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Review article

Monitoring electrical activity of the diaphragm and the ventilation mode NAVA (Neurally Adjusted Ventilatory Assist)

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Abstract

While it is possible to monitor the electrical activity of the heart using an electrocardiogram (ECG), the ability to monitor the electrical activity of the diaphragm (EAdi) in the clinical setting has not been possible until recently. A specialised nasogastric tube is now available which can monitor EAdi in the intensive care units (ICU). The EAdi signal can also be used to control a mechanical ventilator. The mode associated with the EAdi is referred to as neurally adjusted ventilator assist (NAVA). It can be used in spontaneously breathing patients. A NAVA ventilator breath is triggered (beginning of breath), delivered and cycled (end of breath) by the patient's neural control of breathing. This is markedly different from all other modes of ventilation which rely on a pneumatic trigger (flow or pressure change), and where breath delivery and breath termination are generally determined by how the clinician sets the ventilator's controls. This article will include a discussion of patient-ventilator asynchrony, monitoring the electric activity of the diaphragm (EAdi) with a specialised catheter and will provide an explanation of how NAVA operates.

Keywords: Neurally adjusted ventilatory assist, ventilation, electromyography, diaphragm.

Introduction

The respiratory centres of the brain govern our breathing pattern both unconsciously and consciously. They receive information from a variety of receptors in the body including chemoreceptors which respond to changes in blood gases and pH, stretch receptors and volume receptors in the lungs in addition to conscious signals from the brain. A vast amount of information is rapidly processed by the brain which then transmits a neural signal through the phrenic nerve to the diaphragm. Following signal reception, the diaphragm muscle depolarises and contracts. In the clinical setting, this process is analogous to the depolarisation and contraction of the heart. The intensity of the diaphragm's contraction is determined by the intensity of the neural stimulation and the number of neural fibres involved.

In normal individuals, higher the electrical activity during depolarisation, that much stronger will be the muscle contraction as more and more muscle fibres will contract. This is generally not true in patients with respiratory disorders such as chronic obstructive pulmonary disease (COPD) where the patient's lungs maybe in a state of hyperinflation to begin with itself. In such situations, although the EAdi may increase, strength of contraction of the diaphragm may not increase proportionately because of the abnormal position of the diaphragm.[1]

Evaluating diaphragm function using pleural pressure measurements

One way to evaluate diaphragm function in the clinical

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setting is to determine changes in pleural pressure as the thoracic volume changes during ventilation. Measurement of pressure in the pleural cavity can be used to assess changes in thoracic volume as the diaphragm contracts and relaxes. Pleural pressure can be directly measured by inserting a pressure monitoring catheter into the pleural space. However, this is a technique most often reserved for the study of animal models in the laboratory setting because of its invasive nature.

Another method for estimating pleural pressures and diaphragm function in the clinical setting is the monitoring of oesophageal pressure (P_{es}) changes. To indirectly measure pleural pressures, a balloon-tipped catheter with a monitoring lumen is inserted into the oesophagus. The catheter tip can be positioned within the oesophagus either in the thoracic region or in the stomach. Pleural and abdominal pressure values can be estimated from these two positional measurements.

The pressure in the lower third of the oesophagus closely estimates the pleural pressure (P_{pl}) in the area adjacent in an upright individual. Determining P_{pl} during inspiration, expiration and breath holding allows analysis of a variety of measurements including lung and chest wall compliance, work of breathing (WOB) and also the presence or absence of diaphragm movement (*e.g.*, presence of diaphragm paralysis).[2]

Some ventilators can provide an additional pressure port for this purpose. The CareFusion AVEA (Yorba Linda, California, USA) and the Hamilton G5 (Reno, Nevada, USA) have this extra port. Pleural pressures are estimated in the following manner: As intrathoracic pressures change during ventilation, the pressure is measured and displayed. For example, during normal spontaneous breathing, as the diaphragm contracts and descends, the intrathoracic pressure decreases. During relaxation of the diaphragm, the intrathoracic pressures rise as intrathoracic volume decreases.

However, the accuracy of P_{pl} measurements can be affected by several factors such as the volume of air in the catheter balloon, its position within the thorax and the patient's position. Another potential difficulty is inaccuracy associated with the filling of the catheter's balloon, since balloon volume can also affect results.[3] Consequently, the P_{es} may be useful in assessing changes in pleural pressure and diaphragm movement, but these values should be used with caution.

