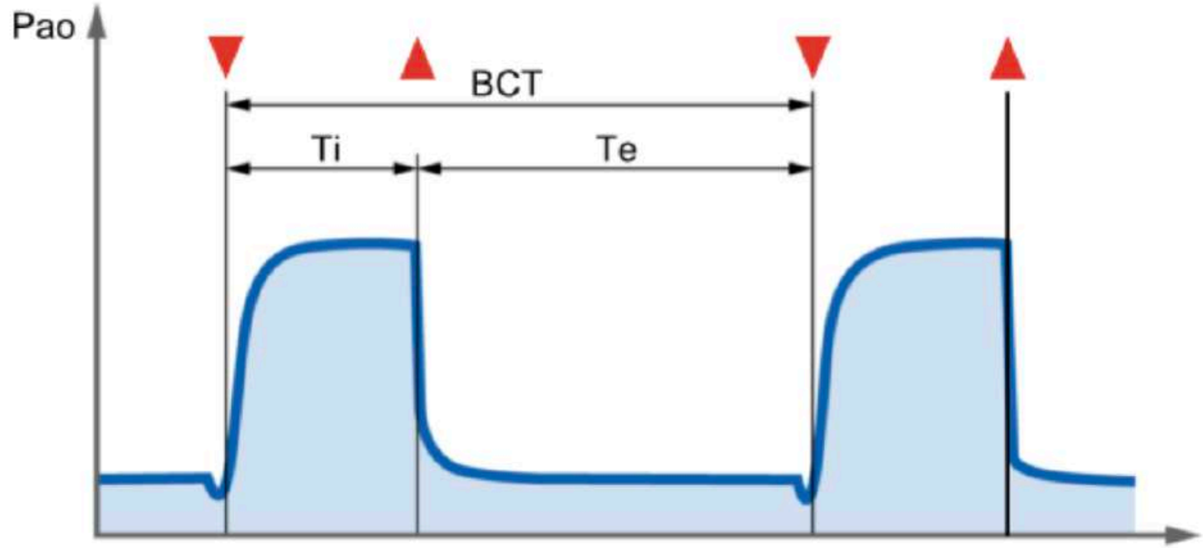
Basic settings



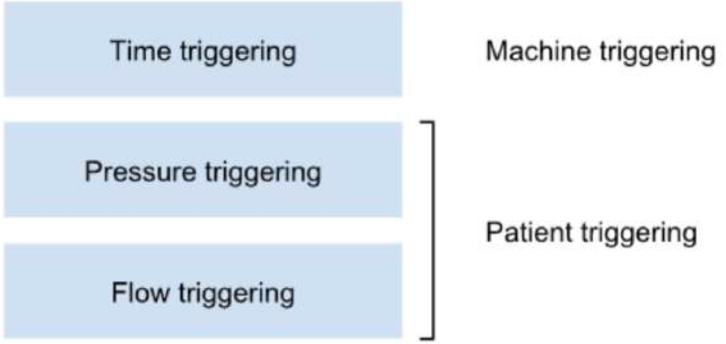
- Inspiration: start & stop
- Expiration: start & stop
- Control of insp. gas delivery
- Size of a mechanical breath
- Baseline pressure

Basic settings

Mechanical breath timing

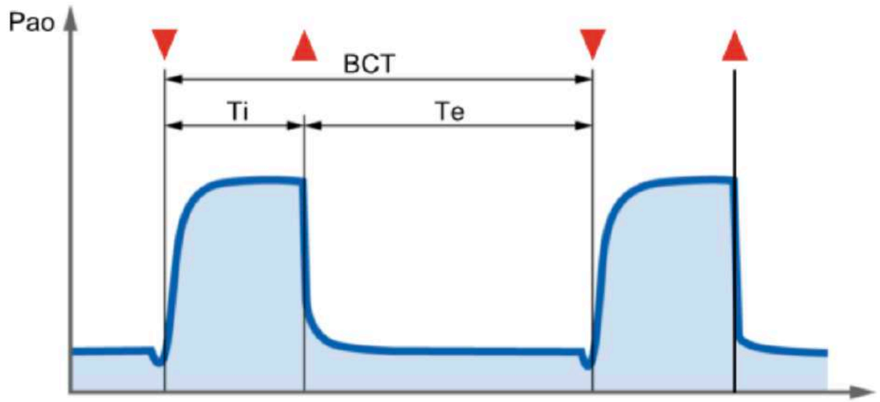


Triggering: refers to the time point when inspiration starts
Cycling: refers to the time point when inspiration ends

