Patient ventilator Asynchrony

Dr Jayabharathi Palanivel

Patient–Ventilator Asynchrony

- Refers to the dynamic coupling between the patient's respiratory drive and the mechanical support delivered by the ventilator.
- **PVA:** a mismatch between the patient's neural respiratory drive and the ventilator's mechanical breath delivery in terms of timing, magnitude, or both.
- Timing-Mismatch between neural inspiratory/expiratory time and mechanical breath delivery
- Magnitude-Ventilator assist level does not match patient's respiratory demand (over or under-assist)
- Both-Combined timing and magnitude errors

RespirCare. 2011 Jan;56(1):39-51.

Ventilatory muscle physiology

- The diaphragm is the primary breathing muscle. It generates pressure to overcome two types of loads:
- **Elastic load** — the stiffness of the lungs and chest wall
- **Resistive load** — airway resistance to airflow

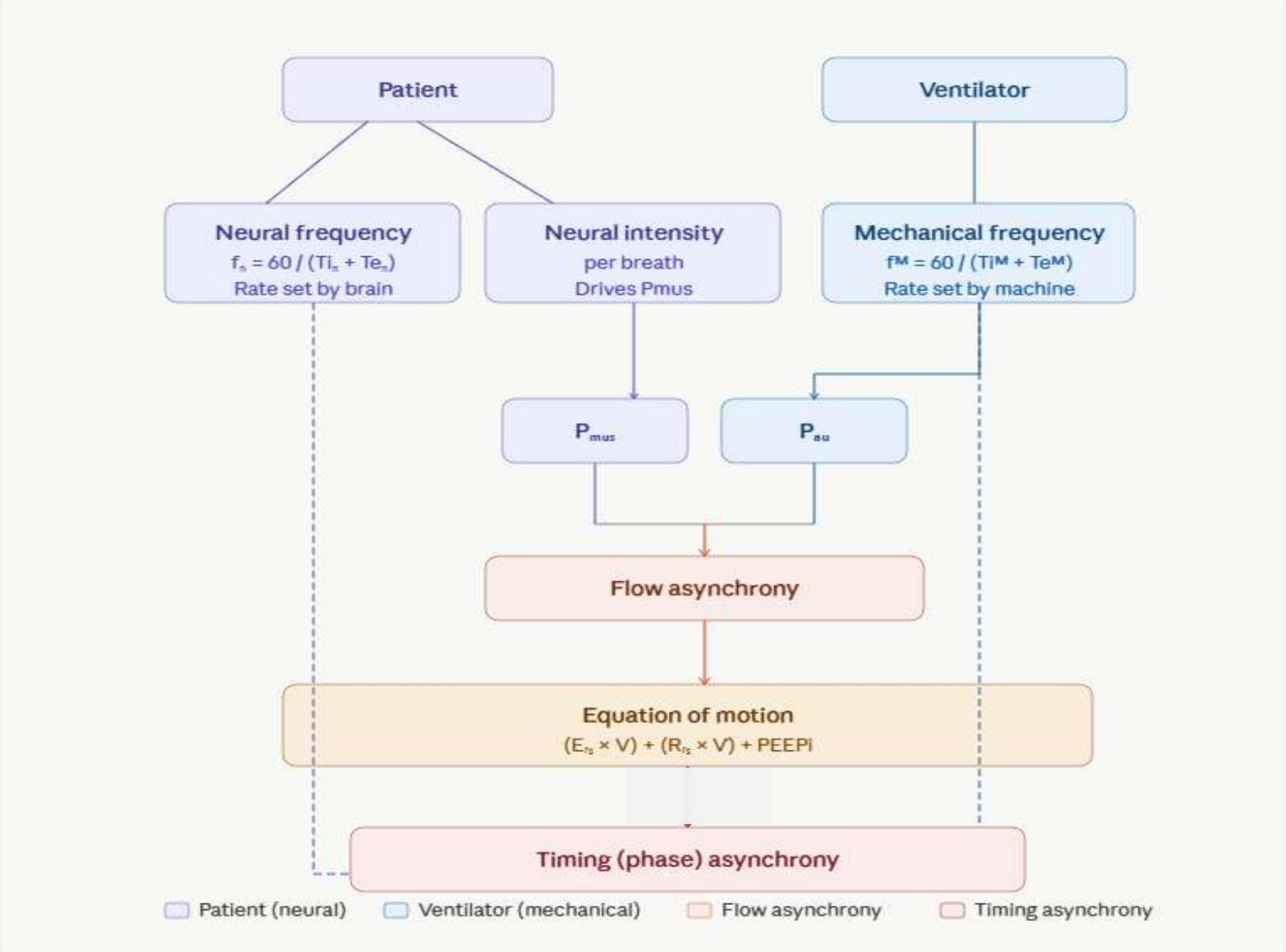
EQUATION OF MOTION

$P_{mus} + P_{vent} = P_{elastic} + P_{resistive}$

$P_{total} = (\Delta Volume / Compliance) + (Resistance \times Flow)$

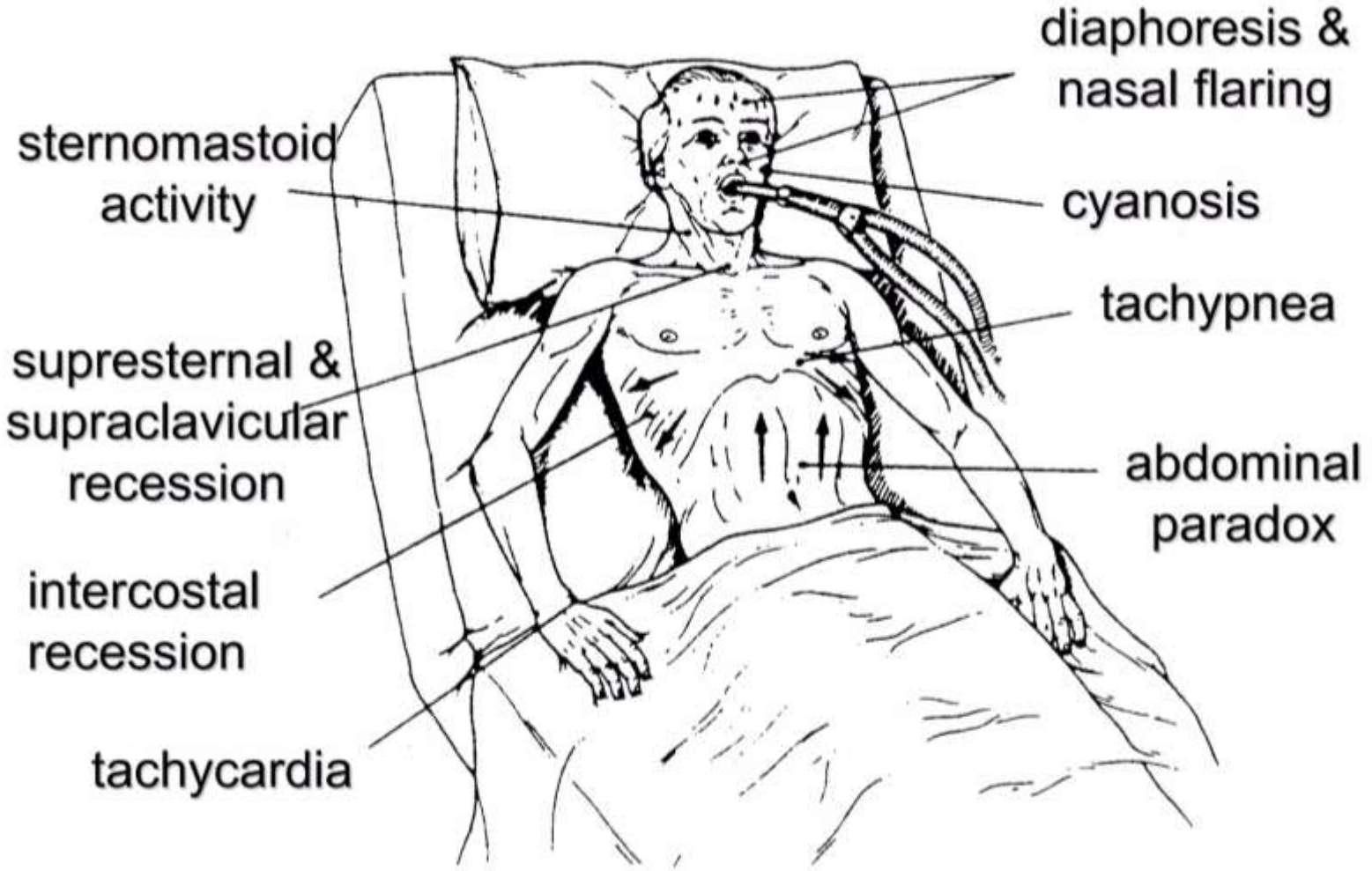
RespirCare. 2011 Jan;56(1):39-51.

Pathophysiology of PVA



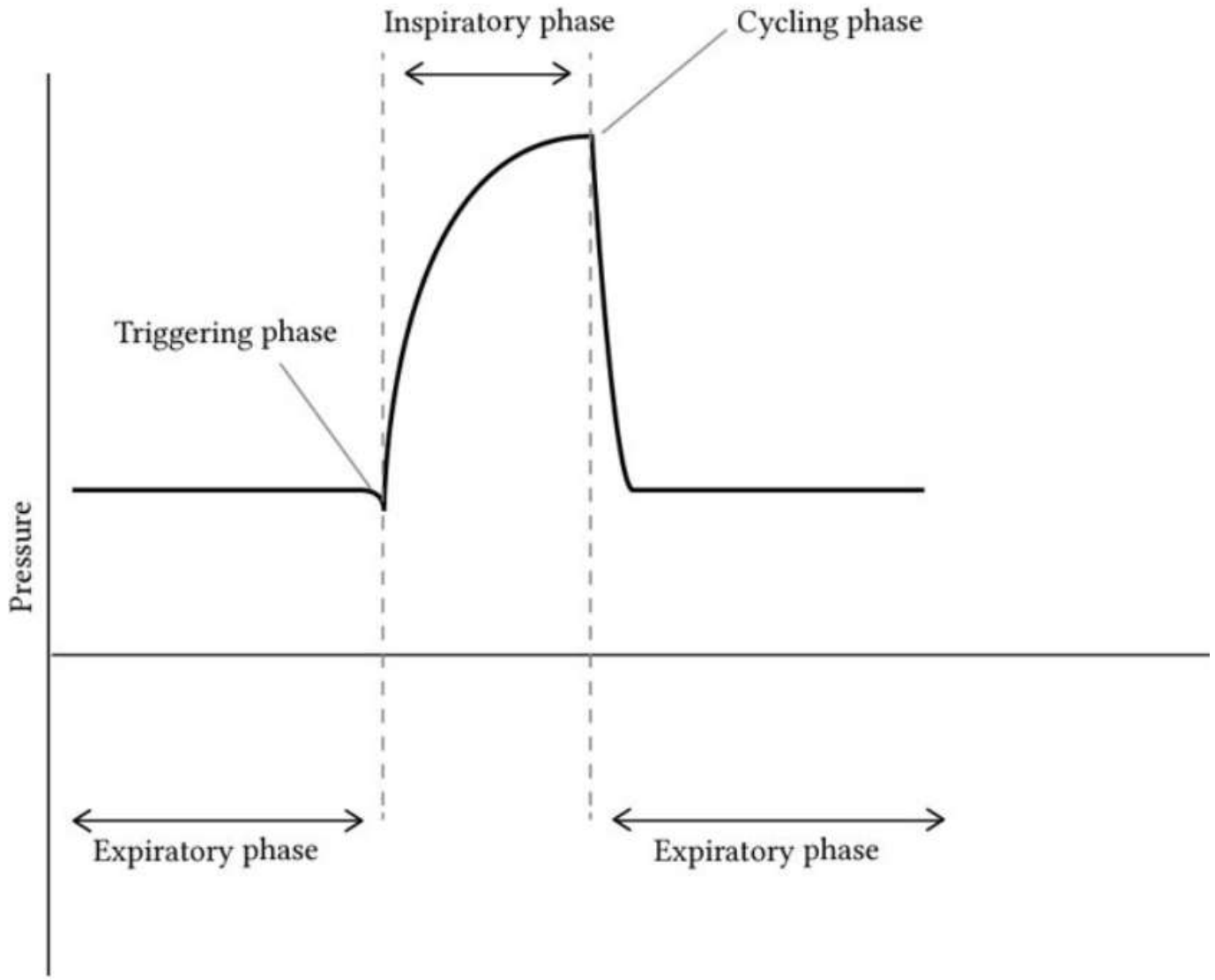
RespirCare. 2011 Jan;56(1):39-51.

Asynchrony between patient and ventilator



Principles and practice of MV.1994

Variables controlling the phases of mechanical breath



Three critical phases of synchrony:

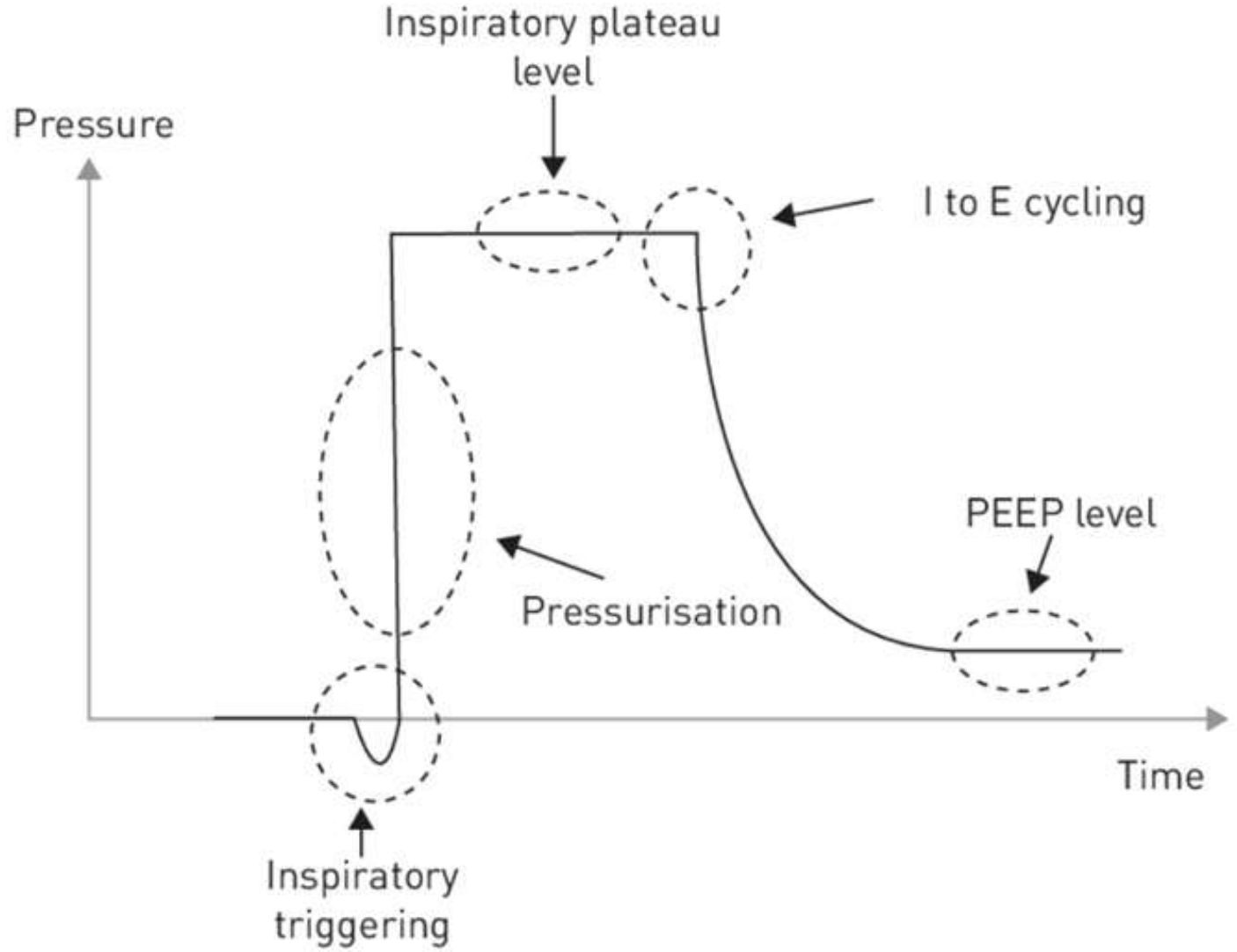
Trigger phase: Ventilator detection of neural inspiration onset

Flow delivery phase: Match between the patient's flow demand and ventilator flow output

Cycling phase: Ventilator termination of inspiration aligned with the end of neural Ti

J Clin Med 2021;10:4550 I Oto et al. 2021

Variables controlling the phases of mechanical breath

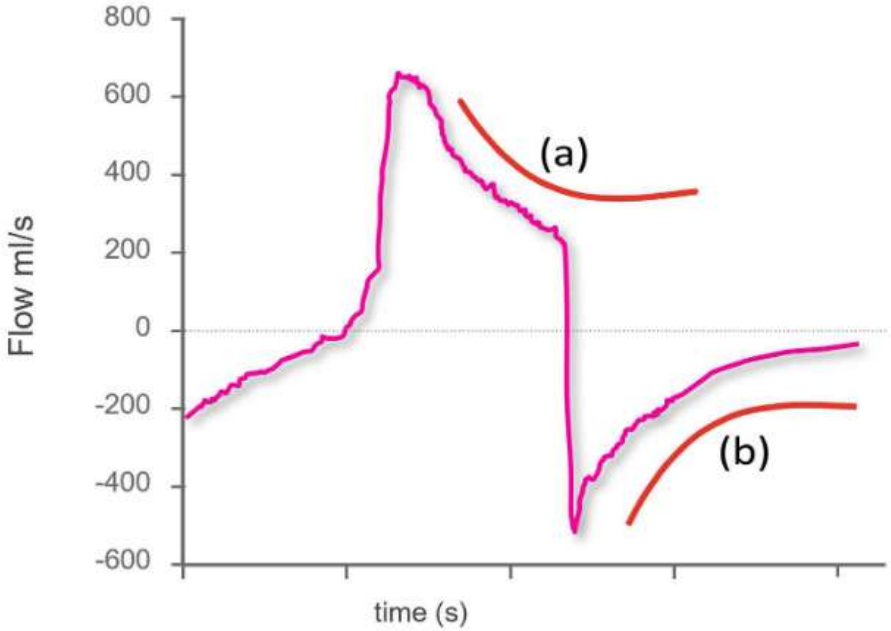


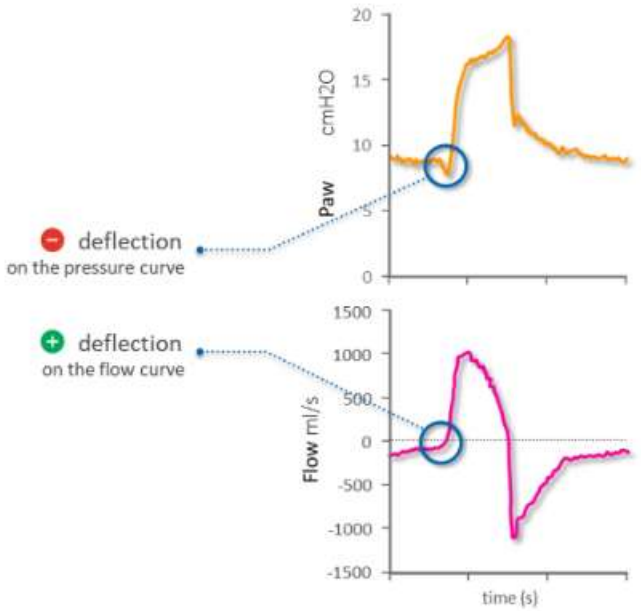
Impact on ICU Patients

- High resistive loads (COPD, asthma)
- High elastic loads (ARDS, pulmonary edema, interstitial lung disease)
- Low muscle strength (malnutrition, neuromuscular disease, shock)
- High minute ventilation requirements
- Intrinsic PEEP — air trapped in lungs that adds extra load

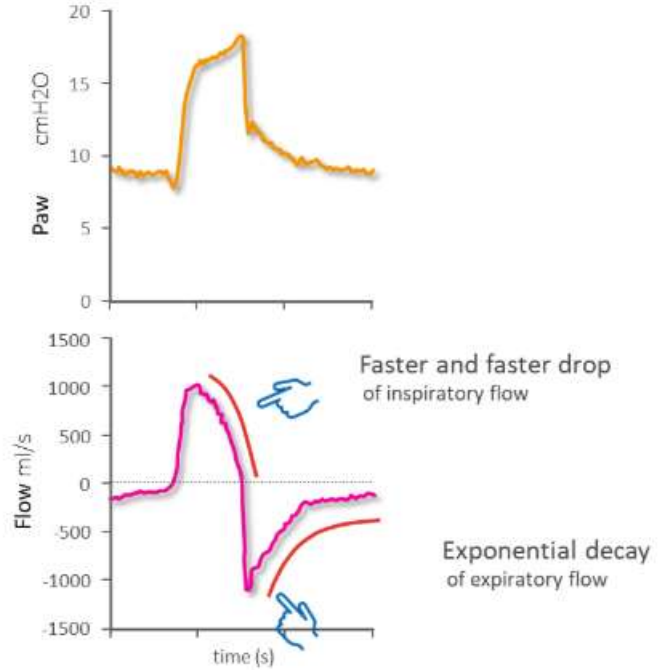
J Clin Med 2021;10:4550 I Oto et al. 2021

Normal Waveform characteristics





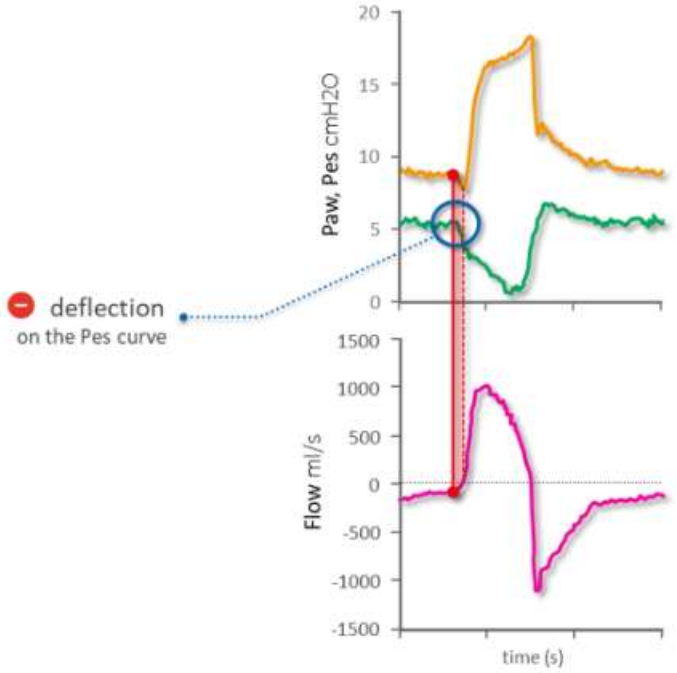
The start of an inspiratory effort on pressure and flow waveforms



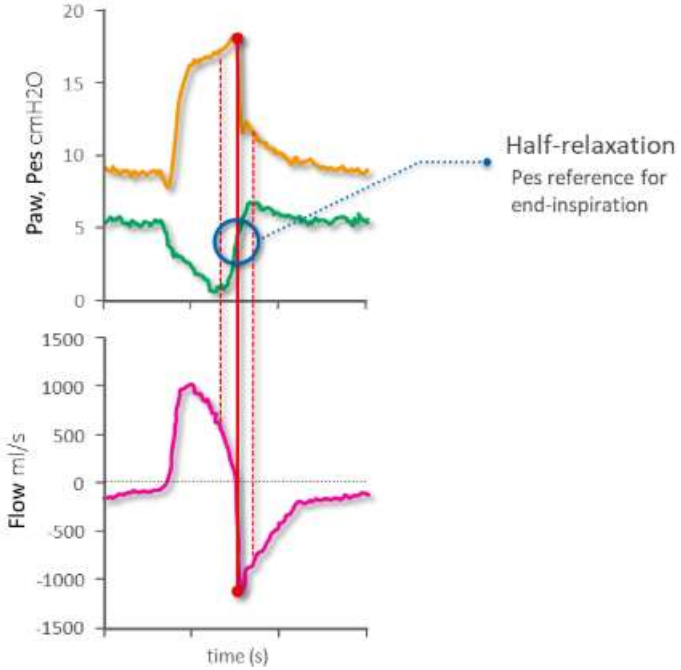
A synchronised end of inspiration on the flow waveform

Mojoli et al. Critical Care (2022) 26:32

Waveform characteristics including Pes waveform



The start of inspiration on the Pes waveform



The end of inspiration on the Pes waveform

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Classification & Types of Dyssynchrony

Trigger · Flow · Cycling · Special Types

Types of PVA

TRIGGER PHASE

- Ineffective triggering (wasted effort)
- Delayed triggering
- Auto-triggering
- Reverse triggering *

INSPIRATORY PHASE (Flow / Pressure)

