

APRV AIRWAY PRESSURE RELEASE VENTILATION

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Acute Respiratory Distress Syndrome ARDS

- An Inflammatory process of the lungs
 - Acute onset of respiratory failure
 - New onset bilateral infiltrates on frontal chest radiograph
 - Absence of left ventricular failure
 - Hypoxemia with a ratio of $\text{PaO}_2/\text{FiO}_2 < 200$

ALI

- Same criteria as ARDS
- $\text{PaO}_2/\text{FiO}_2 < 300 \text{ mmHg}$

- **Severity based on oxygenation index**

- **Three categories ($\text{PEEP} \geq 5 \text{ cmH}_2\text{O}$)**
- **Mild ($200 \text{ mmHg} \leq \text{PaO}_2/\text{FiO}_2 < 300 \text{ mmHg}$)**
- **Mild-moderate ($150 \text{ mmHg} \leq \text{PaO}_2/\text{FiO}_2 < 200 \text{ mmHg}$)**
- **Moderate-severe ($\text{PaO}_2/\text{FiO}_2 < 150 \text{ mmHg}$)**

ALI/ARDS

- An acute inflammatory condition in the lungs due to an underlying disease process
 - Direct (primary)
 - Indirect (secondary)

ALI/ARDS

- Direct (Primary) Injury
 - 70-80% cases
 - Pneumonia the most common 40-50%
 - Aspiration
 - Pulmonary contusion
 - Inhalation of toxic gases
 - Near drowning

ALI/ARDS

- Indirect (Secondary) Injury
- Typically caused by systemic inflammation with generalized activation of mediators
 - Infection (sepsis, peritonitis)
 - Tissue Ischemia (necrosis, pancreatitis)
 - Tissue damage (trauma, CABG, post-op, some intoxications)
 - Possibly COVID₁₉

ALI/ARDS

- Independent Risk Factors
 - Old age
 - Infection
 - Neurological disease



Clinical Picture

- Acute shortness of breath
- Cyanosis
- Tachypnea
- Dyspnea
- Crackles



ALI/ARDS

Phases of ALI/ARDS

- Initial acute phase (days)
 - Noncardiogenic pulmonary edema secondary to capillary leakage
- Second phase (1-2 weeks)
 - Inflammatory
 - Cytokines such as IL-1 β , TNF- α , IL-6, and IL-8 are elevated
 - Organization of edema into hyaline membranes
- Third phase
 - Fibrosis of pulmonary tissue

ALI/ARDS

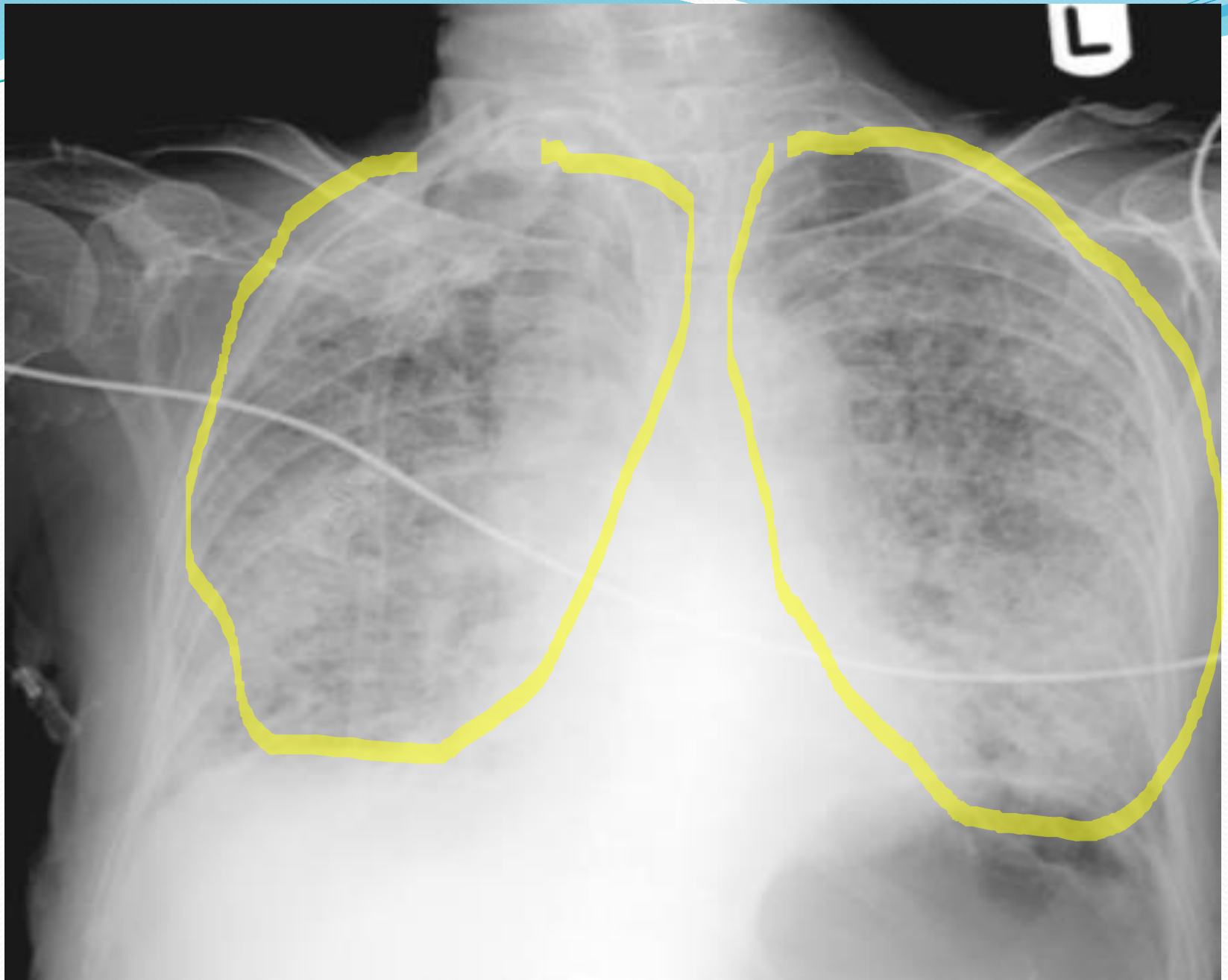
- Interstitial and alveolar edema
- Increase extravascular lung water
- Increased stiffness and weight of lungs
- Compression atelectasis
- Reduction of compliance
- Decrease of functional residual capacity
- Increase intrapulmonary shunting

