Independent Lung Ventilation

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Independent ventilation of each lung in trauma patients is a recently developed technique that specifically addresses the asymmetric nature of certain pathologic lung conditions. The most common conditions in which experience has been accumulated in the trauma patient are unilateral pulmonary contusion,[1] aspiration pneumonia,[2-4] refractory atelectasis,[5,6] and unilateral or asymmetric adult respiratory distress syndrome (ARDS)[7] (see box below). The purposes of this article are to review the principles behind independent lung ventilation (ILV), describe how it is practiced at our institution, and, where appropriate, reference other work in the field.

> **Conditions in which ILV has been used**
> Unilateral lung disease
> - Pulmonary contusion
> - Refractory atelectasis
> - Aspiration pneumonia
> - Bronchopleural fistula and massive air leak
> - Unilateral ARDS
>
> Bilateral asymmetric lung disease
> - ARDS with differential severity
> - Bilateral pulmonary contusions of differential severity

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Indications for ILV

An appreciation of the shortcomings of conventional mechanical ventilation (CMV) provides the physiologic rationale for ILV, also known as differential lung ventilation and simultaneous independent lung ventilation (SILV).

With the exception of bronchopleural fistula, the conditions under treatment have several critical effects in common. All lead to an increase in local interstitial lung fluid, resulting in fluid-filled or atelectatic alveoli. This in turn manifests as decreased compliance, or "stiff lung," and decreased functional residual capacity, the volume remaining in the alveoli at the end of tidal expiration. Because airway resistance may be increased as a result of local edema and inflammation, the time constant for ventilation (airway resistance × compliance) is variable in different regions of the lung. Nonventilated perfused alveoli, low ventilation-perfusion ratio, ($\dot{V}/\dot{Q}$), manifest as right-to-left shunt, causing arterial hypoxemia. The phenomenon of hypoxemic pulmonary vasoconstriction shifts perfusion to better ventilated areas, partially reversing shunt and decreasing the resultant hypoxemia.

The traditional and well-proved[8] treatment for this problem is to maintain increasing levels of positive pressure during the end-expiratory period. When this pressure exceeds the opening pressure of collapsed alveoli, reinflation occurs. $\dot{V}/\dot{Q}$ matching improves and shunt decreases. The lung's elastic properties are improved, compliance increases, and ventilation is easier. This method is quite effective but can fail and can even be deleterious when highly asymmetric lung pathology is encountered.[3,9-13]

Consider, for example, a patient with unilateral severe pulmonary contusion. The injured lung is characterized by poor compliance (prolonged time constant) and a low $\dot{V}/\dot{Q}$ (increased shunt). The healthy lung has a normal compliance (normal time constant) and a relatively equal $\dot{V}/\dot{Q}$. The CMV tidal volumes will divert preferentially to the healthy lung because of its shorter time constant and greater compliance. Similarly, positive end-expiratory pressure (PEEP), applied to treat the injured lung, will exert most of its effect against the more compliant, healthy lung! Thus, the healthy lung serves to "decompress" tidal volume and therapeutic PEEP away from its intended target, the less compliant, injured lung. In one study, applying freely distributed PEEP to patients with unilateral pulmonary contusion resulted in a volume increase of only 35% in the contused lung and of 65% in the noncontused lung.[1]

The result of this process is illustrated in Fig. 1. The alveoli in the compliant lung are overdistended and transmit much of the distending pressure to their local pulmonary capillaries, thereby increasing effective capillary resistance (Starling's resistance effect). If alveolar distending pressure exceeds local pulmonary capillary pressure, that capillary collapses. This effect tends to divert pulmonary blood flow away from the healthy, well-ventilated lung and back toward the injured, poorly ventilated side, working against the compensatory efforts of the hypoxemic vasoconstrictive mechanism. $\dot{V}/\dot{Q}$ increases on the healthy, previously balanced side, resulting in increased dead space. This effect further decreases an already low $\dot{V}/\dot{Q}$ on the injured side, thereby increasing shunt and worsening arterial hypoxemia. In the patient with sepsis, the hypoxemic pulmonary vasoconstriction reflex is often abolished, thus compounding the $\dot{V}/\dot{Q}$ mismatch.

In such unilateral conditions, ILV can allow better matching of mechanical ventilation characteristics to the specific compliances and resistances of each lung. This approach allows more effective application of therapeutic modalities toward the pathologic lung and prevents exacerbation of $\dot{V}/\dot{Q}$ inequality caused by maldistribution of tidal volumes and PEEP to the healthy lung.[3,7,9,14]

In one study in which freely distributed PEEP was applied for lobar bacterial pneumonia, the pneumonic lung experienced increased ventilation of alveoli but also had a 70% increase in perfusion and therefore no improvement in shunt or oxygenation! The extra perfusion had been shifted from the good lung as a result of the effects of the high PEEP there.[18] However, in dogs with left lower lobe pneumonia, administration of PEEP to only the pneumonic lung resulted in a significant decrease in shunt.[20]

When to Start ILV

ILV is indicated for any patient having poor results on CMV with PEEP who can be demonstrated to have an asymmetric lung condition. Though the objective determination of the presence of asymmetric disease is feasible, as described below, the specific indications to start ILV may remain unclear.

