

An Analysis of Desynchronization Between the Spontaneously Breathing Patient and Ventilator During Inspiratory Pressure Support*

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It is common practice to convert patients with acute respiratory insufficiency (ARI) from controlled mechanical ventilation to some form of assisted spontaneous breathing as early as possible. A widely used mode of assisted spontaneous breathing is patient-triggered inspiratory pressure support (IPS). We investigated 11 patients with ARI during weaning from mechanical ventilation using IPS and found that in 9 of these patients, desynchronization between patient and ventilator occurred, *ie*, that the ventilator did not detect and support all the patients' breathing efforts. Five of these 9 patients displayed severe desynchronization lasting at least 5 min and with less than half of all breathing efforts being supported by the ventilator. We present the analysis of gas flow, volume, esophageal pressure, airway pressure, and tracheal pressure of 1 patient with ARI displaying desynchronization under IPS. Our results imply that desynchronization can occur due to the following: (1) inspiratory response delays caused by the inspiratory triggering mechanisms and the demand flow characteristics of the ventilator; (2) a mismatch between

the patient's completion of the inspiration effort and the ventilator's criterion for terminating pressure support; and (3) restriction of expiration due to resistance from patient's airways, endotracheal tube, and expiratory valve. From our analysis, we have made proposals for reducing desynchronization in clinical practice.

(*Chest* 1995; 107:1387-94)

ARI=acute respiratory insufficiency; COPD=chronic obstructive pulmonary disease; FIO₂=fraction of inspiratory oxygen; IPS=inspiratory pressure support; PAV=proportional assist ventilation; PEEP=positive end-expiratory pressure; rr_{patient}=patient's respiratory rate, intrinsic respiratory rate, rate of inspiratory efforts; rr_{ventilator}=respiratory rate, rate of pressure supports; SIMV=synchronized intermittent mandatory ventilation

Key words: respiration, artificial; respiratory insufficiency; therapy; ventilator weaning

Due to the many problems associated with controlled mechanical ventilation, it has become increasingly common practice to introduce assisted spontaneous breathing at a stage where, in the past, the patient was continued to be treated with controlled mechanical ventilation. A currently popular mode of assisted spontaneous breathing is patient-triggered inspiratory pressure support (IPS).

Inspiratory pressure support is used to augment insufficient spontaneous breathing efforts to ensure a sufficient tidal volume. It has been shown that tidal volume during IPS depends on the mechanical

properties of the respiratory system (mainly resistance of airways and the endotracheal tube, and compliance), the level of pressure support, and the driving pressure of the ventilatory muscles. If the driving pressure of the ventilatory muscles decreases (*eg*, due to weakness or fatigue), the tidal volume will decrease correspondingly, assuming all other factors remain constant. However, the patient can keep his minute ventilation constant by increasing his respiratory rate.¹

High respiratory rate with low tidal volume is termed rapid shallow breathing and can be a sign of ventilatory muscle weakness or fatigue.² Patients with rapid shallow breathing can be supported by increasing the level of pressure support.³ As a result of increased pressure support, tidal volume rises and respiratory rate decreases.^{1,3,4}

It is doubtful, however, whether this simple causal relationship between the mechanical properties of the respiratory system, driving pressure, pressure support, and tidal volume is valid under all conditions. Gurevitch and Gelmont⁵ give an example of a

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Presented in part at the joint annual meeting of the Swiss Society of Pneumology and the Swiss Society of Intensive Care Medicine, Lugano, October 7-9, 1993.

Supported by the Swiss National Science Foundation 32-39382.93.

[†]Deceased. Please see the acknowledgment on page 1394.

Manuscript received April 25, 1994; revision accepted October 27.

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ventilator's inconsistent response to the breathing efforts of a patient with chronic obstructive pulmonary disease (COPD) under IPS. Their findings show that it is low inspiratory trigger sensitivity that leads to increased ventilator response delay. Furthermore, they found desynchronization between the patient and the ventilator, *ie*, that the ventilator did not detect (and consequently did not support) all of the patient's breathing efforts. Hubmayr⁶ documented a patient with extremely severe COPD and desynchronization under IPS being related to the level of pressure support. This patient showed desynchronization even at a pressure support level of 5 mbar.