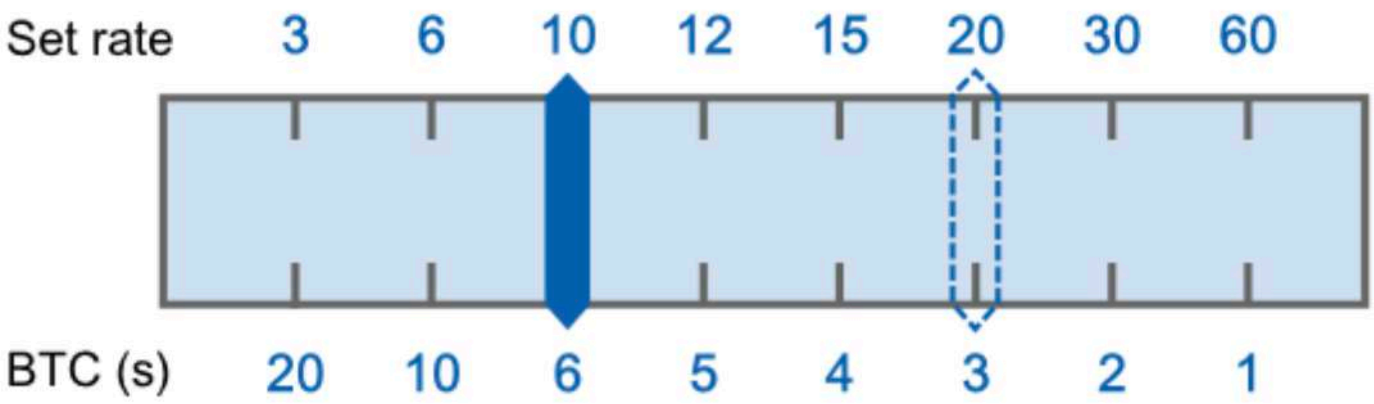


Basic settings

Machine triggering: Time triggering

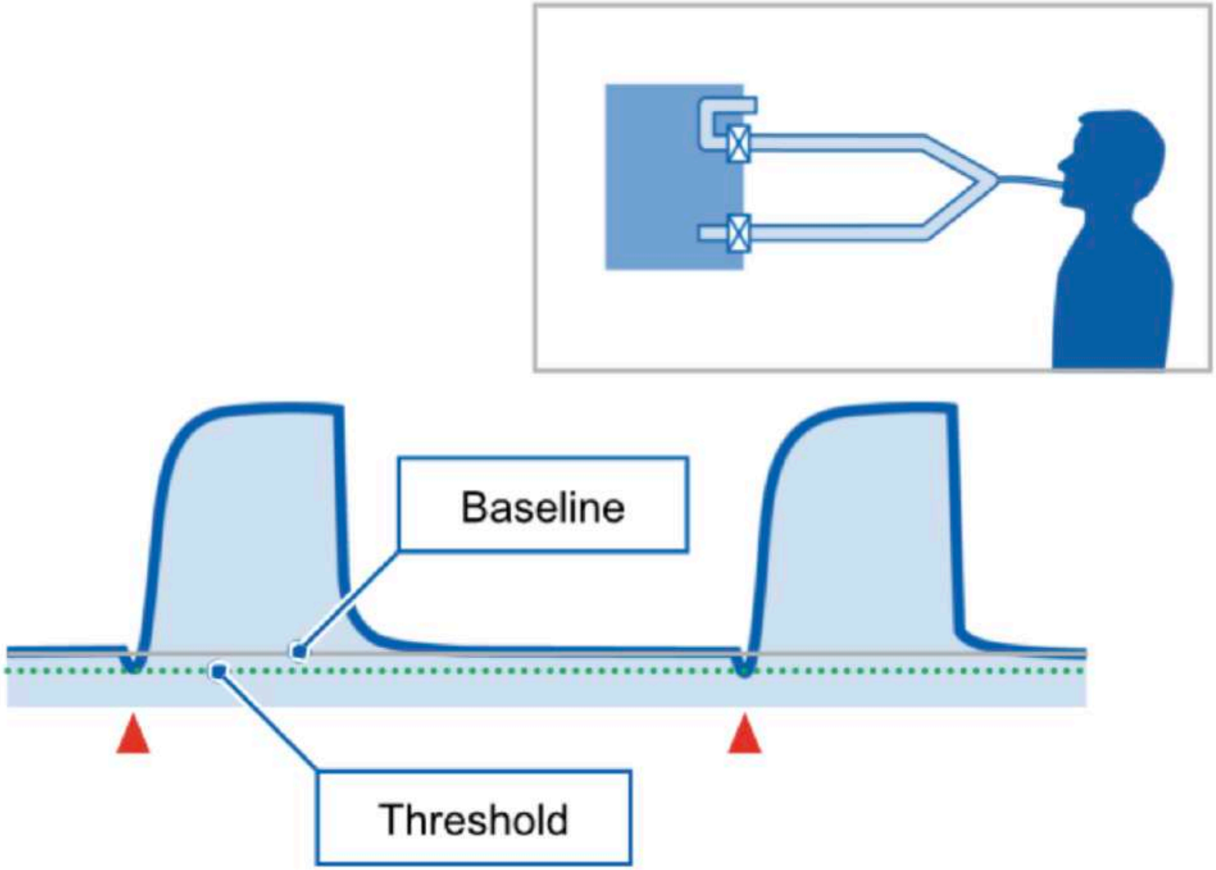


$$BCT\ (s) = \frac{60}{\text{Set rate (b/min)}}$$



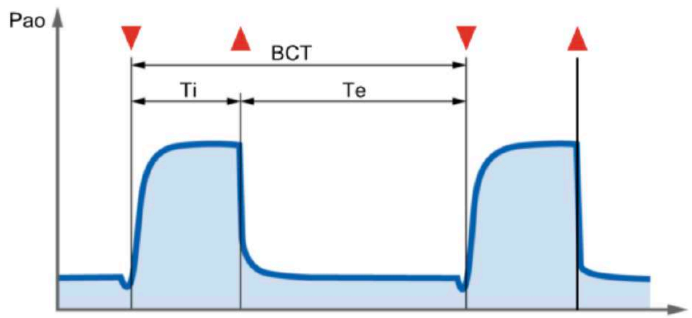
Basic settings

Patient triggering: Pressure triggering



If the pressure drop reaches a set virtual threshold, the ventilator is triggered and starts the delivery of inspiratory gas.

Basic settings
Cycling



Time cycling (machine)

For passive patients

Flow cycling (patient)

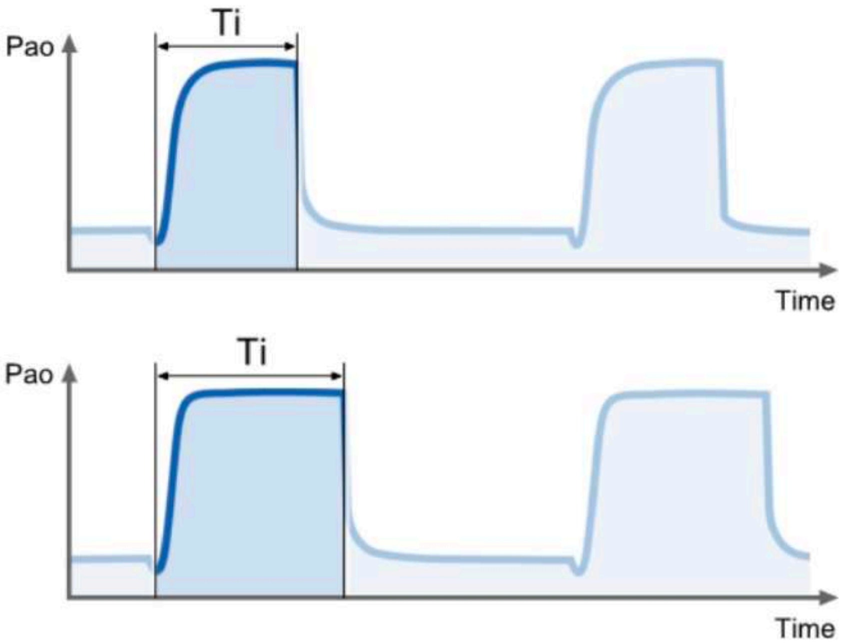
For active patients

Basic settings

Cycling: Ti

Time cycling
(machine)

For passive patients



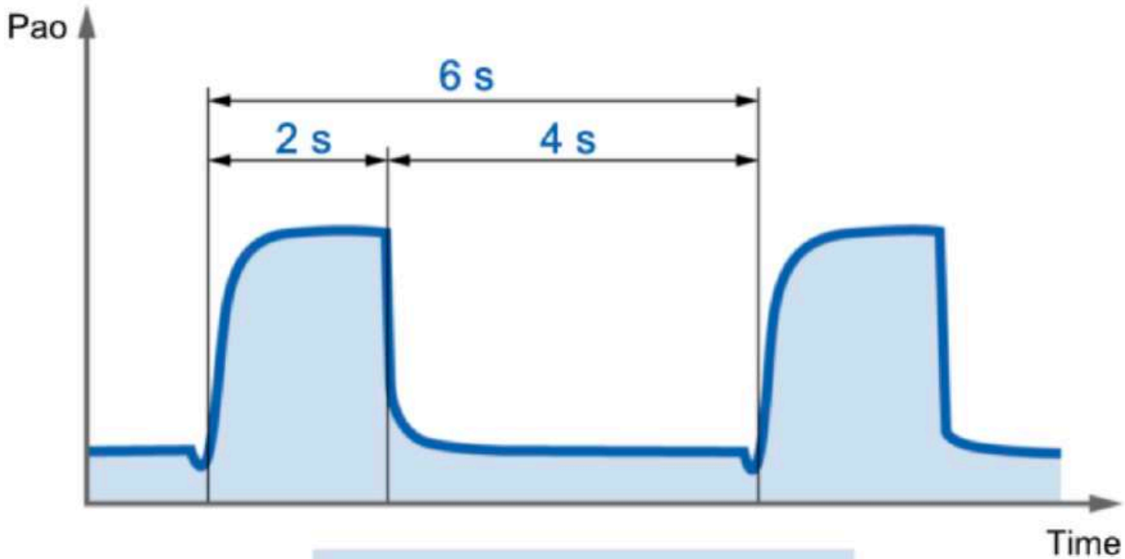
Operator sets directly Ti in seconds. The ventilator switches from inspiration to expiration when the set Ti is over