ALI/ARDS

- With time can progress to fibrosing alveolitis
- Lung compliance is reduced, hypoxemia persists
- Pulmonary hypertension can progress to right heart failure
- Resolution can occur over 6-12 months
- Lung function can return to normal
- Overall mortality is approximately 50%

Diagnostic Workup of ARDS

- Medical History
 - Acute onset of respiratory failure in combination with an underlying insult that initiates the pulmonary inflammation
- CXR
 - Bilateral infiltrates



Diagnostic Workup

- Echocardiography or PA catheterization
 - Documented PCWP $<18\text{mmHg}$
 - Exclusion of significant LV dysfunction
- ABG and FiO_2
 - $\text{PaO}_2/\text{FiO}_2 <200$
 - Marked hypoxemia
 - Initial hypocapnia secondary to:
 - anxiety and increased ventilatory drive
 - Eventual Respiratory Acidosis
 - Typically, late

Diagnostic Workup

- ABG and FiO₂
 - PaO₂/FiO₂ <200 then you have ARDS
 - <300 ALI
- Hypoxemia
- Hypocapnia
 - anxiety and increased ventilatory drive
- Respiratory Acidosis
 - Typically, late

Management

- Ventilatory support with NIV
 - Fully conscious
 - Cooperative
 - Hemodynamically stable
 - Tolerant of short periods off vent support
 - Able to take large breaths
 - Not fatigued
 - **Terminate if no marked improvement in 1 hour**

Management

- Vast Majority of patients will require mechanical ventilation
 - Inadequate gas exchange
- Potential Complications from intubation
 - Prolonged hypoxemia
 - Aspiration
 - Misplacement of tube
 - Hemodynamic compromise

Typical ventilator management

- Mechanical ventilation
- Assist control
- Pressure control
- Inverse I:E ratio Pressure control
- APRV
- HFOV (3100B)
- ECMO

ARDSnet Protocol

- HOB >30 deg
- Tidal volume 6-8 ml/kg (sometimes 4-6)
- Utilization of PEEP
- Plateau pressure goal <30

APRV other names

- APRV
 - Initially by Stock and Downs in 1987
 - Drager Medical
- BiVent - (Servo-i ventilator by Maquet)
- BiLevel - (Puritan Bennett 840 ventilator by Covidien)
- DuoPAP - (C-1 ventilator by Hamilton)

Airway Pressure Release Ventilation

- APRV is CPAP continuous positive airway pressure with an intermittent pressure release
- Features
 - CPAP maintains lung volume (FRC)
 - Prolonged inhalation phase
 - Promotes alveolar recruitment
 - Allows for spontaneous breathing with “floating exhalation valve”
 - Time cycled pressure release to enhance ventilation

Why APRV?

- Advantages
 - Lower P_{aw} for a given tidal volume compared with volume-targeted modes, e.g., AC, SIMV
 - Lower minute ventilation, i.e., less dead space ventilation
 - Limited adverse effects on cardiac output and venous return
 - Spontaneous breathing possible throughout entire ventilatory cycle
 - Decreased sedation use
 - Near elimination of neuromuscular blockade use

Why not APRV?