Younes^{7,8} systematically examined the causes for desynchronization during IPS using a computer model simulation. He found that nonassistance of a patient's inspiratory effort occurs when peak patient effort is less than the sum of intrinsic positive end-expiratory pressure (intrinsic PEEP) and trigger threshold. Intrinsic PEEP can arise when expiratory time is too short (*eg*, due to high respiratory rate) or when expiratory air flow is too low (*eg*, due to high airway resistance) to completely exhale a high tidal volume (which could be caused by a high level of pressure support). Gottfried⁹ documented a severe COPD case, whereby the patient was unable to trigger the ventilator during synchronized intermittent mandatory ventilation (SIMV). He found that trigger failure resulted from dynamic hyperinflation and consequently from an increased intrinsic PEEP. As trigger mechanisms in SIMV and IPS are similar, it is possible that Gottfried's findings are also valid under IPS. However, desynchronization under IPS and SIMV are not comparable in all respects. Under IPS, the ventilator attempts to support every breathing effort of the patient. Under SIMV, however, not all breathing efforts are supported by a volume- or pressure-controlled breath, that is, between the mandatory breaths, the patient can breathe spontaneously. In addition, only the beginning, not the end, of the mandatory breaths are synchronized with the patient's breathing effort. Further, the demand flow of a pressure-targeted breath synchronizes better with the patient's ventilatory drive in comparison to the fixed flow of a volume-assisted breath.^{1,10}

During our long-term study of patients with acute respiratory insufficiency (ARI) being weaned from mechanical ventilation using IPS, we found desynchronization corresponding to that described in other studies⁵⁻⁹ did occur in several patients. Moreover, we observed that the ventilator's nondetection of a patient's breathing effort was not always an isolated event, but that it could occur for hours as sustained desynchronization between patient and ventilator.

The purpose of this study was to investigate the frequency and appearance of desynchronization be-

tween patients with ARI and the ventilator under IPS, and to present a detailed analysis of the origins of desynchronization in one patient who displayed several degrees of desynchronization.

METHODS

Patients suffering from a variety of critical illnesses requiring mechanical ventilation due to ARI (Table 1) were included in our study when the ventilatory mode was switched from mechanical ventilation to IPS. This was done when the patient showed spontaneous breathing efforts being sufficient enough to be able to trigger the ventilator and to maintain a sufficient ventilation at pressure support levels below 20 mbar, independent of PEEP or fraction of inspiratory oxygen (F_IO₂). When F_IO₂ was below 0.6, pressure support level, PEEP, and F_IO₂ were reduced gradually, taking into consideration clinical aspects such as blood gases, respiratory rate, tidal volume, and minute ventilation.

We observed the course of weaning with IPS in the following way: gas flow, airway pressure, and esophageal pressure were recorded by a ventilation monitor (CP-100, Bicore Monitoring Systems, Irvine, Calif) for the 5 min following each change of the ventilator setting selected by the therapeutic team. Computer recordings were analyzed offline and had no impact on therapy or the ventilator setting. All patients were ventilated with a ventilator (Bennett 7200, Puritan Bennett Corp, Carlsbad, Calif). For all patients, the trigger threshold of IPS was set at 2 mbar below PEEP. Gas flow was measured with a variable orifice flowmeter situated between the Y-piece and swivel connector of the endotracheal tube. The monitor (CP-100) measured airway pressure via the patient-side tubing of the flowmeter, digitizing the analog signals with a resolution of 12 bits and sending them via a serial interface (RS232c) at a rate of 50 Hz to a computer (PC) that stored the data. The flow signal of the nonlinear flowmeter was linearized in the PC using the flow-pressure characteristics determined by the manufacturer for each flowmeter individually. Tracheal pressure was calculated from the airway pressure and gas flow using the nonlinear pressure-flow relationship of the endotracheal tube.¹¹ Volume was calculated by numeric integration of flow over time. The respiratory rate of the ventilator (number of pressure supports per minute, $rr_{ventilator}$) was derived from the airway pressure curve. The patient's respiratory rate or, in other words, the intrinsic respiratory rate (number of inspiratory efforts per minute, $rr_{patient}$) was derived from the esophageal pressure, airway pressure, and gas flow curves as described below. The severity of desynchronization was expressed as the percentage of the inspiratory efforts that are not supported by the ventilator.

$$\text{desynchronization} = \left(1 - \frac{rr_{ventilator}}{rr_{patient}}\right) \times 100$$

The measurement was approved by the ethical committee of our institution, and informed consent was obtained from close relatives of the patients.