Basic settings

Cycling: I:E ratio

Time cycling
(machine)

For passive patients



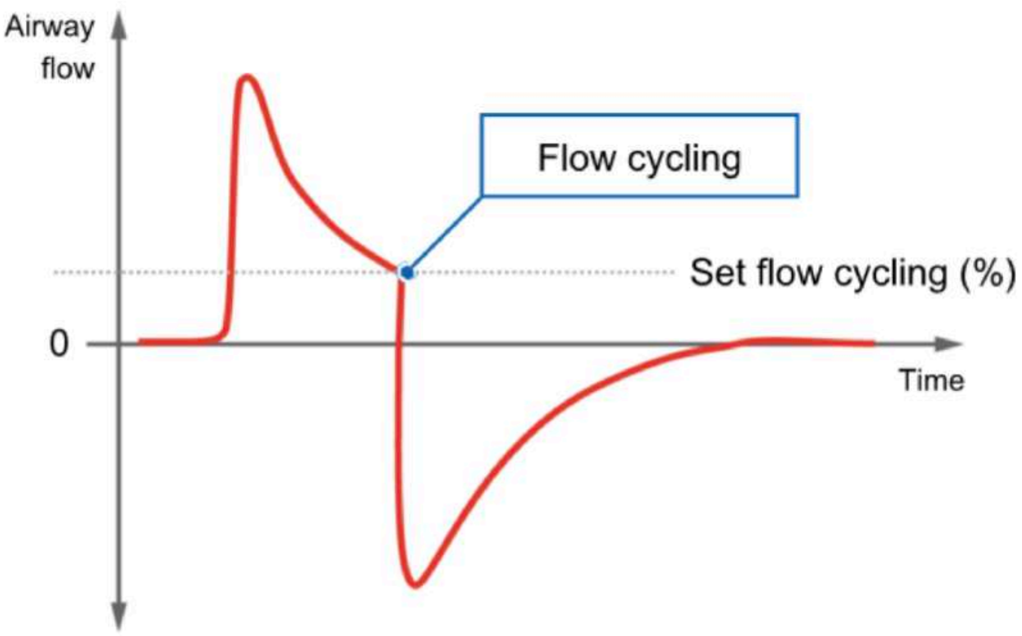
I:E ratio = 1:2

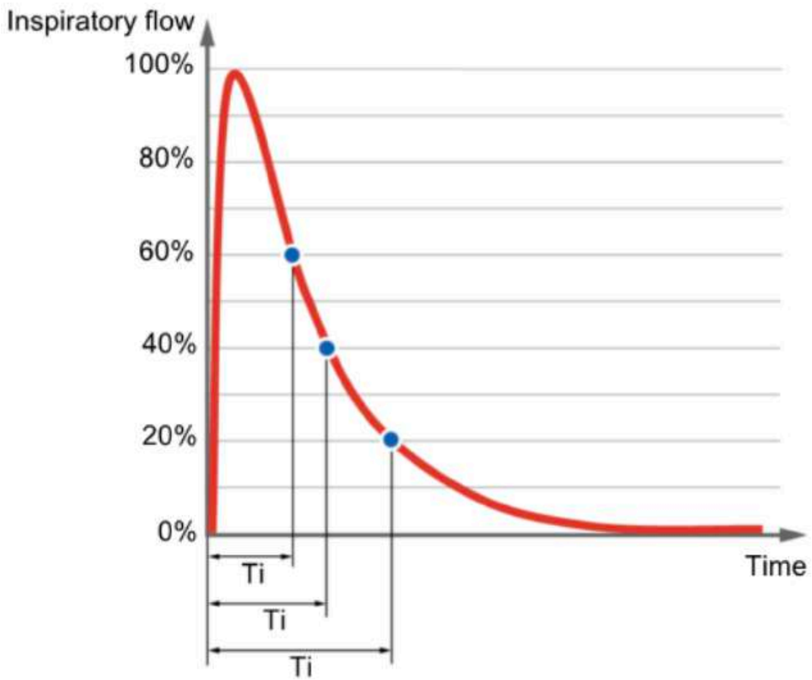
Basic settings

Cycling: Flow cycling

Flow cycling
(patient)

