Patient-Ventilator interaktion

ST-dag om lungsvikt och ventilatorbehandling
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PATIENT-VENTILATORY ASYNCHRONIES

1. Definition and how to manage them
2. Why they occur
3. Differences among ventilatory modalities
The method has the following disadvantages:

(2) When bag ventilation is administered continuously for weeks, there is a risk of emphysema.
(3) If bag ventilation is not administered correctly, the venous return may be reduced, leading to lowered cardiac output and a state of shock.
(4) Hyperventilation with subnormal carbon dioxide values may occur.
(5) The weaning period from positive-pressure ventilation is not infrequently difficult.
(6) The assistance of well-trained personnel all round the clock is essential and costly.
Equation of motion during support ventilation

- Describes interactions between the patient and the ventilator
- Pressure required to deliver a volume of gas in the lungs is determined by elastic and resistive properties of the lung

\[ \text{Paw} = \frac{V_t}{C} + V' \times R + \text{PEEP} \]

\[ P_{\text{vent}} + P_{\text{mus}} = (\text{Volume/Compliance}) + (\text{Flow} \times \text{Resistance}) + \text{PEEP} \]

The optimal assisted mechanical ventilation must:
- Avoid ventilator induced diaphragmatic dysfunction
- Provide sufficient level of ventilatory support to reduce patient’s work of breathing
Figure 3: Patient-ventilator interaction
Transpulmonary pressure

$P_{ao} = 0$
$P_A = 0$
$P_{pl} = -10$

$V_T = 500 \text{ mL}$

$P_{ao} = 10$
$P_A = 10$
$P_{pl} = 0$

$V_T = 500 \text{ mL}$

Transpulmonary pressure

Inspiration
Expiration

Inspiration
Expiration

$P_A$

$P_L = 10$

$P_{pl}$

Spontaneous breathing
Positive pressure ventilation
For perfect synchronization, the period of mechanical inflation must match the period of neural inspiratory time and the period of mechanical inactivity must match the neural expiratory time.
Need of skilled physicians able to optimize the ventilatory settings in order to achieve optimal synchronization
Figure 1. Flow, Airway Pressure, and Inspiratory and Expiratory Muscle Activity in a Patient with Chronic Obstructive Pulmonary Disease Who Received Pressure-Support Ventilation at an Airway Pressure of 20 cm of Water.
Three variables determine the function of a positive pressure ventilator:
(a) the triggering variable
   (the signal of initiating the positive pressure)
   Delay triggering, Auto triggering, Ineffective effort;
(b) the control variable
   (the algorithm that controls the delivered pressure or flow during the mechanical inspiration). Flow starvation;
(c) the cycling off variable
   (the signal of terminating the pressure or flow delivery)
   Delayed cycling, Early cycling, Double triggering;
(a) THE TRIGGERING VARIABLE

*Delay triggering, Auto triggering, Ineffective effort.*
THE TRIGGER  the signal that initiate the positive pressure in assisted ventilation

Controlled breath

Flow Trigger

Pressure Trigger
<table>
<thead>
<tr>
<th></th>
<th>PSV</th>
<th>ACV</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PT</td>
<td>FT</td>
</tr>
<tr>
<td>Time delay, ms</td>
<td>155 ± 56</td>
<td>89 ± 23*</td>
</tr>
<tr>
<td>Airway pressure drop, cm H₂O</td>
<td>3.8 ± 1.6</td>
<td>2.5 ± 1.4†</td>
</tr>
<tr>
<td>PTPes, cm H₂O · s/br</td>
<td>9.0 ± 3.3</td>
<td>8.1 ± 3.2*</td>
</tr>
<tr>
<td>PTPp, cm H₂O · s/br</td>
<td>2.8 ± 2.1</td>
<td>2.4 ± 1.9*</td>
</tr>
<tr>
<td>PTPtr, cm H₂O · s/br</td>
<td>0.26 ± 0.06</td>
<td>0.10 ± 0.04†</td>
</tr>
<tr>
<td>PTPpost, cm H₂O · s/br</td>
<td>5.9 ± 2.9</td>
<td>5.6 ± 2.8</td>
</tr>
</tbody>
</table>

*Definition of abbreviations:* tr = trigger phase; post = post-trigger phase. Other abbreviations as in Table 4.