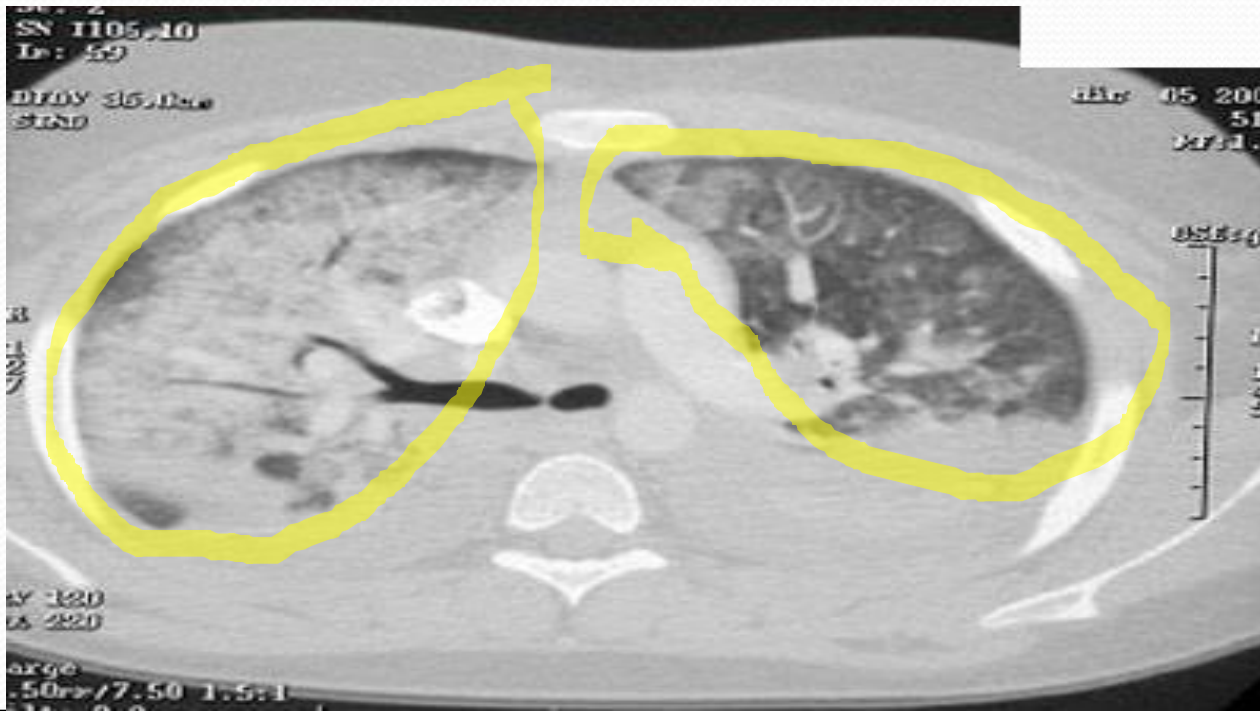
- Potential Disadvantages
 - Volumes change with alteration in lung compliance and resistance
 - Process of integrating new technology
 - Limited access to technology capable of delivering APRV
 - Limited research and clinical experience
 - Different terminology

APRV

- Setting the P-High at plateau pressures ensures you optimize pressure which will provide recruitment
- A PEEP valve in the expiratory limb of the ventilator prevents the patient from generating overdistension
 - Lower peak airway pressures
- Setting the T-High 4-6 seconds optimizes time during oxygenation

Recruitment in APRV

- Enhances oxygenation
- Optimizes ventilation



APRV Spontaneous Breathing

Spontaneous Breaths

- Mimic normal physiology- sinusoidal
- Improves distribution to dependent areas
- Enhances V/Q mismatch
- Reduces the need for sedation