For active patients



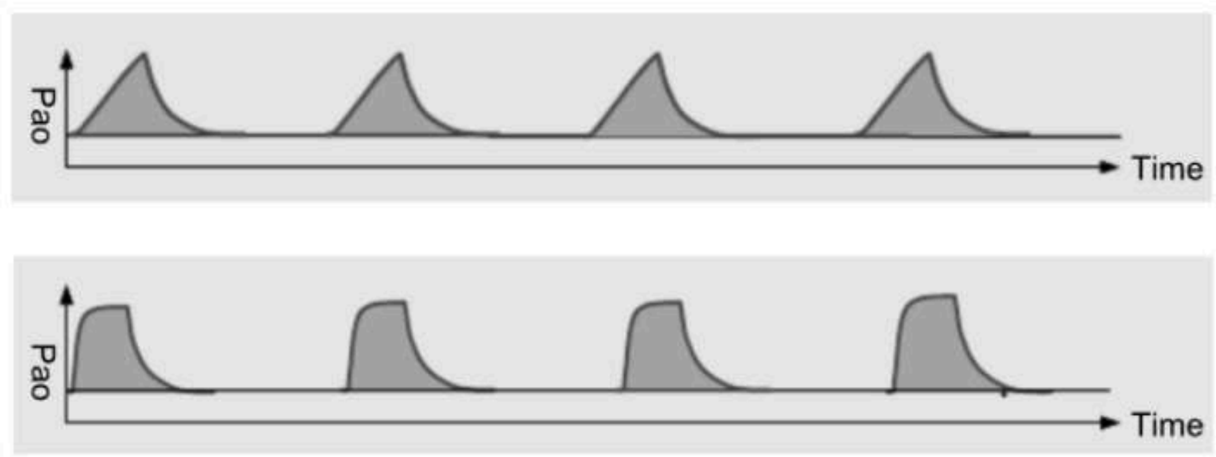


Modes

Passive Patient

Volume-CMV

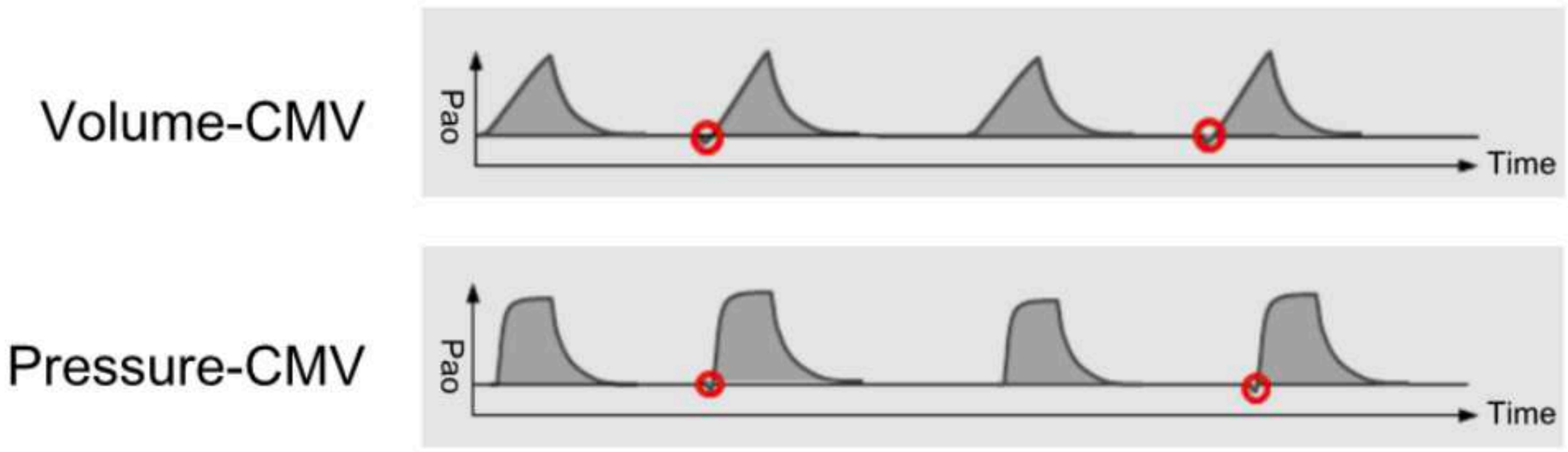
Pressure-CMV



all breaths are VTMB

Modes

Partially active Patient

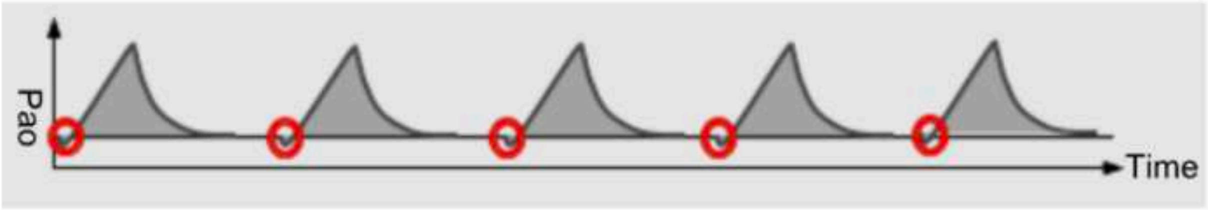


Mix of VTMB and PTMB

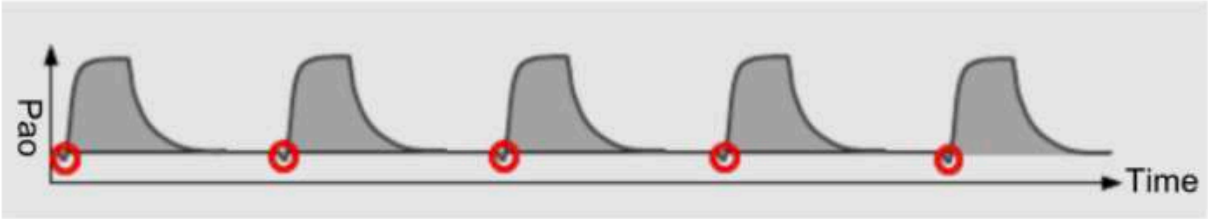
Modes

Active Patient

Volume-CMV



Pressure-CMV

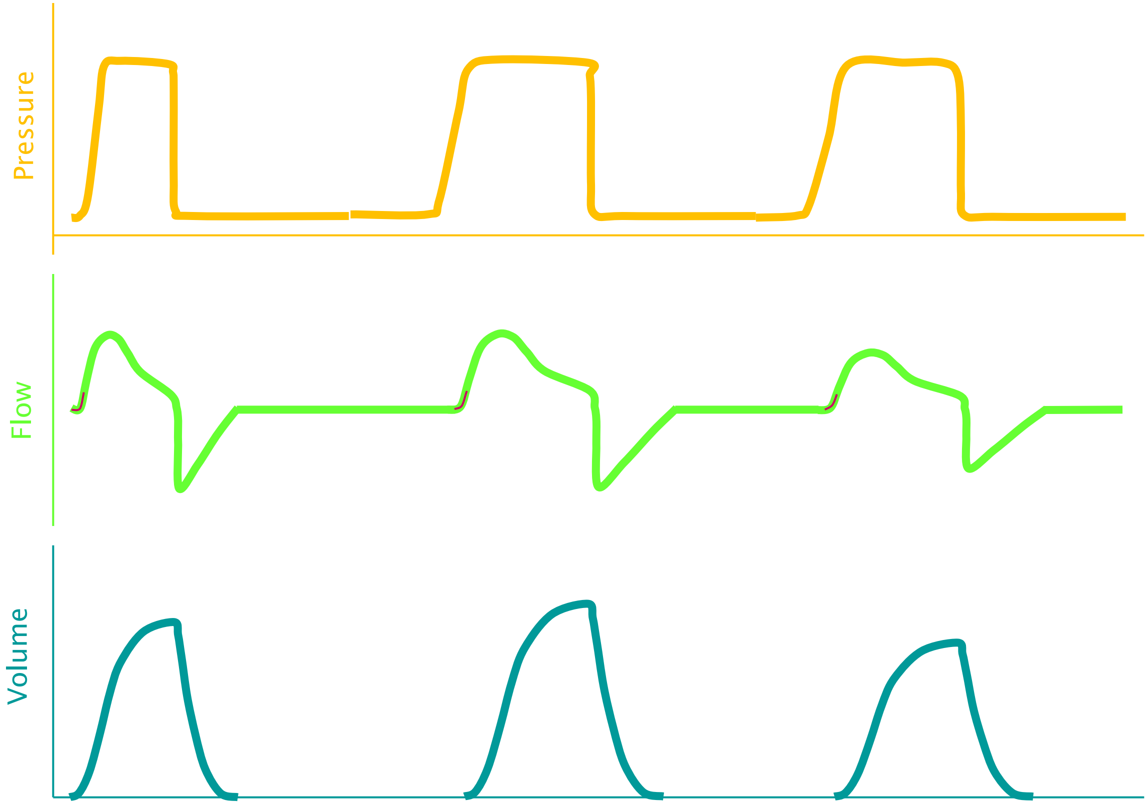


All breaths are PTMB

Pressure support

- Nomenclature
  - Inspiratory assist
  - Assisted spontaneous breathing

Pressure support



Pressure support

- Pressure support of 3.5-14.5 cmH_2O required to overcome the additional work of breathing due to breathing through ETT and demand valve
- Patients who require pressure support of < 6 cmH_2O can probably be extubated

Any questions?