- Flow starvation (demand-flow mismatch)
- Over-assistance

CYCLING PHASE

- Premature triggering
- Delayed cycling

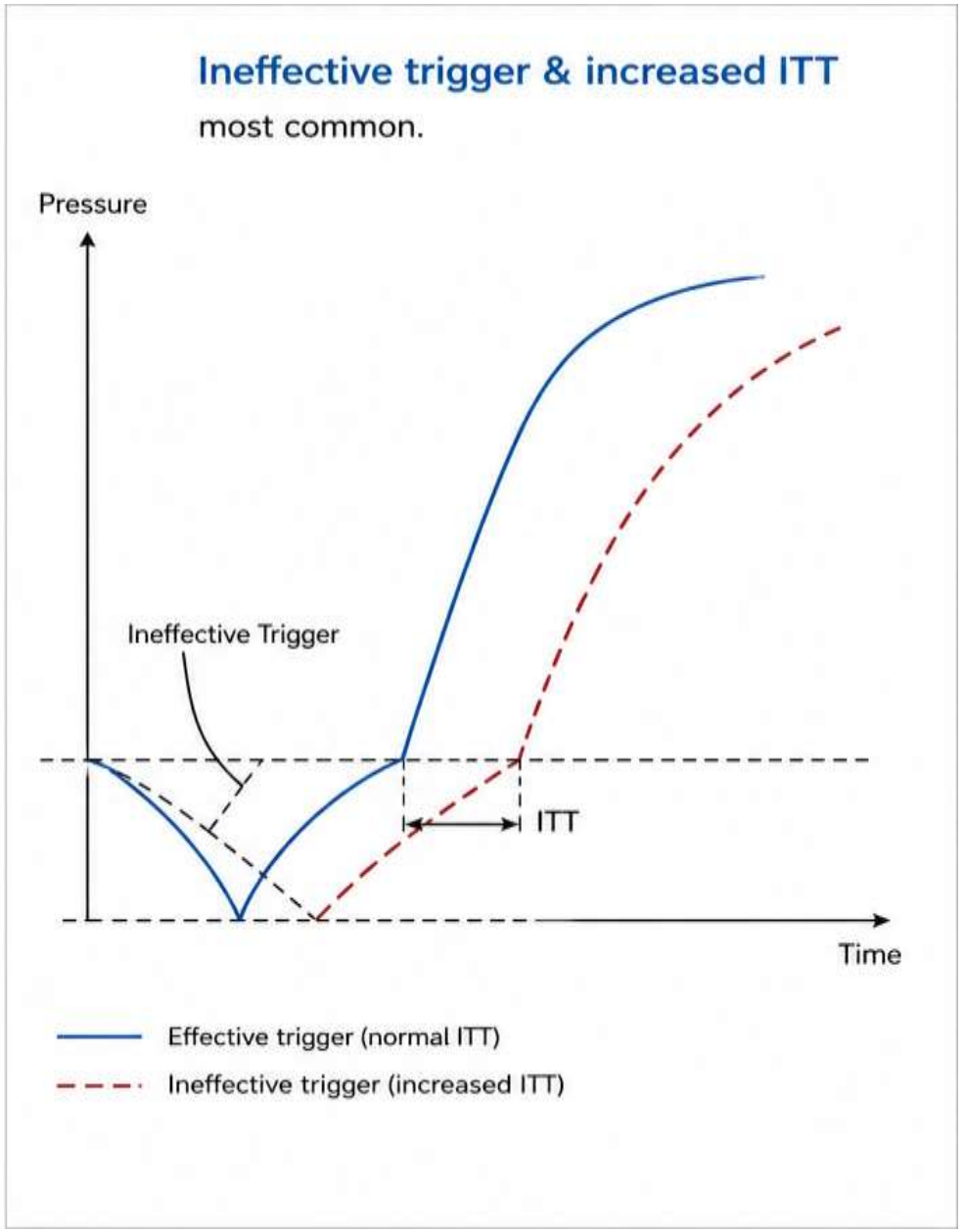
* Special types — require advanced monitoring (EAdi / esophageal manometry) for detection

Anaesthesia and Intensive Care2021, Vol. 49(2) 86–97S

Trigger Dyssynchrony

Types

- Ineffective trigger efforts (missed trigger, delayed trigger)
- Best response time to trigger effort should be < 100 ms



The blue curve shows a normal trigger
The red dashed curve shows increased trigger time → the ventilator responds too late, leading to ineffective trigger
→

J Clin Med 2021;10:4550 I Oto et al. 2021

Ineffective effort

Definition:

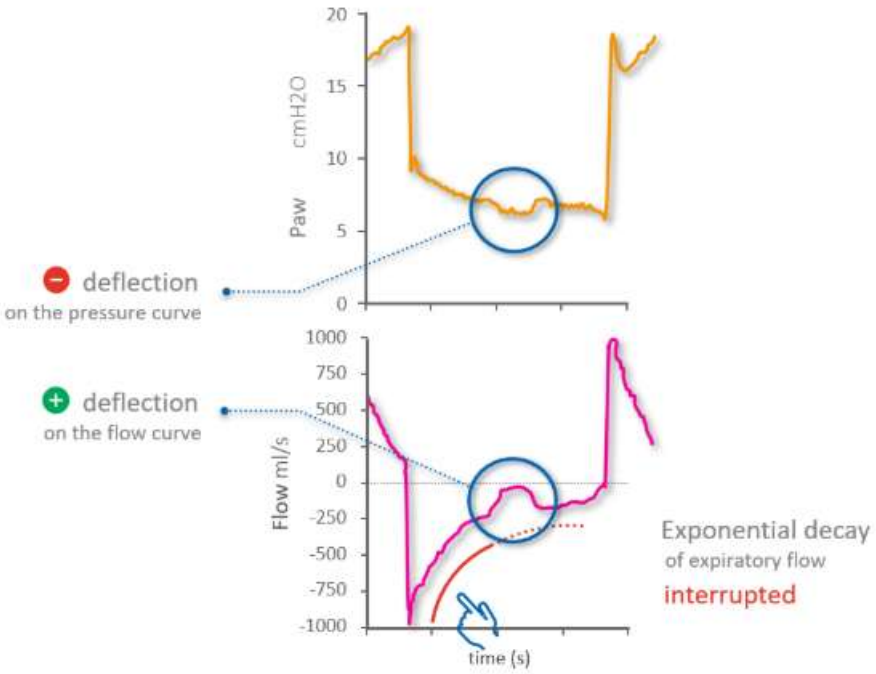
Patient generates an inspiratory effort insufficient to trigger ventilator breath delivery

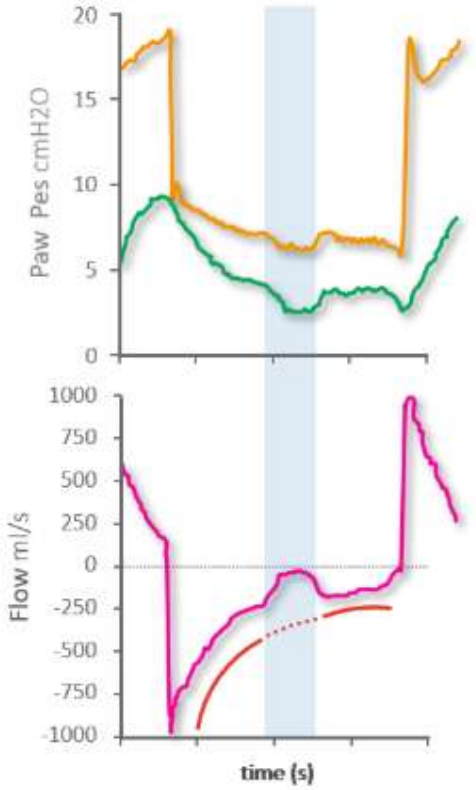
Most commonly occurs in COPD

Causes:

- Trigger sensitivity too insensitive - threshold set inappropriately high
- High respiratory rate with short expiratory time - insufficient time to reach trigger threshold
- Auto-PEEP (dynamic hyperinflation)
- Pressure support too high
- Over-sedation / neuromuscular weakness - effort magnitude simply too low

How to detect ineffective trigger

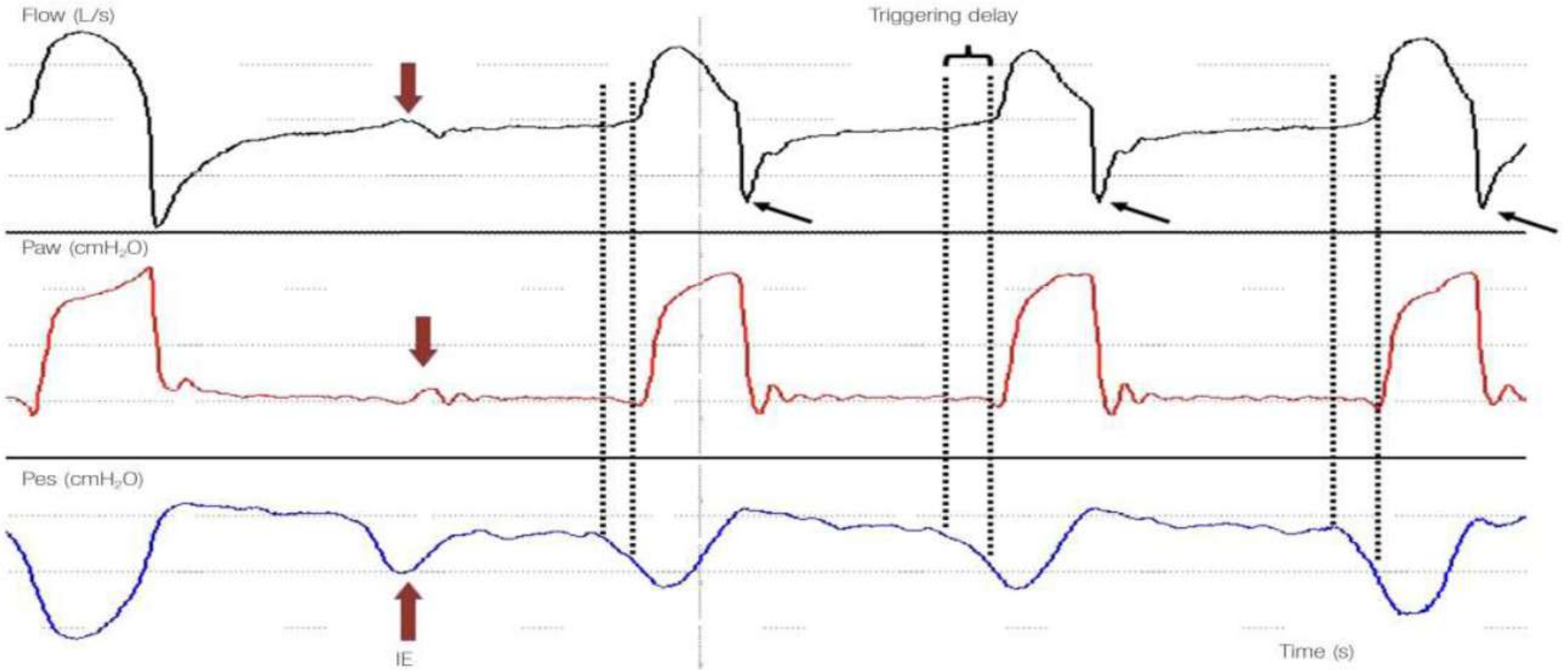




Flow-time curve: Patient effort → small positive deflection
Paw-time curve: Negative dip in pressure (–0.5 to –2 cmH_2O)
Oesophagal Pes: clear negative deflection despite no breath

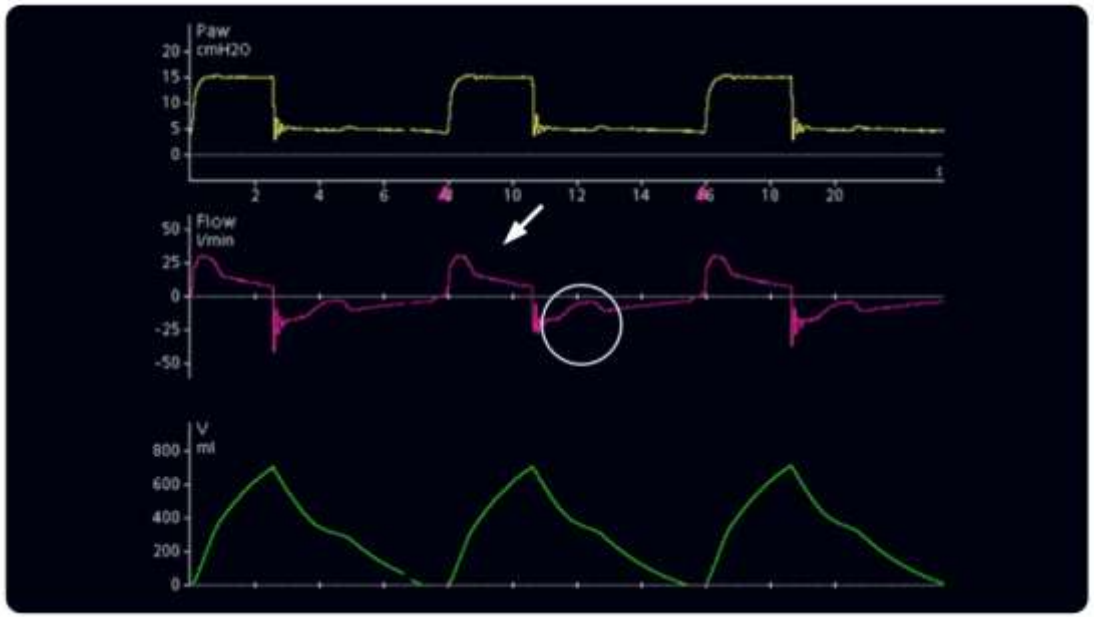
Mojoli et al. Critical Care (2022) 26:32

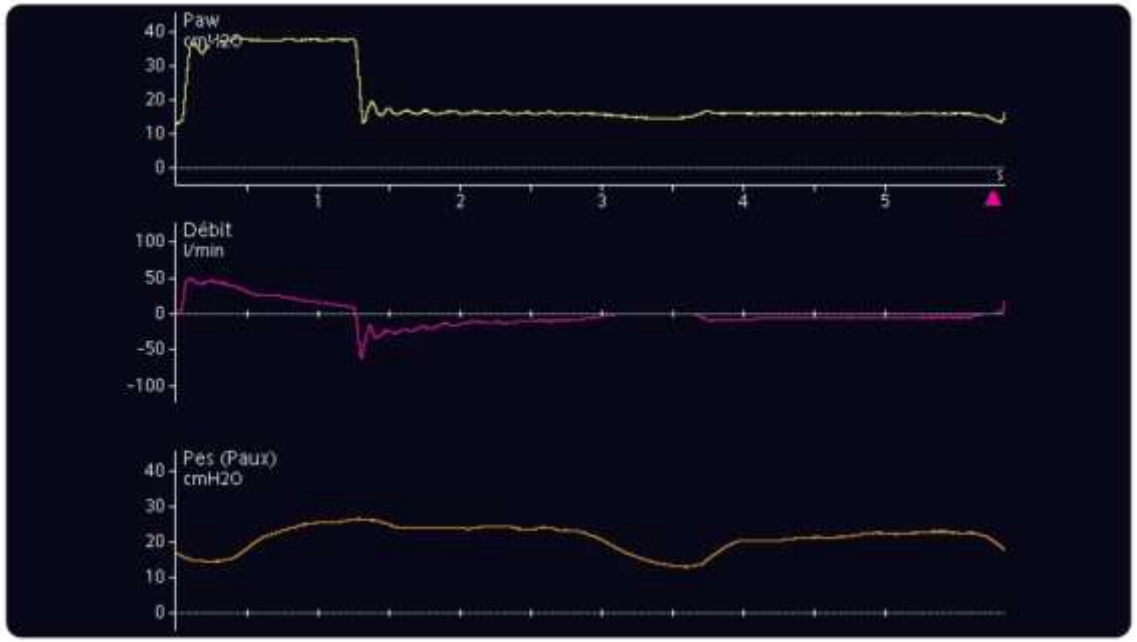
Ineffective trigger and delayed trigger



Korean J Crit Care Med 2017 November 32(4):307-322

Waveform characteristics indicating an ineffective effort

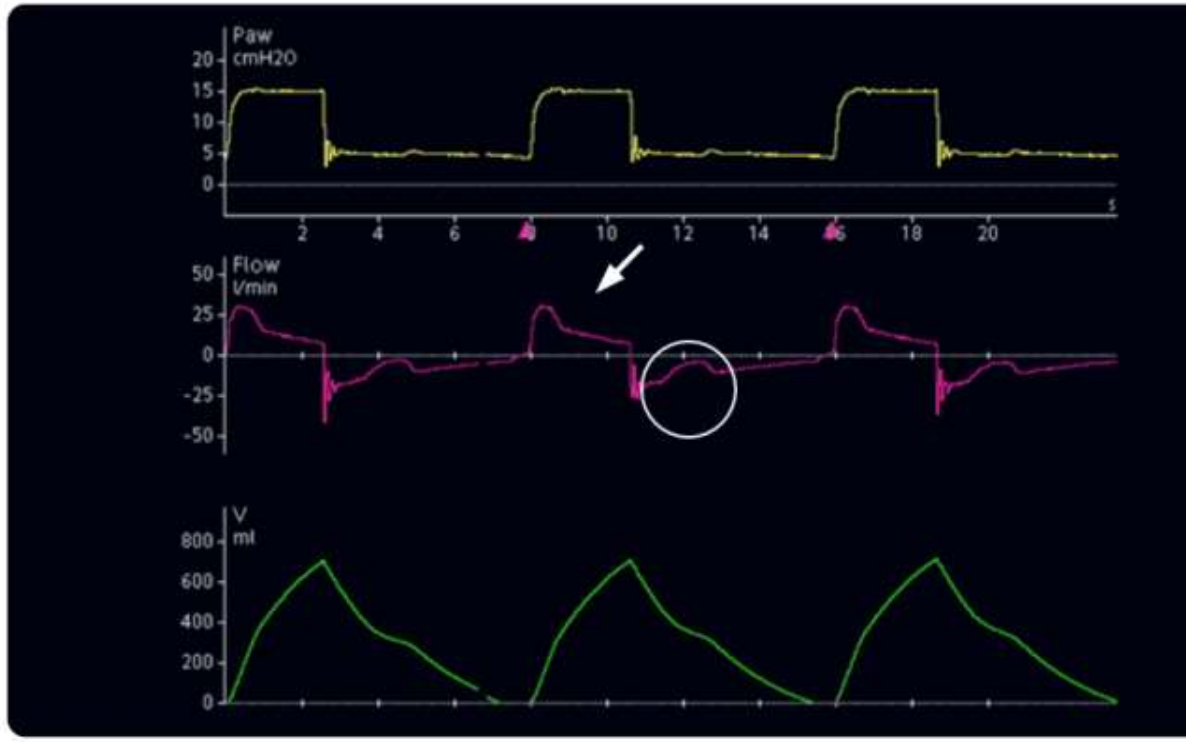




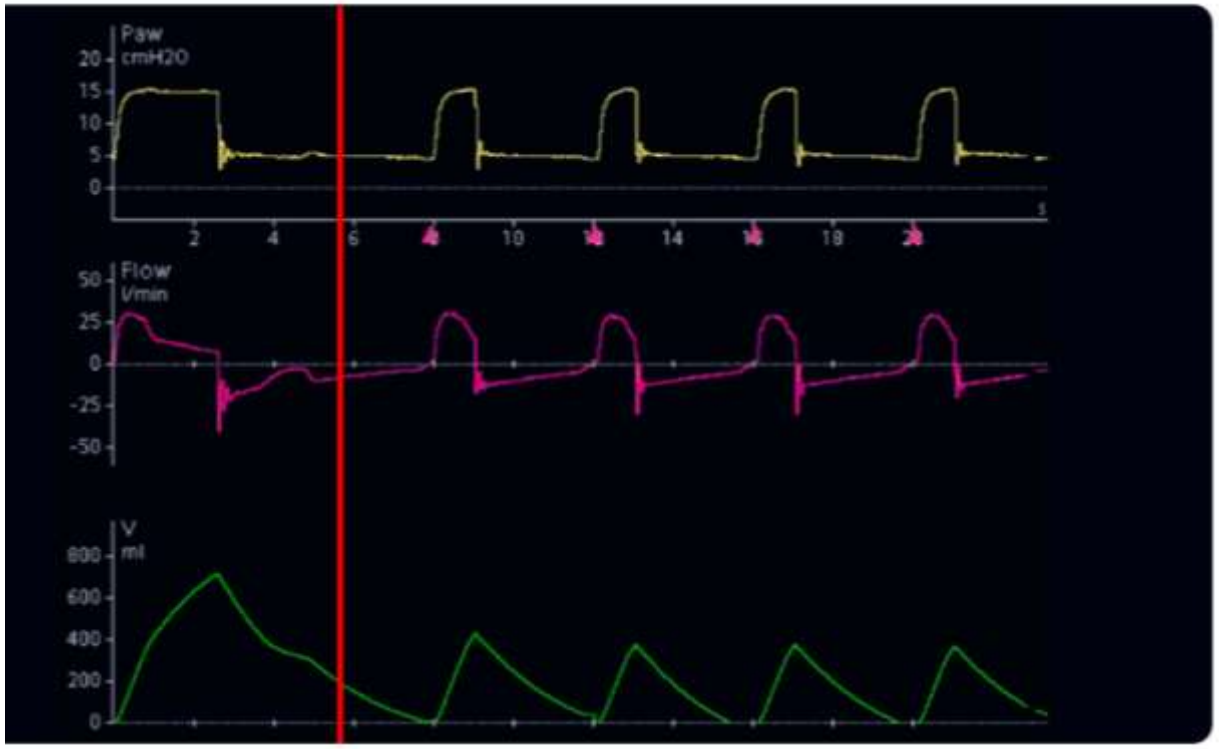
Subtle deflections in expiratory flow or pressure waveform

NO breath delivered — 'missed' triggering

Waveforms before and after intervention

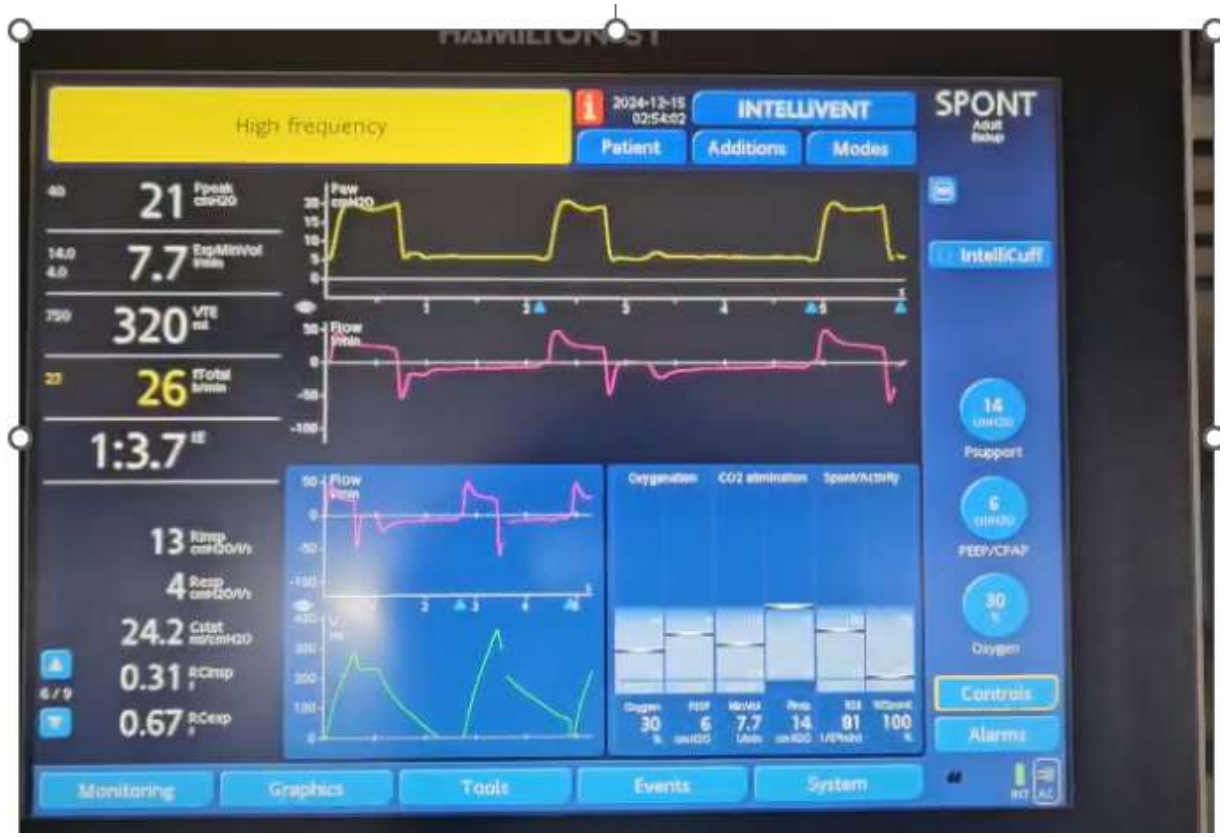


Ineffective effort with ETS set at 25%



ETS increased to 50% at the red line, resulting in elimination of ineffective efforts

Waveform characteristics indicating an ineffective effort



Management

- Measure and address auto-PEEP→ bronchodilators, ↑ expiratory time and set extrinsic PEEP
- Optimise trigger sensitivity
- Reduce PS level if over-assisting
- Review sedation

Auto-PEEP

Factors causing auto PEEP:

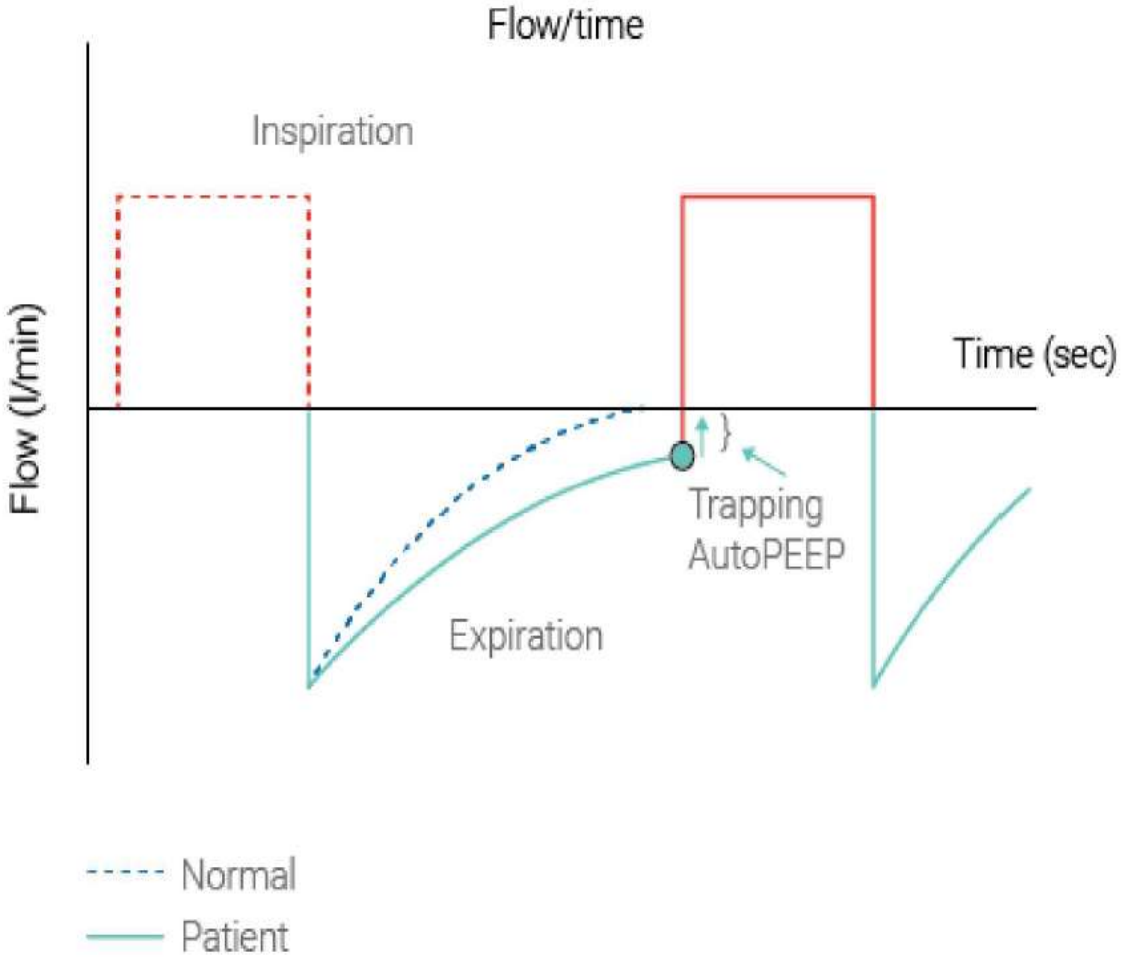
- Increased inspiratory time constant – large tidal volume
- Prolonged expiratory time constant – bronchoconstriction, obstructive diseases
- Shortened expiratory time – higher respiratory rate

Consequence of auto PEEP:

- Hypotension
- VILI
- PV asynchrony

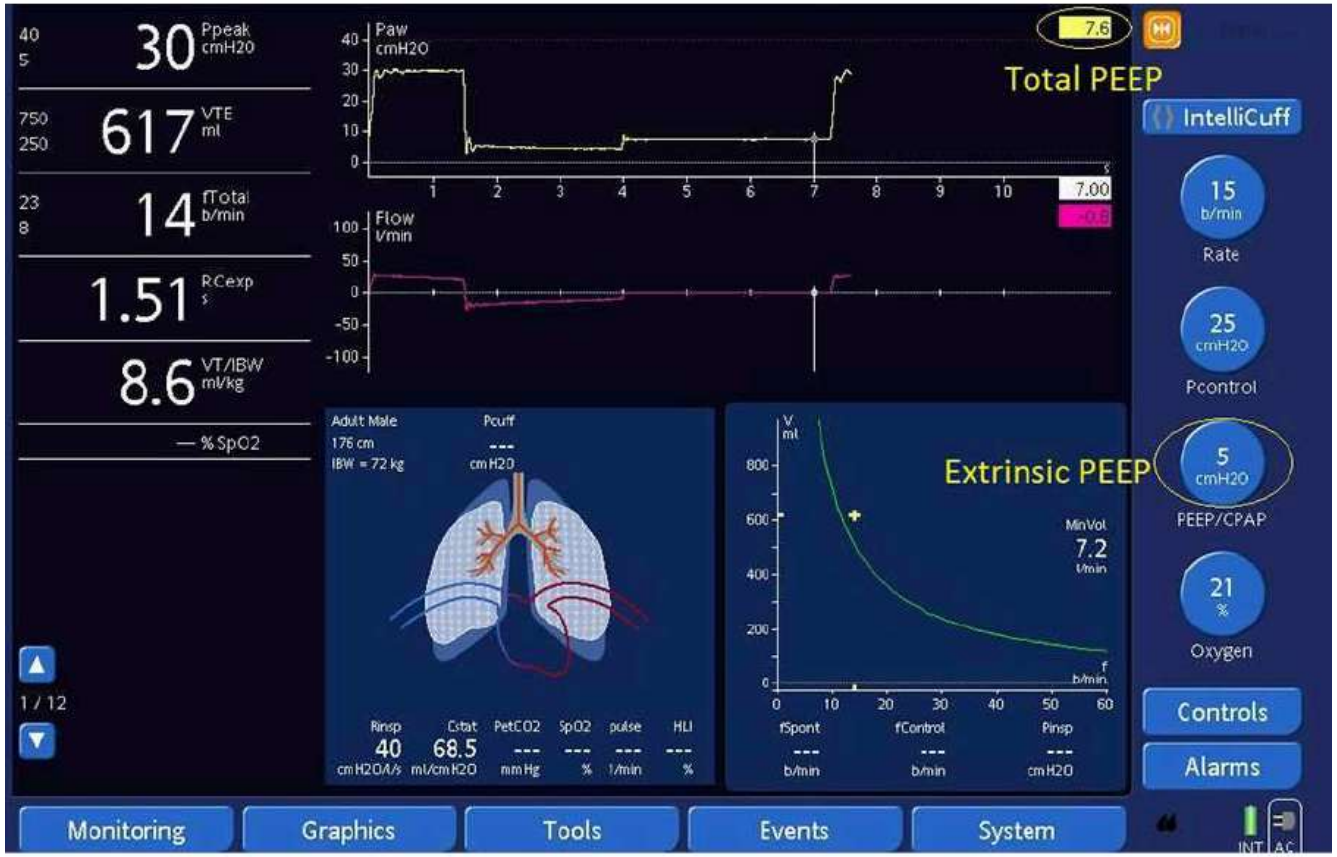
J Clin Med 2021;10:4550 I Oto et al. 2021

Auto PEEP and air trapping



How to detect AUTO PEEP ?

How to measure AUTO PEEP ?



Measuring total PEEP using an expiratory hold maneuver
Calculate AutoPEEP by subtracting extrinsic PEEP from the total PEEP.

Auto-triggering

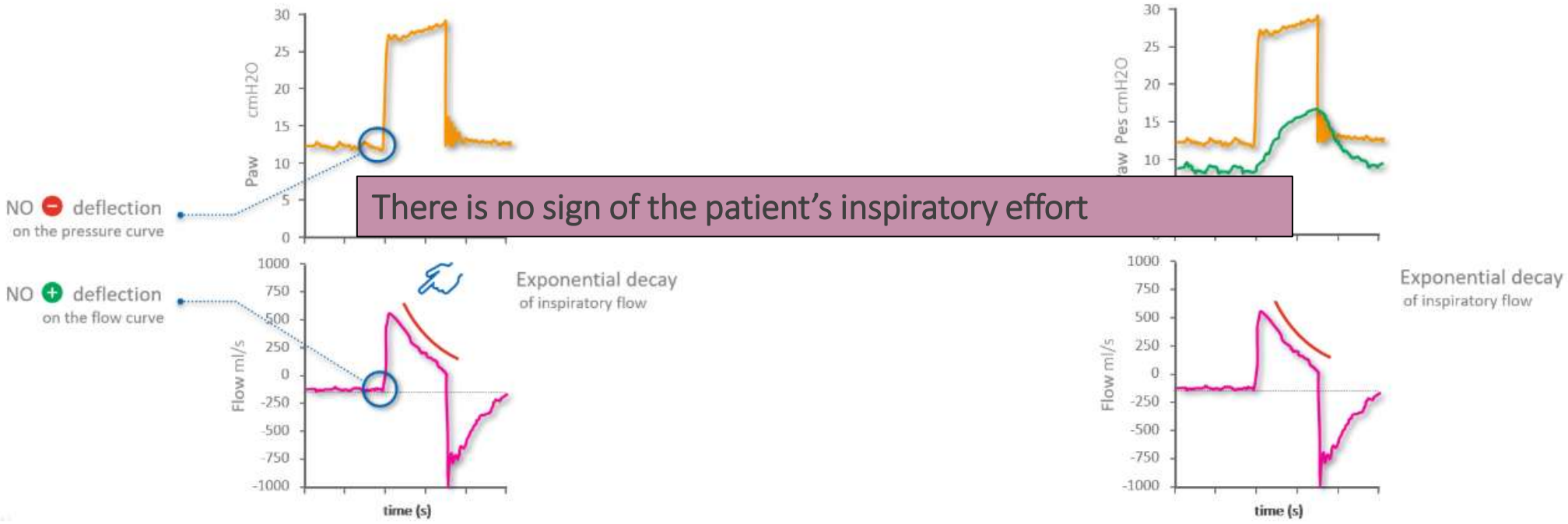
DEFINITION

Mechanical breath delivered WITHOUT a patient effort. The ventilator is triggered by spurious signals mistaken for patient effort.

CAUSES

- Circuit leaks (air leak from ETT cuff)
- Water condensation in circuit
- Trigger threshold set too sensitive
- HFO oscillations
- Cardiogenic oscillations in the circuit

How to detect Autotrigger



No clear negative deflection of Paw
No clear positive deflection of Flow

No negative deflection visible on the Pes reference waveform

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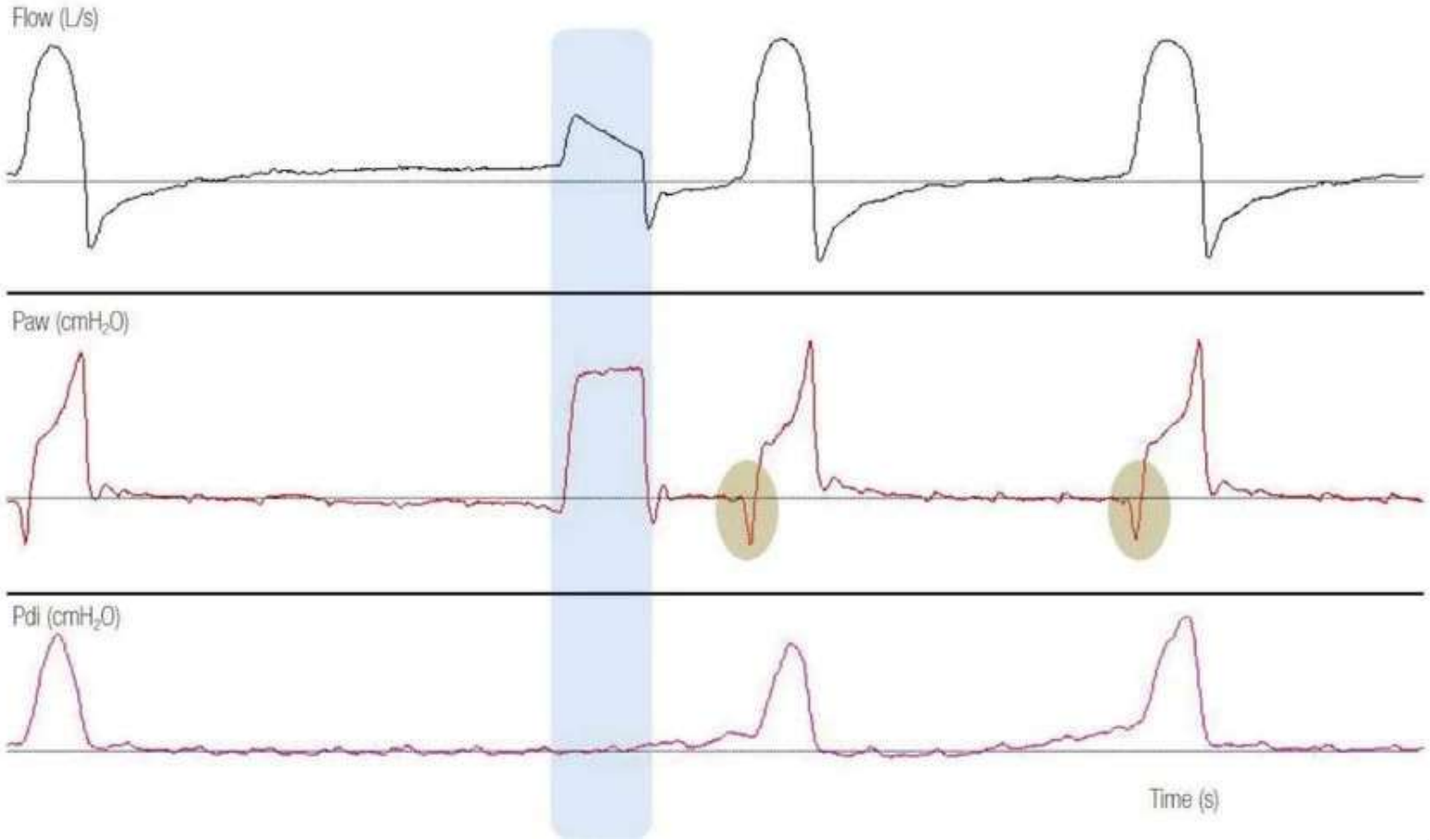
Waveform clues



Korean J Crit Care Med 2017 November 32(4):307-322

Auto-triggering

CLINICAL DANGER

- Breath stacking → ↑ tidal volumes
- Air trapping, auto-PEEP
- Breath delivered in expiration
- Cardiovascular compromise
- Alkalosis from over-ventilation

J Clin Med 2021;10:4550 I Expert Rev Respir Med 2021

Management

- ↓ trigger sensitivity
- Check circuit for leaks/condensation
- Check the heated wire circuit,
- Consider flow triggering if pressure triggering

J Clin Med 2021;10:4550 I Expert Rev Respir Med 2021

Cycling Dyssynchrony: Premature Cycling

Mechanical breath ends BEFORE the patient's neural inspiration ends.

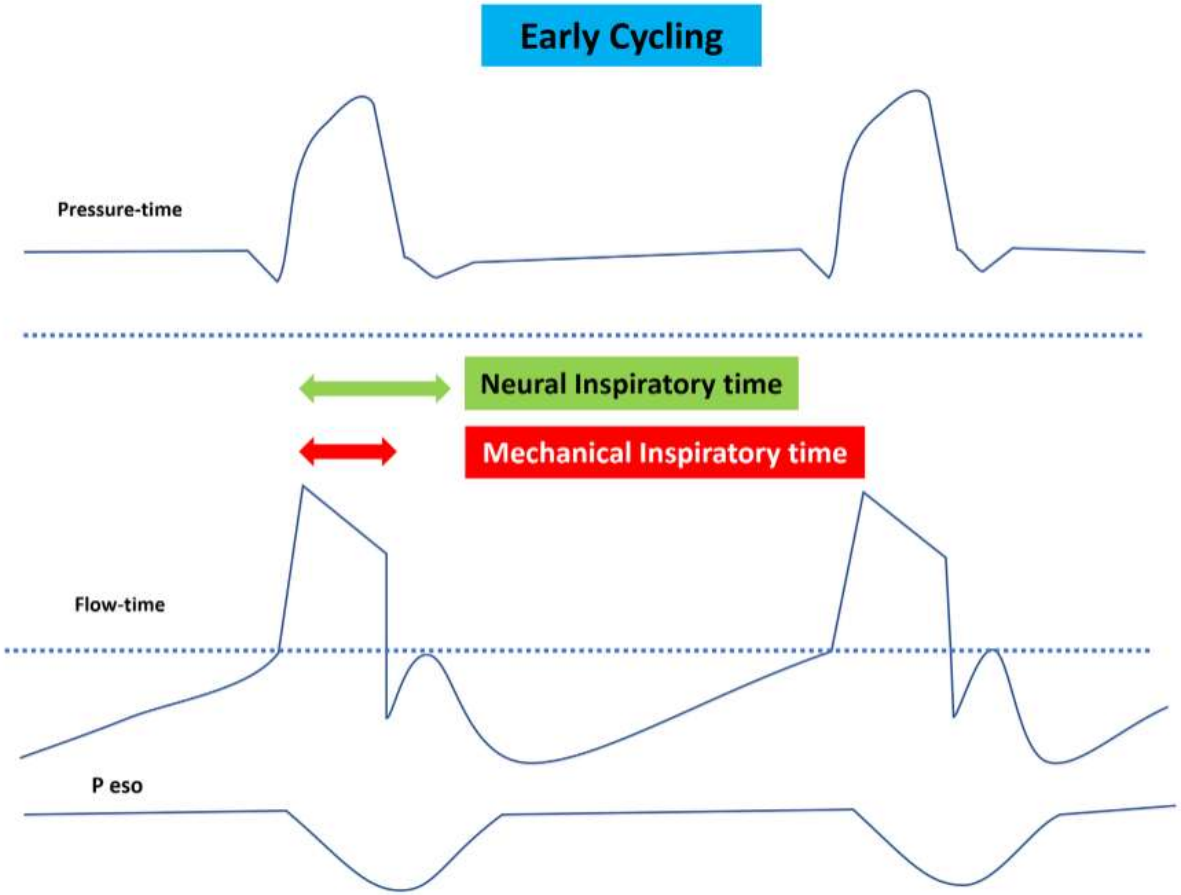
- Common with low compliance(ARDS)

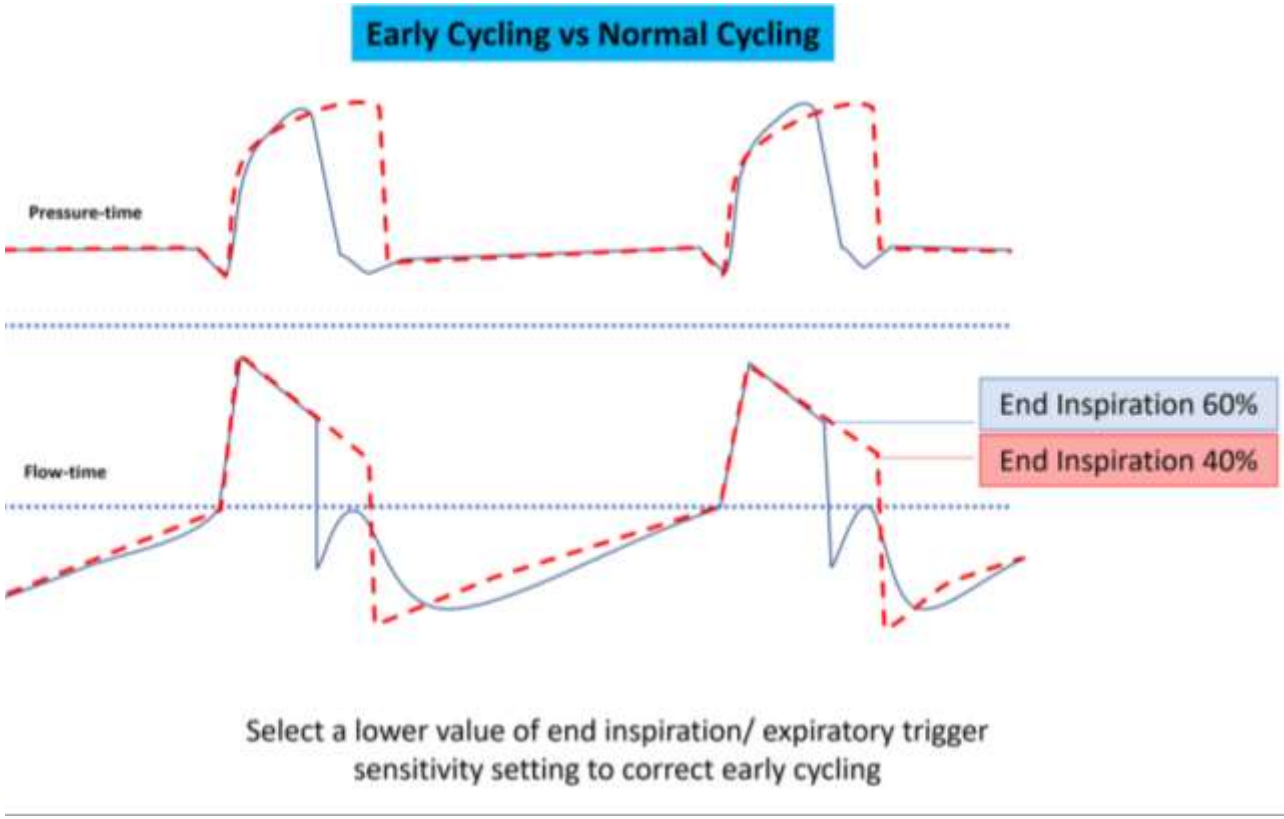
Causes:

- Short Ti setting
- high cycling-off threshold (>25% peak flow in PSV),
- high PS levels
- small leaks.

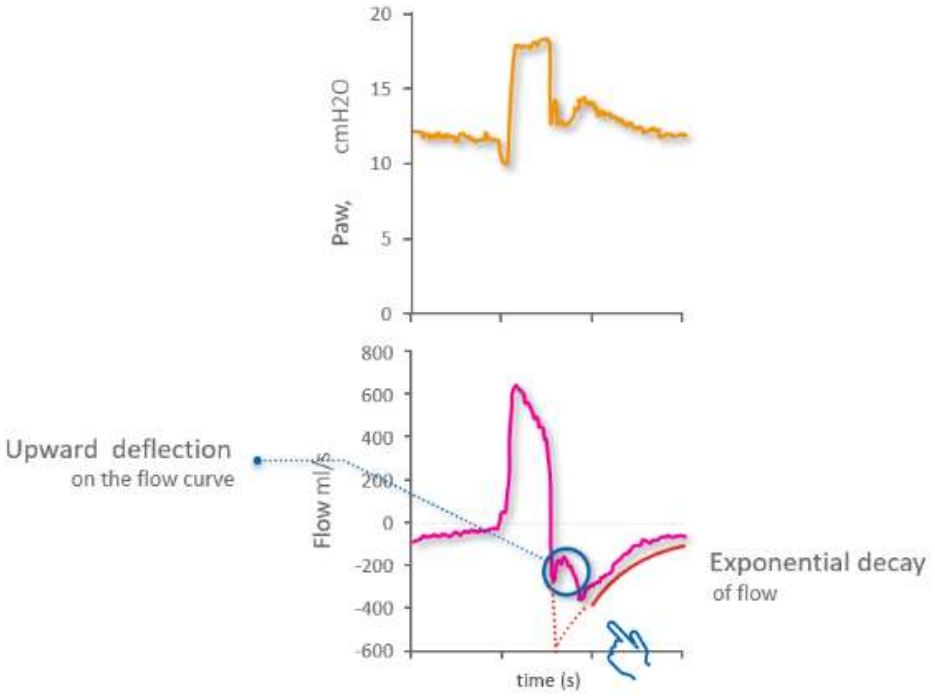
J Clin Med 2021;10:4550

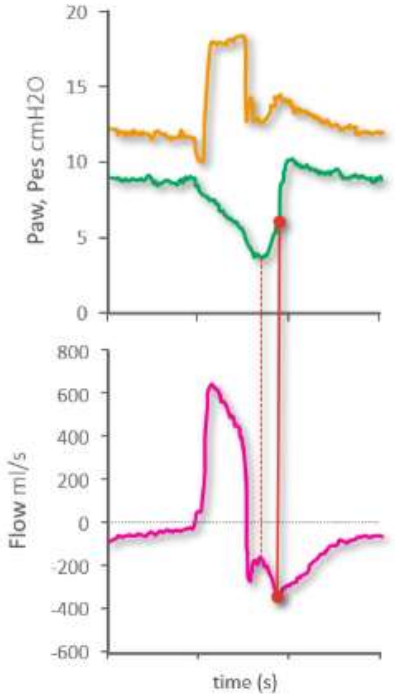
Why it happens ?





How to detect Premature cycling





Normal peak flow is replaced by an upwards deflection, while the normal exponential decay starts later

Point of maximum inspiratory effort in Pes takes place during expiration and corresponds with the upwards deflection of the expiratory flow.