Monitoring electromyograph of the diaphragm in the clinical setting

During the past few decades, researchers have been looking for a method of monitoring the respiratory drive to breathe, phrenic nerve activity and electrical activity of the diaphragm (EAdi). Actual measurement of respiratory drive and phrenic nerve activity have not yielded practical application. For example, early attempts to study phrenic nerve activity required opening of the thoracic wall and placement of wires directly on the nerve in order to evaluate phrenic nerve function. This technique is too invasive and risky for the clinical setting.

In the 1950s, Petit *et al* managed to develop a technique to measure function of the diaphragm muscle with electromyography in human subjects.[1] Four decades of research was required for this to have practical application. Their research demonstrated that the phrenic nerve activity was directly related to the EAdi.[4]

During the years that followed, Christer Sinderby *et al* developed a practical technique for measuring the EAdi.[5] This method uses a catheter, very similar to an orogastric/nasogastric tube, with miniaturised electrodes located at the distal end. These electrodes monitor the electrical activity of the crural portion of the diaphragm where the oesophagus passes through the diaphragm. Their work also demonstrated that the electrical activity of the crural portion of the diaphragm reflects the overall electrical activity of the diaphragm during inspiration and that this is true for normal individuals and in patients with acute or chronic respiratory failure.[1,5,6]

A catheter for monitoring the EAdi

The catheter developed by Sinderby and Beck is

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now commercially available exclusively through Maquet, Inc, a Swedish based manufacturer owned by Gettinge, Inc. It is referred to as the Edi catheter. The SERVOi ventilator (Maquet, Inc) becomes the monitor for electrical activity detected by the catheter. In addition to monitoring EAdi, this can also be used to control ventilator function. This control function is referred to as the mode, neurally adjusted ventilator assist or NAVA. The NAVA mode will be reviewed later in this article.

The Edi catheter is available in neonatal, paediatric and adult sizes and works as a normal nasogastric or orogastric feeding tube. *Table 1* lists the catheter size to use, depending on patient size. It is made of medical grade polyurethane and is phthalate free. As in most nasogastric tubes, there is a lumen for feeding and one for evacuation. The sump lumen is only available in the 12 and 16 French sizes. At the proximal end are connects for the two lumens, plus a connector which goes to an Edi cable attached to the ventilator. The distal end includes side holes for nutrition or evacuation. There is a centimetre scale starting at the proximal tip and running the length of the catheter.

Table 1: Edi catheter selection guide

Patient Height (cm)	< 55	< 55	45-85	76-160	>140
Patient Weight (kg)	0.5 – 1.5	1.0 – 2.0	NA	NA	NA
French/cm	6/49	6/50	8/100	12/125	16/125 or 8/125

NA = not applicable

At the distal end are the electrodes. The 10 bipolar electrodes are made of stainless steel. The most proximal is a reference electrode. The inter-electrode distance (IED) varies based on the size and length of the catheter. Because of the presence of steel, this cannot be used during magnetic resonance imaging.

There is a barium sulphate strip included in the design to assist in radiographic identification, if desired. Radiography is not required to confirm placement, since a ventilator placement screen is available and will be discussed.