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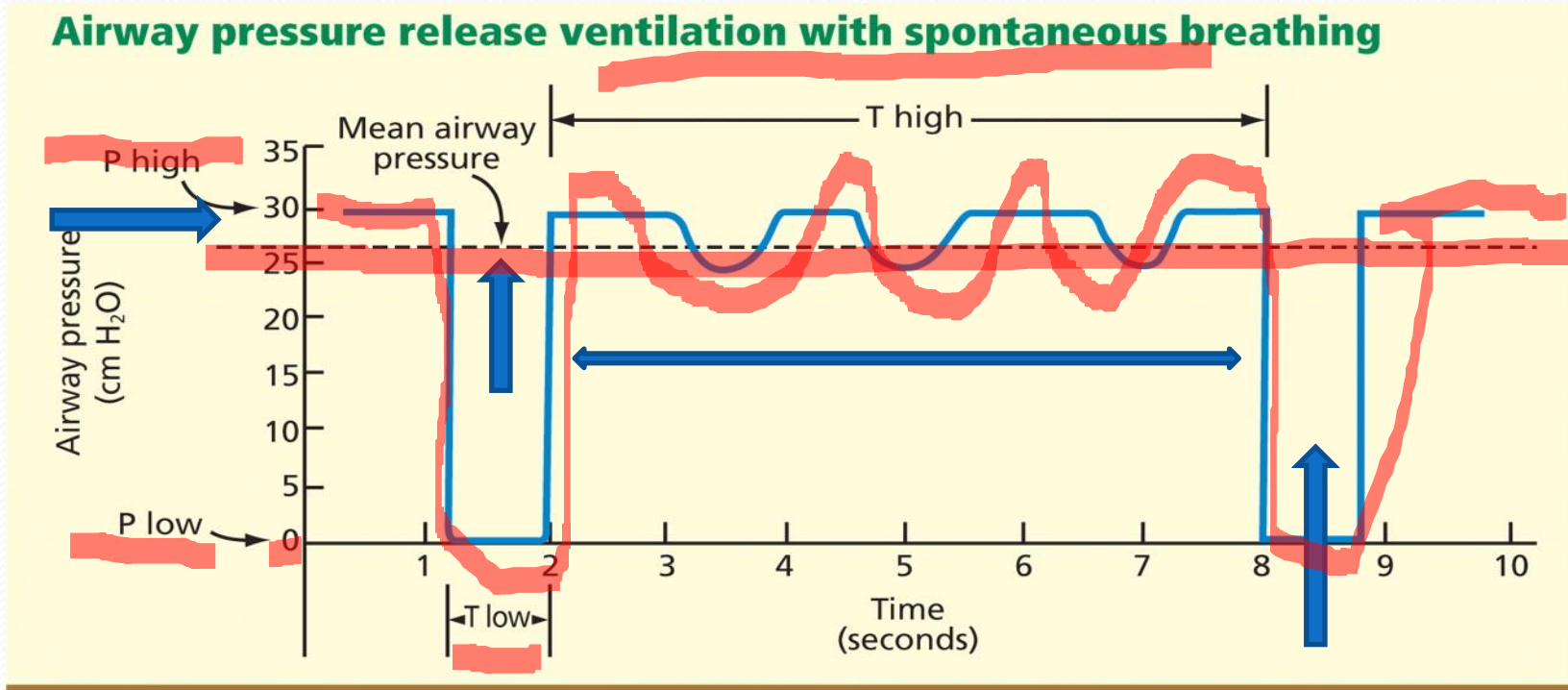