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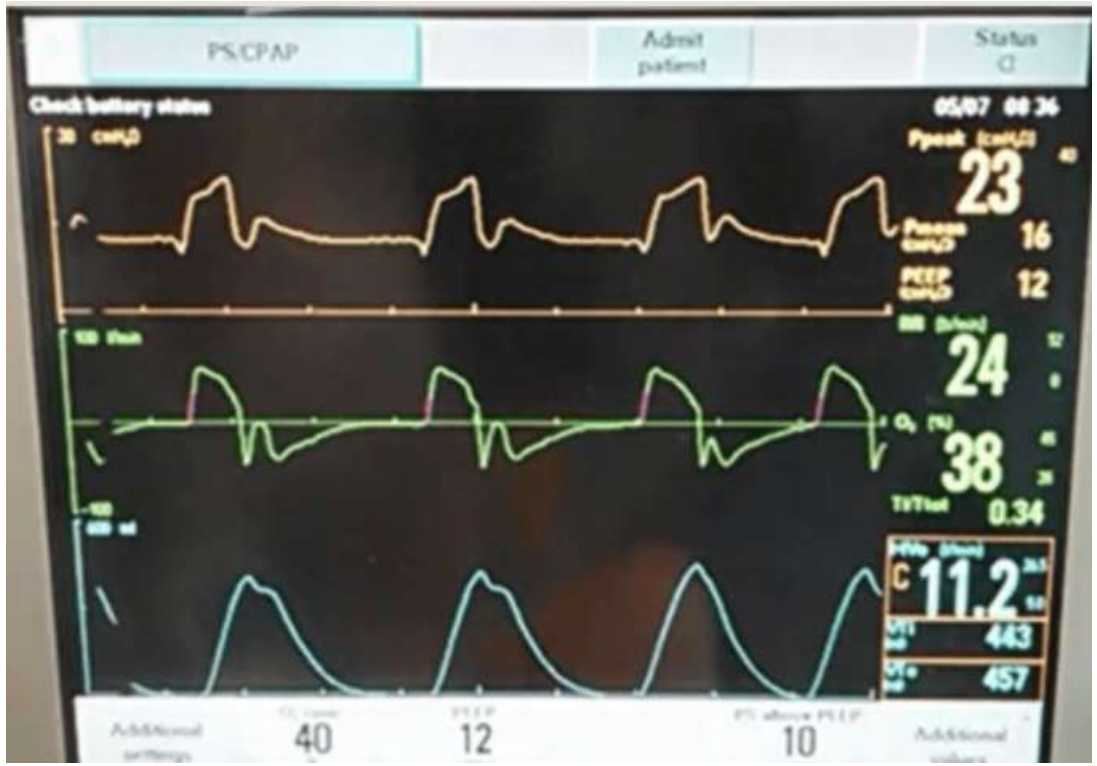
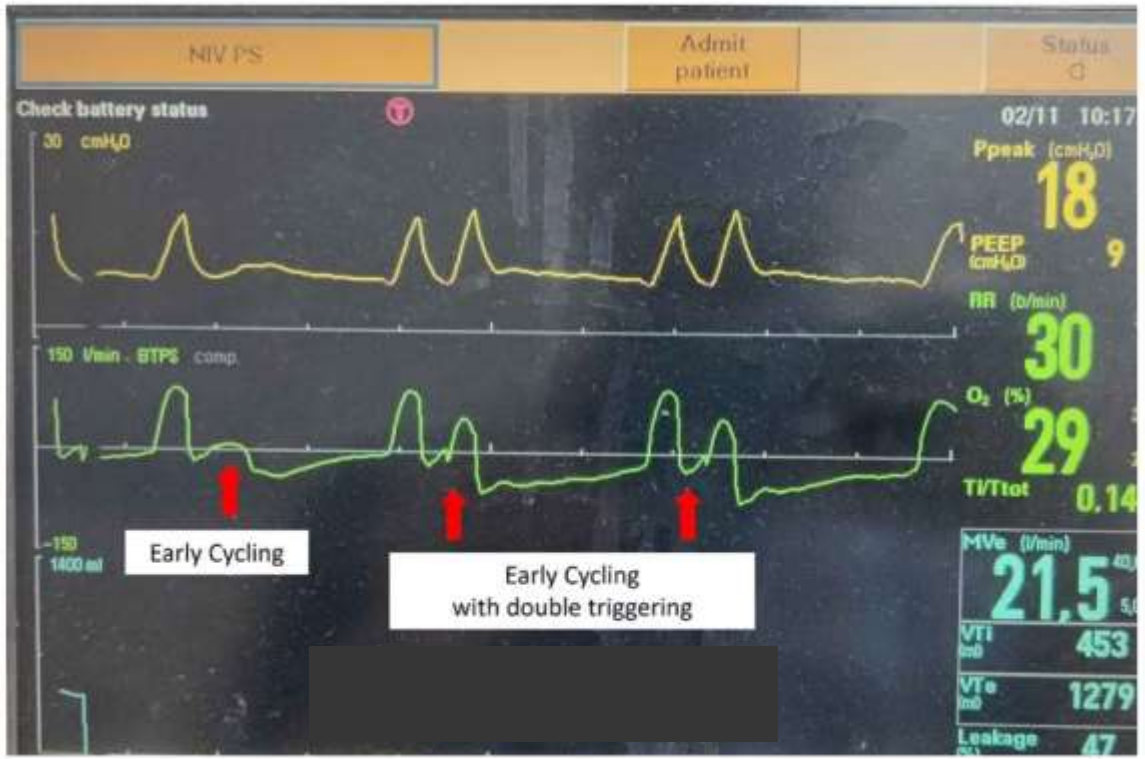
Consequence:

- Ineffective breathing
- ↑ respiratory rate,
- Increased discomfort may progress to double triggering.

Waveforms


PS/CPAP
Admit patient
Status
Check battery status
23
16
12
24
38
0.34
11.2
443
457
40
12
10


NIV PS
Admit patient
Status
Check battery status
02/11 10:17
Ppeak
18
PEEP
9
RR
30
29
TI/TTot
0.14
MVe
21,5
VTI
453
VTe
1279
Leakage
47
Early Cycling
Early Cycling with double triggering

Waveforms before and after intervention

When ETS set at 45%

ETS decreased to 15%

Management of early cycling

- Increase Ti
- Reduce cycle-off threshold in PSV (cycle off at a lower % of peak flow)

Cycling Dyssynchrony: Delayed Cycling

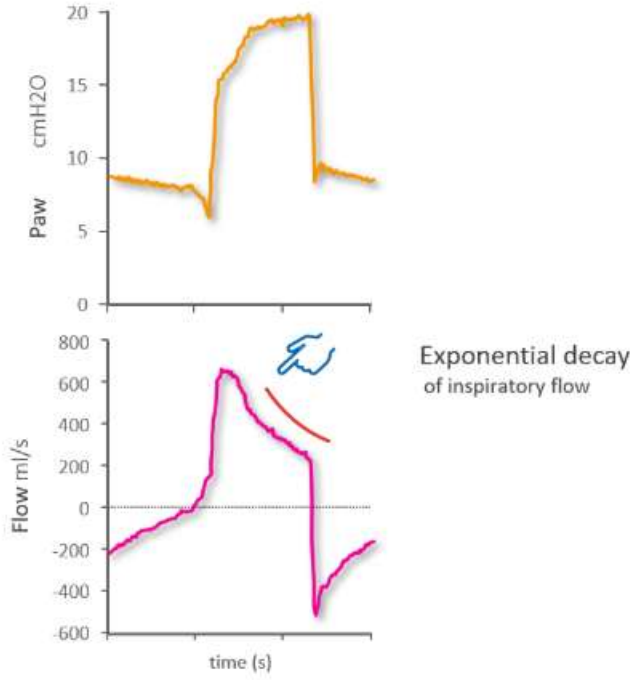
Mechanical breath continues BEYOND the patient's neural inspiration.

- Most common in PSV mode

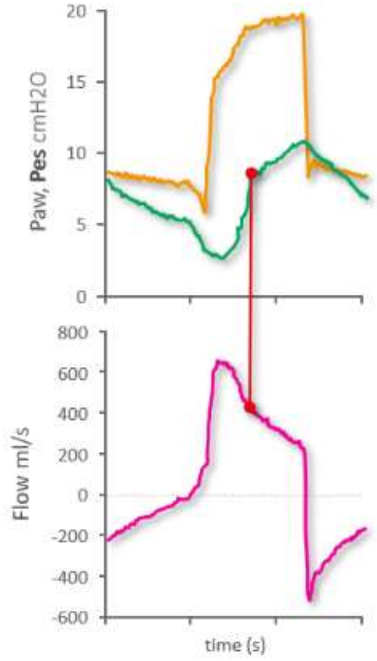
Causes:

- Low cycling-off threshold (<5%)
- high compliance (low recoil)
- obstructive physiology (COPD)
- high PS level.

How to detect Delayed cycling



The Flow waveform from the prolonged exponential decay of inspiratory flow occurring after the first phase with upwards convexity



A point on the Pes rise after its nadir that marks a clear change of slope

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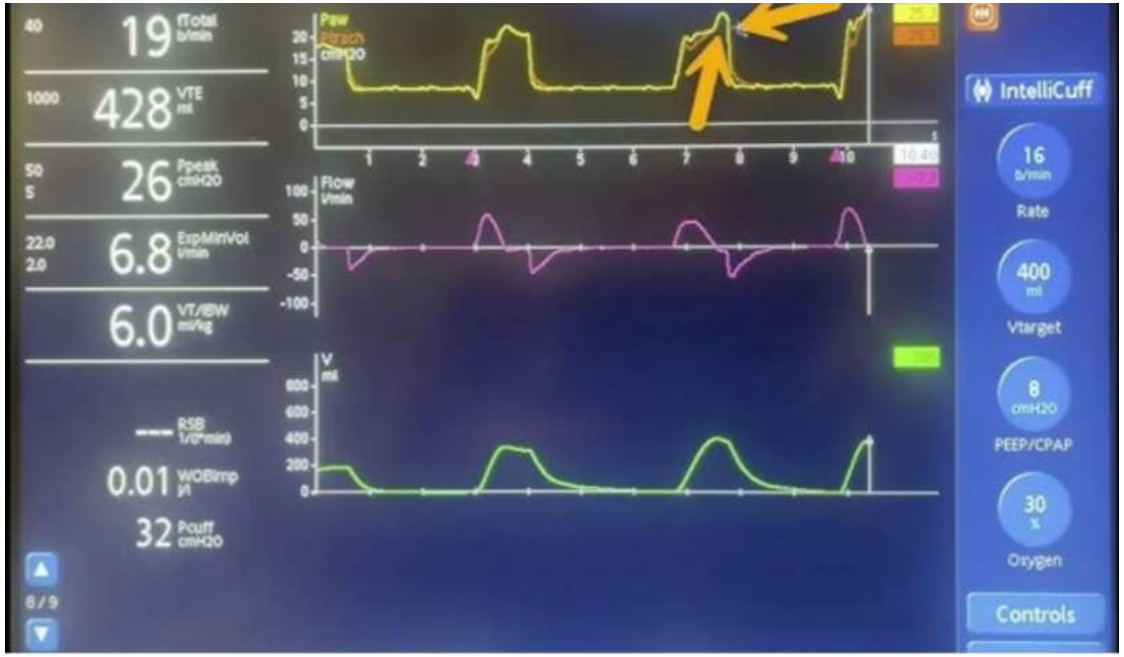
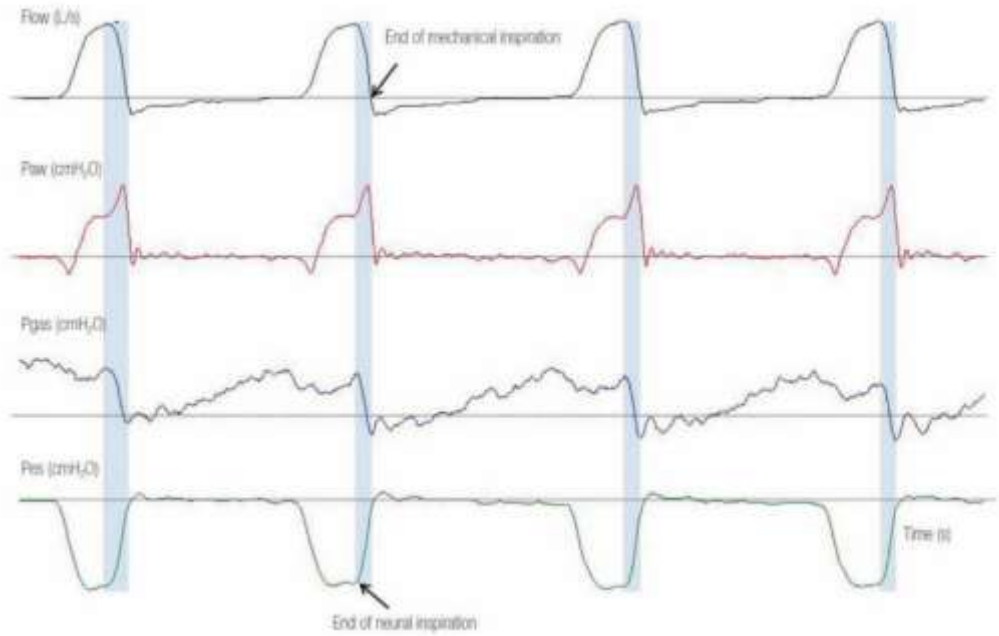
Waveforms



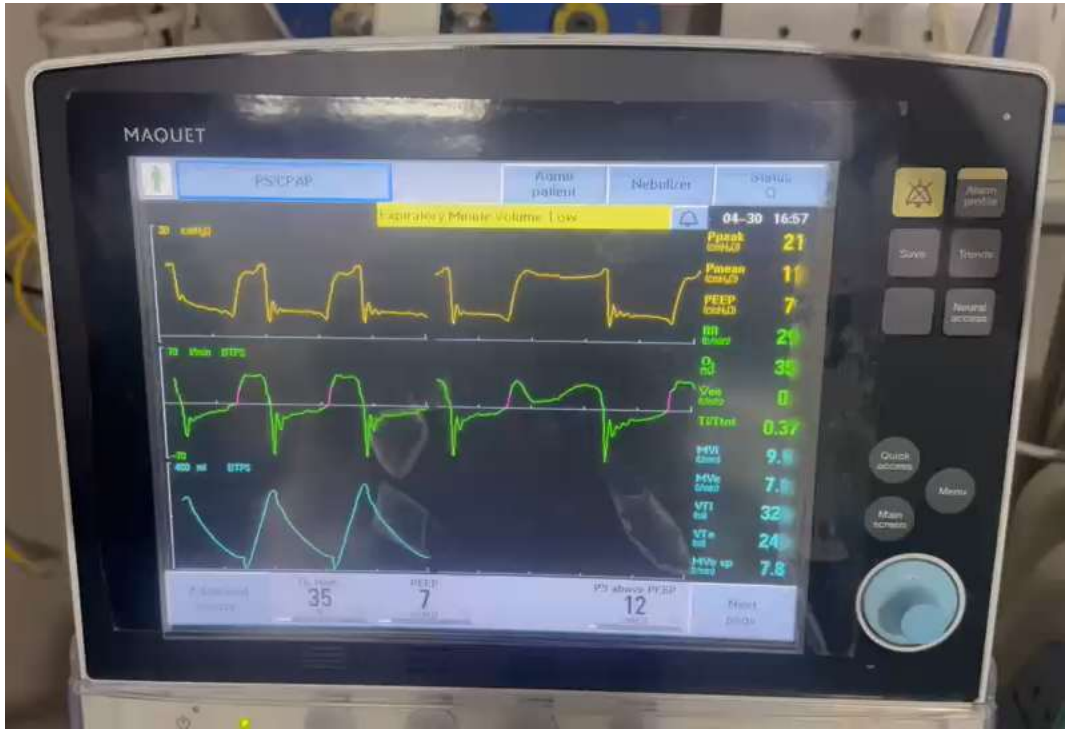


Korean J Crit Care Med 2017 November 32(4):307-322, ,J Clin Med 2021;10:4550

Waveforms


HAMILTON-S1
High frequency
2024-06-21
11:08:36
INTELLiVENT
ASV
Adult
Patient
Additions
Modes
22 Ppeak cmH2O
11.1 ExpMinVol l/min
295 VTE ml
34 fTotal b/min
1:2.1 tE
3 Rinsp cmH2O/l/s
4 Rexp cmH2O/l/s
34.1 Cstat
0.11 RCinsp
0.25 RCexp
Paw
Flow
V
IntelliCuff
200
%MinVol
10
PEEP/CPAP
26
Oxygen
Oxygenation
CO2 elimination
Spont/Activity
Controls
Alarms
Monitoring
Graphics
Tools
Events
System

Waveforms before and after intervention



When ETS set at 15%

ETS increased to 45%

Consequences

- ↑ WOB causing patient discomfort,
- Intrinsic PEEP
- respiratory alkalosis if severe.

Korean J Crit Care Med 2017 November 32(4):307-322, ,J Clin Med 2021;10:4550

How to fix it

- Decrease Ti
- Increase cycle-off sensitivity in PSV
- consider NAVA

J Clin Med 2021;10:4550

Double trigger

Double triggering:

Two ventilator insufflations delivered within one patient inspiratory effort, 2 consecutive breaths triggered by ONE neural effort — effort spans ventilator Ti

Mechanism:

Patient's neural inspiratory time (TiN) > set ventilator Ti → effort continues → triggers second breath

Respir Care. 2011;56(4):460-466

Types of double triggering

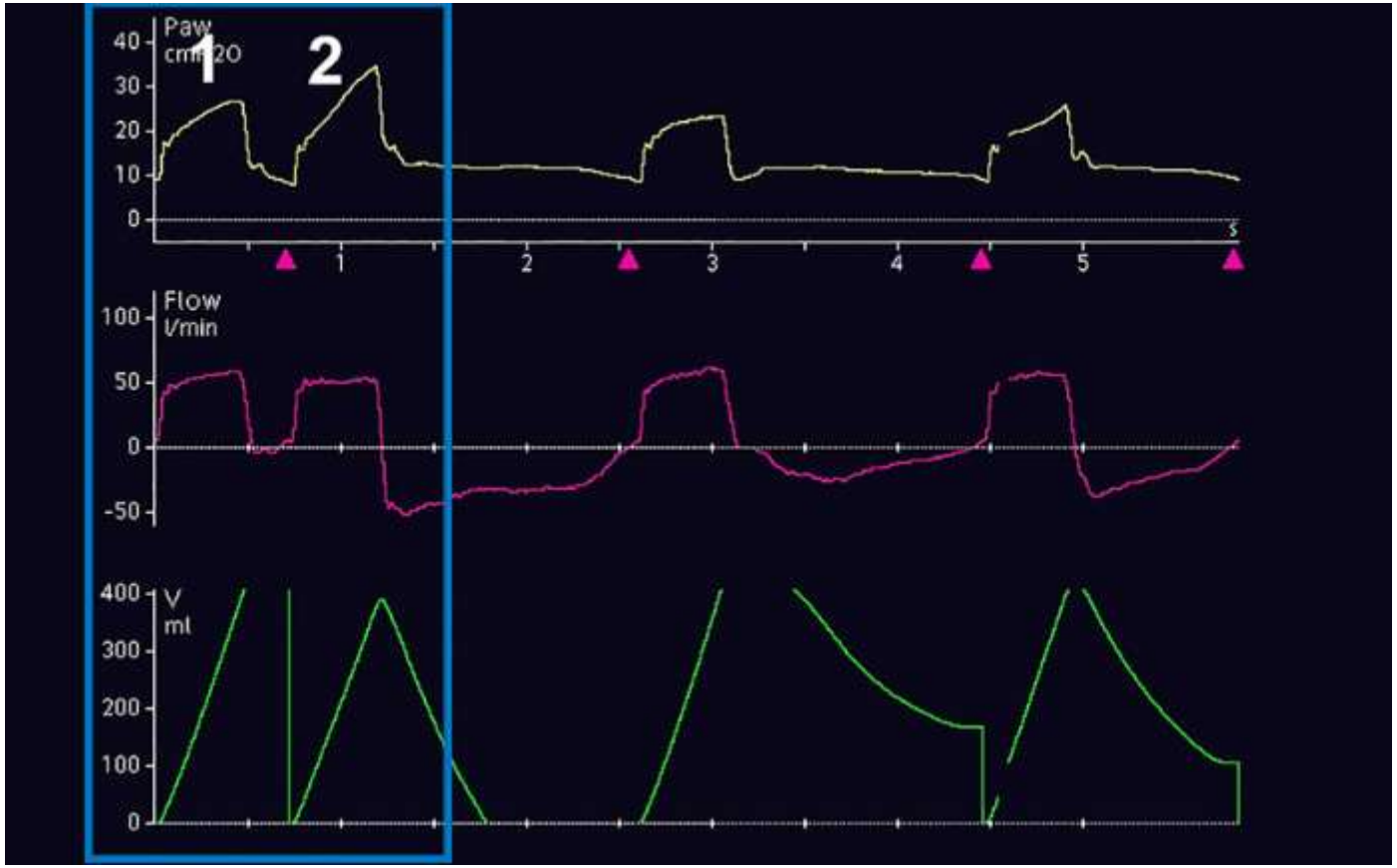
- **Patient-triggered (DT-P):** First triggered breath has an oesophagal pressure decrease of >1 cmH2O and may be associated with a strong inspiratory effort

- **Auto-triggered (DT-A):** First triggered breath occurs earlier than the ventilator-set time trigger, without a concomitant drop in oesophagal pressure

- **Ventilator-triggered (DT-V):** First breath occurs at the ventilator-set time trigger, without a concomitant drop in oesophagal pressure

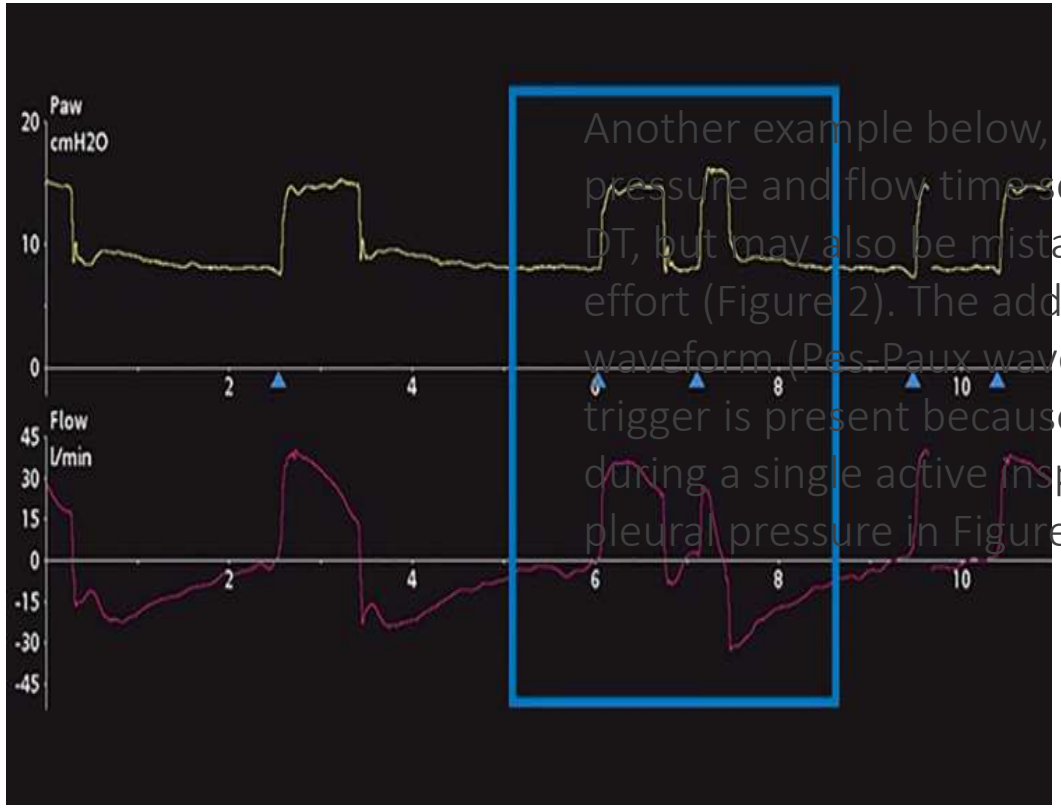
Respir Care. 2011;56(4):460-466

Pressure, flow, and volume waveforms revealing the DT

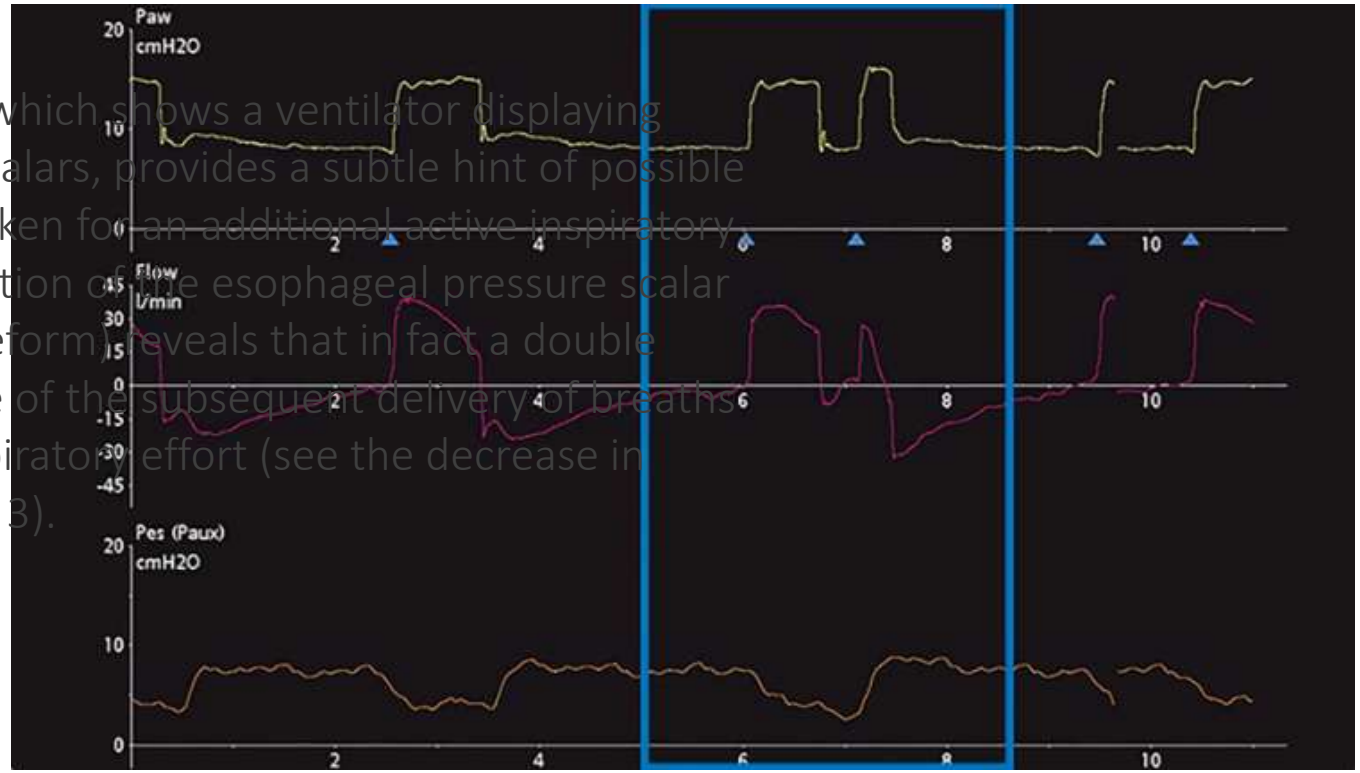


Waveform recognition: Two breaths with inter-breath interval <300–500ms; second breath without normal delay

Pressure, flow, and volume waveforms revealing the DT



Another example below, which shows a ventilator displaying pressure and flow time scalars, provides a subtle hint of possible DT, but may also be mistaken for an additional active inspiratory effort (Figure 2). The addition of the esophageal pressure scalar waveform (Pes-Paux waveform) reveals that in fact a double trigger is present because of the subsequent delivery of breaths during a single active inspiratory effort (see the decrease in pleural pressure in Figure 3).