When is a patient considered to be doing poorly with conventional ventilation? Essentially, progressive, uncorrectable shunt and hypoxemia are the major criteria. No values have been universally agreed on in previous work, but in general, a shunt fraction greater than 30% and progressing or arterial oxygen saturation (Sa_{O_2}) less than 90% on fraction of inspired oxygen (F_{IO_2}) greater than 50% are sufficient. Some groups have used the Pa_{O_2}/F_{IO_2} ratio as a sensitive indicator of shunt. A Pa_{O_2}/F_{IO_2} of less than 180 to 200 indicates severity of disease sufficient to warrant consideration of ILV if the nature of the condition is appropriate. This principle should be qualified by the fact that in certain conditions patients may not be doing so poorly, but ILV may be appropriate nonetheless (see below). For example, some evidence indicates that ILV may play a role in speeding closure of bronchopleural fistulas, beyond the actual maintenance of the patient through the healing period. Acting as a purely mechanical agent, ILV may be helpful in expanding atelectatic lobes.

In which cases of asymmetric lung disease will ILV be beneficial? If the mechanical disparity between the two lungs is so great that the CMV volumes and PEEP necessary to treat the "bad" lung will be deleterious to the function of the good lung, then ILV may be the answer. The following clinical steps can be used when ILV is being considered.

The clinician should be alerted to the possibility of asymmetric lung pathology by one of two means:

1. The chest roentgenogram may show evidence of unilateral pulmonary contusion, pneumonitis, atelectasis, or other disorder and a relatively clear contralateral lung field. Mediastinal shift caused by loss of volume in one lung may also be present.
2. Less obviously, while applying gradually increasing PEEP in small increments during conventional ventilation, a sudden, disproportionate drop in Pa_{O_2} may occur, indicating increased shunt fraction. This situation is most likely caused by overdistension of the more compliant normal lung, with shift of perfusion to the pathologic lung, as described above.

A compliance curve may then be plotted, with plateau pressure as the abscissa and tidal volume as the ordinate. This curve may show two distinct slopes, indicating the response of large areas of differing compliance and hinting that asym-

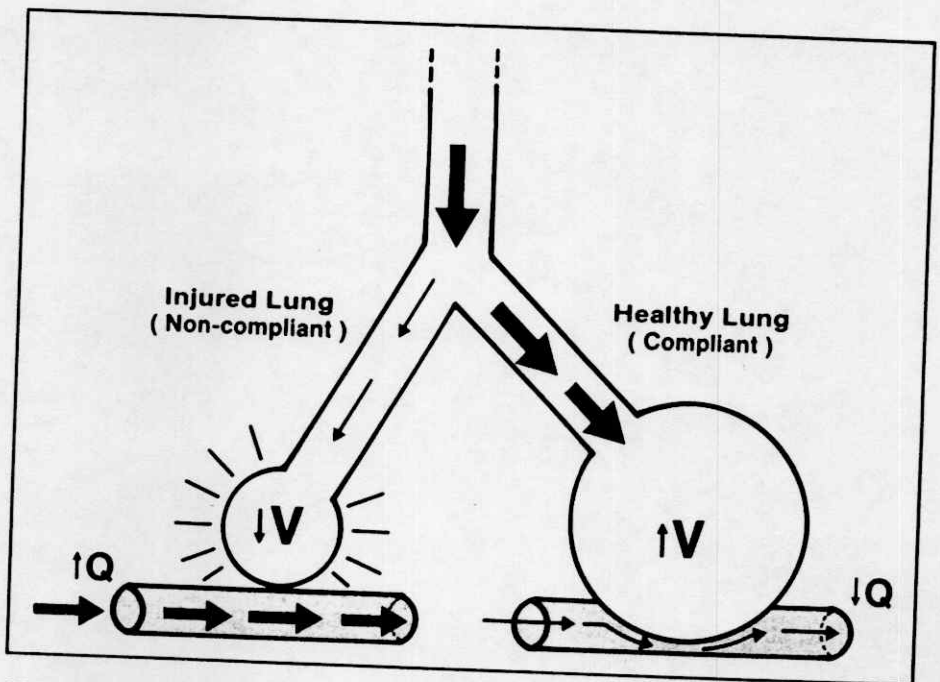

Fig. 1. *Effect of conventional mechanical ventilation on asymmetric lung disease. Large tidal volumes and PEEP are preferentially distributed to healthy, compliant lung at expense of injured noncompliant lung. This in turn extrinsically increases pulmonary vascular resistance on healthy side, shifting pulmonary blood flow to poorly ventilated, injured side. Result is increased $\dot{V}/\dot{Q}$ mismatch with overventilation of healthy lung and overperfusion of injured lung. Shunt and hypoxemia may be exacerbated.*

metric disease may be present. At the MIEMSS Shock Trauma Center, the Servo 900C ventilator (Siemens-Elema, Schaumburg, Ill.) interfaces directly with the critical care computer system, providing continuous volume and pressure data to allow the rapid generation of compliance curves. In the absence of such a system, accurate compliance curves can be created by noting pressures as tidal volume is increased in 100-ml increments.