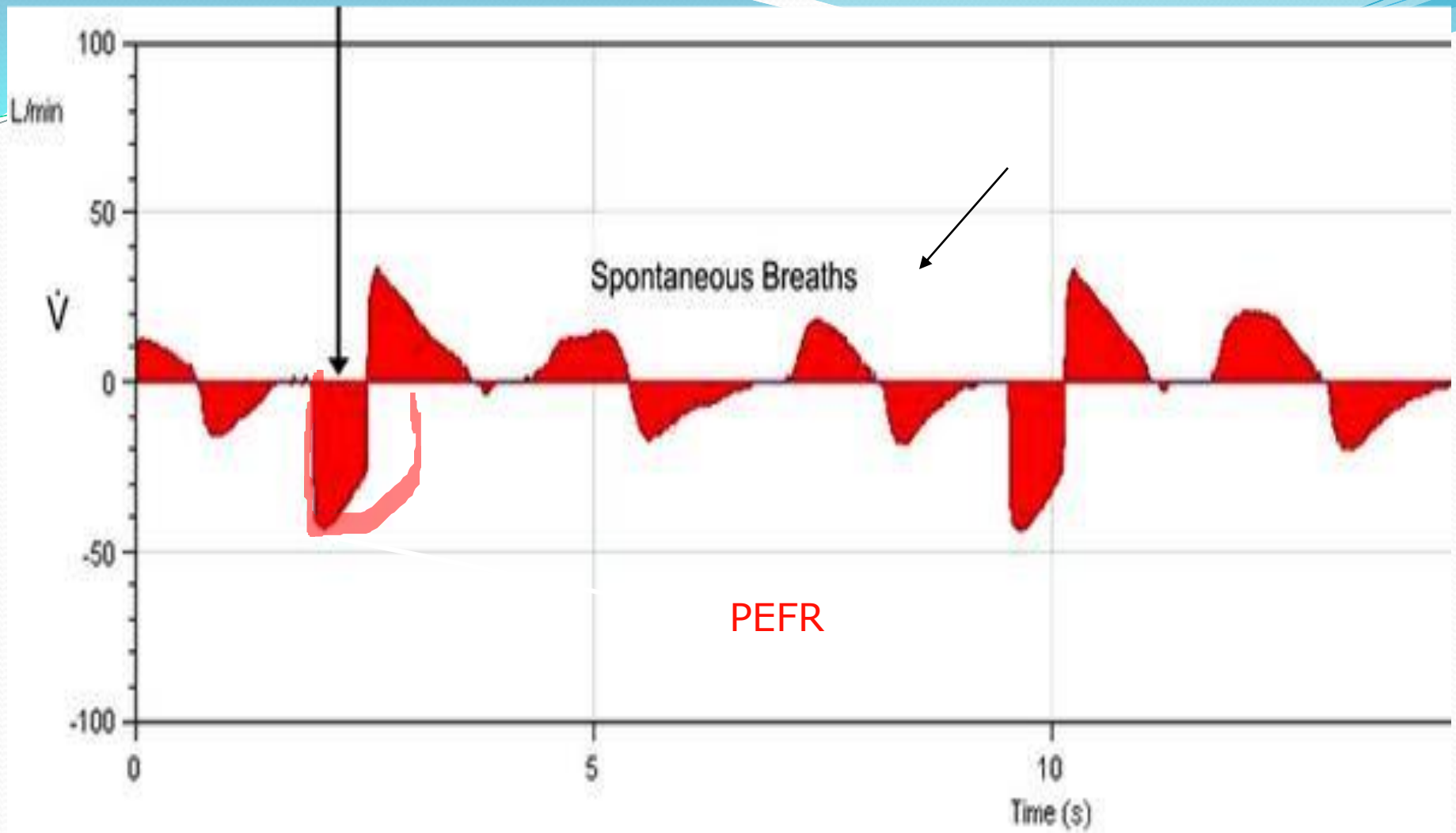
FIGURE 2

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MODRYKAMIEN A et al. Cleveland Clinic Journal of Medicine 2011;78:101-110

Cleveland Clinic Journal of Medicine





HOW DOES APRV WORK

- LUNG RECRUITMENT
- LUNG RECRUITMENT
- LUNG RECRUITMENT

INDICATIONS

- ACUTE LUNG INJURY (ALI)
- ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)
- ACUTE RESTRICTIVE DISEASES

RELATIVE INDICATIONS

- COPD
- SIGNIFICANT BULLOUS DISEASE
- You must increase release phase or T-Low to accommodate increased retention of CO₂