The onset of a rapid decline of esophageal pressure to the nadir, was significantly longer in the first DT-P

Reverse Triggering (RT):

- Definition: Diaphragm contraction ENTRAINED by passive ventilator breath -not primary patient drive
- Mechanism: Stretch-receptor activation (Hering-Breuer reflex) → phrenic nerve stimulation by passive lung inflation
- Epidemiology: ~20% of deeply sedated patients; especially early ARDS during controlled MV

Why Dangerous?

- Eccentric diaphragm loading → diaphragm myotrauma (effort against already-inflating lung)
- May cause pendelluft despite 'controlled' ventilation mode — no protection from RT in volume control
- Can trigger double-triggering if effort is large enough → compound injury

Costa V et al. J Clin Med 2025 | Damiani LF & Goligher EC. Crit Care Med 2023

Waveforms

Reverse triggering

Management

- Assess sedation depth (RASS)
- Consider EAdi monitoring or Pes
- NMB in severe cases

Clinical implication: 'Controlled' modes do NOT guarantee absence of patient effort or injury

Inspiratory Phase: Flow Dyssynchrony

FLOW STARVATION (Under-assist)

- Delivered inspiratory flow is LESS than the patient's demand.
- Occurs in volume control mode with fixed flow settings

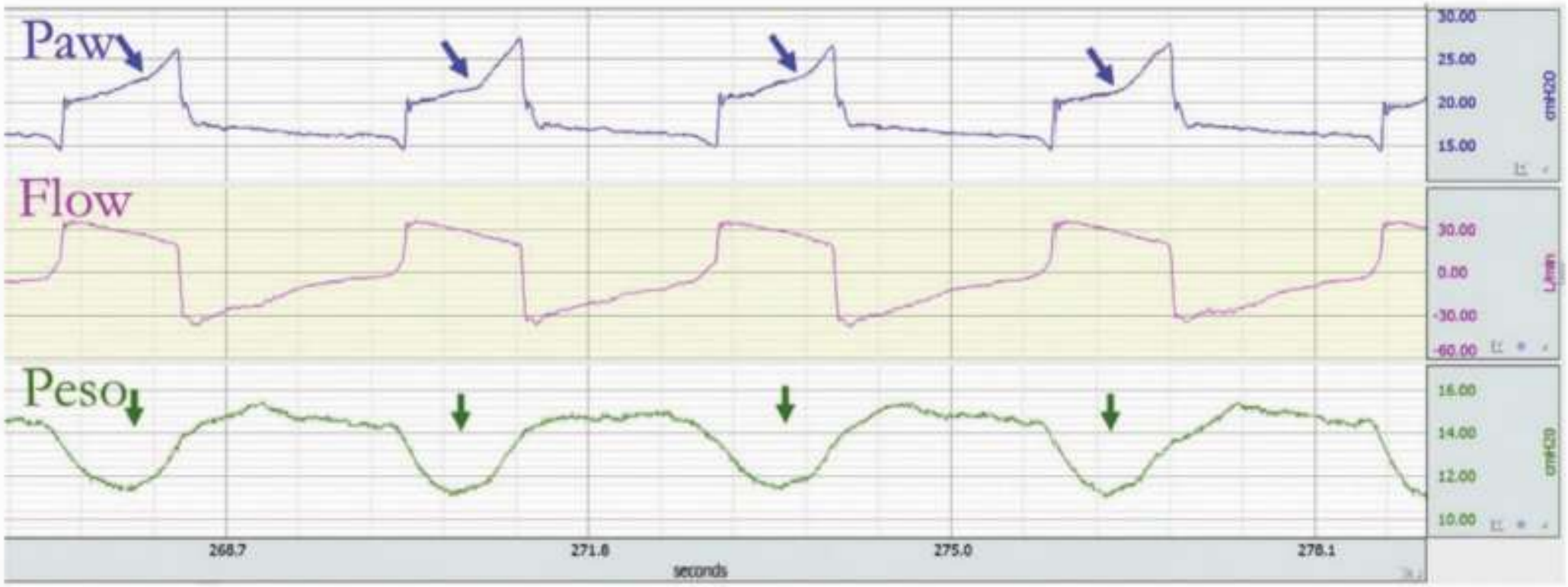
- **Consequence:** Patient works against the ventilator → ↑ WOB, ↑ Pes swings, air hunger, activation of accessory muscles, can lead to double triggering

➢ Patient-related factors - pain, anxiety, fever, metabolic acidosis

➢ Ventilator factors: low tidal volume, high inspiratory time, high rise time

Acute Crit Care. 2022 Nov;37(4):491-501

How to detect?



Waveform: Pressure-time curve shows a characteristic CONCAVITY (scooped shape) during inspiration

Crit. Care Clin. 2018, 34,325–341

Waveforms before and after intervention

After sedation

Waveforms


HAMILTON·S1
INTELLIVENT
(S)CMV
Patient
Additions
Modes
23
7.9
255
31
1:1.3
7
15
25.1
0.18
0.34
Controls
Alarms
Monitoring
Graphics
Tools
Events
System

Management

- ↑ flow rate
- Consider switching to PCV/PSV – flow varies with patient effort, rate of inspiratory pressure rise adjustment
- Address underlying high drive – control of pain, anxiety, fever and metabolic disturbances

Acute Crit Care. 2022 Nov;37(4):491-501

Inspiratory Phase: Flow Dyssynchrony

FLOW EXCESS (Over-assist)

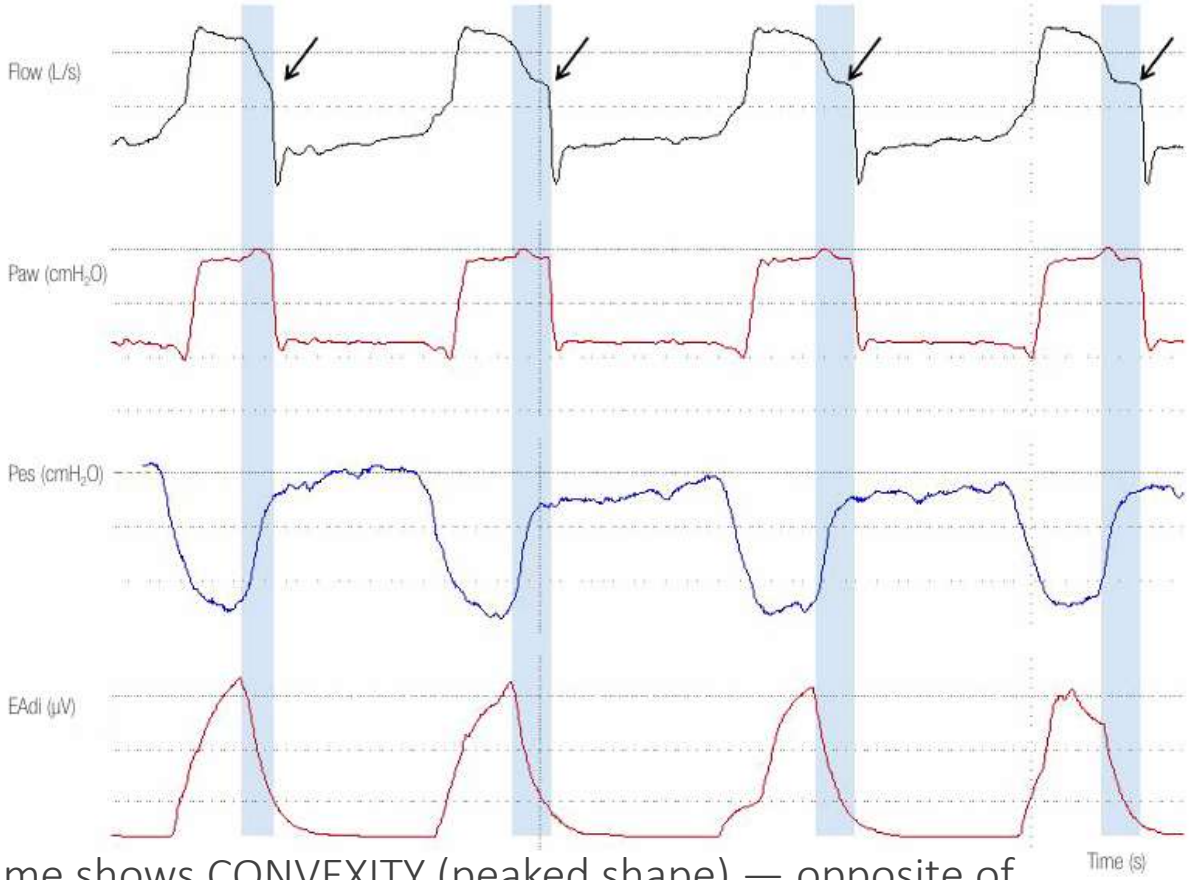
- Delivered flow EXCEEDS patient demand.

- Common in PSV with very high support levels. Patient's own effort rapidly satisfied → premature cycling.

- Waveform:
- **Consequence:** Premature cycling, patient discomfort, breath at peak pressure → expiratory muscles activated during inspiration → delayed cycling.

Acute Crit Care. 2022 Nov;37(4):491-501

Waveforms



Pressure-time shows CONVEXITY (peaked shape) — opposite of starvation. Expiratory flow may start before the patient's neural Ti ends.

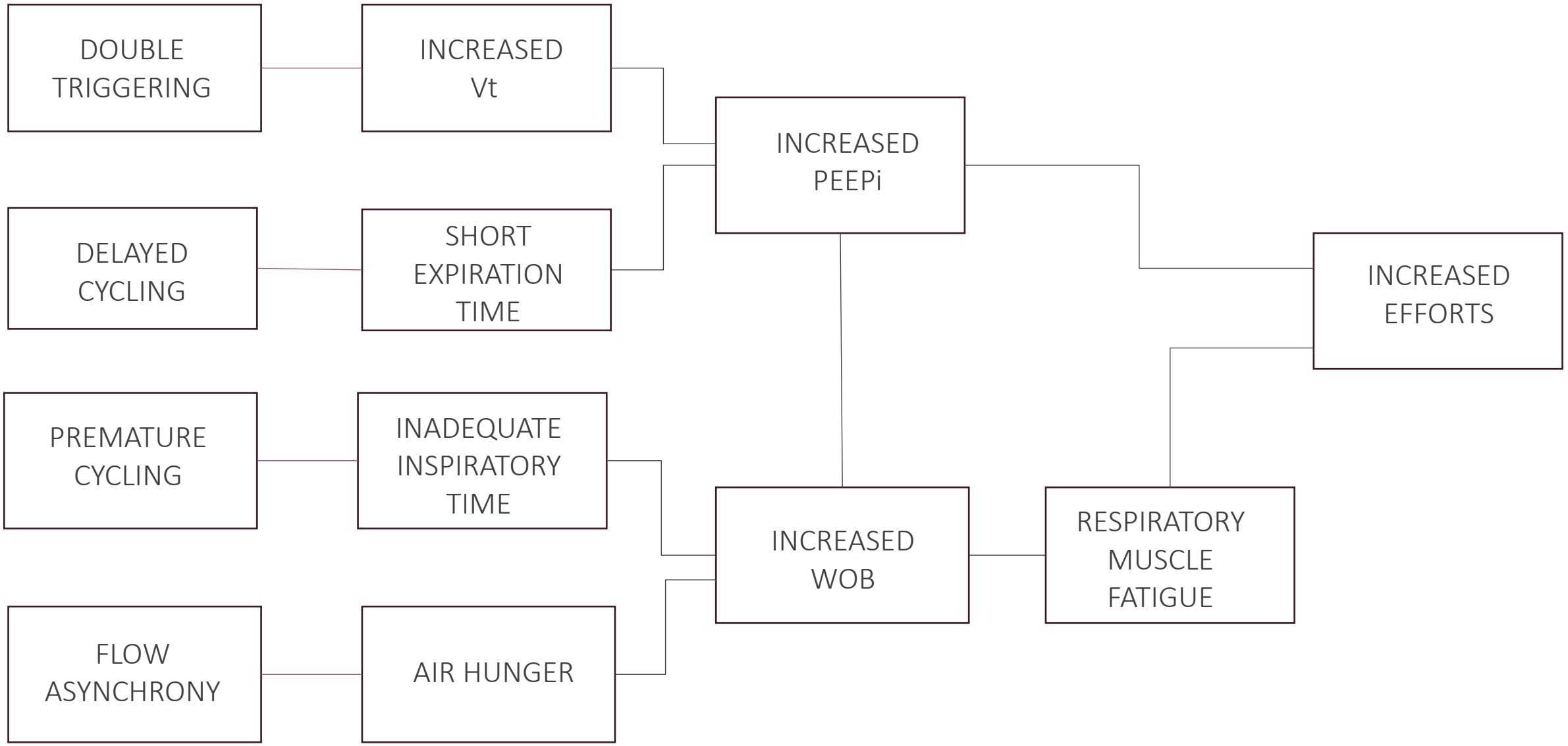
Korean J Crit Care Med 2017 November 32(4):307-322

Management

- ↓ PS level
- ↑ rise time (slow pressurization)
- Adjust cycling criteria

Acute Crit Care. 2022 Nov;37(4):491-501

Asynchrony Begets Asynchrony



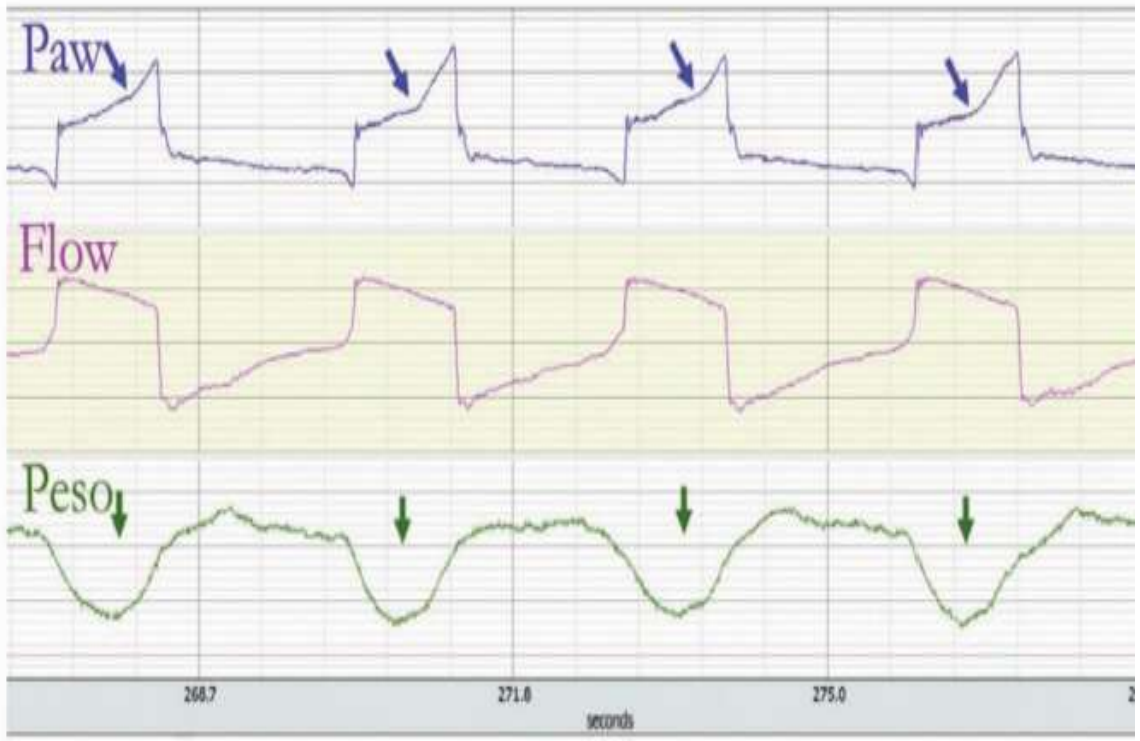
Esophageal waveform monitoring

- Normal values: Delta Pes = 3 -8 cm H20; Edi = 5-15

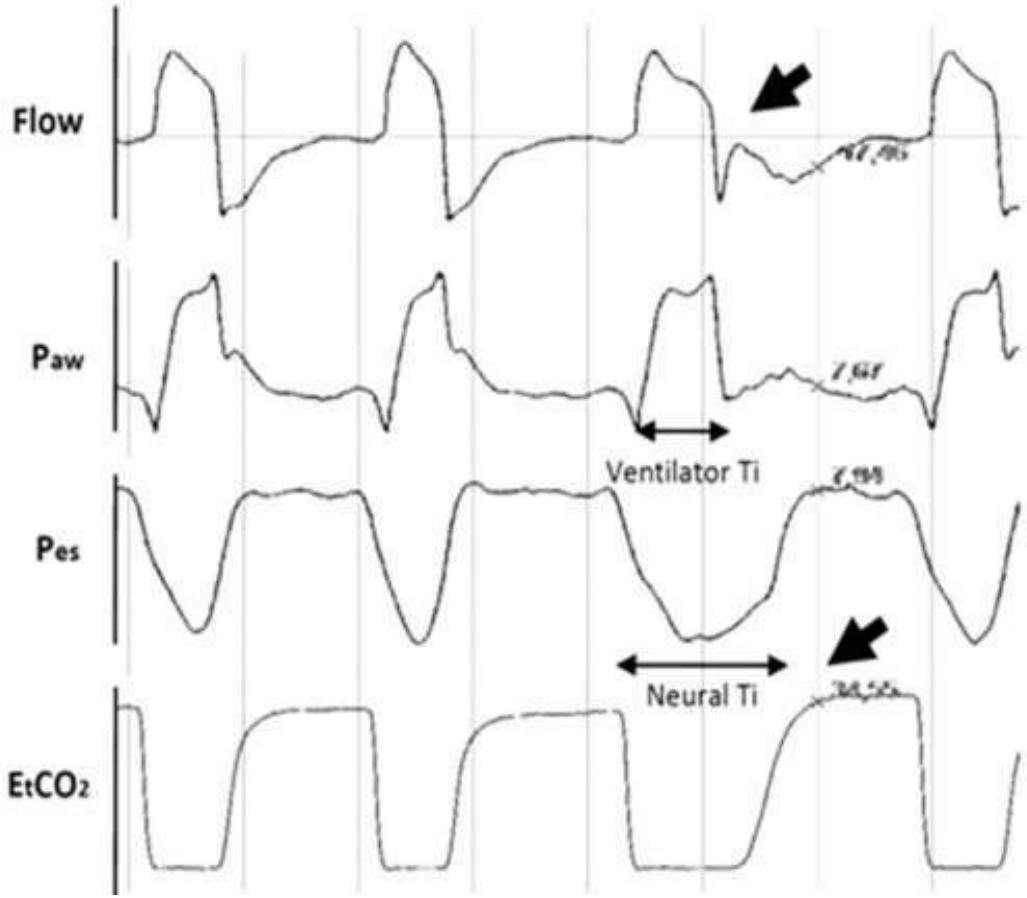
Annemijn H. Jonkman et al.,European Respiratory Review 2023 32: 220186;

Waveforms including oesophageal pressure

Waveforms showing Flow hunger

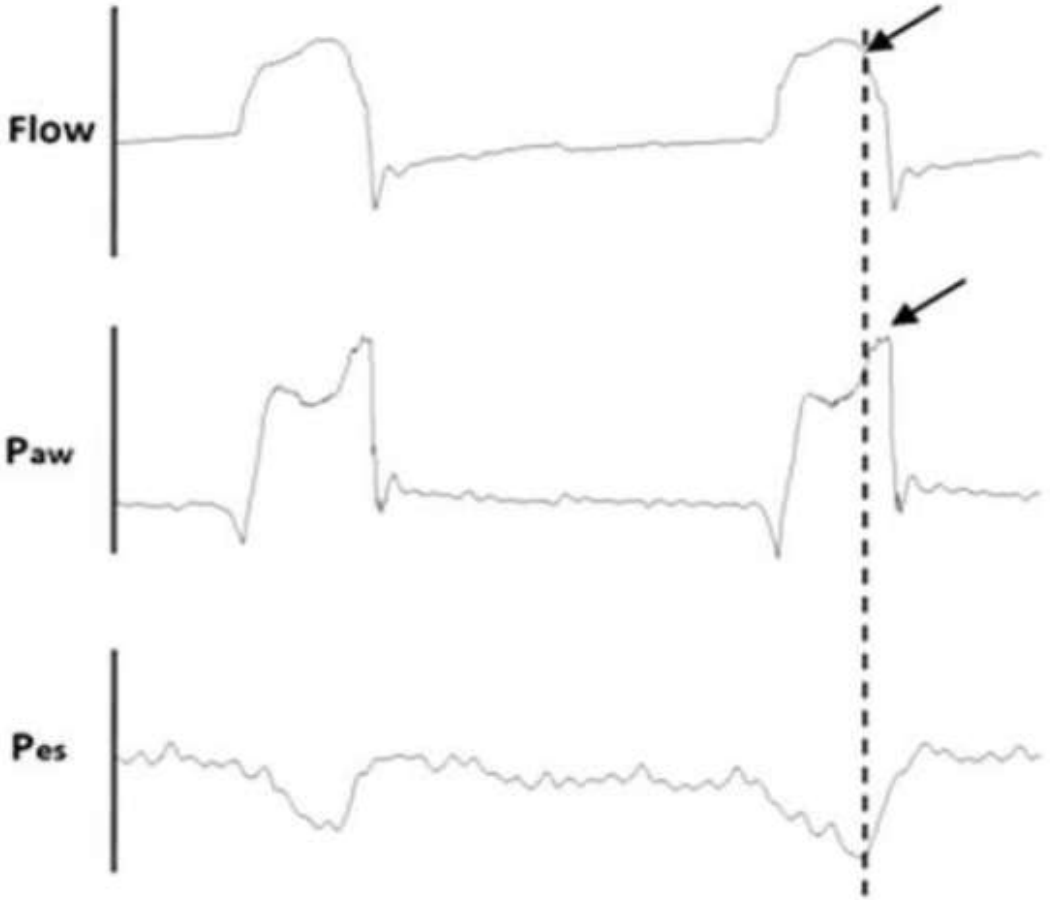


Waveforms showing Premature cycling



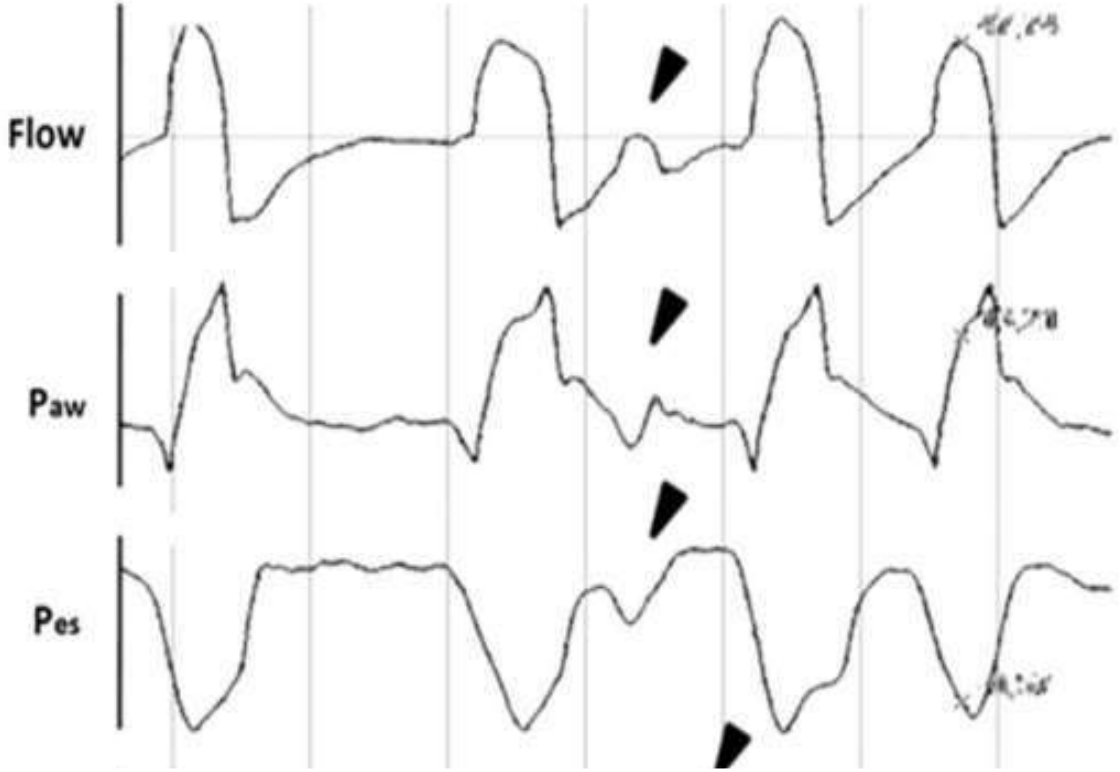
Intensive Care Med2016 Sep;42(9):1360-73