If any of the above suggestive evidence is present, turning the patient onto each lateral decubitus position is indicated for assessing this position's effect on shunt and dead space. The lateral decubitus position shifts perfusion toward the dependent side (zone III) and ventilation toward the nondependent side (zone I). When a patient with "equally bad lungs" (symmetric low $\dot{V}/\dot{Q}$) is placed in either lateral decubitus position, no or minimal change in shunt fraction or PaO_2 should occur because exacerbation of low $\dot{V}/\dot{Q}$ on the down side (decreased ventilation, increased perfusion) is compensated for by improvement of low $\dot{V}/\dot{Q}$ (increased ventilation, decreased perfusion) on the up side. In asymmetric disease, when the bad lung is placed down, its already low $\dot{V}/\dot{Q}$ is further exacerbated by the dependent increase in perfusion, while the high $\dot{V}/\dot{Q}$ in the better lung is worsened by the positional increase in ventilation. Thus, shunt fraction and dead space will increase. If these are the findings, then a trial of ILV is warranted.

Technique

Starting ILV

The first step in ILV is tracheobronchial intubation with a double-lumen endobronchial tube and confirmation of the presence of asymmetric lung pathology. An anesthesiologist who is experienced in this technique should place an appropriately sized left-angled endobronchial tube (Broncho-cath; NCC Division, Mallinckrodt Inc., Argyle, N.Y.). This catheter has two balloons. The "tracheal" balloon seals the distal trachea from the environment and is similar to the balloon on a conventional endotracheal tube. This balloon should be placed in the trachea proximal to the carina. The "bronchial" balloon isolates ventilation to one lung from ventilation to the other. This balloon should be placed in the left mainstem bronchus just distal to the carina. Thus, the proximal lumen, located between the two balloons, will supply the right lung and the distal, or terminal, lumen will supply the left lung. Transcutaneous O_2 or pulse oximetry and end-tidal CO_2 should be monitored during conversion from an endotracheal tube to a double-lumen tube. Monitoring is especially important because severe hypoxemia or hypercarbia may occur when tidal volumes and PEEP are lost during the conversion or if the catheter is seated improperly. Both balloons are inflated and the position is checked by ventilating one lung at a time while auscultating breath sounds. This maneuver gives an initial indication of the tube's position and confirms that each lung is indeed being ventilated. The position of the tube is then checked by roentgenography.

Another technique may be used to confirm complete isolation of both lungs.[10] Each lung may be ventilated independently with a self-reinflating (Ambu) bag while a small balloon is connected over the Broncho-cath lumen to the contralateral lung. Slight inflation and deflation of the balloon with contralateral ventilation is normal. Any continuous increase in the size of the balloon indicates that the lungs are incompletely isolated and that leakage is occurring. In this case the tube should be repositioned until leakage does not occur.

Next, the Broncho-cath lumens are each connected to a ventilator. At our center, we use Siemens 900C Servo ventilators (SV-900C) because of their flexibility with different modes of ventilation and because they include ILV synchronizing circuits (Fig. 2).

For final confirmation of asymmetric lung pathology appropriate for ILV, the previous CMV settings are continued unchanged and half the tidal volume is administered by each ventilator to its respective lung. For example, if the patient had been maintained with CMV so that tidal volume = 800 ml, rate = 12 breaths per minute, PEEP = 16 cm H_2O, and FiO_2 = 0.5, then each lung on ILV would initially receive a tidal volume of 400 ml still at a PEEP of 16 cm H_2O, rate of 12 breaths per minute, and FiO_2 of 0.5. Initial static compliance is measured. Compliance curves are then plotted for each lung individually by using pressure and volume data taken from the appropriate ventilator as tidal volume is gradually increased (Fig. 3). Significantly different initial compliances and differing compliance curves, indicating different optimal tidal volumes, confirm that "differential ventilation" of each lung should be attempted.

Conducting ILV

The following decisions need to be made before starting ILV:

1. Should synchronous or asynchronous ILV be used?
2. Which mode of ventilation is best for each lung, and how will the optimal ventilatory parameters be determined?
3. How will the effectiveness and safety of treatment be monitored?
4. How, and by what criteria, will ILV be discontinued?

These questions are still unresolved among groups working with ILV, but some experience has accumulated.

The major theoretic objection to asynchronous ILV is that, because no true expiratory phase is involved, venous return and cardiac output may be compromised. Indeed, the mediastinum can be noted on fluoroscopy to "swing" during asynchronous ILV (AILV).

However, no studies have demonstrated cardiac compromise with AILV, and many patients have had successful outcomes with this method of support with no evidence of cardiovascular depression.[15,16] Animal studies have indicated no difference in gas exchange or hemodynamics between dogs ventilated with AILV or SILV.

The major objection to SILV is that it does not allow as much flexibility in adjusting the ventilator in response to each lung's needs. In reality, the only constraint in SILV is that both lungs begin inspiration at the same time. With the Siemens Servo 900C ventilators, tidal volume, PEEP, pause time, inspiratory time, and F_{IO_2} may still be selected independently for each lung.

Another objection is that SILV requires expensive, complicated synchronizing equipment that can fail. Though this may have been the case several years ago, modern ventilators, including the SV-900C, can function in a synchronized manner using inexpensive, commercially available cables. With the SV-900C, one ventilator acts as "master," initiating ventilation for itself and for the "slave" ventilator. Other than this, all parameters can be set individually on each machine. At the MIEMSS Shock Trauma Center we use synchronized ILV.

For completeness, the box below lists the different modes of ventilation that have been used with ILV. Any permutations of these are possible, but, for the most part, volume control (VC) or pressure control (PC) is used. PC is a mode of ventilation in which the tidal volume delivered is determined by the lung compliance and airway resistance and is limited by a maximum peak airway pressure as set on the ventilator. Inspiratory waveform is characterized by a decreasing (asymptotic) flow as the preset pressure limit is reached. PC can often deliver a given volume at lower airway pressures than can VC. Inability to obtain adequate ventilation with reasonable airway pressures on VC at the point of optimal compliance may be an indication to switch to the pressure control mode. Administering VC to the healthy lung and VC or PC to the injured lung is common.