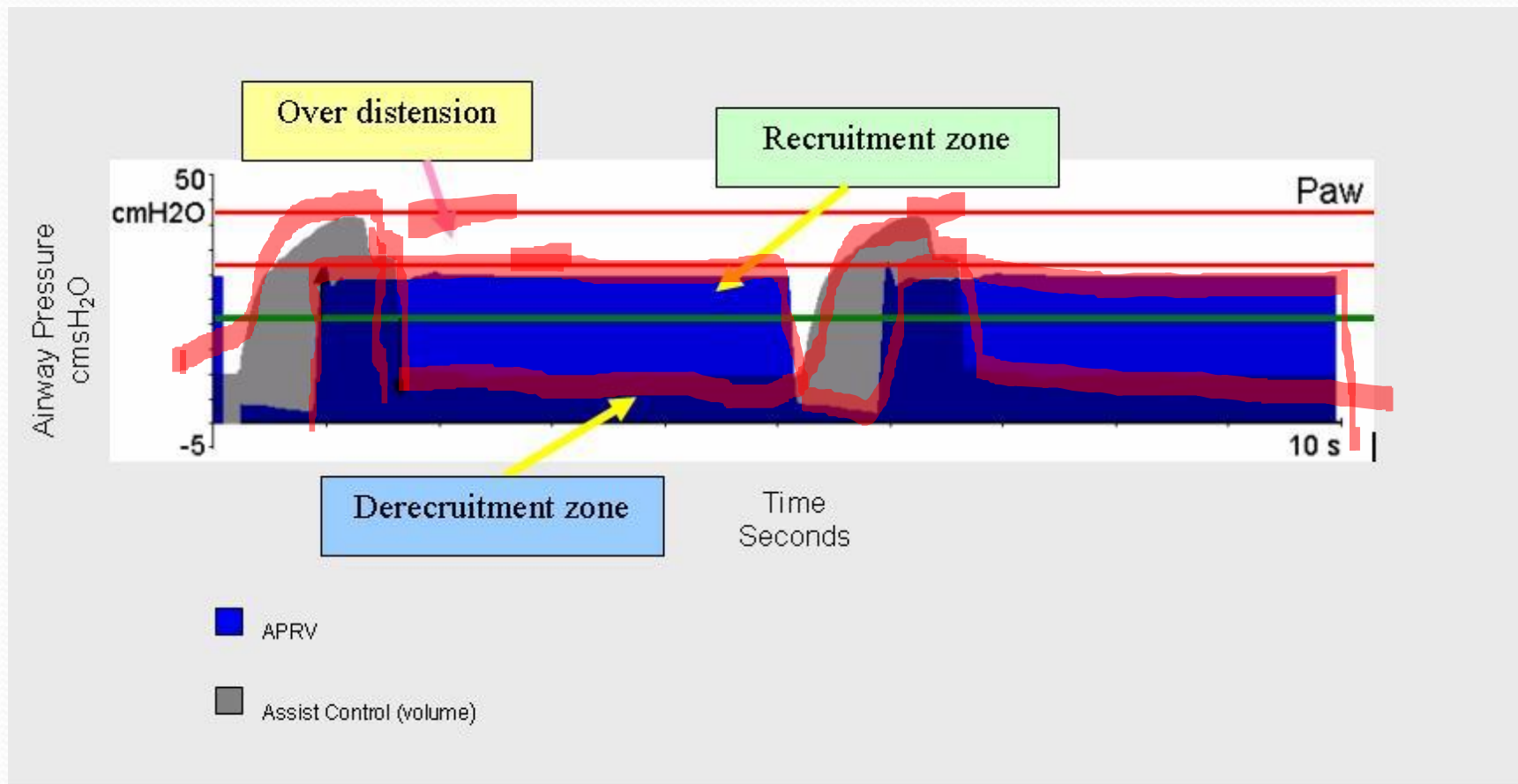
BENEFITS

- SPONTANEOUS BREATHING
- IMPROVES VENTILATION-PERFUSION MISMATCH
- REDUCE PULMONARY SHUNT
- LOWER PEAK AIRWAY PRESSURES
- DECREASED NEED FOR SEDATION
- IMPROVES HEMODYNAMICS*****

POTENTIAL RISK

- TIDAL VOLUME CHANGE WITH ALTERATION OF COMPLIANCE AND RESISTANCE (SAME AS PCV)
- LEARNING CURVE ASSOCIATED WITH INTRODUCTION OF NEW TECHNOLOGY

HOW DOES IT WORK



HOW DOES IT WORK CONTINUED

- PROVIDES CONTINUOUS AIRWAY PRESSURE TO FACILITATE LUNG RECRUITMENT, LUNG VOLUME, AND OXYGENATION
- FACILITATES SPONTANEOUS BREATHING AND PATIENT COMFORT
- DECREASE NEED FOR SEDATION

HOW TO SETUP THE DRAGER XL FOR APRV

- OBTAIN A PLATEAU PRESSURE
- MUST BE IN A VOLUME MODE
- NO AUTOFLOW
- DO INSPIRATORY HOLD FOR 10 SECONDS

P - HIGH

- CONVERT PLATEAU PRESSURE TO P-HIGH (you can add 2 to the plateau pressure obtained)
- MAXIMUM GOAL IS 35 CMH₂O
- P HIGH > 35CMH₂O IS NEEDED WITH DECREASED THORACIC AND ABDOMINAL COMPLIANCE