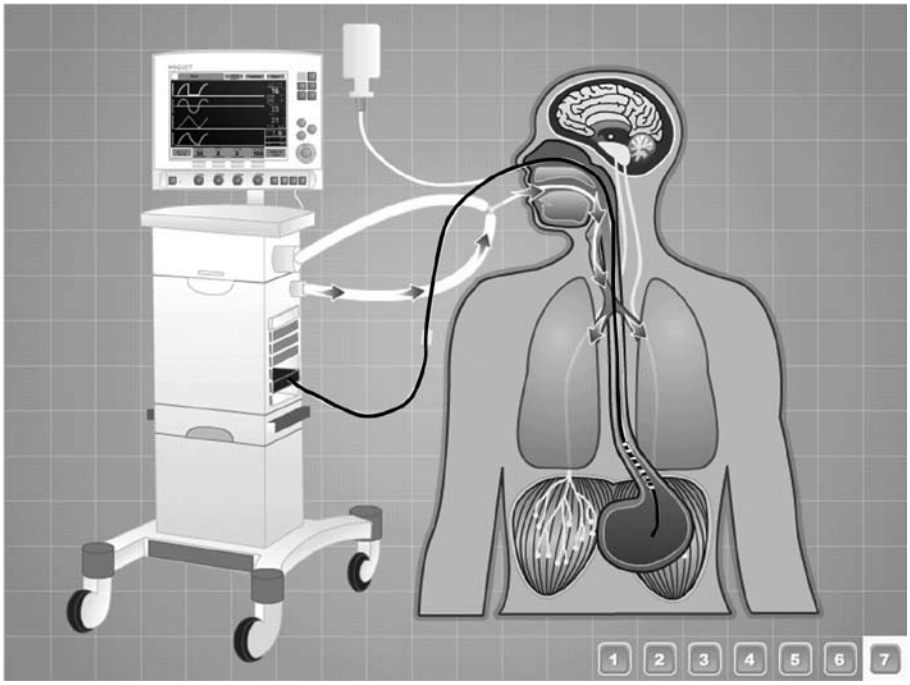

Figure 1: The catheter is inserted nasally and positioned so that the catheter electrodes (visible as white bands on the catheter in this image) is at the level where the oesophagus passes through the diaphragm (Courtesy Dr. C Sinderby, St-Michael's Hospital, Toronto, Canada).

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The catheter is inserted in the oesophagus so that the electrodes are positioned in the oesophagus at a level which is adjacent to the diaphragm (crural muscle of the diaphragm) (*Figure 1*). The depth of insertion can be estimated using a NEX (nose to ear lobe to xiphoid process of the sternum) measurement in centimetres. The NEX value is then adjusted based on an equation provided by the manufacturer. The equation uses the following information: oral or nasal insertion, catheter size and a correction factor.

Once inserted, position of the catheter can be confirmed by several techniques. Use of the NEX measurement along with manufacturer's recommended calculation confirms acceptable placement in about two-thirds of patients. Use of the ventilator's positioning screen further verifies the catheter's position (Figure 2).[7]

In the 'Edi Catheter positioning' screen seen in *Figure 2*, there are five waveforms. The first four represent ECG tracings obtained from the catheter electrode. When the catheter is correctly positioned, the electrodes which provide the top two waveforms are located above the diaphragm and adjacent to the heart. The electrodes for the next two waveforms are located below the diaphragm and further from the heart. The last (bottom) waveform represents the electrical activity of the diaphragm (Edi in microvolts).

When the Edi waveform is present and the catheter is in correct location, the middle two ECG waveforms will illuminate in a blue colour during the highest peak of the Edi waveform. Occasionally the blue colour might migrate up and down the four ECG waveforms when there is slight movement of the catheter. This is not unusual. Clinicians have demonstrated the use of the Edi catheter positioning screen in confirming a catheter's placement.[8]

In addition to the two methods for confirming Edi catheter position described above, there are a few additional techniques that are sometimes used to confirm the position of any nasogastric or orogastric tube. The centimetre marking at the nose or mouth should correspond with the value calculated from the NEX measurement used to determine the depth of insertion. Clinicians can also use the technique of injecting air into the catheter while listening

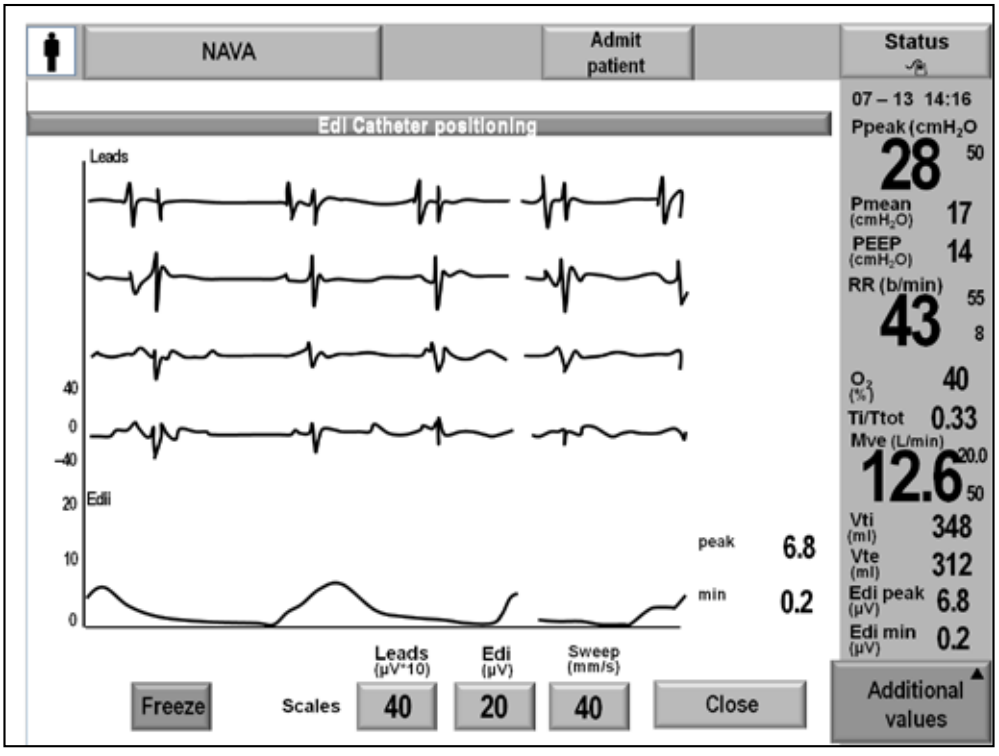

Figure 2: The catheter positioning screen on the SERVOi ventilator (see text for description).