The patient (No. 1 in Table 1) whose gas flow and pressure recordings are presented in this article is a 59-year-old man with a body weight of 85 kg. He had an acute myocardial infarction with cardiogenic shock and was tracheally intubated following severe pulmonary edema complicated by bronchopneumonia. The endotracheal tube had an inner diameter of 8 mm and a length of 28 cm. The patient was mechanically ventilated for 24 h after which sedation was reduced and the ventilatory mode was switched to assisted spontaneous breathing with IPS. The data presented were taken on the third and fourth day after intubation. The patient was successfully extubated on the sixth day.

Table 1—Clinical Situation of the Patients and Severity of Desynchronization

Patient No./ Age, yr/ Weight, kg	ETT* ID/ Length	PaO ₂ , kPa	FIO ₂	IPS,† mbar	VT†	PEEP,† mbar	Desyn- chroni- zation,†‡ %	rr _{patient} ,† br/min	Intuba- tion Dura- tion,§ d	Indication for Mechanical Ventilation, ARI Due to	Relevant Diseases
1/59/85	8.0/28	12.2	0.5	15	786	7	40	36	3	Pulmonary edema and bronchopneumonia	Septic shock; coronary heart disease with myocardial infarction
2/69/85	7.5/26	13.8	0.4	14	445	3	14	31	2	Impaired respiratory drive	Perforated brain abscess with secondary seizures
3/58/98	8.0/28	20.1	0.4	15	516	5	31	48	10	Impaired respiratory drive and pulmonary edema	Near-drowning, systemic inflammatory response syndrome; epilepsy
4/74/74	7.5/26	13.7	0.4	14	391	5	40	29	4	Bronchopneumonia	History of bronchopneumonia (3x); paralysis of diaphragm
5/68/60	8.0/29	9.3	0.4	7	506	5	21	23	5	COPD and pneumonia	Severe COPD; history of thromboembolism
6/65/71	8.0/28	11.6	0.3	8	448	5	21	21	2	Amyotrophic lateral sclerosis	Bronchopneumonia
7/42/70	8.0/26	11.4	0.35	15	622	10	5	23	7	Drug-induced toxic pulmonary edema	Acute myelogenous leukemia, pancytopenia due to chemotherapy
8/68/119	7.5/26	9.7	0.3	13	637	8	20	15	2	Pulmonary edema and aspiration pneumonia	Coronary heart disease with left ventricular failure
9/60/100	8.5/26	9.3	0.6	17	851	8	2	12	2	Pulmonary edema and COPD	Acute myocardial infarction with repeated ventricular fibrillation; heart failure
10/82/58	8.0/28	10.6	0.6	11	805	10	0	12	4	Alveolar hemorrhage and pulmonary fibrosis	Systemic vasculitis with renal failure; history of COPD
11/30/61	7.5/30	12.4	0.21	14	467	5	0	25	1	Impaired respiratory drive	Ishemic stroke with secondary epilepsy due to diabetic complications

*Mallinckrodt hi-lo tubes; ETT=endotracheal tube; ID=internal diameter in millimeters, length in centimeters.

†Mean values taken from all data recordings of each patient.

‡Percentage of nonsupported breathing efforts= $(1 - [rr_{ventilator}/rr_{patient}]) \times 100$.

§Total numbers of days of intubation prior to commencement of investigation.

RESULTS

Table 1 lists clinical data of all patients and their ventilatory situation. We found desynchronization of several levels of severity in 9 of the 11 patients with ARI studied.