Sedation, Analgesia, Nutrition

Continuing development of BASIC is supported by an unrestricted educational grant from

MAQUET Dräger medical A Dräger and Siemens Company

„Der intensivmedizinisch behandelte Patient soll wach, aufmerksam, schmerz-, angst- und delirfrei sein, um an seiner Behandlung und Genesung aktiv teilnehmen zu können.“

Sedation

Sedation: Why?

- Enhance tolerance of endotracheal tube & mechanical ventilation
- Allow therapeutic & monitoring procedures
- Control cerebral oxygen demand
- Relieve anxiety

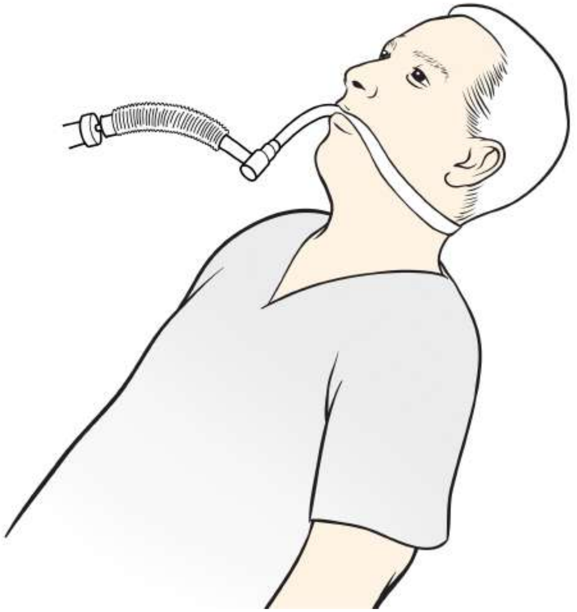
Sedation

- Pain
  - Analgesia not sedation
- Delirium
  - Treat cause
  - Anti-psychotic if necessary
  - Not sedation !!

Eine der bedeutendsten Präventionsstrategien des Delirs stellt die Vermeidung einer Sedation dar.

Sedation

- Appropriate level
  - Frequent repeated re-appraisal
  - In general lighter (but calm) better than deeper
  - Exceptions:
    - Difficult to ventilate
    - High ICP



Continuous infusion of sedative drugs…

- provides a more constant level of sedation
- may increase patients' comfort

… but

- prolong the duration of mechanical ventilation
- prolong the ICU lengths of stay
- increase costs
- increase the risk for developing delirium
- increase mortality

Kress JP. et al. (2000). Daily interruption of sedative infusion in critically ill patients undergoing mechanical ventilation. New England Journal of Medicine 342:1471-1477

ZIEL

Patientenorientiertes Behandlungskonzept zur bedarfsadaptierten Analgesie und Sedation zur Vermeidung von Angst und Delir mit individueller patientenspezifischer Festlegung von Therapiezielen

Systemic evaluation of pain and agitation...

- decreases
  - the incidence of pain and agitation,
  - the duration of mechanical ventilation
  - the incidence of nosocomial infection
  - ICU lengths of stay
  - mortality

Jakob, S.M., et al., Sedation and weaning from mechanical ventilation: effects of process optimization outside a clinical trial. J Crit Care, 2007. 22(3): p. 219-28

Changes G et al. Impact of systematic evaluation of pain and agitation in an intensive care unit. Crti Care Med. 2006 Jun. 34(6):1691-9

Sedation

- Titrate sedation to achieve appropriate level
  - Target sedation score may help
  - Beware decreased elimination due to organ failure
  - Consider drug pharmacokinetics

Evaluation of sedation

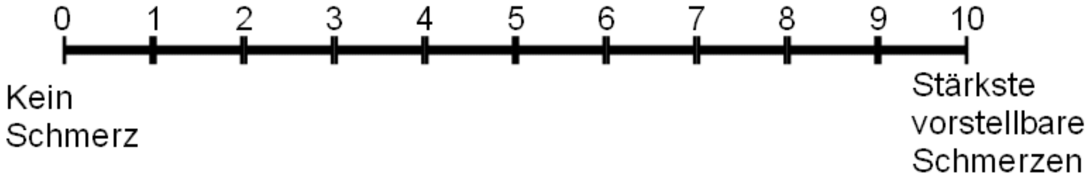
Medscape

Richmond Agitation and Sedation Scale (RASS)		
+4	Combative	violent, immediate danger to staff
+3	Very Agitated	Pulls or removes tube(s) or catheter(s); aggressive
+2	Agitated	Frequent non-purposeful movement, fights ventilator
+1	Restless	Anxious, apprehensive but movements not aggressive or vigorous
0	Alert & calm	
-1	Drowsy	Not fully alert, but has sustained awakening to *voice* (eye opening & contact ≥ 10 sec)
-2	Light sedation	Briefly awakens to *voice* (eye opening & contact < 10 sec)
-3	Moderate sedation	Movement or eye-opening to *voice* (but no eye contact)
-4	Deep sedation	No response to voice, but movement or eye opening to *physical* stimulation
-5	Unarousable	No response to *voice or physical* stimulation

Source: Pain Manag Nurs © 2009 W.B. Saunders

Evaluation of pain

- Numeric rating scale
- Behavioral pain scale
- ZOPA Score….



Sedation

- Consider adverse effects
  - Caution in haemodynamically unstable patients

Sedative Drugs

- Midazolam
  - Boluses: 1-2 mg
  - Infusion: 0-10 mg/h
- Propofol
  - Boluses: 10-20 mg
  - Infusion: 0-4 mg/kg/h
- Dexmedetomidin
- Clonidine, Ketamine…

Drugs: Analgetics

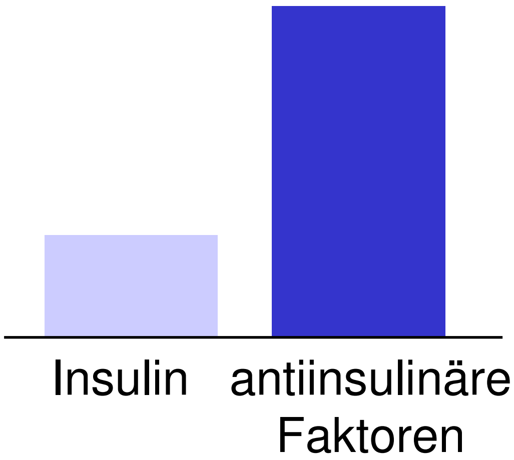
- Non-Opioid Analgetics
  - Paracetamol
  - Metamizole
  - NSAID
- Opioid Analgetics
  - Morphine
  - Sufentanil
  - Fentanyl
  - Remifentanil

Nutrition

How, what, when, how much?

Akutphase → Übergangsphase → Reparationsph.