As with most critically ill patients maintained with high ventilatory support in a "control" mode, our patients maintained with ILV are pharmacologically paralyzed with a neuromuscular blocking agent. We do this to prevent any spontaneous respiratory efforts that might hamper machine ventilation. Concomitant with neuromuscular blockade, the patients are sedated for comfort with a continuous infusion of either a narcotic, if they are believed to be in pain, or a benzodiazepine.

After commencing ILV as indicated, with equal VC ventilation to each lung, compliance curves are constructed as tidal volumes are varied in 100-ml increments (Fig. 3). The point of maximum compli-

Ventilatory modes used with ILV

- Healthy lung
  - VC + PEEP
  - PC + PEEP
- Injured Lung
  - VC + PEEP
  - PC + PEEP
- High-frequency jet ventilation
- Continuous positive airway pressure
- Apneic air insufflation

VC, Volume control ventilation; PC, pressure control ventilation; PEEP, positive end-expiratory pressure (see text).

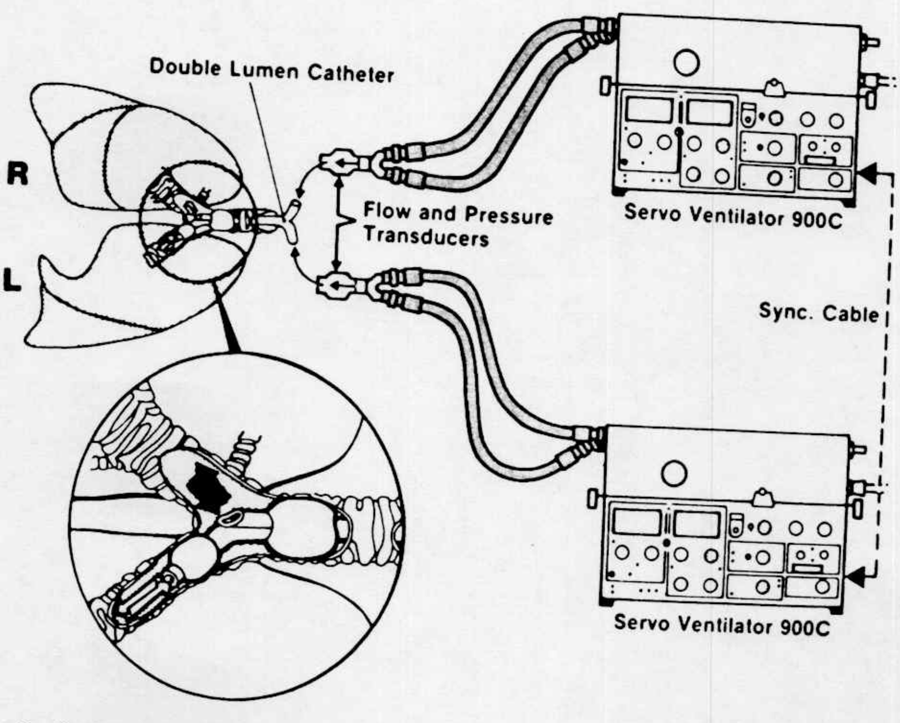

Fig. 2. *System used for independent lung ventilation at MIEMSS Shock Trauma Center. Patient is intubated with a double-lumen endobronchial catheter. Separate Siemens 900C Servo ventilators supply each lung and are synchronized through a cable available from manufacturer. Flow and pressure information is available from in-line transducers. Through appropriate analog-to-digital converter and computer interface, the 900C will, in future, provide information directly to critical care computer system. (From Siegel JH, Stoklasa JC, Borg U, et al. Ann Surg 1985;202:425-39.)*

ance represents the optimal tidal volume for that lung. Incremental changes ideally should be followed by observing trends in pulse oximetric oxygen saturation and end-tidal CO_2 to confirm the maintenance of adequate oxygenation and ventilation, respectively. Frequent arterial blood gas levels should be checked as the optimal tidal volume is sought.

Similarly, initial optimal PEEP is selected by assessing compliance as PEEP is increased for each lung. Oxygen saturation and end-tidal CO_2 are monitored for trending. A decrease in end-tidal CO_2 may indicate overdistension of alveoli with decreased perfusion and increased dead space. The inflection point on the compliance curve, or point of sudden increase in compliance, represents the initial optimal PEEP for that lung. This value is believed to coincide with the point at which critical opening pressure is reached and recruitment of atelectatic alveoli occurs. The use of different levels of PEEP for each lung, as appropriate, is referred to as selective PEEP.