Figure 1 shows the course of desynchronization over time in all patients who displayed desynchronization. Figure 1 contains the results for all data recordings.

The following results analyze and explain in detail our findings in relation to patient 1. Figure 2 shows gas flow, volume, airway pressure, intratracheal

pressure, and esophageal pressure of the patient. Recordings of the esophageal pressure enabled us to estimate the timing of the patient's inspiratory effort. The onset of the fast drop in esophageal pressure was taken as the initiation of inspiratory effort, the end of the fast rise in esophageal pressure as termination of inspiratory effort. The inspiration and expiration of the ventilator, in contrast, is read from the fast rise and fast drop of airway pressure. When the duration of the inspiratory effort of the patient and the inspiration delivered by the ventilator are marked (Fig 2), a time lag between them (*ie*, resulting from a delay

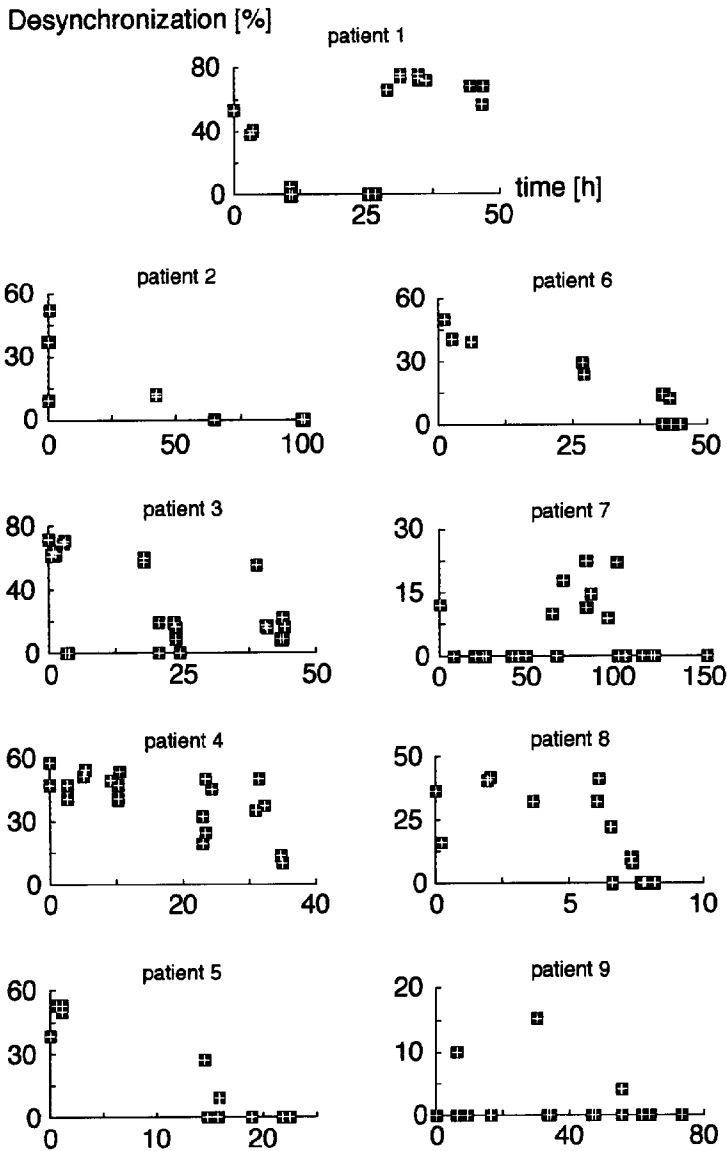


FIGURE 1. Course of desynchronization over time for all data recordings in all patients in which desynchronization was identified.

in response by the ventilator to both the beginning and end of the patient's inspiratory effort) or even a complete lack of response by the ventilator becomes visible.