P - LOW

- SET TO ZERO
- PRODUCES MINIMAL EXPIRATORY RESISTANCE
- ACCELERATES EXPIRATORY GAS FLOW

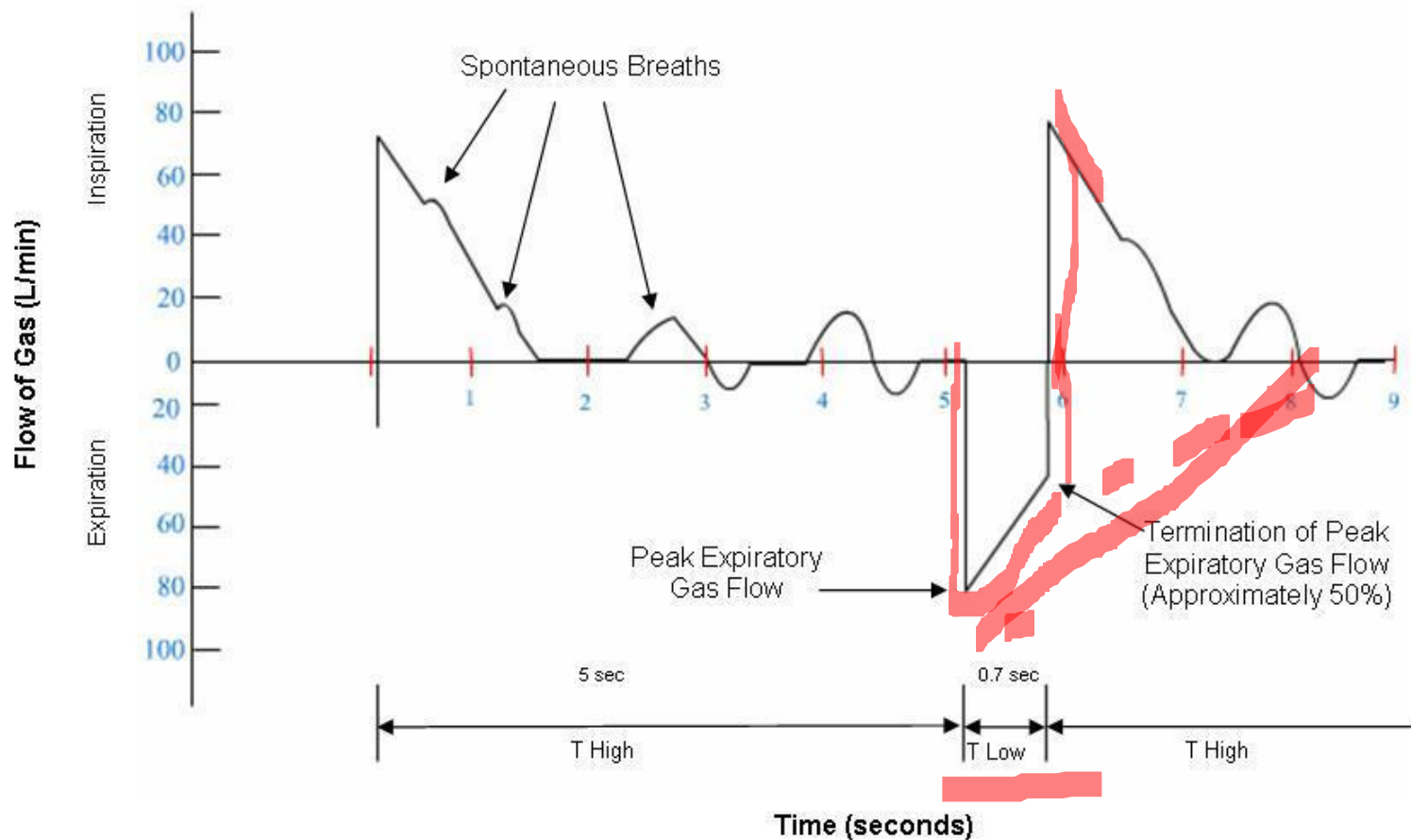
T- HIGH

- SET TO 4-6 SECONDS
- AS PATIENT WEANS EXTEND THE T-HIGH AND LOWER THE P - HIGH

T-LOW

- **MOST CHALLENGING SETTING WITH APRV**
- TYPICALLY, .4 - .6 SECONDS
- MOST OFTEN .5 SECONDS
- ADJUST TO CHIEVE AN END-EXPIRATORY LFLOW EQUAL TO 75% OF THE PEAK EXPIRATORY FLOW RATE
- DETERMINED BY EXPIRATORY GAS FLOW CURVE

EXPIRATORY GAS FLOW

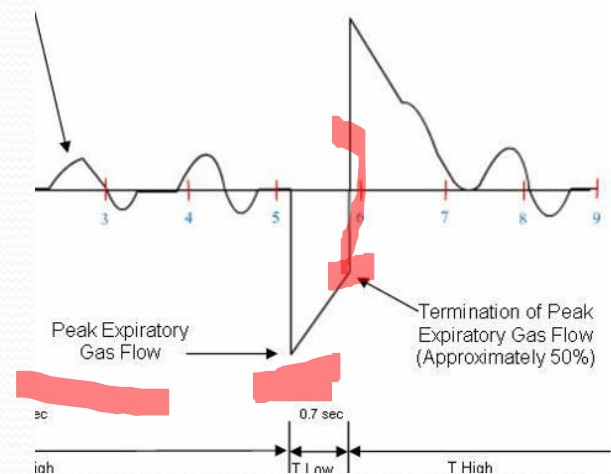


EXPIRATORY GAS FLOW

- LUNG DERECRUITMENT STRATEGIES
- TERMINATE AT 50% TO 75% OF PEAK EXPIRATORY FLOW
- PAY ATTENTION TO CHANGES IN COMPLIANCE
 - EXPIRATORY WAVEFORM CHANGES WITH COMPLIANCE AND RESISTANCE

HOW TO SET EXPIRATORY WAVEFORM

- DISPLAY FLOW WAVEFORM
- FREEZE WAVEFORM
- MEASURE PEAK EXPIRATION WITH TOGGLE SWITCH
- ADJUST T-LOW TO GET 50% TO 75% OF EXPIRATORY FLOW RATE