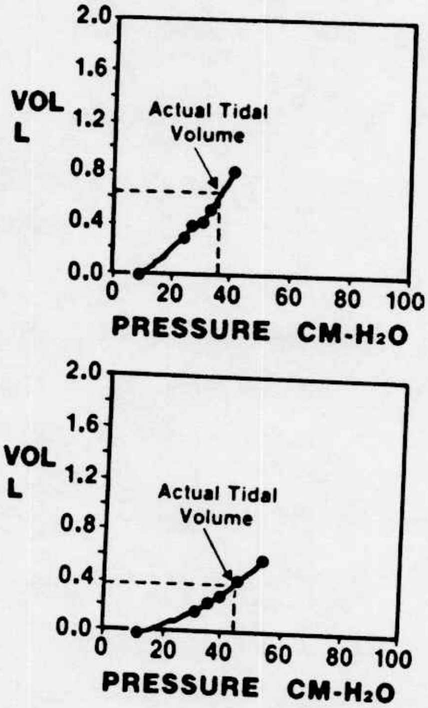

Fig. 3. *Differing compliance curves for right (top box) and left (bottom box) lungs after endobronchial intubation. Specific tidal volumes for maximal compliance for each lung are indicated.* FIO_2 = 0.50; PaO_2 = 110 mm Hg; $PaCO_2$ = 38 mm Hg; pHa (arterial pH) = 7.41; C.O. (cardiac output) = 6.8 L/min; $\dot{Q}_s/\dot{Q}_t$ = 25%. *(Respiratory function consultation, MIEMSS, University of Maryland.) (From Siegel JH, Stoaklosa JC, Borg U, et al. Ann Surg 1985;202:425-39.)*

Some of the parameters monitored on ILV are listed in the box at right. Arterial blood gases are, of course, monitored frequently and pulse oximetry can provide moment-to-moment information on the adequacy of oxygen saturation. Static compliance, which is the parameter used to guide initial ventilatory parameters, is monitored throughout the course of ILV.

By far, the most relevant parameters in judging the therapeutic effect of ILV are shunt fraction ($\dot{Q}_s/\dot{Q}_t$) and alveolar dead space (V_d/V_t). Because the ultimate goal of ILV is to better match ventilation to perfusion by the selective application of tidal volume and PEEP, shunt and dead space should decrease if the treatment is effective. Consequently, these values are measured frequently—and certainly after every ventilator change.

Continuous individual lung end-tidal CO_2 measurements are also most useful. Overdistension of alveoli will cause decreased perfusion and increased dead space in that lung. This situation may manifest by a sudden decrease in individual end-tidal expiration CO_2 content ($ETCO_2$) as ventilation is adjusted and should raise the possibility that overdistension is occurring in that lung. Trew et al.[18] believe that $ETCO_2$ is the most important parameter to monitor. They select the ventilation that produces equal $ETCO_2$ concentrations from both lungs, which they believe gives the best therapeutic results in terms of increased PaO_2 and decreased shunt. We do not follow this practice although we monitor $ETCO_2$ routinely.

Worsening of oxygenation, shunt, or dead space is an indication to check the chest roentgenogram for status of pulmonary pathology and to recalculate optimal compliance curves, if necessary.

To ensure optimal cardiac function, filling pressures (central venous pressure and pulmonary artery occlusion pressure) and hemodynamics (cardiac output, stroke index, systemic vascular resistance index, and pulmonary vascular resistance index) are measured routinely. Because the ultimate goal of all ventilatory support is optimal delivery of oxygen to peripheral tissues, indexes of peripheral oxygen availability (O_2 delivery, O_2 con-

Parameters monitored during ILV

Respiratory parameters

- ABG (Arterial blood gas)
- SaO_2 (Oxygen saturation of arterial blood)
- Ċst (Static compliance)
- $\dot{Q}s/\dot{Q}t$ (Venoarterial admixture ["shunt fraction"])
- $ETCO_2$ (End-tidal expiration CO_2 content)
- V_d/V_t (Dead space fraction of tidal volume)

Cardiac filling pressure and hemodynamics

- CVP (Central venous pressure)
- PAOP (Pulmonary artery occlusion pressure)
- CI (Cardiac index)
- SI (Stroke index)
- SVRI (Systemic vascular resistance index)
- PVRI (Pulmonary vascular resistance index)

Peripheral oxygen availability

- CaO_2 (Arterial oxygen content)
- O_2 del (Oxygen delivery [CI × CaO_2])
- VO_2I (Oxygen consumption index)
- A-VDO_2 (Arteriovenous oxygen content difference)

sumption index, and arteriovenous O_2 difference) are determined routinely and maximized.

Discontinuing ILV

The criteria for discontinuing ILV are the least firmly established aspect of the technique. The first requirement is that the mechanical differences between the two lungs have minimized to the point where some type of conventional ventilatory support would allow adequate function of the worse lung without having untoward effects on the better lung. This requirement usually translates to minimal differences in compliance and PEEP requirements. The total static lung compliance is the sum of the left and right static compliances. This indication that the asymmetric nature of the disease is resolving is probably the most compelling reason to discontinue ILV. The second requirement for stopping ILV is that respiratory function should have improved to the point where oxygenation is adequate on reasonable levels of PEEP and nontoxic F_{IO_2} (0.5 or less). These requirements are opposite sides of the same coin and, as indicators of improving disease, often occur at or about the same time. One group's criteria for weaning from ILV are shown in the box, below left.

Among a group of eight patients maintained with ILV recently studied at the MIEMSS Shock Trauma Center,[7] the time of ILV use ranged from 46 to 91 hours, with a mean of 66.75 hours. Before attempted termination of ILV, if possible bring the high levels of PEEP and tidal volume of the worse lung down to the levels of the better lung while observing the effect on oxygenation and compliance. Then, for trial termination of ILV, a single ventilator is set for a tidal volume and PEEP midway between the values most recently being used on ILV. The double-lumen bronchial catheter is left in place, and the single ventilator is connected to both lumens via a Y connector. This arrangement allows a trial of "whole lung" ventilation while preserving the option of returning easily to ILV. All respiratory parameters are followed continually, including Pa_{O_2}, Sa_{O_2}, shunt fraction, dead space, and ET_{CO_2}. If no deterioration occurs after several hours, the Broncho-cath is replaced with a conventional single-lumen endotracheal tube and conventional ventilation is continued.