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over the epigastric area with a stethoscope to assess for air entry into the stomach. A carbon dioxide (CO_2) detector can be placed at the proximal end of the catheter to ensure that no CO_2 is present. The presence of CO_2 would indicate the catheter is in the trachea and not the oesophagus. Some hospitals have protocols which require a chest radiograph to confirm catheter placement. Finally, the practitioner can use the ventilator screen with a spontaneously breathing patient and observe the pressure waveform and the Edi waveform while performing an expiratory hold manoeuvre. A drop in pressure as the patient actively inspires should coincide with an increase in the Edi indicating increased respiratory drive.

The Edi waveform and Edi values (normal and abnormal)

As illustrated in *Figure 2* (bottom waveform) the Edi is basically a sinusoidal wave and is measured in microvolts (μV). The Edi signal is measured 62.5 times/s.[9] The Edi is a very low intensity signal compared to the electrical signal of the heart. An ECG measured in millivolts may have an electrical amplitude 10-100 times that of the diaphragm.[1] Because of the ECG signal strength, electrodes can be placed on the skin outside the body. The Edi measured in μV is not a strong enough signal to be registered from outside the body. Edi monitoring requires that the Edi catheter be placed in the oesophagus, close to the diaphragm for the signal to be detected, filtered and amplified.

The Edi varies in intensity with each breath and even during a breath, which is how we normally breathe. Natural breathing is very noisy and does not have a constant rate or tidal volume, which is the kind of breathing pattern typically imposed by a mechanical ventilator. Studies have shown that noisy ventilation can be beneficial to patients with respiratory failure.[10] Occasionally, a very high Edi will appear corresponding to a sigh breath from the patient.

Until the Edi catheter technology became available, it was very difficult to determine what an Edi signal value would be in a normal individual. In this author's experience, the Edi value in normal individuals without respiratory or neuromuscular problems is about 5-10 μV during its peak (Edi peak). The Edi minimum value (Edi min) tends to be 0-1 μV in normal adults. Edi peak will increase when the diaphragm has to work harder, such as when respiratory demand increases.[11] Patients with chronic obstructive pulmonary disease and other forms of chronic respiratory insufficiency have been shown to have an Edi 5-7 times higher than normal. [5,11-13]

It has been observed that the Edi min tends to be higher in infants. The Edi min measured during expiration is believed to be involved in maintaining end-expiratory lung volume (EELV) in infants. A study of intubated and mechanically ventilated infants found that the Edi at end exhalation increased whenever PEEP was reduced to zero and increased when PEEP was returned to previous values. These findings suggest that the diaphragm remains partially active during expiration. Removal of the PEEP affects this tonic activity (Edi min). Thus, the evaluation of Edi min may have application in the management of PEEP in intubated infants.[14]

Sometimes when the Edi catheter is in the correct position, based on clinical findings, an Edi waveform is flat or not visible. This poses the question, what kind of patient condition would cause an absence of a signal from the respiratory centre, through the phrenic nerve and to the diaphragm. The problem could be the absence of respiratory centre activity. This might occur with brainstem injury, heavy sedation or similar problems. Even hyperventilation or excessive ventilator support, such as too much pressure or volume and too high a rate will induce apnoea and absence of the Edi signal. The presence of central apnoea in premature infants will also result in no signal.[15] It may be caused by a severed or damaged phrenic nerve, which might occur following thoracic surgery. The signal from the phrenic nerve to the diaphragm may also be blocked by neuromuscular blocking agents or a neuromuscular disorder which inhibits or blocks neuromuscular transmission.[13] The absence of a signal may be diagnostic for the clinician. In fact, the Edi signal can be used to evaluate recovery from paralysis.[16]

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Using the Edi to monitor patient-ventilator interaction

In addition to its value in monitoring diaphragm activity, the Edi catheter can also be used to evaluate patient-ventilator synchrony in patients being mechanically ventilated. Again this function is available only on the SERVOi ventilator (Maquet, Inc). The SERVOi has the ability to display the Edi waveform superimposed over the pressure waveform on the ventilator's main screen when appropriate software and hardware are installed. *Figure 3* shows an example of this function.