* p < 0.05, paired t test versus PT for the same ventilatory mode.
† p < 0.001, paired t test versus PT for the same ventilatory mode.

ACV: assist-control ventilation
PSV: pressure support ventilation
PT: pressure trigger
FT: flow trigger
(clinical study + model with nine ICU ventilators)
Bench testing of pressure support ventilation with three different generations of ventilators


Fig. 8  Inspiratory trigger time delay ($TD_{tg}$, expressed in ms) according to the simulated level of inspiratory drive ($P_{0.1} = 2$ cmH$_2$O and $4$ cmH$_2$O). Same ventilators as in Fig. 2. No asterisk indicates new generation, * indicates previous generation, ** indicates piston and turbine-based ventilators, # indicates $P<0.05$ versus all new generation ventilators.

Pressure trigger:
- Delay (TD)
- Negative pressure ($\Delta P_{aw}$)
INSPIRATORY EFFORTS DURING ACV

Volume-control: TransPulmonary Pressure is controlled

Pressure-control: TransPulmonary Pressure is NOT controlled
TRIGGERING DELAY

Effective trigger: $P_{mus} = - (\text{elastic recoil pressure} + \text{trigger threshold})$. 

**INTRINSIC PEEP (PEEPi)** favors also ineffective efforts.
MISSED EFFORTS

flow trigger 5 l/min.  flow trigger 2 l/min.
MEASURES TO REDUCE PEEPi

Reduce resistance in airways
  • Bronchodilators and steroids
  • Tube patency – remove secretions

Prolong expiratory time
  (only in case of not-flow limitation PEEPi) hyperdynamic
  • Reduce inspiratory time (cycle sooner)
  • Decrease respiratory rate

Increase external PEEP
  (only in case of flow limitation: reduces the pressure difference between Palv and Paw)

Decrease Tidal Volume
  • Decrease pressure support
CAUSES OF DELAYED TRIGGERING AND MISSED EFFORTS

✓ **Intrinsic PEEP** (common in COPD patients)
✓ **Ineffective respiratory drive** (including sedation)
✓ **Muscle weakness**
✓ **Insensitive trigger**
✓ **Dependency to the previous breath**
Ineffective triggering

<table>
<thead>
<tr>
<th>Component</th>
<th>Breaths Preceding Nontriggering Attempts</th>
<th>Breaths Preceding Triggered Breaths</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inspiratory time, s</td>
<td>1.38 ± 0.05</td>
<td>1.24 ± 0.03</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Expiratory time, s</td>
<td>1.02 ± 0.04</td>
<td>1.46 ± 0.08</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Respiratory cycle duration, s</td>
<td>2.39 ± 0.07</td>
<td>2.70 ± 0.09</td>
<td>&lt; 0.0005</td>
</tr>
<tr>
<td>T_i/T_e</td>
<td>1.87 ± 0.26</td>
<td>1.01 ± 0.05</td>
<td>&lt; 0.005</td>
</tr>
<tr>
<td>Tidal volume, ml</td>
<td>486 ± 19</td>
<td>444 ± 16</td>
<td>&lt; 0.02</td>
</tr>
<tr>
<td>Dynamic PEEPi, cm H_2O</td>
<td>4.22 ± 0.26</td>
<td>3.25 ± 0.23</td>
<td>&lt; 0.0001</td>
</tr>
</tbody>
</table>

* Values are means ± SE.

Ineffective triggering increased with increasing level of assistance as a result of the decreased in the neural drive. (but note that higher assistance reduce PTP and dyspnea)
PSV (Pinsp 16 cmH$_2$O)

PSV (Pinsp 12 cmH$_2$O)

COPD patient.
Prolonged weaning process
AUTO-TRIGGERING

![Graphs showing Paw (cmH₂O), Flow (l/sec), and Pes (cmH₂O) over time.](image)
CAUSES OF AUTO-TRIGGER

✓ Too low trigger threshold
✓ Leaks
✓ Water in the circuit
✓ Hyper-dynamic cardiac state
(b) THE CONTROL VARIABLE

*Flow starvation*
FLOW STARVATION  delay in pressurization
In **volume-based** ventilatory modes. When the flow is fixed.