A flow chart for the conduct of ILV is shown in Fig. 4.

Criteria for weaning from ILV

- Difference in differential PEEP less than 5 cm H_2O
- Difference in differential compliance less than 10 ml/cm H_2O
- Stable Pa_{O_2} on equalizing SPEEP (Pa_{O_2}/F_{IO_2} greater than 300)
- Disappearing asymmetry on chest x-ray film
- $V_{E\,left} + V_{E\,right}$ less than 12 L/min
- Difference in differential Raw_{insp} less than 3 cm H_2O/L/sec

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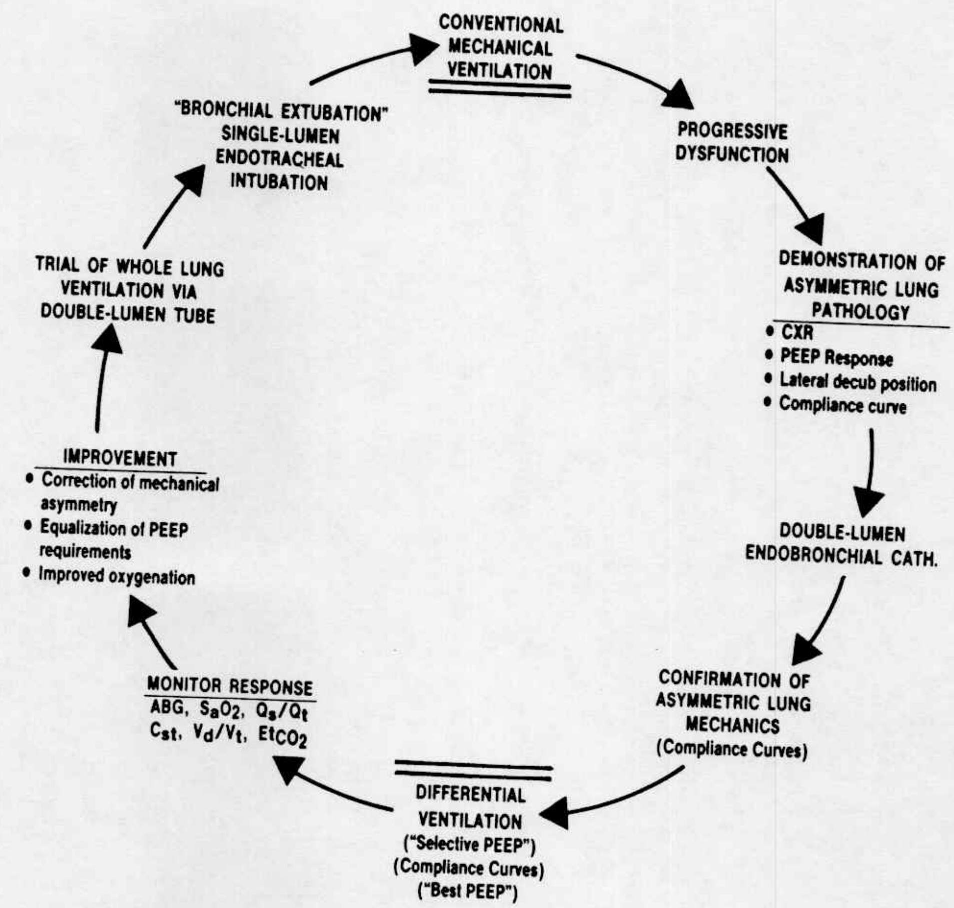

Fig. 4. *Conceptualization of initiation, conduct, and termination of independent lung ventilation. CXR, Chest x-ray; ABG, arterial blood gases; Q_s/Q_t, intrapulmonary shunt fraction; V_d/V_t, dead space to tidal volume ratio.*

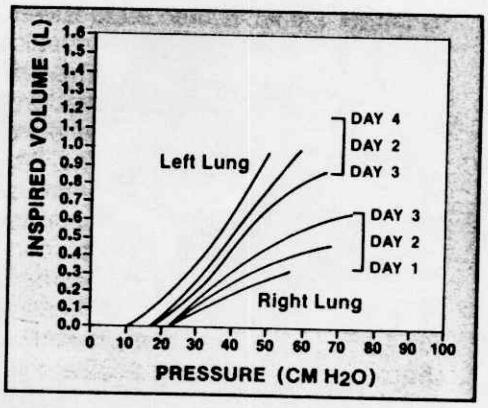

Fig. 5. *Changes in static compliance during course of SILV treatment in one patient. (From Siegel JH, Stoklosa JC, Borg U, et al. Ann Surg 1985;202:425-39.)*

Table I. *Response of patient to SILV therapy*

	Before ILV	ILV			
		Day 1	Day 2	Day 3	Day 4
FIO_2	1.0	1.0	0.70	0.60	0.50
PaO_2	59	71	84	102	81
$\dot{Q}s/\dot{Q}t$	47	40	27	21	31
Cst (right)	—	0.009	0.012	0.015	0.015
Cst (left)	—	0.022	0.023	0.021	0.024
Cst (R + L)	0.023	0.031	0.035	0.036	0.039