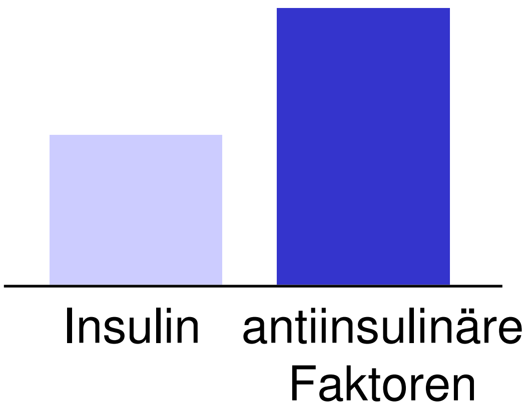
"Aggressionsphase"
Stunden



Insulin supprimiert
antiinsulinäre Faktoren überwiegend

Keine Ernährung

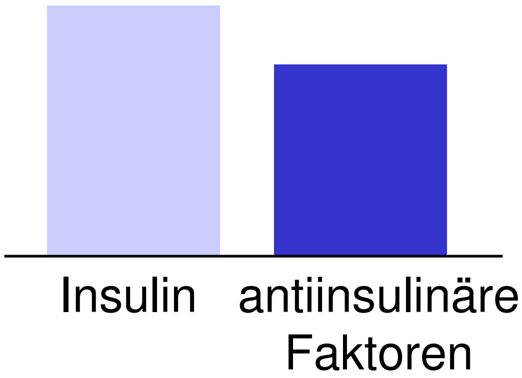
"Postaggressionsphase"
Tage



Insulin stimuliert
antiinsulinäre Faktoren überwiegend

Stufenweiser Nahrungsaufbau

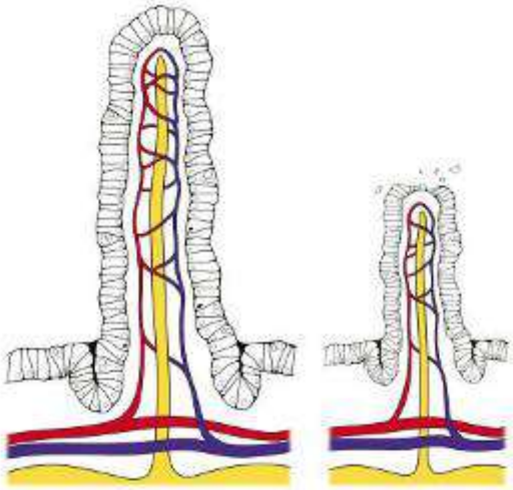
"Rekonvaleszenzphase"
Wochen



Insulin dominiert
antiinsulinäre Faktoren normalisiert

Volle Ernährung

How?



- Enteral
  - May prevent atrophy and possible loss of barrier function
  - Gastroesophageal reflux
- Parenteral
  - Intravenous
  - Higher complication rate ??
  - indicated when enteral nutrition is not possible or has failed

How much?

- Basal energy expenditure (BEE, kcal/day) = 25 x Body weight (kg)
- Adjustment in hypermetabolic conditions
  - Fever: BEE x 1.1 (for each ^{0}C above the normal body temperature)
  - Mild to moderate stress: BEE x 1.2
  - Moderate to severe stress: BEE x 1.4
- Daily protein requirements
  - 1-2 g/kg
  - Hypercatabolism: 2-3 g/kg

How much?

- Commercial feed
  = 1-1.3 kcal/ml

Example: 70 kg x 25 kcal/d = 1750 kcal/d

(1750 kcal/d/1.3 kcal/ml)= 1340 ml/d

Parenterale Ernährung niereninsuffizienten Patieten

- **Ohne Nierenersatzverfahren**

Überlege höher konzentrierte Sondennahrung im Sinne einer Volumeneinsparung

- **Mit Nierenersatzverfahren**

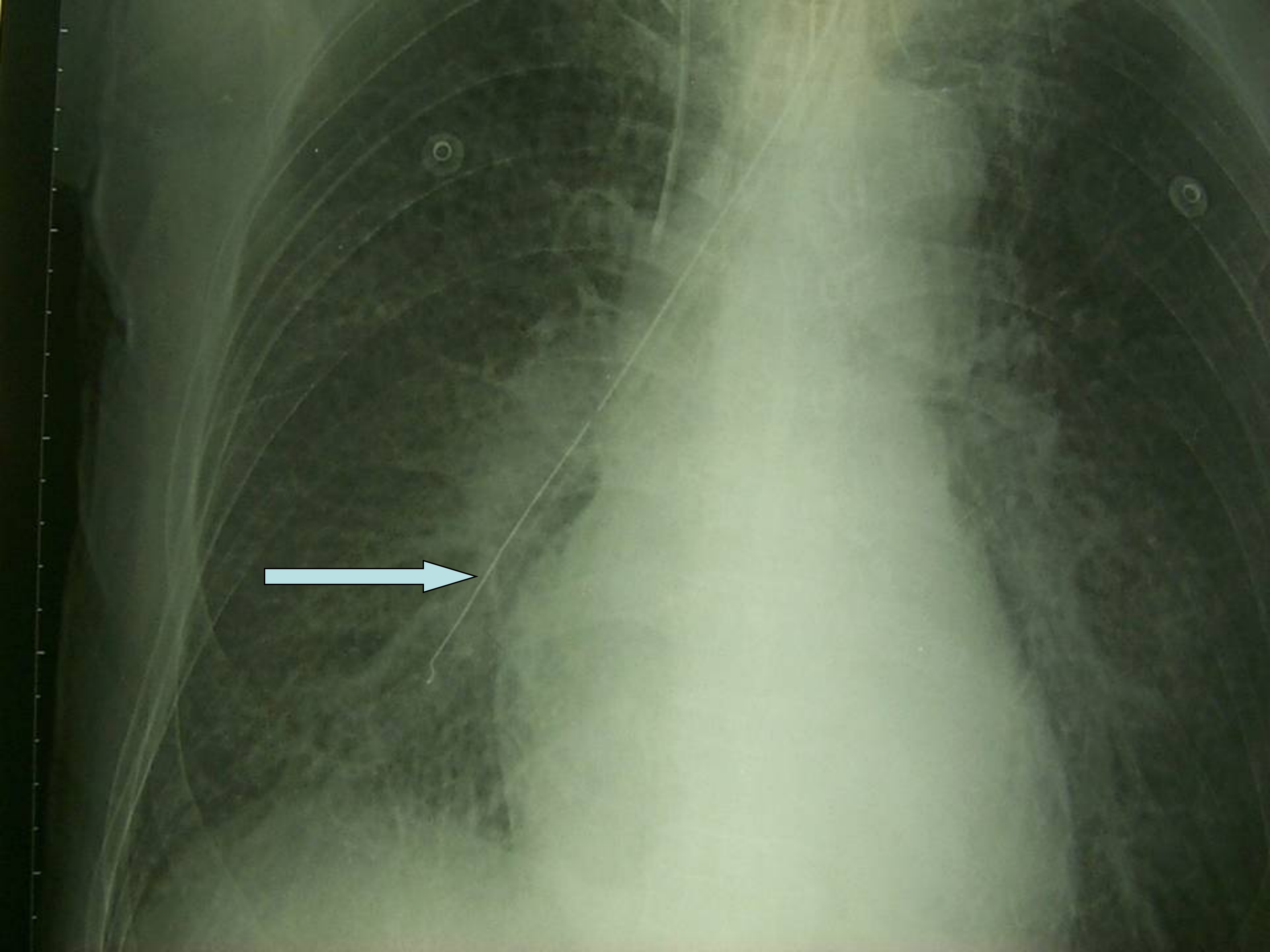
Denke an Elektrolyt- Spurenelement-, Vitaminverlust….