Specifically, the patient's third breath in Figure 2 begins 700 ms after opening of the expiratory valve, *ie*, after the start of the ventilator's expiration. (Time measurements in our study are rounded to 100 ms because the beginning and end of a patient's inspiratory effort can be roughly estimated only from esophageal pressure.) At this point, airway pressure is still 5 mbar above trigger threshold and tracheal pressure is 6 mbar above trigger threshold. It takes 400 ms for the patient to lower airway pressure to trigger threshold. One hundred milliseconds later, the ventilator increases airway pressure. After an-

other 200 ms, the patient terminates his inspiratory effort, after which the resulting inspiratory flow pattern is not determined by an inspiratory movement of the patient's ventilatory muscles but by the airway pressure and mechanics of the respiratory system. As a result, the ventilator reaches the termination criterion of pressure support only 1,300 ms after the patient's completion of inspiratory effort. After only 400 ms, the patient attempts to inspire again, but tracheal pressure (11 mbar) and airway pressure (9 mbar) are now so high that they cannot be lowered to the trigger threshold. Our measurement of volume shows a progressive dynamic hyperinflation during the first 3 breaths leading to intrinsic PEEP, which must first be overcome by the patient before an inspiration can be triggered. Tra-

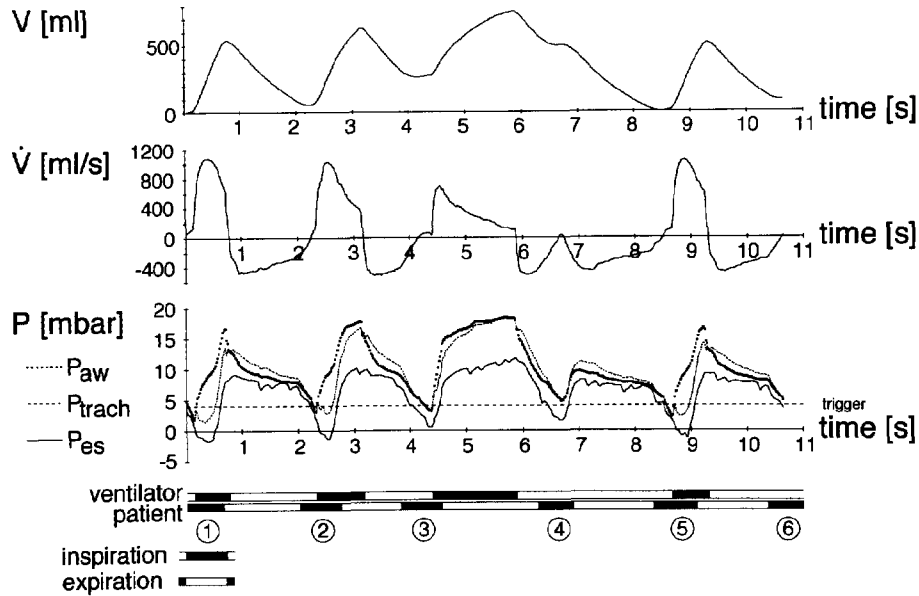


FIGURE 2. Volume, gas flow, tracheal pressure, airway pressure, and esophageal pressure in a patient with ARI with IPS=12 mbar, PEEP=6 mbar, trigger threshold=4 mbar. Tracheal pressure is calculated by the method described by Guttman et al.¹¹ Increasing time lag between the patient's inspiratory effort and the ventilator's response leads to nondetection of the patient's fourth inspiratory effort.

cheal pressure is 5 mbar above PEEP at the onset of the patient's fourth inspiratory effort and intrinsic PEEP must be even higher and thus greatly exceeds the trigger threshold of 2 mbar below PEEP.

Figure 3 shows sustained severe desynchronization when IPS=16 mbar. The patient's respiratory rate is very high (48 breaths per min); however, only every second or third inspiratory effort made by the patient is supported by the ventilator.

Figure 4 shows airway pressure and esophageal

pressure at a pressure support of 14 mbar. The patient's respiratory rate was 38 breaths per min with a tidal volume of 367 mL and a minute volume of 14.1 L/min. The esophageal pressure amplitude was extremely large, indicating strong inspiratory efforts, and correspondingly, the ventilator responded with pressure support to each of the patient's inspiratory efforts.