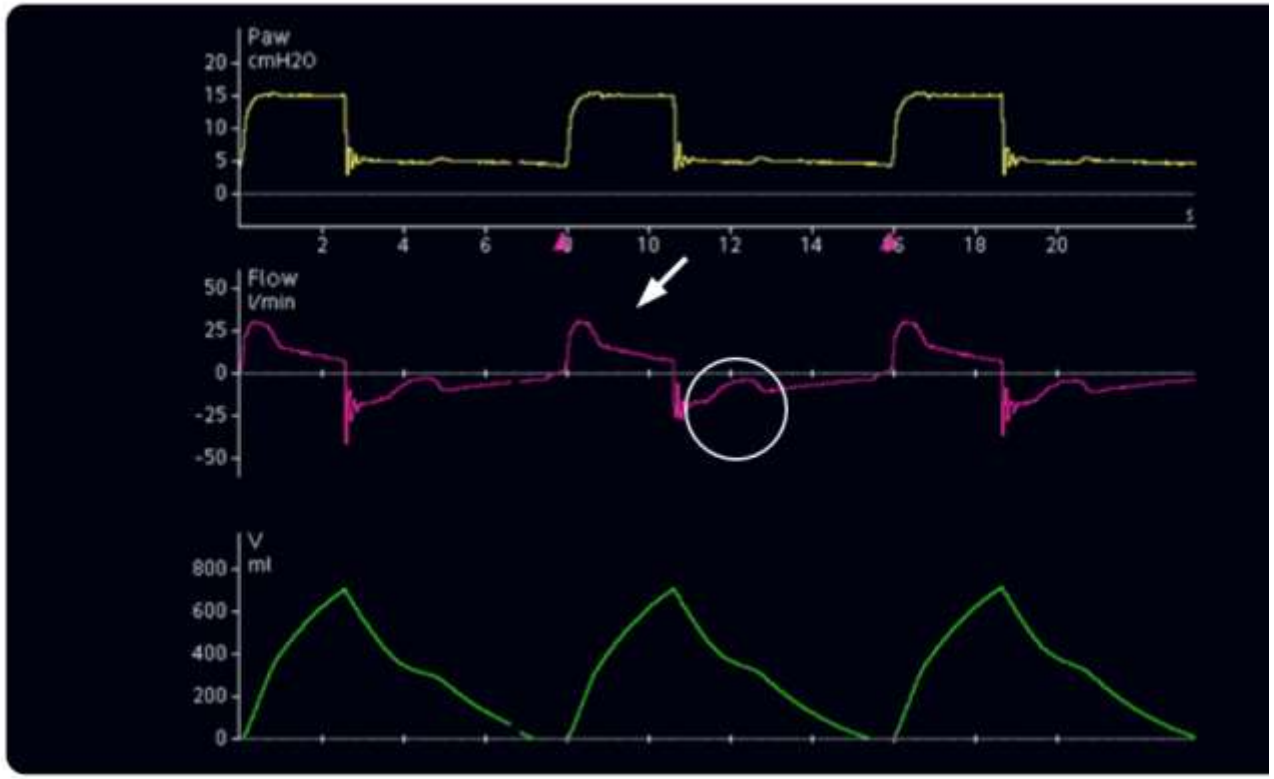
Waveforms showing Delayed cycling



Intensive Care Med2016 Sep;42(9):1360-73

Waveform characteristics indicating an ineffective effort

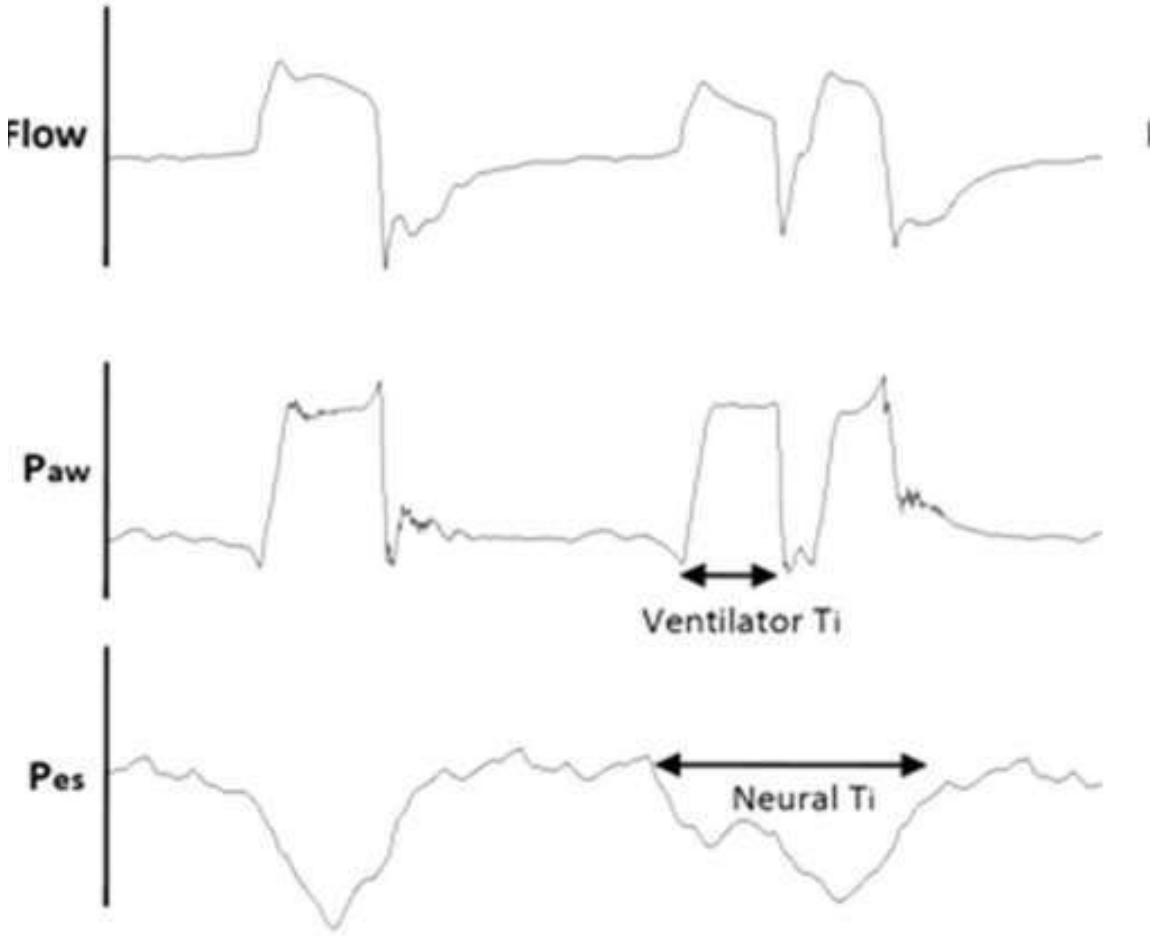




Intensive Care Med2016 Sep;42(9):1360-73

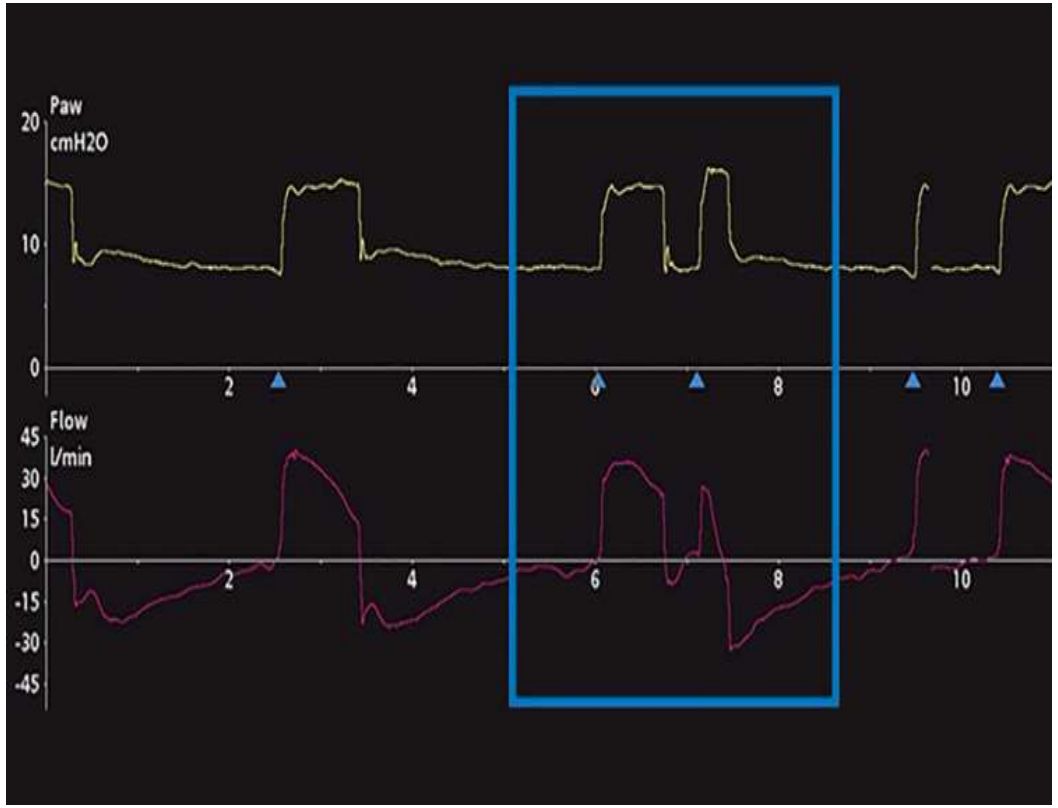
How to differentiate Double triggering by the patient or the ventilator?

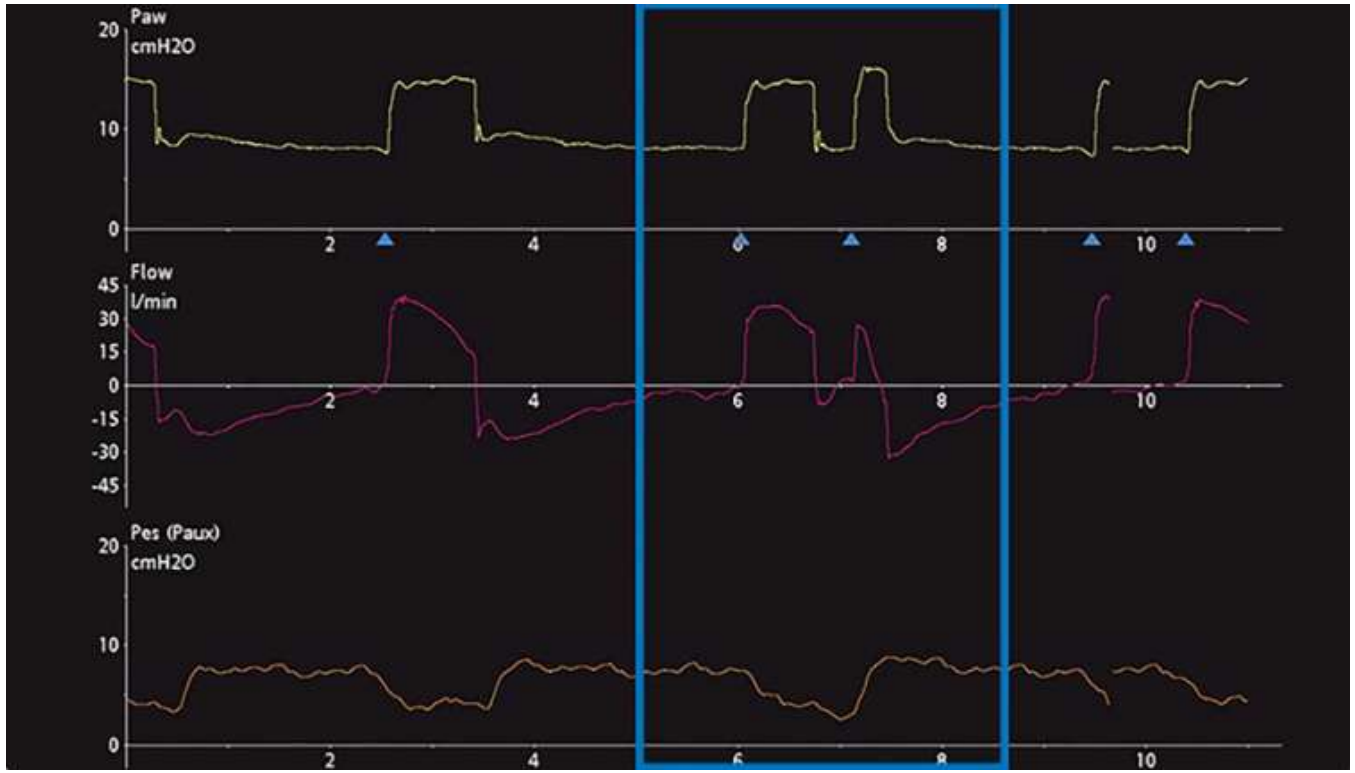
Double triggering (with breath stacking)



Intensive Care Med2016 Sep;42(9):1360-73

Pressure, flow, and volume waveforms revealing the DT

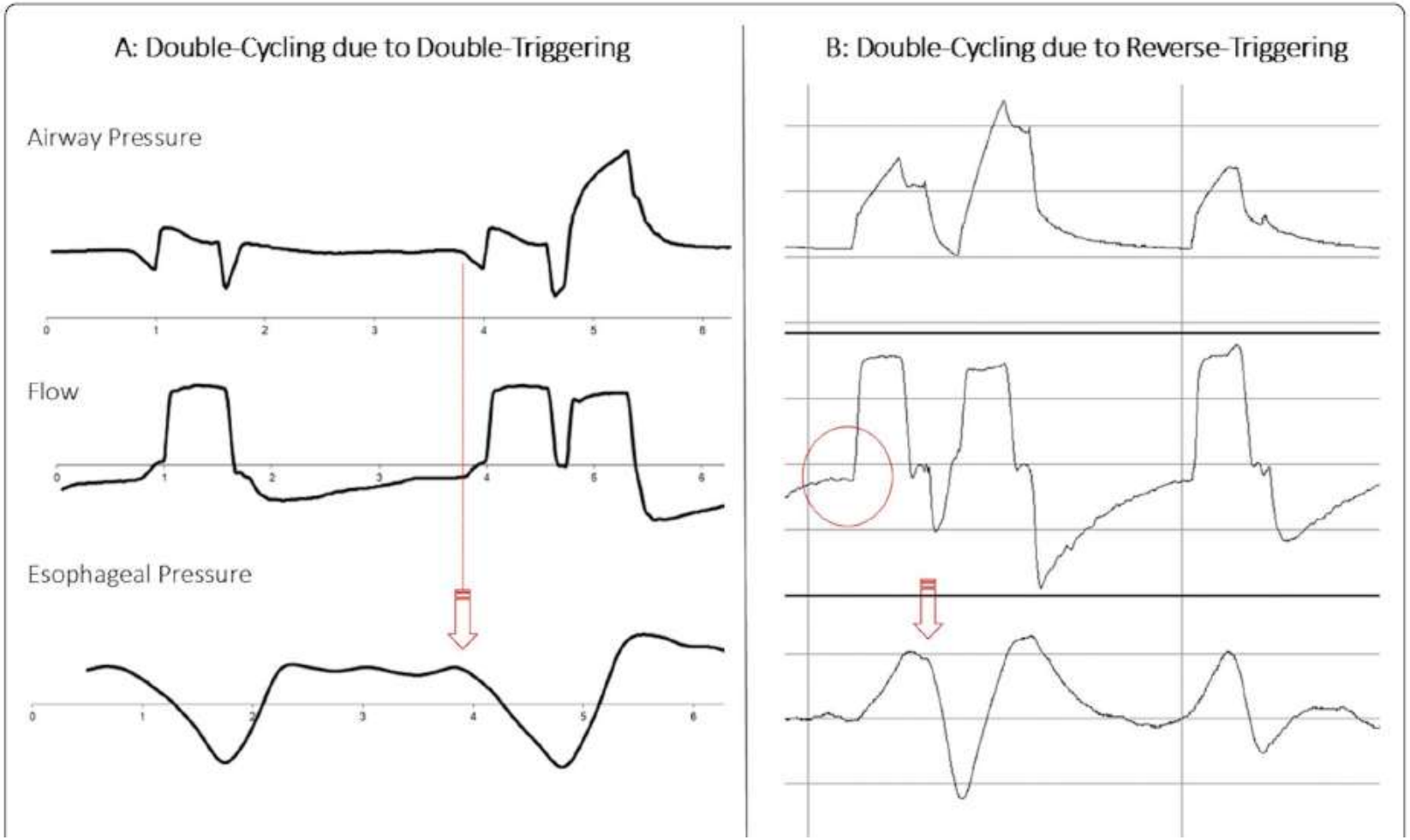




The onset of a rapid decline of esophageal pressure to the nadir, was significantly longer in the first DT-P

EDITORIAL

When the ventilator and the patient disagree: a Delphi roadmap to clarity



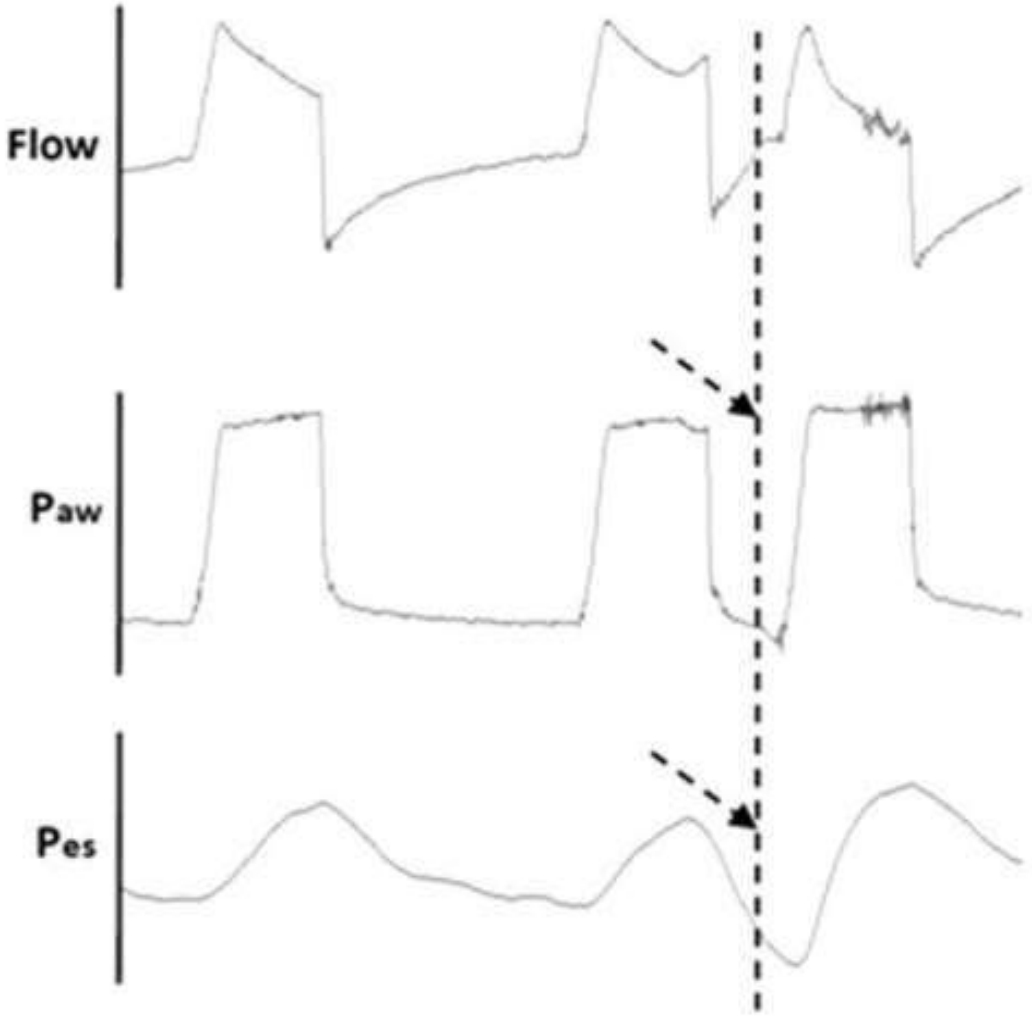
Signals of airway pressure (at the top), flow (in the middle), and oesophagal pressure (at the bottom), illustrating double-cycling (two ventilator insufflations with inexistent or incomplete expirations between them) due to double-triggering on the left or due to reverse-triggering on the right. Regardless of the mechanisms involved, double-cycling may have harmful effects due to the delivery of large tidal volumes and increased airway pressure

Intensive Care Med (2026) 52:1315–1318

Waveforms

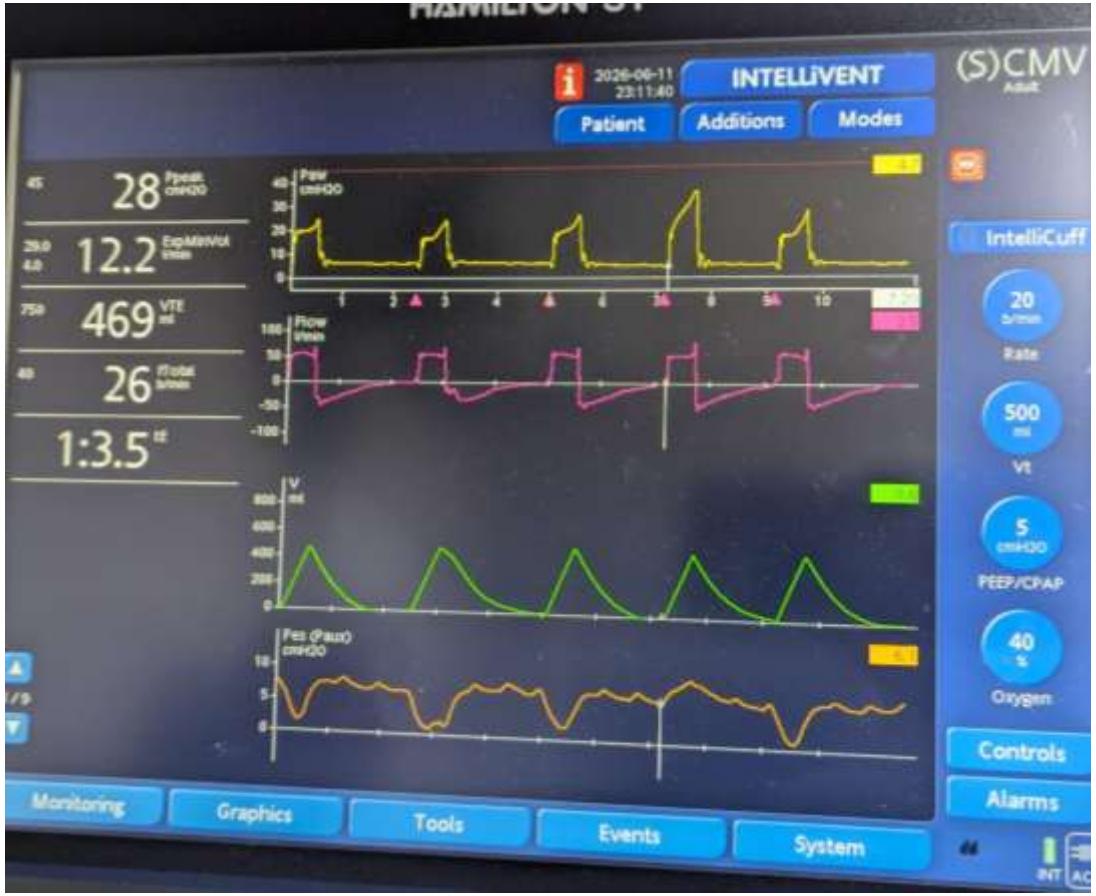
Reverse triggering

Reverse triggering (with breath stacking)



Intensive Care Med2016 Sep;42(9):1360-73

Find out dyssynchrony?



Find out dyssynchrony?