When?

- Enteral as soon as possible (24-48h)
- Early feeding
  - Reduced infection
  - Better wound healing
  - Prior malnutrition - feed earlier (1-2d)
- Parenteral – can wait 7 days

Practical aspects

- Insert feeding tube
  - Usually nasogastric
  - Check position on CXR

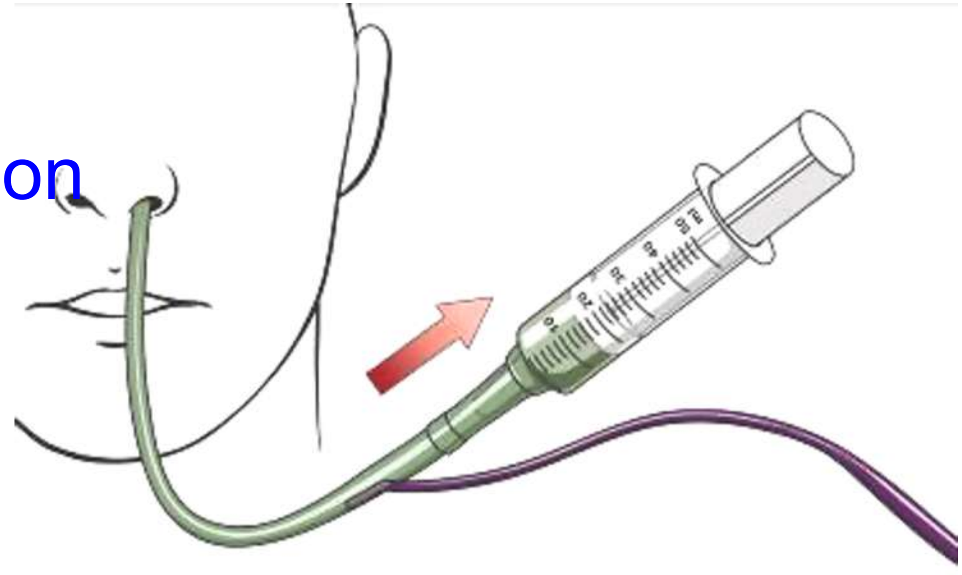


Practical aspects

- Start with 5 kcal/kg/h
- Aspirate NG every 4 hours
- Stop feeding if aspirate >200-400 ml
- Otherwise return aspirate to patient & continue feeding
- Full feeding within 48h

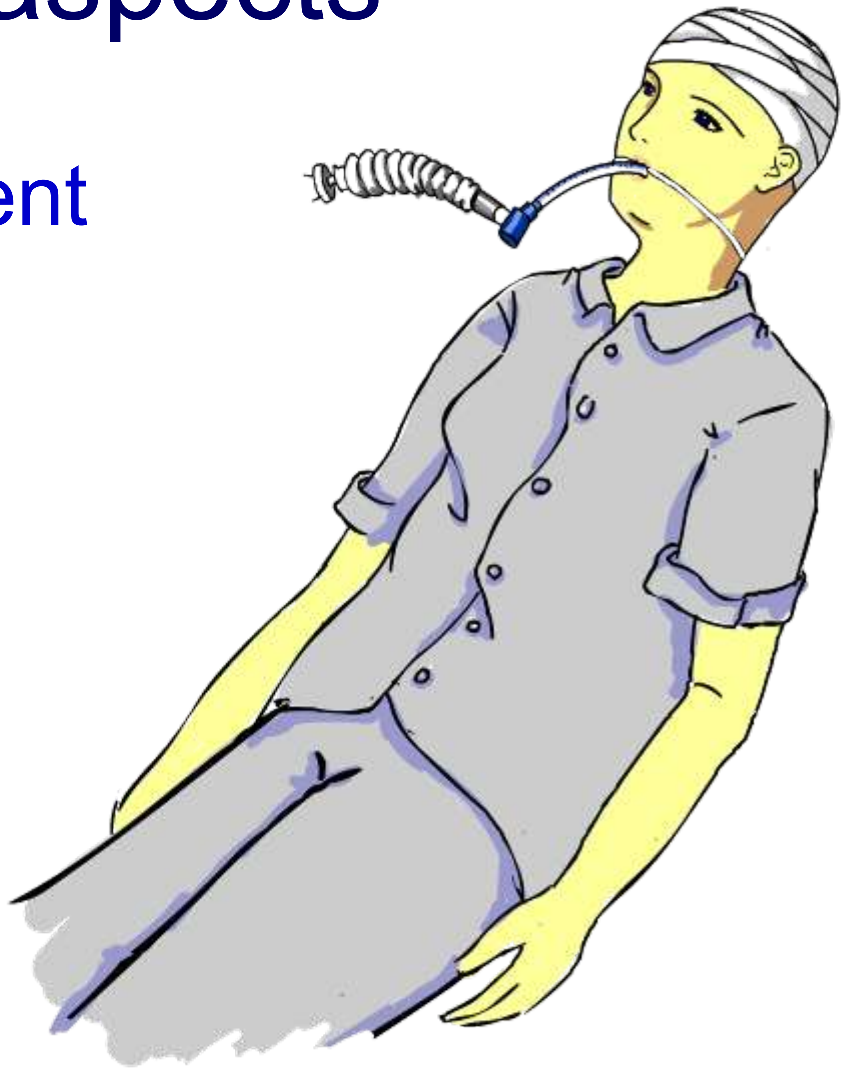
Practical aspects

- Signs of feed intolerance (poor specificity)
  - Poor gastric emptying
    - High residual volume
  - Abdominal pain
  - Abdominal distension
  - Diarrhoea



Practical aspects

- Feed in semi-recumbent position
  - 30° head up
  - Decrease aspiration/nosocomial pneumonia risk



Practical aspects

- Diarrhoea
  - Usually not due to feed
  - Consider drugs, *Clostridium difficile* colitis
  - If feed related may be due to:
    - Osmolality
    - Malabsorption

Any questions?

Transport of the critically ill

INTENSIVMEDIZIN BASICPLUS 2018
DR. MED. JOLANDA CONTARTESE
KANTONSSPITAL BADEN

Indikation für Transport

- Transporte von kritisch kranken Patienten erhöhen die Morbidität und Mortalität.