To treat the high respiratory rate and low tidal volume of this patient, pressure support was in-

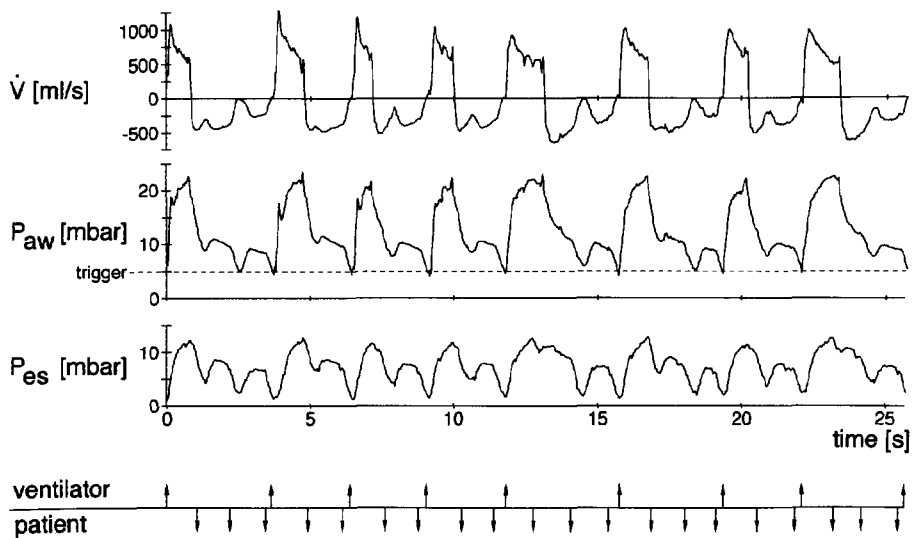


FIGURE 3. Gas flow, airway pressure, and esophageal pressure at IPS=16 mbar, PEEP=7 mbar, trigger threshold=5 mbar. Although the patient attempts to inspire 48 times per minute, the ventilator responds with a pressure support of only 20 times per minute. Downward-pointing arrows indicate an inspiratory effort by the patient; upward-pointing arrows indicate the onset of pressure support.

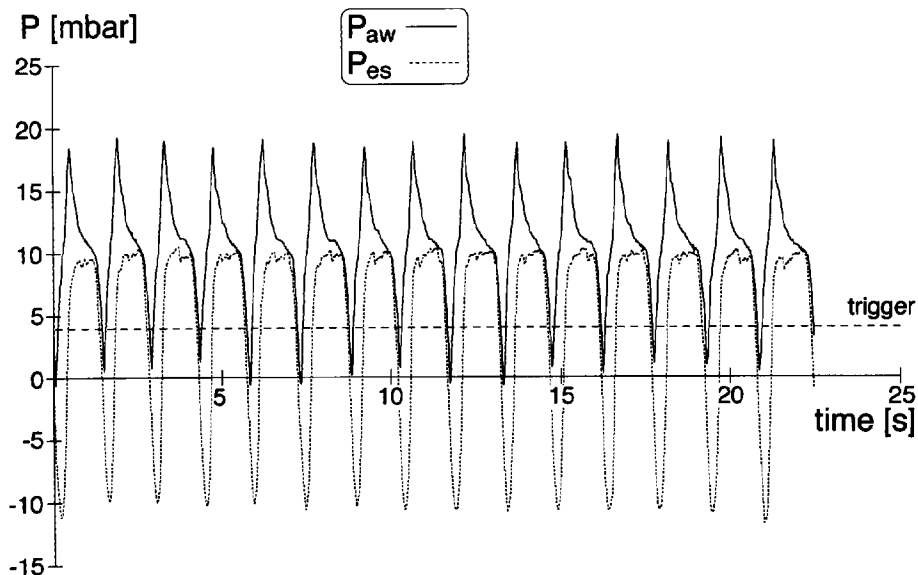


FIGURE 4. Airway pressure and esophageal pressure at a pressure support of 14 mbar. Respiratory rate was 38/min. The ventilator responds with pressure support to each of the patient's inspiratory efforts.

creased by 4 mbar. Figure 5 shows the airway pressure and esophageal pressure 2½ hours later. Tidal volume increased to 806 mL, but minute ventilation decreased to 8.7 L/min due to the respiratory rate, measured by the ventilator, having decreased from 38 breaths per min to 11 breaths per min. However, observing the esophageal pressure, it is apparent that the patient's rate of inspiratory efforts is virtually unchanged (only a slight decrease from 38 breaths per min to 34/min).