CLINICAL OUTCOMES OF PVA

Asynchronies during mechanical ventilation are associated with mortality

- Prospective observational study aimed to assess the prevalence of asynchronies, the time course during MV and association with outcomes
- Continuous waveform analysis using: Better Care™ software and parameters recorded were Airflow, Airway pressure and Tidal volume
- Population-N = 50 MV patients with acute respiratory failure, 7027 hours of MV analyzed and 8,731,981 breaths analyzed
- Primary Outcome: Association between Asynchrony Index (AI) and mortality
- Secondary Outcomes: Duration of MV, Reintubation, Tracheostomy, ICU mortality, Hospital mortality

Blanch L et al. Intensive Care Med 2015;41:633–641]

Table 1 Prevalence of different asynchronies in the different modes studied

Modes	PCV	PSV	VCV
Hours	692	2,141	2,886
Aborted inspirations	0.08 [0.000; 0.31]	–	0.06 [0.00; 0.29]
Short cycling	–	0.27 [0.06; 0.70]	–
Prolonged cycling	–	0.07 [0.00; 0.24]	–
Autotriggering	–	1.01 [0.13; 4.60]	–
Double-triggering[b,c]	0.11 [0.00; 0.44]	0.12 [0.00; 0.32]	0.06 [0.00; 0.29]
Ineffective inspiratory efforts during expiration[a,c]	0.98 [0.23; 3.32]	1.18 [0.49; 2.96]	0.91 [0.15; 3.36]
Asynchrony index[a,c]	1.69 [0.54; 4.37]	2.15 [0.90; 4.74]	1.49 [0.32; 4.68]

Table 2 Relationship between AI and duration of MV, reintubation, tracheostomy, and ICU and hospital mortality by comparing patients AI $\leq$ 10 vs AI $>$ 10 %

	AI $\leq$ 10 % ($n = 44$)	AI $>$ 10 % ($n = 6$)	p value
Length of MV (days)	6 [5.0; 15.0]	16 [9.7; 20.0]	0.061
Reintubation	9 (20 %)	0 (0 %)	0.57
Tracheostomy	14 (32 %)	2 (33 %)	0.999
ICU mortality	6 (14 %)	4 (67 %)	0.011*
Hospital mortality	10 (23 %)	4 (67 %)	0.044*

Data are expressed as numbers and percentages or as medians and interquartile ranges
MV mechanical ventilation, *ICU* intensive care unit, *AI* asynchrony index
* Significant at $p < 0.05$

Blanch L et al. Intensive Care Med 2015;41:633–641]

Patient–ventilator asynchrony, impact on clinical outcomes and effectiveness of interventions: a systematic review and meta-analysis

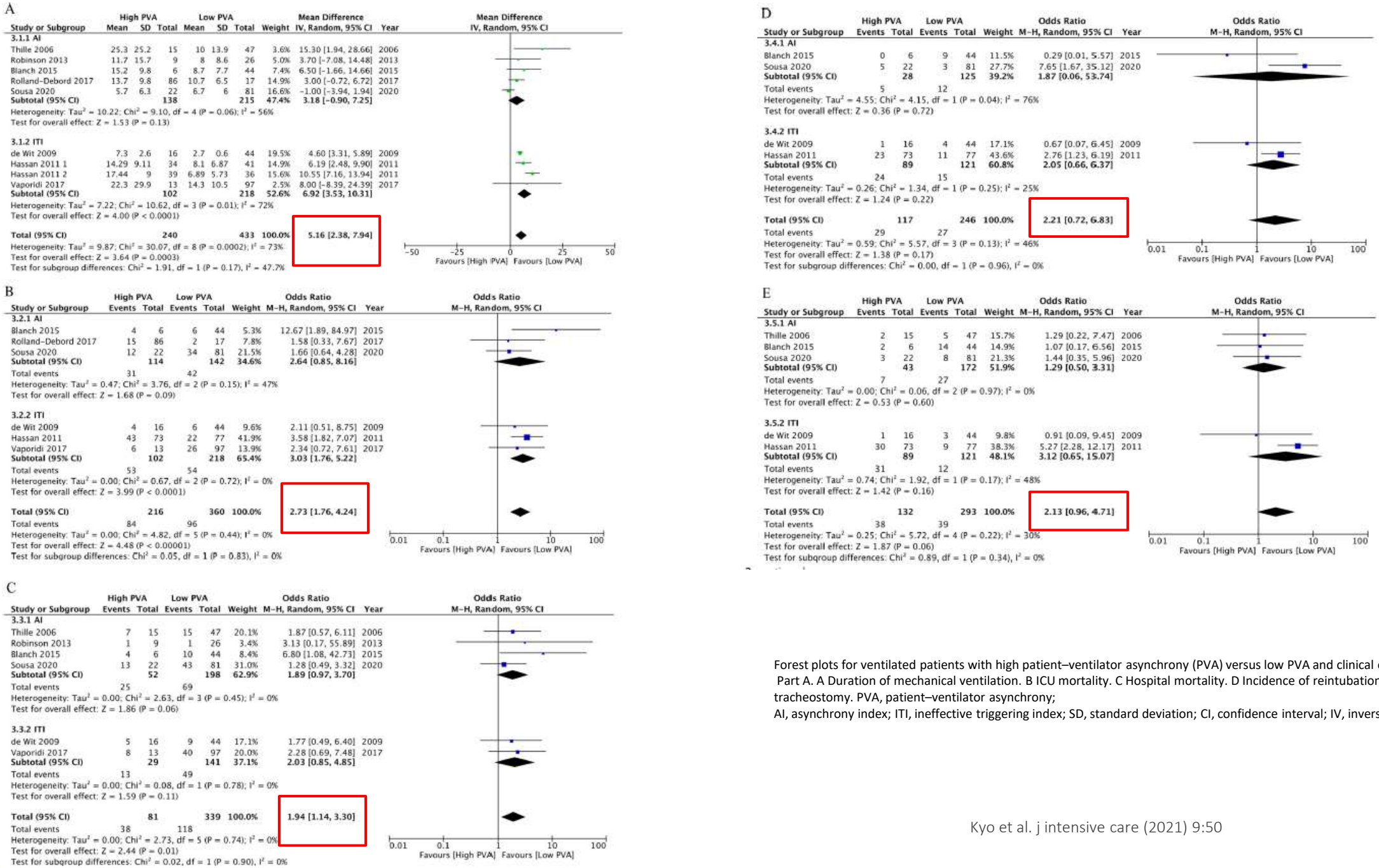
Michihito Kyo[1*], Tatsutoshi Shimatani[1], Koji Hosokawa[2], Shunsuke Taito[3,5], Yuki Kataoka[4,5,6,7], Shinichiro Ohshimo[1] and Nobuaki Shime[1]

- Part A: Impact of PVA on outcomes and Part B: Effect of interventions on PVA
- Sample Size Part A: 8 studies (673 patients) and Part B: 8 Studies
- Population: Adult ICU patients on invasive mechanical ventilation
- Inclusion: Adults on MV AI ≥10 or ITI ≥10 and exclusion: Part A: Post-surgical only, BPF/air leak, age <18 and Part B: Closed-loop ventilation (NAVA, PAV)
- Outcomes:

1) Part A: MV duration, ICU mortality, hospital mortality

2) Part B: PVA incidence, MV duration, secondary Reintubation, tracheostomy, ICU & hospital mortality

Kyo et al. j intensive care (2021) 9:50



Forest plots for ventilated patients with high patient–ventilator asynchrony (PVA) versus low PVA and clinical outcomes in Part A. A Duration of mechanical ventilation. B ICU mortality. C Hospital mortality. D Incidence of reintubation. E, Incidence of tracheostomy. PVA, patient–ventilator asynchrony;
AI, asynchrony index; ITI, ineffective triggering index; SD, standard deviation; CI, confidence interval; IV, inverse variance;

PART B: Interventions to Reduce Patient–Ventilator Asynchrony (PVA)

Study / Intervention	Study population	Inclusion criteria	What was changed	Result
Luo 2015			SIMV+PS vs ACV	No significant difference in PVA, MV duration, hospital mortality
Bassuoni 2012			No sedation vs daily interruption	No sedation → lower AI
Vaschetto 2014			Awake vs light vs deep sedation	ITI: 5.9% (awake), 7.6% (light), 21.8% (deep) (P<0.0001)
Chanques 2013			Ventilator change vs sedation	AI ↓ −99% (−92 to −100) vs −41% (−66 to 7) (P<0.001)

Kyo et al. j intensive care (2021) 9:50

Study / Intervention	Study population	Inclusion criteria	Intervention/ compariosn	Outcome	Result
Thille 2008	12 Prospective observation al study	Intubated patients with greater than 10% ineffective breaths while receiving PSV	Pressure support level, inspiratory time	Aysnchrony index	AI ↓ from 45% (36–52) to 0% (0–7) (P<0.01) Ti adjustment → 45% to 7% (3–15) (P<0.01)
Doorduin 2015	12 Randomized cross over trial	ARDS patients received MV	Mode: 1) PCV 2) PSV 3) NAVA Observation: 30min	Dyssnchrony index	Transpulmonary pressure reduced grom 11.2 cmh20 (PV) to 9.4cmh20 Tidal volume remained protective at 6ml/kg in allmodes AI: 29% (PCV), 12%(PSV) to 6% (NAVA) P value > 0.50
Luo 2015	40 RCT	Patients with ARDS who received MV	SIMV+PS ACV Observation: From intubation to SBT	1) PVA 2) Duration of mechanical ventilation 3) Hospital mortality	1) Duration of MV : 11.6 vs 12.1 days P value= 0.84 2) Hospital mortality : 30% (SIMV+PS) and 40% (ACV); P value-0.507 3) PVA : 1.1±1.3 and 1.3 ±2.3 ,no significant difference P=0.339

Pathophysiology:
Lung & Diaphragm Injury

P-SILI · Pendelluft · VILI · VIDD

P-SILI: Definition

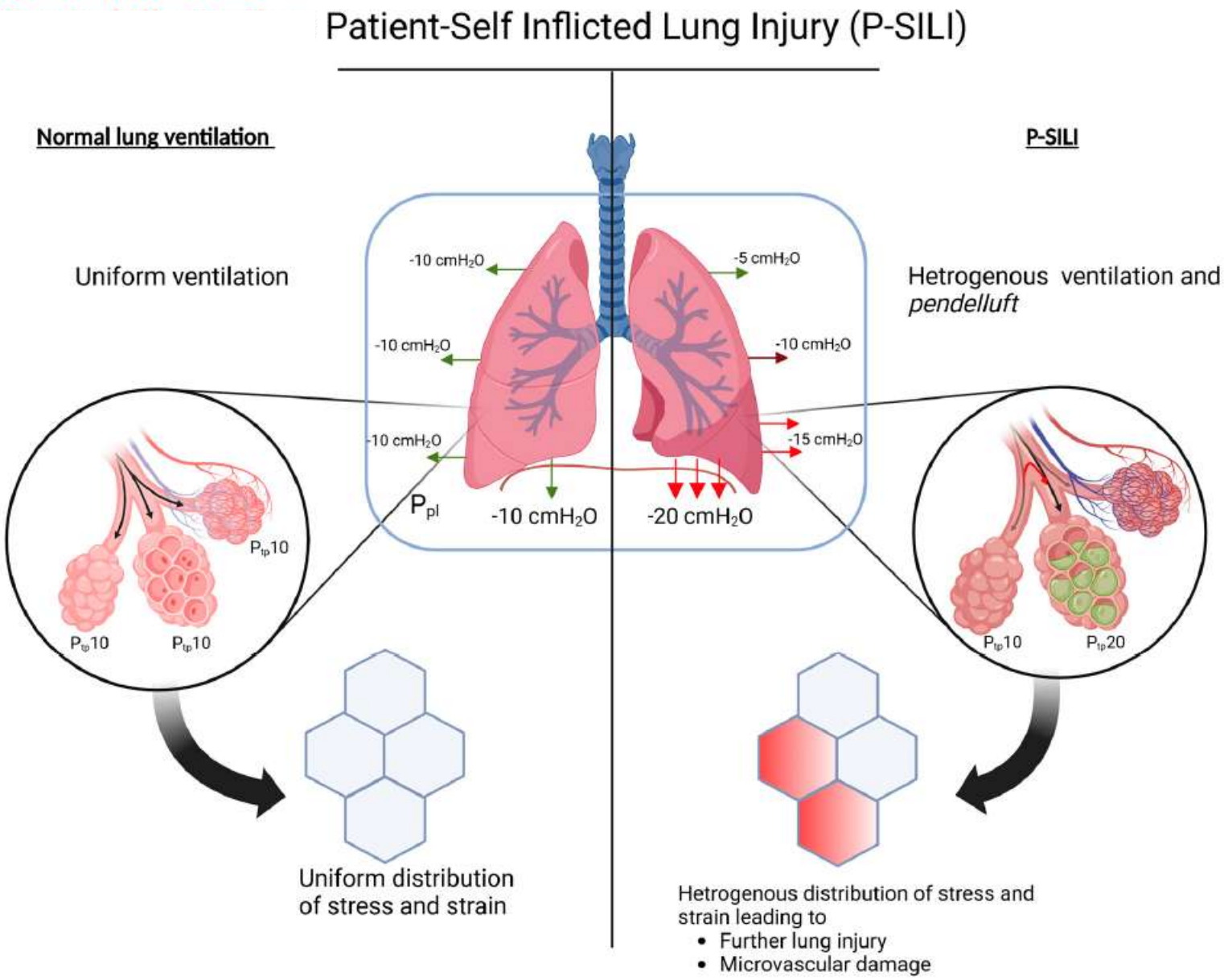
- P-SILI: Lung injury arising from the patient's own excessive spontaneous breathing effort, as distinct from ventilator-delivered injury (VILI)

Clinical relevance:

- P-SILI explains in part why some ARDS patients fail NIV or high-flow oxygen — uncontrolled respiratory drive generates injurious effort
- Debate continues regarding the exact threshold of effort that is injurious vs. beneficial

Deshwal et al., J Clin Med 2025

The pathophysiology



Deshwal et al., J Clin Med 2025

Pathophysiology of P-SILI

Four major injurious mechanisms :

1. **Pendelluft**: Regional redistribution of air from non-dependent to dependent lung during early inspiration → regional overdistension of dependent zones without change in global VT

2. **Increased transpulmonary pressure (PL):** Strong inspiratory effort generates highly negative pleural pressure → local stress amplification at atelectatic-aerated interfaces → local tidal recruitment/decruitment

3. **Increased transvascular pressure:** Negative pleural pressure → increased pulmonary vascular transmural pressure → fluid flux into alveoli (stress failure of pulmonary microvasculature)

4. **Eccentric contraction injury:** High inspiratory effort against lung inhomogeneity → sarcomere disruption in diaphragm

- These mechanisms can operate even when global VT appears 'safe' (e.g., 6 mL/kg PBW) — the distribution of stress matters

Deshwal et al., J Clin Med 2025; Lin et al., Sci Rep 2023

P-SILI: Risk Factors

- Severe acute respiratory failure (ARDS, pneumonia)
- High respiratory drive (hypoxaemia, hypercapnia, acidosis, fever, pain, agitation)
- Poor lung compliance (low Crs → small VT per effort but high effort-generated PL)
- Awake, poorly sedated patients with vigorous effort

Deshwal et al., J Clin Med 2025; Damiani & Goligher, Crit Care Med 2023

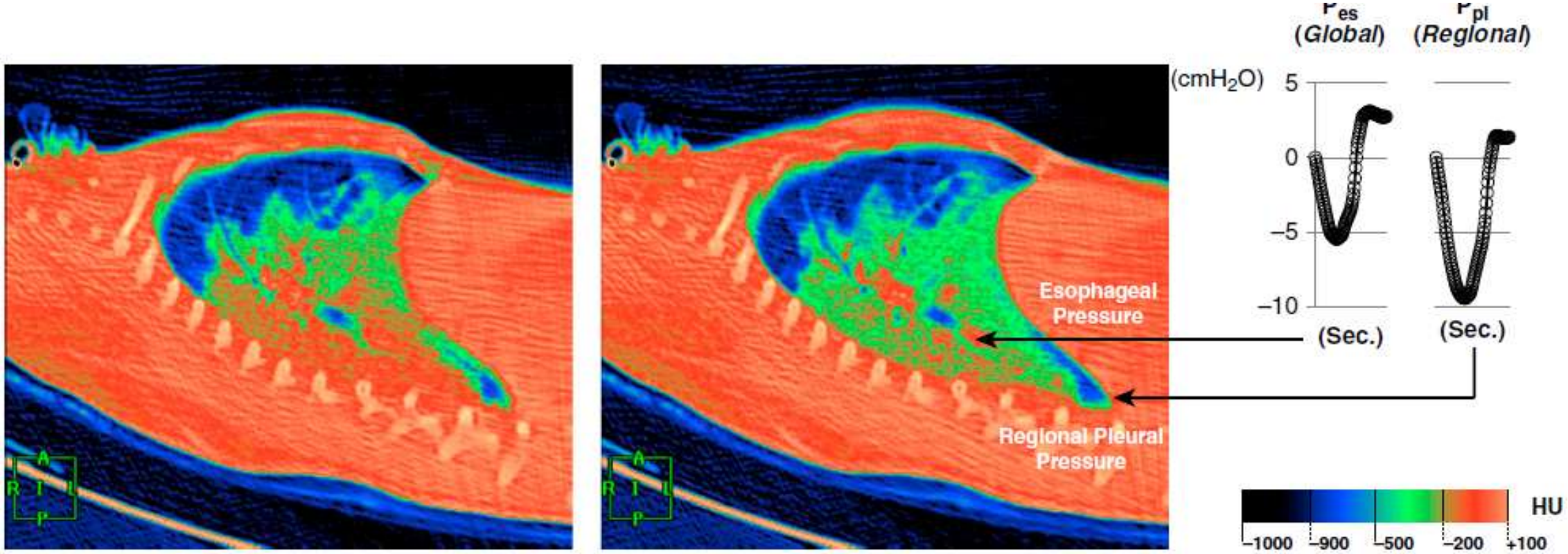
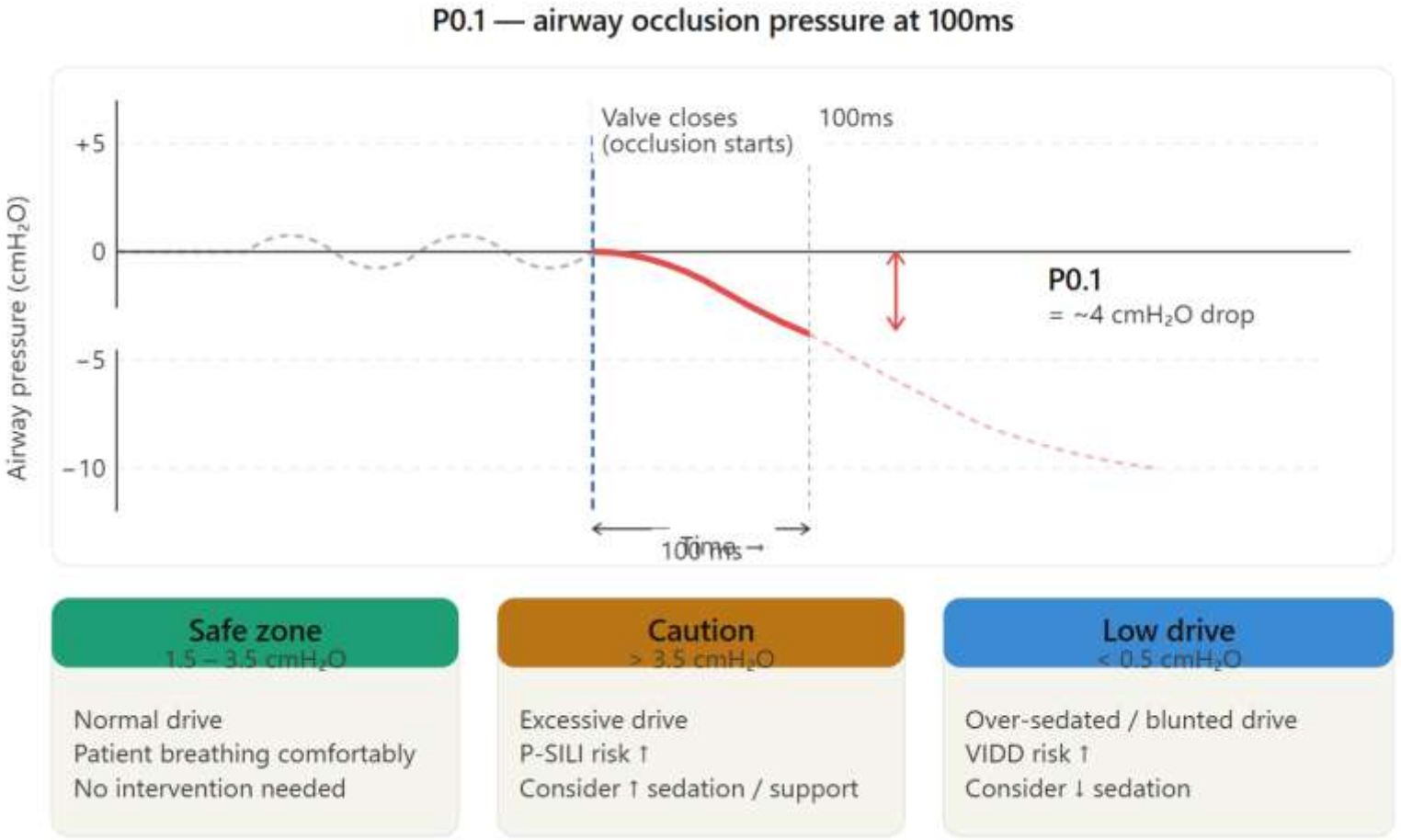


Figure 2. Spontaneous effort and distribution of regional ventilation and pleural pressure. Dynamic computed tomographic scan in end-expiration (*left*) demonstrates that the aerated lung (*blue*) is nondependent, while the dependent lung is densely atelectatic (*red*). At end-inspiration during a spontaneous breath (*middle*), there is little change in the nondependent aerated lung (*blue*); the dependent lung, previously densely atelectatic (*red*), is now partially aerated (*green/red*) (i.e., tidal recruitment). The inspiratory pleural pressure traces (*right*), measured at the *arrow tips*, show the negative deflections ("swings") in regional Ppl and global Pes during inspiration. However, the "swing" in regional Ppl is greater (by twofold) than the "swing" in Pes, indicating that diaphragm contraction results in greater distending pressure applied to the regional lung near the diaphragm, compared with the pressure transmitted to the remainder of the lung (i.e., Pes). A = anterior; HU = Hounsfield units; I = inferior; L = left; P = posterior; Pes = esophageal pressure; Ppl = pleural pressure; R = right.