The Edi waveform can be superimposed over the pressure/time waveform in any of the commonly used modes of ventilation such as volume control, pressure control, synchronised intermittent mandatory ventilation (SIMV) and pressure support. *Figure 3* illustrates Edi monitoring in pressure support. Notice from the top waveform that the Edi and pressure do not correspond. The Edi activity in the diaphragm in this example is occurring before the ventilator begins to deliver the breath. In other words, the patient wants a breath, but the ventilator is delayed in providing flow to the patient. This is referred to as *trigger-asynchrony*. Following delivery of a breath, Edi begins to decrease from its peak value indicating the beginning of exhalation by the patient. However, the ventilator continues to deliver a constant pressure after the time the patient's drive to breath has ended. This represents *cycle-asynchrony*.

Using the Edi waveform during patient ventilation, the practitioner can identify the presence of patient-ventilator asynchrony (PVA) and can try to adjust ventilator parameters to better match what the patient wants. For example, adjusting ventilator trigger sensitivity may allow the ventilator to detect patient effort earlier and delivers the breath without a trigger delay or adjusting the inspiratory time may better fit the actual needs of the patient.

The Edi catheter can also be used postextubation to monitor the patient's tolerance of weaning and their response to any respiratory therapy treatments that

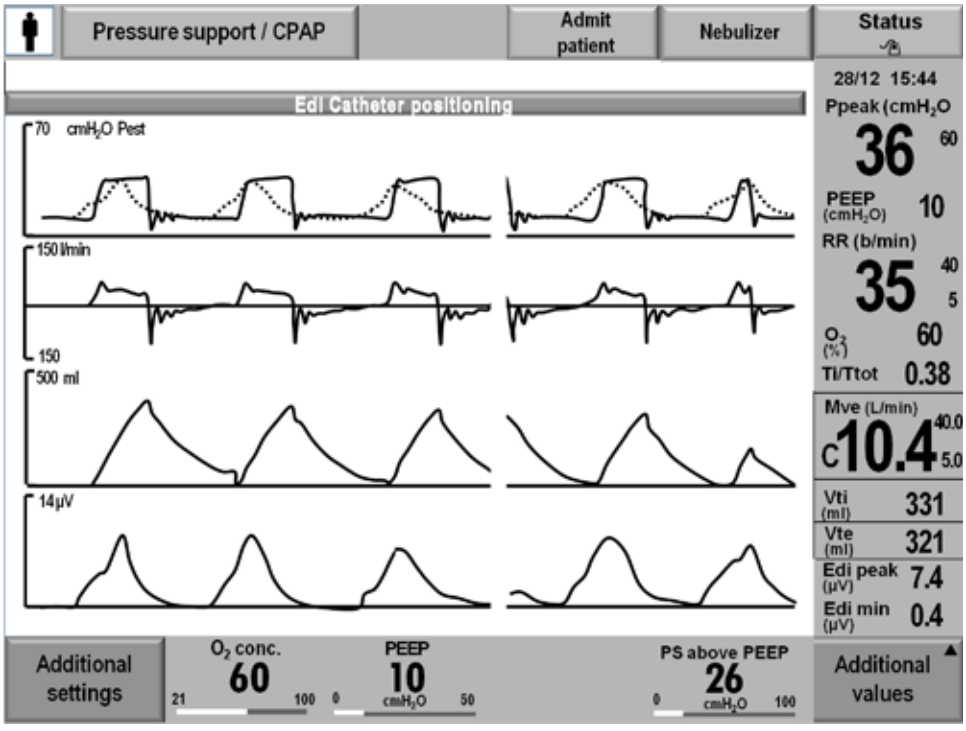

Figure 3: A drawing of the main screen of the SERVOi ventilator. This represents an adult patient in pressure support/CPAP mode. The top waveform is the pressure/time waveform (solid) and the Edi waveform (dashed line). The second waveform is flow/time, the third is volume/time and the fourth (bottom) is Edi/time. Note that the Edi signal from the diaphragm and the pressure waveform from the ventilator do not correspond. This is an example of patient-ventilator asynchrony (See text for further description).