From Siegel JH, Stoklosa JC, Borg U, et al. Ann Surg 1985;4:425-39.
$\dot{Q}s/\dot{Q}t$, Shunt fraction; *Cst*, static compliance

Example of ILV

An 18-year-old male motorcyclist was admitted to the Shock Trauma Center of MIEMSS after being struck by a motor vehicle. He had sustained a closed-head injury with an occipital lobe contusion, a myocardial contusion, a right pulmonary contusion, and a fractured right humerus. He was maintained initially with conventional mechanical ventilation, but on the fourth hospital day he had a deterioration in shunt fraction from 28% to 47% and a decrease in static compliance from 0.047 to 0.023 L/cm H_2O. Bleeding continued from the right lung, with further opacification of the chest roentgenogram indicating asymmetric disease. For this reason, and to prevent entrance of blood into the healthy left lung, endobronchial intubation was performed and the patient began ILV.

Initially, each lung was ventilated with a tidal volume equal to one half that with which the patient had most recently been maintained on conventional ventilation and a PEEP of 20 cm H_2O. Compliance curves were then constructed to determine the optimal tidal volumes and level of PEEP for each lung. The resultant curves (similar to those in Fig. 3) indicated significantly different compliance properties for each lung. The injured right lung had an optimal compliance of 0.011 at a tidal volume of 0.300 L on a PEEP of 20 cm H_2O; ILV was continued for the right lung at these parameters. The healthy left lung had an optimal compliance of 0.022 at a tidal volume of 0.677 L and a PEEP of 15 cm H_2O. The compliance in the left lung on 15 cm H_2O PEEP was higher than the value on 20 cm H_2O PEEP (0.017). ILV was instituted for the left lung at these parameters.

ILV was continued for 91 hours in this patient. Compliance studies were done at regular intervals (representative curves are shown in Fig. 5). Applying optimal tidal volumes at the "best PEEP" of 20 cm H_2O realized significant improvements in the compliance properties of the injured right lung. Similarly, optimal compliance was maintained in the healthy left lung by individual tailoring of its ventilatory parameters.

On the third day of treatment, an attempt was made to assess the remaining mechanical disparity between the two lungs in hopes of discontinuing ILV. PEEP was increased to 20 cm H_2O in the left lung, which was the value necessary to prevent alveolar collapse in the injured right lung. This increase produced a marked change in slope of the compliance curve, indicating decreased compliance in the healthy left lung caused by overdistension of alveoli. In the reciprocal test, the PEEP in the injured right lung was decreased to the 15-cm H_2O level of the left lung. This effect produced a marked decrease in oxygenation as a result of increased shunting consequent to alveolar collapse.

ILV was discontinued after 4 days. Table I shows the changes in oxygenation, shunt, and compliance during the course of treatment. Thus in the appropriate patient with asymmetric lung disease, ILV can achieve superior matching of ventilation to perfusion while optimizing the differing mechanical properties of each lung.

Complications of ILV

The majority of problems encountered during ILV relate to the use of the double-lumen bronchial tube (Fig. 2) and are obstructive in nature.

First, positioning of the tube is critical and an anesthesiologist experienced in the technique should perform the insertion. Positioning is confirmed by roentgenogram and by auscultation. Appropriate positioning of either lumen can be confirmed further by checking for equality of inhaled and exhaled volumes on each ventilator or with a spirometer placed in the circuit. Some groups assess positioning bronchoscopically, though this is of no proven benefit and may be detrimental in a patient with a large shunt and severe restrictive pulmonary disease. Once adequate positioning is confirmed, leakage around the tracheal balloon can be assessed by listening for air leak as with a standard endotracheal tube. The tracheal balloon is then inflated to obliterate the leak. Leakage between each lung can be assessed by manually ventilating one lung while a balloon remains attached over the lumen from the contralateral lung, as previously described. Lost inspiratory volume in one lung may show up as extra expiratory volume from the other lung. The bronchial balloon is then inflated until inspiratory and expiratory volumes are equal for each lung. Cuff pressures should be monitored at all times. As with the placement of a conventional endotracheal tube, the final word on tube placement comes only with the analysis of an arterial blood gas sample and review of a chest roentgenogram. The entire sequence of assessment should be repeated whenever the endobronchial tube is moved or replaced.

During use, the endobronchial tube can become obstructed for several reasons. First, the proximal lumen may become obstructed against the tracheal wall. Second, the tube has less tolerance for changes in position than a conventional endotracheal tube, and small shifts with patient movement may be significant. Third, the left-sided (bronchial) lumen, or its cuff, may obstruct the take-off of the left upper lobe bronchus in a small patient or in patients in whom it is positioned too far distally. The bronchial cuff takes significantly less air than the tracheal cuff, and overinflation may cause it to prolapse upward and obstruct the proximal lumen. Finally, because the lumens are each smaller than a conventional endotracheal tube, obstruction by secretions is easier. Whatever the cause, obstruction will manifest as a sudden increase in the peak inspiratory pressure for that ventilator. This appearance indicates the need for a closer look at the tidal volume of each machine, a check of cuff pressures, auscultation of the lungs, and a chest roentgenogram. Despite all the possibilities for obstruction, if the appropriate care is taken, maintaining proper endobronchial cannulation is quite feasible.