It occurs if the delivery of a mechanical breath is inappropriately low for the patient’s demand.
1) the lowest pressurization rate caused the lowest tidal volume, highest respiratory rate and highest work of breathing (no differences for all the other pressurization rates);
2) the patients comfort was worse at the lowest and highest pressurization rates.
(c) THE CYCLING OFF VARIABLE

Delayed cycling, Early cycling.
DELAYED CYCLING

FIGURE 4. Phase asynchrony (TIP < TIV). When patients arrive at end-inspiration, they expect the airway to be free of resistance and ready to allow expiration. However, if the ventilator’s inspiratory time (TIV) is longer than the patient’s inspiratory time (TIP), the airway is still being pressurized. If
Delayed cycling occur mostly in COPD patients.

When airway resistance increases, the flow curve becomes flatter. The cycling point will be reached later, which in turn increases the likelihood of delayed cycling.
FIGURE 5. Phase asynchrony ($T_{Ip} > T_{Iv}$). If the patient’s inspiratory time ($T_{Ip}$) is longer than the ventilator’s inspiratory time ($T_{Iv}$).
MEASURES TO REDUCE DOUBLE TRIGGERING

Reduce ventilatory demand
  Increase Vt
  Sedation

Increase the inspiratory time (Ti)
  Reduce cycling criterion
  Increase Ti
ASYNCHRONIES DURING DIFFERENT VENTILATORY MODES

✓ PAV
✓ NAVA
✓ NIV
Pmus = (Volume*Elastance) + (Flow*Resistance)

PAV provides ventilatory assistance in terms of flow assist (FA, cmH₂O/l/s) and volume assist (VA, cmH₂O/l)

Pmus = Volume* (Elastance - VA) + Flow* (Resistance - FA)

The clinician set the % of ventilatory support

PAV improves patient-ventilator **synchrony during the onset of inspiration**, by matching the patient’s inspiratory demand. However, triggering is not directly improved with PAV because the patient still has to generate sufficient flow to trigger the ventilator.
THE “RUNAWAY” PHENOMENON

delayed ventilator flow ending

✓ ELASTANCE AND RESISTANCE OVERESTIMATION
✓ LEAKS

\[ P_{mus} = \text{Volume} \times (\text{Elastance} - VA) + \text{Flow} \times (\text{Resistance} - FA) \]

COPD pat with **PEEPi**, VA 90%
THE TRIGGER
The EAdi exceed a threshold deflection EAdi at the beginning of the neural inspiration. Only if before pressure and flow increase.
NO: Missed efforts; Delay triggering, Auto triggering,
YES: accessory muscles contraction

THE CYCLES OFF
70% of the EAdi peak

THE NEUROMECHANICAL COUPLING
affected by dynamic hyperinflation, muscle weakness, NAVA level

Advantages of NAVA

Edi monitoring
Standardized Edi catheter position procedure allows reliable feed tube placement
Monitoring presence or not of spontaneous breathing during conventional ventilation
Monitoring patient-ventilator interaction during conventional ventilation

NAVA Mode
Improved timing between patient effort and assist delivery independent of
a. Leaks
b. Properties of interface
c. Liquid in respiratory circuit
d. Cardiogenic oscillations in the airway flow
e. Intrinsic PEEP

Neurally integrated with timing of upper airway activity and function
Matching of magnitude assist delivery to patient effort
a. Preservation of respiratory drive
b. No runaway
c. Prevention of excessive assist delivery
d. Responds to changes in respiratory demand
NAVA IMPACT ON PATIENT-VENTILATOR INTERACTION

TRIGGERING DELAY:
Shorter trigger delay (13% of Ti vs 35% in PSV)
always < 150 ms.
even in case of severe air trapping or leaks, triggering is not compromised.

WASTED EFFORT:
Always absent, even for high NAVA levels

CYCLING-OFF DELAYS:
Low, even for high NAVA levels

ASYNCHRONY INDEX:
Significantly lower with NAVA.

WOBtot AND WOBtrigger:
Lower during NAVA

Same advantages even during NIV-NAVA
With PAV the most promising closed-loop ventilation modes.
A very high pressurization rate (i.e. 200 cmH₂O·s⁻¹) is associated with increased air leakage and a poor tolerance of ventilation, despite the diaphragmatic effort being reduced. An individual titration should be targeted.
Air leaks are very likely during NIV. Leaks from the mask may impair the expiratory trigger cycling mechanism during PSV.