BLOOD GAS ADJUSTMENTS

- To Improve oxygenation
 - MAP
- To Improve hypercapnia
 - Minute Ventilation

TO IMPROVE OXYGENATION

- INCREASE P-HIGH BY INCREMENTS OF NO MORE THAN 2 CMH₂O
- MAINTAIN A SPO₂ OF AT LEAST 93%
- KEEP FIO₂ ≤ 60%

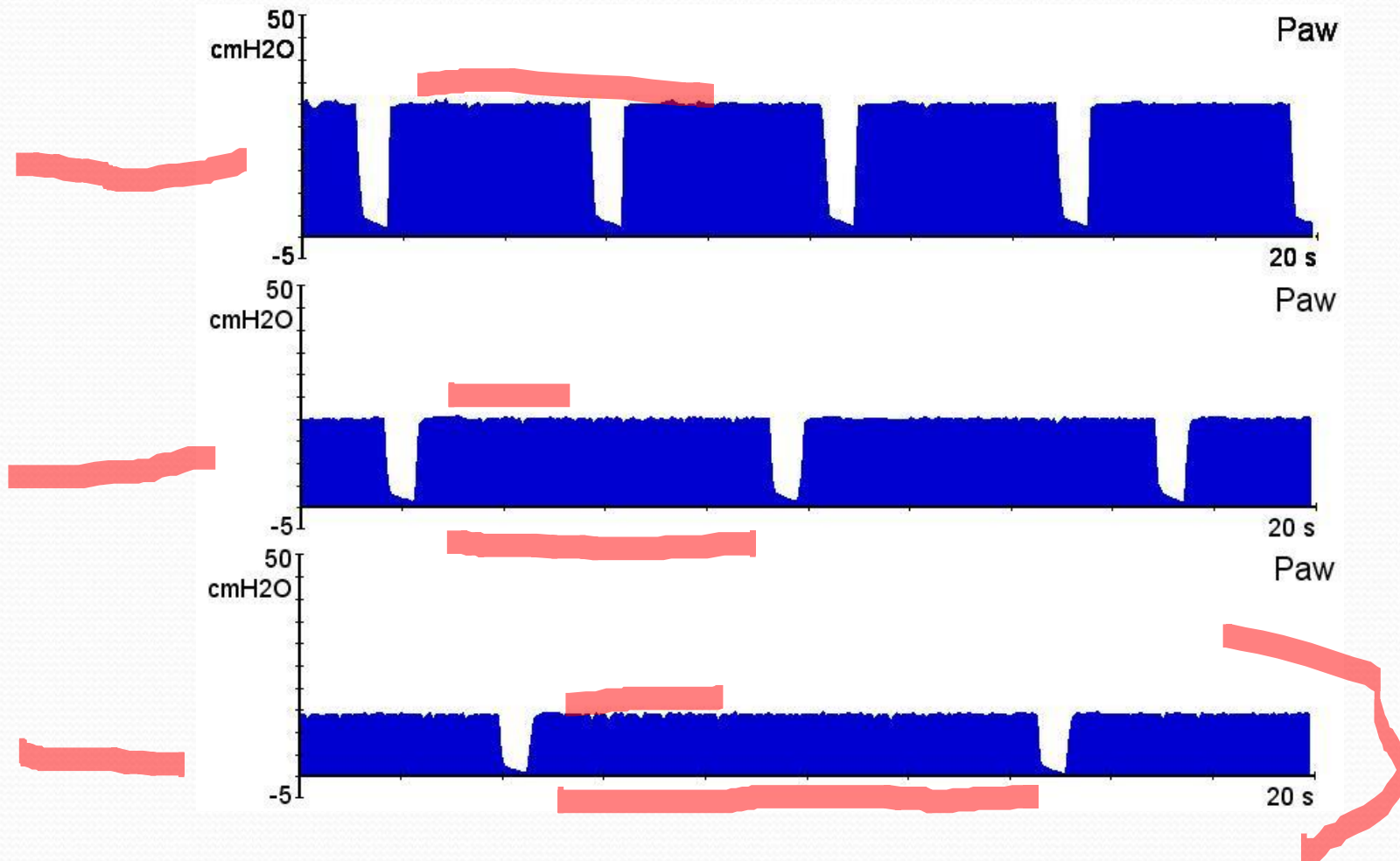
TO DECREASE PCO₂

- STEP 1
 - INCREASE P-HIGH ENHANCE RECRUITMENT
- STEP 2
 - INCREASE T-HIGH IN .5 SECONDS INCREMENTS
- STEP 3
 - INCREASE T LOW TO ACHIEVE LARGER EXPIRATORY TIDAL VOLUMES (DO NOT EXCEED 50-60% OF PEAK EXPIRATORY FLOW)

THE PRIMARY METHOD TO WEAN SUPPORT FROM APRV

- DROP AND STRETCH TECHNIQUE
- DECREASE P HIGH BY INCREMENTS OF 2 CMH₂O
- INCREASE T HIGH BY .5 SECONDS INCREMENTS
- GOAL IS TO REACH A CPAP OF 8-14 CMH₂O
- ONCE A P HIGH OF 10 CMH₂O IS REACHED USE ATC

DROP AND STRETCH TECHNIQUE



Summary

- APRV is a great tool for ARDS
- Don't wait till it becomes a rescue ventilation strategy
- Use it for obese patients with compliance issues due to abdominal fat (EXTEND T-LOW TIMES)
- Don't be afraid to speak up

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