DISCUSSION

The main advantage of patient-triggered IPS over other modes of assisted spontaneous breathing is that the patient controls inspiratory and expiratory time. Therefore, IPS is expected to increase the patient's breathing comfort and synchrony with a ventilator.¹ Ideally, the ventilator would provide pressure support in the airways during the entire inspiratory effort, and on completion of the patient's inspiratory effort, the airway pressure would then be reduced to

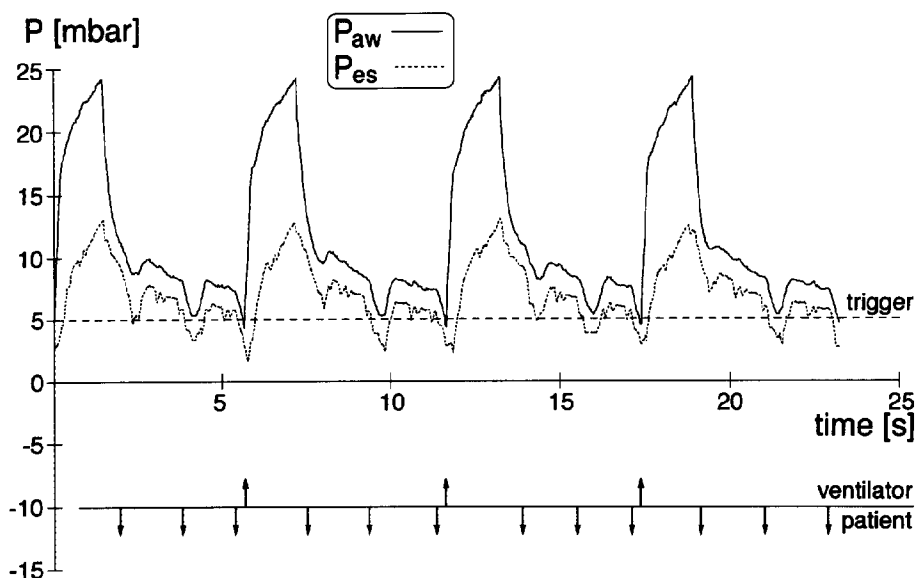


FIGURE 5. Airway pressure and esophageal pressure at a pressure support of 18 mbar. The ventilator responds only to every third patient inspiratory effort with pressure support.

PEEP level.¹ In practice, however, there is a delay between the initiation and termination of a patient's inspiratory effort, the detection of this effort, and reaction by the ventilator,^{5,10} as shown in Figure 2. The delay between the initiation of the patient's inspiratory effort and the onset of pressure support is called inspiratory response delay, that is, the time that the patient needs to decrease airway pressure below trigger threshold and the ventilator's response delay. The delay between the completion of the patient's inspiratory effort and termination of pressure support is called expiratory response delay, which arises mainly from the arbitrariness of the inspiratory termination criterion. Most ventilators terminate inspiration when inspiratory flow falls below a certain percent of inspiratory peak flow. Inspiratory termination criterion can be reached before the patient's inspiratory effort is actually terminated.^{8,10} This type of asynchrony would be most likely to occur at high inspiratory peak flow (*eg*, due to a short pressure rise time);⁸ however, we did not observe this in our patients.

Expiratory response delay is increased due to the following ventilator settings and behavior: (1) a lower inspiratory peak flow due to a longer pressure rise time or a higher endotracheal tube resistance; (2) a higher level of pressure support; and (3) a less sensitive criterion (*ie*, a lower percentage of inspiratory peak flow) for the termination of pressure support.

Inspiratory response delay is increased when the following occur: (1) the inspiratory trigger threshold is lowered (becoming less sensitive); (2) there is a larger tidal volume to be exhaled (resulting from higher levels of pressure support and/or prolonged inspiration); and (3) the expiratory gas flow is restricted, in particular by endotracheal tube resistance or by expiratory flow limitation due to bronchial constriction and collapsing airways.