How to detect P- SILI

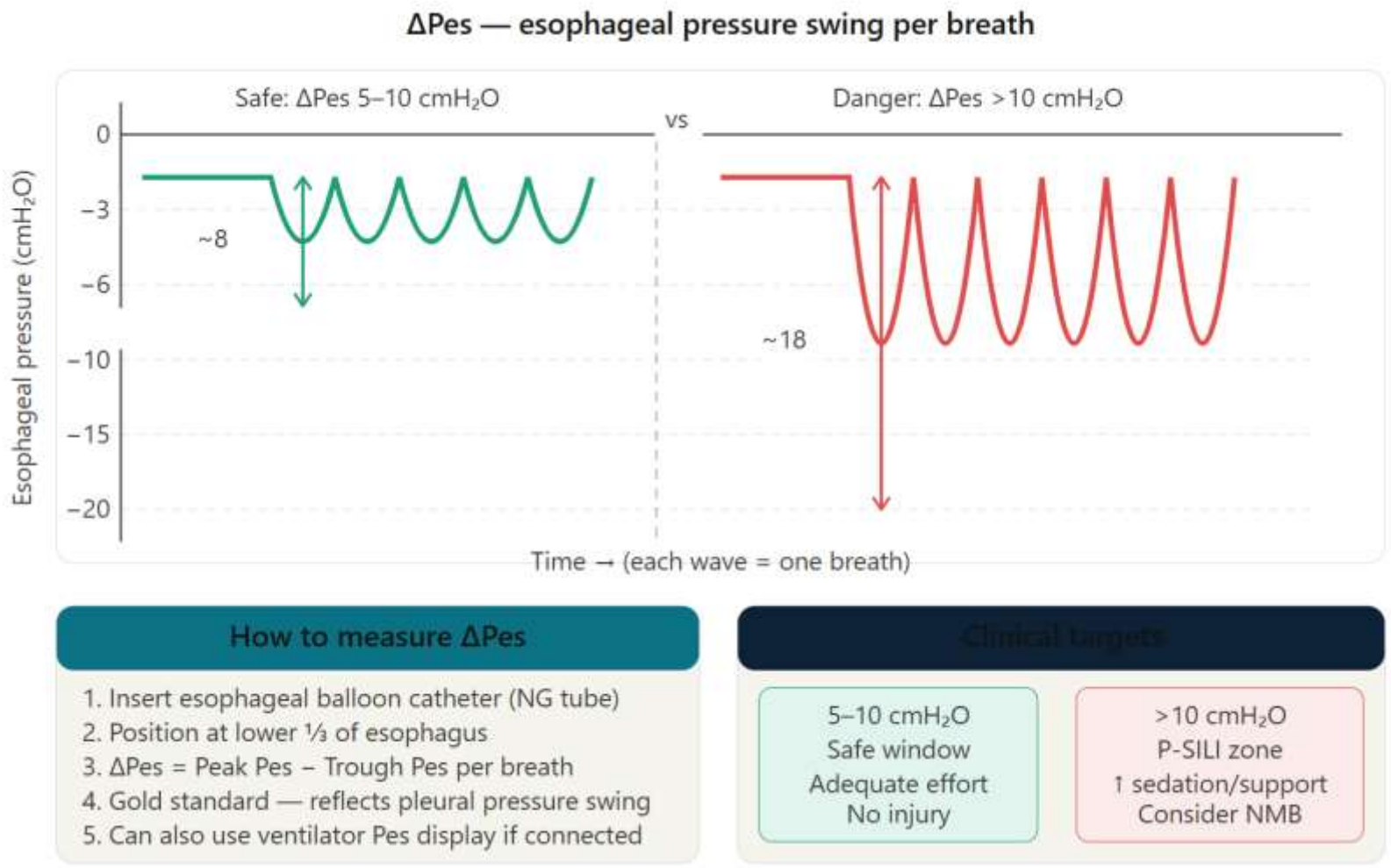


Targets

- P0.1 <3.5 cmH2O reassess before spontaneous breathing progression

Deshwal et al., J Clin Med 2025; Damiani & Goligher, Crit Care Med 2023

How to detect P- SILI



Targets

- ΔPes <10 cmH2O

Deshwal et al., J Clin Med 2025; Damiani & Goligher, Crit Care Med 2023

Diaphragm Protective ventilation

- OVER-ASSISTANCE → VIDD:
  - Controlled ventilation: Diaphragm atrophy begins within 18–48 hours of complete inactivity
  - VIDD: Diaphragm thickness decreases 3–11%/day; reduced contractile force; prolonged weaning

- UNDER-ASSISTANCE → Fatigue & Myotrauma:
  - Excessive patient effort: Contractile fatigue, P-SILI amplification
  - Eccentric loading (reverse triggering): Diaphragm myotrauma despite 'passive' mode

- OPTIMAL ZONE: Moderate, regulated effort — ΔPes 5–10 cmH2O, DTF 20–30%

- Guide titration: P0.1 + DTF (routine) + ΔPes (when available) to maintain optimal assist level

Damiani LF & Goligher EC. Crit Care Med 2023

The Dual Hazard: P-SILI vs VIDD

P-SILI

OVER-EFFORT

- Strong drive
- High Pes swings
- Under-assistance
- Pendelluft
- ↑ Transpulmonary pressure
- Regional overdistension
- Capillary stress failure
- Worsens ARDS

THE NARROW SAFE WINDOW

Adequate drive
Adequate assistance
Synchrony

VIDD

UNDER-EFFORT

- Over-assistance
- Diaphragm inactivity
- Rapid atrophy (18–69 hrs)
- ↑ MV duration
- Difficult weaning
- Iatrogenic from deep sedation & NMB
- ICU-acquired weakness

Damiani LF, Goligher EC. Crit Care Med 2023;51:1618–1621 · Deshwal H et al. J Clin Med 2025;14:1632

Neurally Adjusted Ventilatory Assist (NAVA)

Principles, Physiology, and Technical Basis

NAVA

NAVA developed by Sinderby et al. (Maquet/Getinge) as a fundamentally different mode of assisted ventilation

Core concept:

- Use the electrical activity of the diaphragm (Edi) — the direct neural output of the respiratory centre — as the control signal for ventilator support delivery
- Edi signal acquired via nasogastric catheter with esophageal electrode array (Edi catheter)
- Support proportional and synchronous with neural effort

Tian et al., J Thorac Dis 2025

Advantages over flow/pressure-triggered modes:

- Trigger based on neural signal — eliminates trigger delay from chest mechanics
- Cycle-off based on neural signal — eliminates delayed and premature cycling
- Support proportional to effort — prevents over-assistance and under-assistance
- Auto-triggering eliminated: Edi is specific to diaphragm; cardiac oscillations and leaks do not generate Edi

Tian et al., J Thorac Dis 2025

Neurally adjusted ventilatory assist versus pressure support ventilation in patient-ventilator interaction and clinical outcomes: a meta-analysis of clinical trials

Study or Subgroup	NAVA Mean	NAVA SD	NAVA Total	PSV Mean	PSV SD	PSV Total	Weight	Mean Difference IV, Random, 95% CI
3.1.1 Randomized								
Demoule 2016	16.23	7.16	53	29.2	22.36	50	15.4%	−12.97 [−19.46, −6.48]
Ferreira 2017	11.8	12.37	20	21.63	22.34	20	13.0%	−9.83 [−21.02, 1.36]
Kuo 2016	0	0	14	11.9	11.2	19		Not estimable
Longhini 2017	0	0	14	10	19.28	14		Not estimable
Longhini 2019	0	0	10	23	43.26	10		Not estimable
Mauri 2012	20	13	10	74	43	10	5.9%	−54.00 [−81.84, −26.16]
Mussi 2016	5.84	3.8	13	12.53	12.87	12	14.9%	−6.69 [−14.26, 0.88]
Patroniti 2011	0	0	14	0.23	0.59	14		Not estimable
Piquilloud 2014	5.97	6.64	13	23.63	36.63	13	8.5%	−17.66 [−37.90, 2.58]
Schmidt 2012	9.87	8.89	17	37.2	25.54	17	12.1%	−27.33 [−40.19, −14.47]
Subtotal (95% CI)			**178**			**179**	**70.0%**	**−16.79 [−25.35, −8.24]**
Heterogeneity: $Tau^2 = 69.19$; $Chi^2 = 16.27$, df = 5 (P = 0.006); $I^2 = 69\%$								
Test for overall effect: Z = 3.85 (P = 0.0001)								
3.1.2 Non-randomized								
Schimidt 2015	1.6	1.88	16	0.64	1.01	16	16.9%	0.96 [−0.09, 2.01]
Yonis 2015	7.32	15.38	30	12.61	26.83	30	13.1%	−5.29 [−16.36, 5.78]
Subtotal (95% CI)			**46**			**46**	**30.0%**	**0.36 [−3.24, 3.96]**
Heterogeneity: $Tau^2 = 3.45$; $Chi^2 = 1.21$, df = 1 (P = 0.27); $I^2 = 18\%$								
Test for overall effect: Z = 0.20 (P = 0.84)								
Total (95% CI)			**224**			**225**	**100.0%**	**−12.82 [−21.20, −4.44]**
Heterogeneity: $Tau^2 = 107.58$; $Chi^2 = 60.15$, df = 7 (P < 0.00001); $I^2 = 88\%$								
Test for overall effect: Z = 3.00 (P = 0.003)								
Test for subgroup differences: $Chi^2 = 13.12$, df = 1 (P = 0.0003), $I^2 = 92.4\%$								

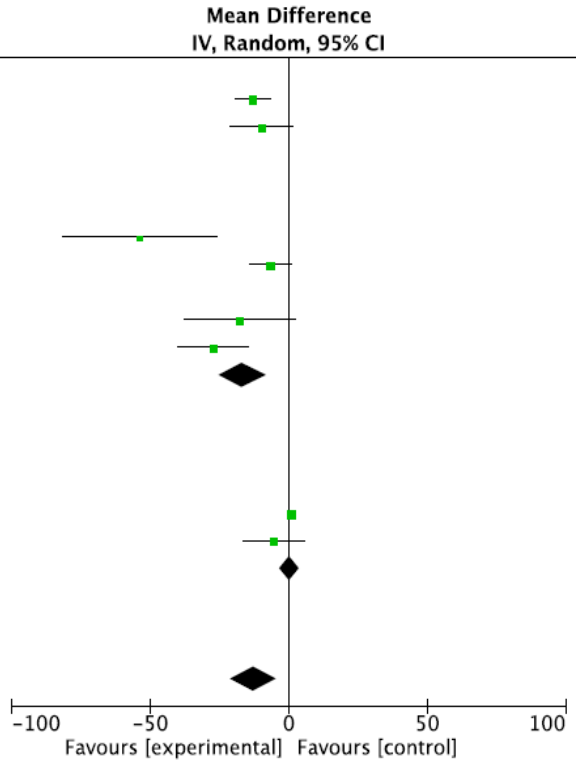


Figure 5 Subgroup analysis of divided by randomization of asynchrony index.

Systematic review & meta-analysis of 12 prospective clinical trials

331 adult ventilated patients, both invasive and non-invasive ventilation, ICU patients with respiratory failure

Chen.et.al,Ann Transl Med 2019;7(16):382

Study or Subgroup	NAVA Mean	NAVA SD	NAVA Total	PSV Mean	PSV SD	PSV Total	Weight	Mean Difference IV, Fixed, 95% CI
Mauri 2012	0	1	10	0	1	10	7.2%	0.00 [−0.88, 0.88]
Mussi 2016	0.23	0.4	13	0.17	0.2	12	92.6%	0.06 [−0.19, 0.31]
Piquilloud 2014	0	0	13	0.77	1.5	13		Not estimable
Schimidt 2015	0	0	16	0.1	0.21	16		Not estimable
Schmidt 2012	0	0	17	0	0	17		Not estimable
Yonis 2015	2.62	5.95	30	5.73	13.2	30	0.2%	−3.11 [−8.29, 2.07]
Total (95% CI)			**99**			**98**	**100.0%**	**0.05 [−0.19, 0.28]**

Heterogeneity: $Chi^2 = 1.45$, df = 2 (P = 0.48); $I^2 = 0\%$
Test for overall effect: Z = 0.41 (P = 0.68)

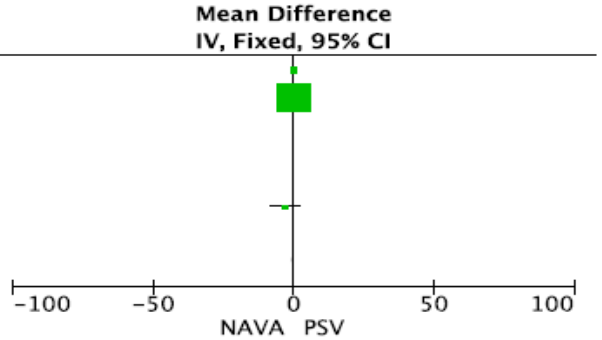


Figure 7 Ineffective efforts of patients.

Study or Subgroup	NAVA Mean	NAVA SD	NAVA Total	PSV Mean	PSV SD	PSV Total	Weight	Mean Difference IV, Fixed, 95% CI
Mauri 2012	1	1	10	3	5	10	0.5%	−2.00 [−5.16, 1.16]
Mussi 2016	0	0	13	0	0	12		Not estimable
Piquilloud 2014	1.17	1.74	13	1.13	1.41	13	3.5%	0.04 [−1.18, 1.26]
Schimidt 2015	0	0	16	0.01	0.02	16		Not estimable
Schmidt 2012	0.07	0.16	17	0.3	0.49	17	87.3%	−0.23 [−0.48, 0.02]
Yonis 2015	0.17	0.37	30	0.98	2.15	30	8.6%	−0.81 [−1.59, −0.03]
Total (95% CI)			**99**			**98**	**100.0%**	**−0.28 [−0.51, −0.05]**

Heterogeneity: $Chi^2 = 3.33$, df = 3 (P = 0.34); $I^2 = 10\%$
Test for overall effect: Z = 2.39 (P = 0.02)

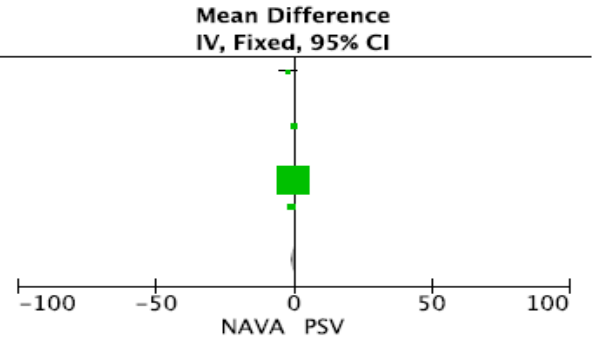


Figure 8 Auto triggering of patients.

Study or Subgroup	NAVA Mean	NAVA SD	NAVA Total	PSV Mean	PSV SD	PSV Total	Weight	Mean Difference IV, Fixed, 95% CI
Mauri 2012	1	1	10	3	6	10	0.1%	−2.00 [−5.77, 1.77]
Mussi 2016	0.17	0.23	13	0.09	0.08	12	81.0%	0.08 [−0.05, 0.21]
Piquilloud 2014	0.4	0.5	13	0.6	1.25	13	2.7%	−0.20 [−0.93, 0.53]
Schimidt 2015	0.45	0.67	16	0.06	0.15	16	12.7%	0.39 [0.05, 0.73]
Schmidt 2012	0.97	1.21	17	1.2	0.73	17	3.2%	−0.23 [−0.90, 0.44]
Yonis 2015	1.65	3.71	30	1.61	3.67	30	0.4%	0.04 [−1.83, 1.91]
Total (95% CI)			**99**			**98**	**100.0%**	**0.10 [−0.02, 0.22]**

Heterogeneity: $Chi^2 = 5.71$, df = 5 (P = 0.34); $I^2 = 12\%$
Test for overall effect: Z = 1.63 (P = 0.10)

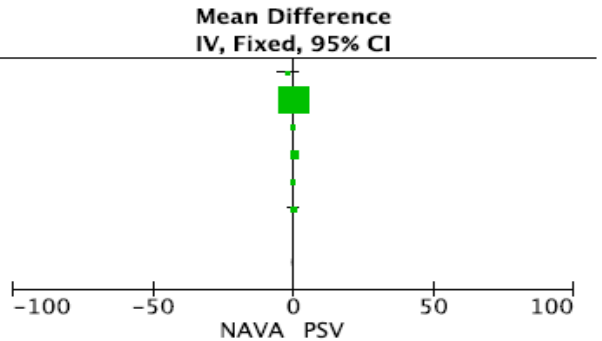


Figure 9 Double triggering of patients.

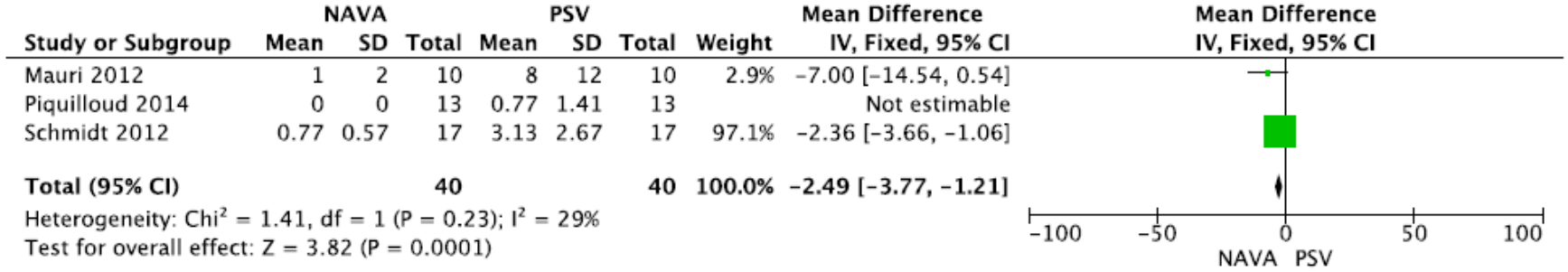


Figure 10 Premature triggering of patients.

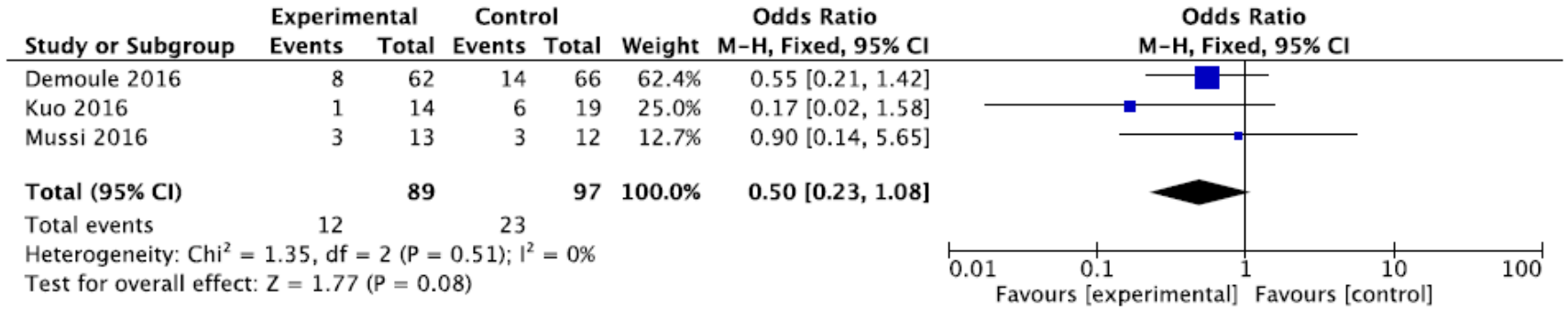


Figure 11 ICU mortality of patients.

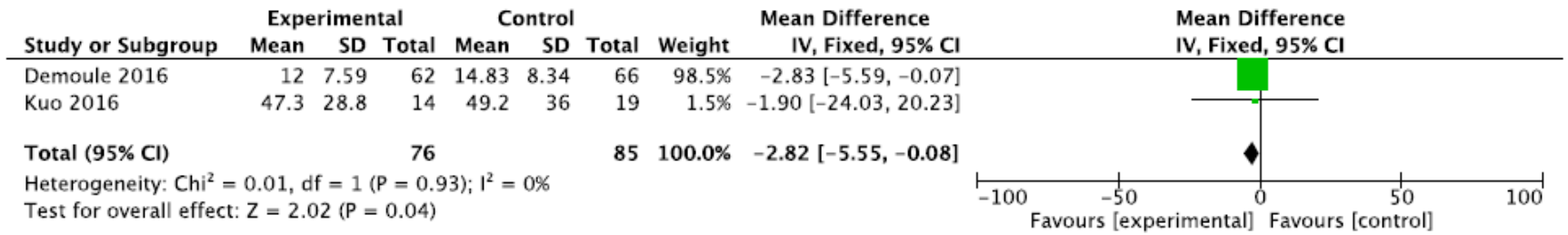


Figure 12 Duration of ventilation of patients.

Closed-Loop Ventilation

Closed-Loop Ventilation:

Closed-loop (CL) ventilation: Computer-controlled, automated adjustment of ventilator settings using real-time patient data feedback

Rationale:

- Conventional MV requires frequent clinician/RRT adjustments to maintain lung-protective targets
- Studies show significant time outside protective ranges even in well-resourced ICUs (up to 30–50% of ventilator time)
- Human response to alarms and parameter drift is delayed and intermittent
- Automated systems can adjust continuously (every breath or every few minutes)

Botta et al., Expert Rev Resp Med 2021

JAMA | Original Investigation | CARING FOR THE CRITICALLY ILL PATIENT

Effect of Automated Closed-Loop Ventilation vs Protocolized Conventional Ventilation on Ventilator-Free Days in Critically Ill Adults
A Randomized Clinical Trial

Jante S. Sinnige, MD; Laura A. Buiteman-Kruizinga, PhD; Janneke Horn, MD, PhD; Frederique Paulus, PhD; Marcus J. Schultz, MD, PhD; Ary Serpa Neto, MD, MSc, PhD; for the ACTiVE Investigators and the Protective Ventilation Network

Multicentre, randomized clinical trial (The Netherlands)

Intervention: INTELLiVENT-ASV (automated CL ventilation) vs. protocolized conventional ventilation

Population: Critically ill adults requiring invasive MV in the 7 ICUs of sample size 1201

Primary endpoint: Ventilator-free days (VFD) at day 28

Sinnige et al., JAMA 2026

Table 2. Primary and Secondary Outcomes[a]

Outcomes	Closed-loop ventilation (n = 602)	Conventional ventilation (n = 599)	Absolute difference (95% CI)[b]	Effect estimate (95% CI)[b]	P value
Primary outcome					
Ventilator-free days at day 28[c]				OR, 0.91 (0.77 to 1.06)	.23
Median (IQR)	16.7 (0.0-26.1) [n = 601]	16.3 (0.0-26.5) [n = 595]	0.4 (−4.0 to 5.8)		
Mean (SD)	13.1 (12.2) [n = 601]	13.4 (12.3) [n = 595]	−0.3 (−1.7 to 1.0)		
Secondary outcomes					
28-Day mortality, No./total (%)	224/602 (37.2)	218/597 (36.5)	0.7 (−4.8 to 6.1)	HR, 1.04 (0.86 to 1.25)	.69
Duration of ventilation in survivors, median (IQR), d	3.3 (1.0-9.4) [n = 377]	2.6 (1.0-8.7) [n = 377]	0.7 (−0.0 to 1.4)	MdD, 0.7 (−0.0 to 1.4)	.05
Time spent in each ventilatory zone, mean (SD), %[d]	n = 78	n = 64			
Optimal	48.4 (40.2)	36.7 (38.4)	12.9 (−0.2 to 25.9)	MD, 12.9 (−0.2 to 25.9)	.05
Acceptable	40.1 (36.6)	27.5 (31.6)	12.5 (1.2 to 23.9)	MD, 12.5 (1.2 to 23.9)	.03
Critical	11.6 (26.9)	35.8 (45.0)	−24.6 (−36.6 to −12.6)	MD, −24.6 (−36.6 to −12.6)	<.001
Acute respiratory distress syndrome, No./total (%)[e]	15/599 (2.5)	12/596 (2.0)	0.5 (−1.2 to 2.2)	AD, 0.5 (−1.2 to 2.2)	.57
Ventilator-associated pneumonia, No./total (%)[f]	13/599 (2.2)	21/596 (3.5)	−1.4 (−3.3 to 0.4)	AD, −1.4 (−3.3 to 0.4)	.13
Severe hypercapnia: $Paco_2$ >55 mm Hg and pH <7.35, No./total (%)	119/599 (19.9)	144/596 (24.2)	−4.1 (−8.7 to 0.5)	AD, −4.1 (−8.7 to 0.5)	.08
Severe atelectasis, No./total (%)[g]	23/599 (3.8)	36/596 (6.0)	−2.2 (−4.7 to 0.3)	AD, −2.2 (−4.7 to 0.3)	.08
Severe hypoxemia: Pao_2 <55 mm Hg, No./total (%)	96/599 (16.0)	126/596 (21.1)	−5.1 (−9.4 to −0.7)	AD, −5.1 (−9.4 to −0.7)	.02
Pneumothorax, No./total (%)[h]	6/599 (1.0)	2/596 (0.3)	0.7 (−0.3 to 1.6)	AD, 0.7 (−0.3 to 1.6)	.16
Need for rescue strategies, No./total (%)[i]	86/599 (14.4)	121/596 (20.3)	−6.0 (−10.2 to −1.7)	AD, −6.0 (−10.2 to −1.7)	.006
Recruitment maneuvers	31/599 (5.2)	30/596 (5.0)	0.1 (−2.4 to 2.5)	AD, 0.1 (−2.4 to 2.5)	.95
Prone positioning	55/599 (9.2)	83/596 (13.9)	−4.8 (−8.4 to −1.2)	AD, −4.8 (−8.4 to −1.2)	.009
Bronchoscopy for atelectasis	33/599 (5.5)	44/596 (7.4)	−1.9 (−4.7 to 0.9)	AD, −1.9 (−4.7 to 0.9)	.19
Extubation failure (reintubation within 24 h of extubation), No./total (%)	32/413 (7.7)	30/412 (7.3)	0.5 (−3.0 to 4.1)	AD, 0.5 (−3.0 to 4.1)	.77
Length of stay, median (IQR), d					
Intensive care unit	4.5 (1.8-11.9) [n = 595]	4.4 (1.8-12.4) [n = 594]	0.1 (−0.9 to 1.1)	MdD, 0.1 (−0.9 to 1.1)	.85
Hospital	12.2 (3.5-27.5) [n = 564]	12.0 (4.0-25.3) [n = 581]	0.3 (−0.8 to 1.4)	MdD, 0.3 (−0.8 to 1.4)	.60
Mortality, No./total (%)					
In intensive care unit	208/558 (37.3)	205/556 (36.9)	0.1 (−5.5 to 5.8)	AD, 0.1 (−5.5 to 5.8)	.97
In hospital	241 (40.0)	233 (38.9)	1.1 (−4.4 to 6.6)	AD, 1.1 (−4.4 to 6.6)	.70
By day 90	246/600 (41.0)	237/592 (40.0)	0.9 (−4.6 to 6.5)	HR, 1.04 (0.87 to 1.24)	.66

Closed-loop ventilation improved physiological control but not clinical outcomes

Sinnige et al., JAMA 2026

Emerging Technologies in Ventilator Monitoring

Electrical Impedance Tomography (EIT):

- Continuous, radiation-free real-time regional lung ventilation monitoring
- Enables pendelluft detection, PEEP optimisation, and regional overdistension quantification at bedside
- Still not routine; device cost and technical expertise limit uptake

Oesophagal pressure (Pes) monitoring:

- Increasingly incorporated into clinical practice for drive assessment (ΔPes) and transpulmonary pressure measurement
- Recommended by some experts for all ARDS patients receiving assisted MV

AI-augmented waveform analysis:

- Continuous, automated PVA detection; pilot systems being evaluated

Let's Get in Sync, ICM Exp 2025; Damiani & Goligher, Crit Care Med 2023

Summary: The PVD Management Spectrum

- RECOGNIZE: Waveform analysis + AI calculation — systematic, intentional, twice daily
- CLASSIFY: IE vs DT vs RT vs Flow vs Cycling — management is type-specific
- QUANTIFY: AI >10% = significant; ΔPes >15 = P-SILI territory; DTF as daily monitor
- TREAT ROOT CAUSE: Auto-PEEP → PEEP; High drive → analgosedation; Mode mismatch → change mode
- PROTECT BOTH: Dual lung AND diaphragm protection — the central principle of modern MV (Damiani 2023)
- ESCALATE APPROPRIATELY: NAVA for persistent PVD; NMB only for life-threatening refractory cases

THANK YOU