  -> **Indikation für Transport muss stimmen**

- Mögliche Indikationen:
  - Diagnostische Untersuchungen (intrahospitale Transporte)
  - Verlegung in ein Zentrumsspital zur adäquaten Versorgung (interhospitale Verlegung)
  - Regionalisierung

Fallbeispiel 1

- 50 jähriger intubierter Patient im kardiogenen Schock bei St.n. anteriorem Myokardinfarkt an der ECMO mit neu akutem Abdomen.
- Sediert, hochdosiert Vasopressoren und Inotropika

- > Abdomen-CT

Fallbeispiel 2

- 48jähriger Patient mit Schädelhirntrauma nach Sturz aus 5m Höhe im Kantonsspital Baden.
- GCS 14, BD 130/70mmHg, Sp02 95% mit Raumluft

-> Verlegung ins KSA für neurochirurgische Versorgung

Vorbereitung des Transportes

- **P**ersonalbesetzung und **P**atientenzustand
- **A**usrüstung und Medikamente
- **C**hecken der Ausrüstung
- **K**omplikationen
- **T**ransportweg

-> Akronym : **PACKT**

Personalbesetzung

- Mindestens 1 Pflegefachperson und 1 Arzt
- Abhängig von
  - Schweregrad der Erkrankung des Patienten
  - Transportmittel
  - Personal braucht entsprechende Ausbildung bzw. Erfahrung
    - z.B. Airway-Management
    - Wo kann man Hilfe holen, wenn etwas schief geht? Telephonnummern für Notsituationen (REA, Kaderarzt)
- Aufgabenzuteilung

Personalbesetzung

Fallbeispiel 1	Fallbeispiel 2
• 1 Arzt mit Anästhesieerfahrung und 2 Pflegefachpersonen • ECMO: Kardiotechniker	• Rettungsdienst mit Notarzt mit Anästhesieerfahrung

Patientenzustand

- Stabil, instabil
- Organversagen

Patientenzustand

Fallbeispiel 1	Fallbeispiel 2
• Instabil • Beatmungsgerät • ECMO • ZVK, Arterie, DK	• Stabil • Sauerstoffmaske • Infusion

Vorbereitung des Transportes

- **P**ersonalbesetzung und **P**atientenzustand
- **Ausrüstung und Medikamente**
- **C**hecken der Ausrüstung
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-> Akronym : **PA**CKT

Ausrüstung und Medikamente

- Welche Geräte?
- Welche Medikamente?

- Abhängig von:
  - Dauer des Transportes
  - Transportmittel
  - Patientenzustand

Geräte

- Transportmonitor
- Transportables Beatmungsgerät, Kapnographie
- Sauerstoff
- Absauggerät
- Defibrillator
- Perfusoren
- Etc.

Medikamente: the big Five

1. REA
2. Intubation
   - Bronchospasmus
3. Hypo-oder Hypertonie
4. Agitation/Schmerz
5. Anaphylaxie (KM-Untersuchungen)

- Spezielle Situationen berücksichtigen: z.B: Epilepsie, SHT mit Hirndruck, Antiarrhythmica

Ausrüstung und Medikamente

Fallbeispiel 1	**Fallbeispiel 2**
• Beatmungsgerät/Sauerstoffflasche • Ambubeutel • Intubationsset • ECMO • Defibrillator • Medikamente: Analgosedation, Vasopressoren, Inotropika, Muskelrelaxanz, Infusion, Medikamente für REA	• Sauerstoffflasche • Intubationsset • Medikamente für Intubation aufziehen • Absaugvorrichtung • Ambubeutel griffbereit

Vorbereitung des Transportes

- **P**ersonalbesetzung und **P**atientenzustand
- **A**usrüstung und Medikamente
- **Checken der Ausrüstung**
- **K**omplikationen
- **T**ransportweg

-> Akronym : **PAC**KT

Checken der Ausrüstung und Medikamente

- Wie funktionieren die Geräte?
- Kontrolle des Beatmungs- und Absauggerätes
- Sauerstoffreserve in Sauerstoffflasche überprüfen
- Batteriestand
- Sauerstoffmaske, Wendel-oder Güdel (Grösse)
- Defibrillator
- Haben wir die richtigen und genügend Medikamente dabei?

Vorbereitung des Transportes

- **P**ersonalbesetzung und **P**atientenzustand
- **A**usrüstung und Medikamente
- **C**hecken der Ausrüstung
- **Komplikationen**
- **T**ransportweg

-> Akronym : **PACKT**

Komplikationen

- des Patienten
- während Transport und Untersuchung

Komplikationen am Patienten

Fallbeispiel 1	Fallbeispiel 2
• Agitation/Schmerz • Bronchospasmus • Rhythmusstörungen • Abdominelles Kompartement	• Bewusstseinsverlust bei steigendem Hirndruck • Epilepsie • Pneumothorax • Hämorrhagischer Schock

Komplikationen während Transport oder Untersuchung

- Dislokation oder Entfernung von Kathetern oder Drainagen, Dekanülierung der ECMO, Extubation
- Falsche, ungenügende Ausrüstung je nach Zielort z.B im MRI, fehlender Sauerstoffanschluss
- Platzmangel z.B Lift
- Untersuchungsspezifische Komplikationen

Ready to go?

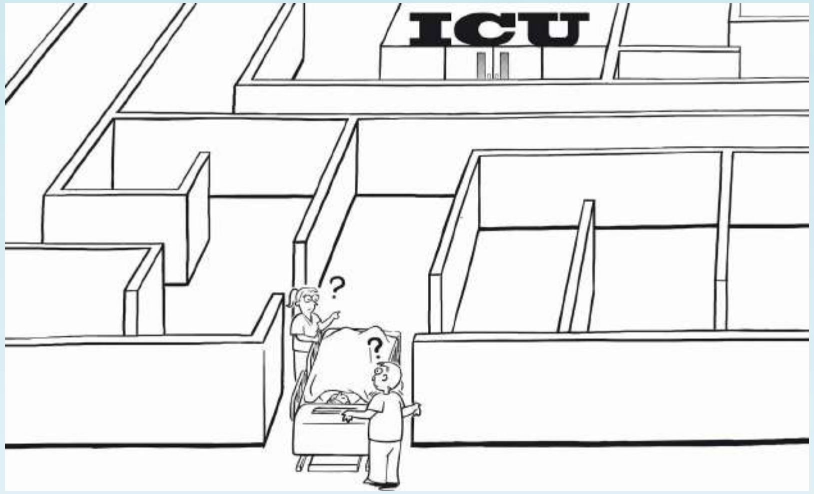
Vorbereitung des Transportes

- **P**ersonalbesetzung und **P**atientenzustand
- **A**usrüstung und Medikamente
- **C**hecken der Ausrüstung
- **K**omplikationen
- **Transportweg**

-> Akronym : **PACKT**

Transportweg, Planung

- Distanz und Dauer
- Transportmittel
  - Zu Fuss, Ambulanz, REGA
- Rahmenbedingungen
  - Verkehr
  - Wetter
  - Lift
- Zielort informieren über Abfahrt



Zusammenfassung

- Transportindikation muss stimmen
- Sorgfältiges Planen der Ausrüstung und der Medikamente
- Remember: **PACKT**


Also, ich muss schon sagen: Von dieser Reise ins Blaue bin ich einigermaßen enttäuscht. Jetzt latschen wir schon seit mehreren Stunden durch die Gegend, und es gab noch nicht eine einzige Überraschung!