- time-cycled (NIPSVtc)
- flow-cycled (NIPSVfc)

6 pat (mild ARDS, pneumonia AIDS-related. No COPD)
PSV: P_{insp} 10 \text{ cmH}_2\text{O}, \text{ PEEP} 5 \text{ cmH}_2\text{O}.
DELAYED CYCLING OFF can cause patient-ventilator mismatch and NIV failure in COPD patients.

1. Augmenting off-cycling reduced expiratory cycle latency ($P < .001$), decreased intrinsic PEEP, and avoided non-supported breaths.

2. Setting cycling to 50% of peak inspiratory flow achieved best synchronization.

3. Using the helmet interface increased expiratory cycle latency in almost all settings ($P < .001$).
PREVALENCE, PROGNOSIS AND INFLUENCES ON PATIENT–VENTILATOR ASYNCHRONY

Incidence
Detection
Influencing factors (Ventilator settings and Sedation)
24%-27% exhibited asynchrony in greater than 10% of breaths.
Lower IA for NAVA ventilation

**Ineffective triggering** and **double-triggering** contributed more than 98% of the total number of asynchrony events.

Ineffective triggering accounts for 85% of asynchronies.

Patients with an **ineffective triggering (ITI) ≥ 10%** showed:
- 4 days more in ventilation time;
- 4 days longer ICU and 13 days hospital stays;
- Not clear differences in reintubation, tracheostomy, mortality (different pat population)

ICU staff physicians are able to detect less than one-third of asynchronies

**Thille** et al. ICM 2006. 32:1515-22
**De Wit** et al. Crit Care Med 2009 Vol. 37, No. 10
**Colombo** et al. Crit Care Med 2011 Vol. 39, No. 11
**Blanch L et al.** ICM 2015, 41:633–641
Two methods of asynchrony detection

103 patients post ARF ventilated for a median duration of 5 days

Patient–Ventilator Asynchrony during Weaning

7% exhibited severe asynchrony (AI > 10%) using the flow-and-pressure method and 86% when using the EAdi-based method (P < 0.0001).

Severe patient–ventilator asynchrony was not associated with adverse clinical outcome.
The value of dynamic preload variables during spontaneous ventilation

Respiratory efforts
Patient-ventilator asynchrony
Respiratory rate
Tidal volume
Upper airway obstruction
High abdominal pressure during exp

DPOP (%)
PLET respiratory variations as difference between the maximal and minimal amplitude divided by mean;

sPVI (%)
PLET respiratory variations as difference between the maximal and minimal amplitude divided by the maximal amplitude;

PPV/SVV/SPV
pulse pressure variation
Stroke volume variation
Systolic pressure variation

The observed magnitude of the Pleth variations and of the PPV were much higher than the values that are usually used to determine fluid responsiveness in mechanically ventilated patients (around 13%).
severe breath-stacking asynchrony (DOUBLE TRIGGERING)

asynchrony index > 10% of total breaths.

Breath-stacking asynchrony index
44 [27–87]% at baseline.
66 of 254 (26%) mechanically ventilated patients

3 categories:  
- no intervention,  
- increase of sedation–analgesia,  
- change of ventilator setting.

the decrease of asynchrony index

(4 [−4, 12]%)  
(−41 [−66, 7]%)  
(−99 [−92, −100]%)  

Multiple Regression Analysis Assessing Predictors of Breath-Stacking Decrease After Intervention

Increase of inspiratory time (A)
Coef. 50.7; SE 20.3; T 2.5; p 0.01

Pressure-support ventilation (B)
Coef. 46.4; SE 13.6; T 3.4; p 0.001
SEDATION
PSV or NAVA

PROPOFOL

✓ Not variation in respiratory timing;
✓ Significant decrease of neural drive (PSV, NAVA) : reduced Eadi_{peak};
✓ Significant decrease of effort (PSV) : ineffective triggering.

REMIFENTANIL

✓ Prolongation of the expiratory time (if >0.05 μg/(Kg·min));
✓ Not variation in respiratory drive;

Costa R. et al. Respiratory Physiology & Neurobiology 244 (2017) 10–16
"He's resting comfortably."