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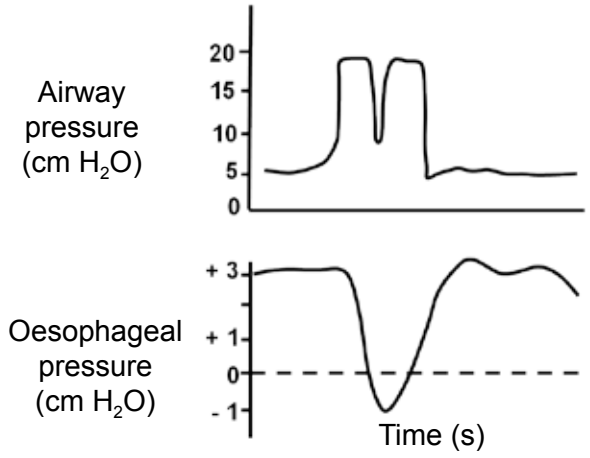

Figure 4A: An illustration of an airway pressure/time waveform and an oesophageal pressure/time waveform during a double triggering event (See text for description). The decrease in oesophageal pressure (patient effort) occurs after the first mandatory breath begins. The rise in oesophageal pressure indicates the beginning of patient exhalation, at the same time that a second mandatory breath is being given.

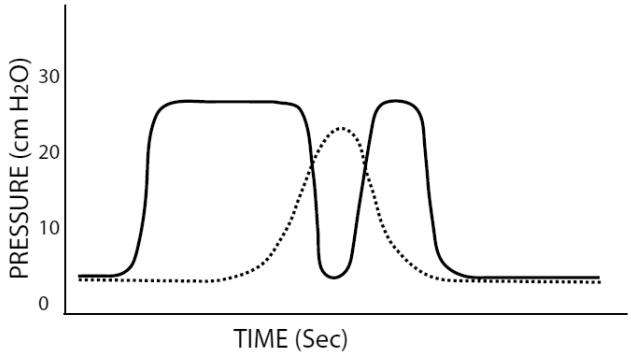

Figure 4B: The solid line represents airway pressure delivery. The dashed line represents estimated pressure delivery from the Edi. As with 4A, the patient effort (rise in diaphragm activity) begins after the ventilator has already started a breath. Patient effort is ending and the ventilator is delivering another mandatory breath.

are administered following extubation. For example, the Edi can monitor an infant's response to use of nasal CPAP or a high-flow nasal cannula.[17]

Patient-ventilator asynchrony

Patient-ventilator asynchrony (PVA) is a common problem in mechanically ventilated patients. Studies have demonstrated that an estimated 25 percent of patients receiving mechanical ventilation, regardless of the brand of ventilator in use or the mode employed, have PVA.[18-21]

PVA is associated with a number of potential complications including diaphragm atrophy and diaphragm dysfunction. These can occur in as short a time as one to two days in the ventilated patient.[22-25] A common practice is to sedate patients who are 'fighting the ventilator'. However, the use of sedatives can reduce diaphragm activity which increases the risk of diaphragm atrophy. Use of sedation also can increase cost and length of stay.[26-27]

In addition to trigger and cycle asynchrony, another form of asynchrony is called double triggering. It is thought by some practitioners that when double triggering occurs it is because the ventilator is unable to provide the amount of gas flow demanded by the patient.[28,29] However, use of oesophageal pressure monitoring and Edi monitoring have elucidated what is actually occurring. (*Figures 4 A* and B) In *figure 4A*, the ventilator triggers the breath based on time (rate setting) but the patient's inspiratory effort occurs during breath delivery from the ventilator. The ventilator ends the first breath, detects the patient effort and gives a second breath. But the second breath occurs when the patient is trying to exhale. The phenomena of double trigger can occur regardless of the type of ventilator being used.[28] *Figure 4B* illustrates the same phenomena, using a pressure/time and an Edi/time waveform shown together. During double triggering the ventilator is not coordinated with what the patient's respiratory centre wants.

Other types of asynchrony that may be evaluated include wasted efforts, where the patient wants a breath and does not receive one from the ventilator. This is a common form of asynchrony. The neural rate is different from the ventilator delivery rate. Another type of asynchrony occurs when the amount of assist provided, either as pressure or volume is more or less than the patient actually desires. The level of assist does not correspond to the variable drive of the patient.

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Neurally Adjusted Ventilatory Assist (NAVA)

In addition to its use as a monitoring tool, the Edi signal can also be used to control the ventilator. As defined earlier, the mode governed by the Edi signal is referred to as NAVA. With NAVA, the patient is actually controlling the trigger, delivery and cycling of every breath. The ventilator becomes completely synchronous with the patient avoiding the undesirable outcomes of patient-ventilator asynchrony. Since the Edi is a reflection of neural respiratory output, ventilator breath delivery is in synchrony with neural control of ventilation.[1] The clinician no longer has to judge appropriate settings for trigger sensitivity, flow delivery, volume or pressure delivery and inspiratory time. Improved synchrony may provide better outcomes for patients. It has also been shown, for example, to be more comfortable.[30]

NAVA was released for clinical use in Europe in late 2006 (Maquet, Inc; Solna, Sweden). It was approved by the United States Food and Drug Administration for use in the U.S. in early 2007. Since this is relatively new mode, research regarding its use and function is somewhat limited.