Both higher tidal volume and restricted expiratory flow increase intrinsic PEEP and thus have a similar effect to a lower trigger threshold.

During inspiratory response delay, the patient's inspiratory effort is not supported by the ventilator, and during expiratory response delay, the patient is unable to exhale. Consequently, the tidal volume decreases when the inspiratory or expiratory response delay increases. This becomes more apparent at higher respiratory rates. Figure 4 illustrates such a situation. The high amplitude of the esophageal pressure in Figure 4 indicates that an enormous inspiratory effort is necessary to trigger pressure support 38 times per minute.

If the response delay exceeds a critical value, the ventilator is unable to detect every inspiratory effort by the patient, and thus desynchronization between patient and ventilator occurs. Figures 3 and 5 illus-

trate such a type of desynchronization. We detected desynchronization in 9 of 11 patients with ARI. In five of these patients, desynchronization was severe, *ie*, less than 50% of the inspiratory efforts were supported by the ventilator. Thus, severe desynchronization seems to be quite a frequent event in patients with ARI.

This raises the question how desynchronization can be detected and how it can be eliminated. Desynchronization can be clearly detected not only by comparison of esophageal pressure and airway pressure, but also by analyzing gas flow curves vs time. A nonassisted breathing effort by the patient during the ventilator's expiration results in a typical temporary fall in expiratory gas flow. Correspondingly, the associated airway pressure curve shows a less marked temporary fall during the nonassisted breathing effort.

Inspiratory pressure support is commonly used in patients with severe acute respiratory failure. As these patients often need a large minute ventilation and show a reduced efficiency of ventilatory muscles, detection and elimination of desynchronization are clinically very important. Synchronization between patient and ventilator could be improved by the following measures: (1) increasing the inspiratory trigger sensitivity;^{5,7,8} (2) minimizing the inspiratory pressure rise time;¹⁰ (3) reducing the level of pressure support as much as possible;⁶ (4) increasing the sensitivity of the termination criterion of pressure support; (5) using an endotracheal tube with a large inner diameter; (6) avoiding resistive elements inserted between Y-piece and endotracheal tube (*eg*, bacterial filter); (7) repeating thorough suctioning of tracheobronchial mucus as often as necessary; (8) treating bronchial constriction and mucus production effectively; and (9) treating expiratory flow limitation in patients with COPD by increasing PEEP.^{9,12}

Unfortunately, pressure rise time and the termination criterion for pressure support are adjustable only in few ventilators. Furthermore, pressure support with weak patients cannot be lowered too much, as alveolar ventilation may become insufficient, and increasing trigger sensitivity may cause self triggering.

Inspiratory and expiratory response delay is inherent in the principle of the patient-triggered IPS. Consequently, the occurrence of desynchronization between patient and ventilator cannot be completely avoided. In a computer model simulation, Younes^{7,8} found that desynchronization can be avoided when pressure support is applied in proportion to the inspired volume and flow (proportional assist ventilation, PAV). In one patient, we were able to demonstrate that desynchronization can be completely and immediately eliminated even at high

respiratory rates by continuous compensation of the pressure difference across the endotracheal tube in combination with PAV of the tracheal pressure.¹³ Under this mode, excessively high pressure support is avoided, inspiratory and expiratory response delays are minimized, and expiratory flow restriction due to the resistances of the endotracheal tube and expiratory valve is eliminated.


Our study has shown that desynchronization is accompanied by dynamic hyperinflation. By improving synchronization and thereby reducing dynamic hyperinflation, it is thus reasonable to assume that there will be positive effect on a patient's comfort and breathing freedom.

ACKNOWLEDGMENT: Professor Gunther Wolff died unexpectedly on October 24th, 1994. He was one of the pioneers in the field of intensive care medicine and dedicated a major part of his scientific career to research of mechanical ventilation and lung pathophysiology. Dr. Wolff's energy and enthusiasm, his innovative ideas, and his personality are irreplaceable.

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