The Puritan Bennett 840 ventilator has a similar mode available that is referred to as proportional assist ventilation (PAV). In comparing NAVA and PAV, both ventilators deliver an inspiratory breath that is proportionate to the patient's demand.[31] The primary difference between the two is that PAV uses a pneumatic signal for trigger. It uses measurements and calculations to estimate the amount of breath delivery desired by the patient. PAV does not require an invasive nasogastric tube to function. NAVA does require nasogastric or orogastric tube placement and this may be contraindicated in some patients.[32] For example placement of a nasogastric or orogastric tube may have relative contraindications in patients with facial or head injury or in patients with oesophageal varices or similar problems.

Both PAV and NAVA are only functional in spontaneously breathing patients. While the Edi catheter can be used in monitoring most types of patients, NAVA can only be used in patients who have an Edi signal.[1]

The following is a list of patients who are not eligible for NAVA:

- Patients with a brain injury or tumours affecting the respiratory centre
- Patients who are paralysed or heavily sedated
- Patients in whom both branches of the phrenic nerve have been damaged or are otherwise not functional
- Patients with certain neuromuscular disorders
- The presence of apnoea for whatever reason

There is an exception to the presence of apnoea. In premature infants who experience periods of apnoea, NAVA has a backup mode of ventilation that allows its use in this patient population. Recent software upgrades also reduce the occurrence of nuisance alarms when NAVA is used either invasively or noninvasively in premature infants.[15]

Using NAVA

With the Edi catheter correctly positioned, the catheter is connected to the ventilator by means of a cable that attaches at the other end to a module located on the side of the ventilator *(Figure 1)*. Once connected, the operator can open the 'Edi Catheter positioning' screen by touching the Neural Access key. As shown in *Figure 2* and described earlier, this screen can be used to help determine the correct depth of the catheter. In clinical practice the positioning screen is checked on a regular basis to confirm that the catheter has not moved during use. From the ventilator's mode screen the operator can then select the NAVA mode.

The ventilator parameters that can be adjusted by the operator in NAVA include the FiO_2, PEEP, trigger sensitivity (Trigg Edi), and the NAVA level which is similar to setting a pressure support level. The NAVA trigger begins delivery of inspiration when the Edi is 0.5 μV above the Edi min of the previous breath. The Trigg Edi is adjustable from 0-2 μV. Trigger synchrony can be achieved with NAVA compared to other modes where trigger asynchrony

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may occur.[33] There is also a backup pneumatic trigger if the ventilator is unable to detect the Edi signal.

The NAVA level can be adjusted from 0-15 μV/cm H_2O. A typical range for most patients is about 0.5-3 μV/cm H_2O. The cycling or end of inspiratory flow delivery with NAVA occurs when the Edi decreases to 70 percent of the Edi peak. The decrease in Edi occurs as the patient's inspiratory phase is ending and exhalation is beginning.

There is a screen available on the ventilator called the "NAVA preview" screen, which can help estimate an initial NAVA setting for the patient. What the NAVA preview allows is for the operator to adjust the starting NAVA level so that the estimated pressure delivery of a NAVA breath (P_{est}) will approximate the delivery pressure of the current mode. The Ppeak est is based on the following equation:

$$\text{NAVA Ppeak}_{est} = \text{NAVA level x (Edi peak} - \text{Edi min)} + \text{PEEP}$$

As shown in the equation the pressure delivery in NAVA is based on the Edi signal strength. P_{est} is the pressure from the Edi that is superimposed over the airway pressure as seen in *Figure 3*. As in pressure support, the higher the NAVA level, the higher the potential pressure delivery. Edi intensity varies both between breaths and within a breath and delivered pressure follows suit.

The pressure varies constantly during the breath delivery as the Edi varies (*i.e.*, it varies in proportion to changes in the Edi and the neural demand for a breath).[34-36] As an example, suppose the NAVA level is set at 0.5 cm H_2O/μV, the Edi peak is 11 μV and the Edi min is 1 μV and the PEEP is 5 cm H_2O, the Ppeak est delivered will be equal to 0.5 cm H_2O/μV x (10 μV × 1 μV) + 5 cm H_2O = 9.5 cm H_2O.

The actual pressure delivery has a physiological safety back up in the form of a negative feedback loop. Suppose the ventilator is ready to target a $Ppeak_{est}$ of 10 cm H_2O, but during delivery of the breath the lungs expand and the pulmonary stretch receptors are excessively stretched (Hering-Breuer Reflex). Neural impulses are sent from the receptors through the vagus nerve to the neural respiratory centre. The respiratory centre then suppresses the signal transmission through the phrenic nerve and the electrical activity of the diaphragm decreases or ceases. The ventilator rapidly senses the decrease in Edi and reduces pressure delivery within the same breath.[37]

An important factor in both the initial setting of the NAVA level and the adjustment of NAVA is the patient's response to the mode. Clinicians should carefully assess their patient for changes in work of breathing and level of comfort in addition to the normally monitored vital signs and ventilator parameters. The NAVA level is increased to provide more ventilator support. Generally, the Edi will tend to decrease as support is increased. Conversely, as the NAVA level is decreased, the Edi will generally increase but again this can vary among patients. Not all patients respond the same.[38] Responses vary widely depending on the patient's medical problems. For example, a patient with a cardiac condition and Cheyne-Stokes respiration will have a fluctuation in Edi from zero to much higher values. In this case the Edi might actually help identify the abnormal respiratory pattern. A patient with metabolic acidosis will tend to have a high respiratory drive and Edi might remain high after slight increases in the NAVA level.

Backup ventilation modes for NAVA

In the 'Set Ventilation Mode' window that displays the parameters to set NAVA parameters, there are also displayed two backup modes. One is pressure support and the other is Backup ventilation (pressure control mode). Ventilation parameters for these two modes can also be set from this screen.

If the Edi signal is lost, as might occur if the catheter is disconnected or pulled out or moves within the oesophagus, the ventilator will alarm and provide a warning that the catheter position needs to be checked. At the same time the ventilator will switch to pressure support [NAVA(PS)] and provide

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the pressure and cycle criteria selected during setup. NAVA(PS) will also be activated if the pneumatic signal and Edi are not synchronised. (Note: a few additional criteria will initiate NAVA(PS). This information is available in the manufacturer's literature).[9]

If the patient becomes apnoeic, the ventilator switches to the backup mode which is pressure control mode (PC). The operator can set the pressure, rate and inspiratory time (or I:E ratio) for backup ventilation.

In addition to providing backup ventilation with either PS or PC, the operator can also set the usual available ventilator alarms such as upper pressure limit, high and low minute volume, high and low rate alarm, and high and low PEEP.

Comparing NAVA to traditional modes of ventilation

When compared to traditional modes, the use of NAVA tends to result in lower peak pressures, lower tidal volumes, higher rates, similar minute ventilation values, lower FiO_2 requirements, lower compliance and similar mean airway pressures. These results will vary from one population to another, for example from neonates to adults. Results can also vary based on the underlying pathology.[15,39,40] A study of neonatal patients showed that blood gases appear to normalise when patients are ventilated with NAVA.[15] In addition, patients establish their own transpulmonary pressures.[1] In addition, NAVA allows patients' respiratory centres to maintain their normal biological rhythm compared to pressure support ventilation.[41]

Another potential advantage of the NAVA is that it is not affected by leaks. In pneumatically triggered systems or modes, the ventilator relies on measuring a change in flow or pressure to identify a patient's inspiratory effort. The presence of a leak in either invasive or noninvasive ventilation can interfere with flow and pressure change detection. Leaks can also lead to patient-ventilator asynchrony. Since NAVA uses the physiological signal from the diaphragm's electrical activity, leaks do not affect the ventilator's ability to identify the Edi trigger. NAVA can be used either with invasive or noninvasive interfaces.

Summary

The use of NAVA and the Edi catheter introduces a novel approach to mechanical ventilation. Rather than the operator, it is the patient who controls breath rate, tidal volume, flow and even occurrence of sigh breaths. NAVA has been shown to be a safe mode of ventilation in various patient populations from adults to premature infants.[15,42] It is tempting to speculate that the use of the Edi as a monitor and NAVA as a mode might reduce need for sedation and require less time on the ventilator. However, additional studies are needed to substantiate improved outcomes with NAVA.

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DISCLOSURE: The author has worked full-time for Maquet, Inc. and now works for them as a consultant. Maquet is the exclusive manufacturer and distributor of the Edi catheter, NAVA ventilation and the SERVOi ventilator.