In terms of other problems relating to the double-lumen bronchial catheter, "suctioning hypoxemia" can be induced much more readily when suctioning the good lung, as more total obstruction of the tube's lumen occurs as a consequence of its smaller cross-sectional area.

One anecdotal report of bronchial rupture occurred from overinflation of a bronchial cuff, even though these cuffs are of the "low pressure" variety. Tracheomalacia and tracheobronchial stenosis have also been reported. Though their incidence cannot be assessed reliably, they appear to be rare. These occurrences reinforce the need for frequent determination of cuff pressures during ILV. Tolerating a small air leak is probably preferable to accepting high cuff pressures. Finally, we do not know how long a Broncho-cath should remain in position, in regard to the effect of the balloon on the bronchus, but apparently it should stay in longer than the period currently used.

Finally, in terms of the physiologic complications of ILV, some have expressed concern related to its asymmetric effects on intrathoracic pressures with unequal pressure on each side, possible impairment of venous return, and decreased cardiac output. The greatest concern occurs with asynchronous ILV, in which no true expiratory phase exists and the mediastinum has been noted to swing during ventilation. Both animal and human studies have failed to demonstrate a significant decrease in cardiac output with either synchronous or asynchronous ILV.[15,16,21] Compromise to cardiac output caused by high PEEP, as can happen with conventional ventilation, is treated similarly, with volume loading under the guidance of pulmonary artery catheter monitoring. If necessary, inotropic support is added.

Other Applications of ILV

ILV is also used in managing unilateral massive air leak (MAL) or bronchopleural fistula[11,22] and reexpanding refractory lobar atelectasis. The possible benefits of ILV for treating respiratory failure caused by acute bilateral disease have been investigated.[3] These uses highlight the flexibility and possible future benefits from this technique.

Bronchopleural fistula

Bronchopleural fistula occurs as a result of lung laceration from blunt or penetrating trauma, pneumonia, lung abscess, and empyema. MAL without bronchopleural fistula occurs as a result of closed chest trauma or pulmonary barotrauma during treatment of parenchymal lung disease. Thus, MAL can be associated with a healthy or a diseased contralateral lung.

Even in MAL with a healthy contralateral lung, conventional ventilation can lead to alveolar hypoventilation and hypoxemia because the PEEP effect and tidal volumes follow the route of least resistance and bypass the alveoli. In other words, ventilation is dissipated out of the leak or fistula. Attempts to reduce this loss by decreasing tidal volumes and PEEP similarly cause contralateral hypoventilation. Here ILV can provide adequate ventilation to the healthy lung to ensure gas exchange. At the same time, ventilation of the injured lung can be tailored to the specific pathology, so as to minimize the air leak, which may actually speed healing of the fistula. Several combinations have been used: The healthy side can be ventilated with CMV with PEEP at a specific selected minute ventilation and the side with the leak can be treated with pressure-controlled ventilation or "apneic air insufflation" to augment oxygenation while minimizing air leak. With the apneic air insufflation technique, a small catheter is placed down one lumen of the endobronchial tube and air with varying FIO_2 is insufflated continuously. Alternatively, the side with the leak can be treated with continuous positive airway pressure. The continuous positive airway pressure level is adjusted to just below that pressure necessary to produce air leakage in the leak chamber of the chest water-seal system (critical opening pressure of the fistula).[11]

The problem becomes more difficult when massive air leak or bronchopleural fistula occurs as a consequence of parenchymal lung disease. Usually compliance is reduced bilaterally with compromised ventilation and shunting. High airway pressures necessary to treat the parenchymal disease exacerbate loss through the fistula. Attempts to treat the air leak by decreasing tidal volume and PEEP cause hypoventilation and hypoxemia. Here, the nonleaking side can be treated with tidal volume and PEEP appropriate for its altered compliance, while the leaking side is treated with smaller volumes and minimal or no PEEP to decrease the leak. If this combination cannot provide adequate ventilation without large leakage of volume through the fistula, then switching to high frequency jet ventilation on the leaking side may be necessary.

Lobar atelectasis

ILV has been used successfully to reinflate atelectatic lobes after lobectomy[23] and in cases of incompletely treated, inoperable lung cancer.[23] The principle here is that, after removal of obstructing secretions or lesions, a PEEP greater than the critical opening pressure of the alveoli must be applied continuously and effectively. With CMV, achieving this goal is difficult, as the complaint contralateral lung distends, thus dissipating the pressures intended for the atelectatic lobe. ILV allows normal ventilatory pressures to be maintained on the contralateral lung while reinflation pressures can be applied to the atelectatic lobe without dissipation.

Other uses

Recently, ILV has been used to treat symmetric disease, specifically bilateral acute respiratory failure. The physiologic basis relates to the accentuation of the classic lung zones in the disease state. In the normal, supine person, perfusion is slightly increased and ventilation decreased to the dependent portions of the lungs (zone III) with slightly increased ventilation to the superior portions of the lungs (zone I). Artificial ventilation further causes dependent parts of the lung to decrease their volume and approach their residual volume.[25] Because acute respiratory failure is also characterized by reduced functional residual capacity,[26] the result is significantly decreased ventilation to the dependent parts of the lungs (also the naturally best perfused), which can result in a significant shunt. Here, ILV has been used with the patient in the lateral decubitus position. This positioning converts the upper lung into a better ventilated unit (increased $\dot{V}/\dot{Q}$) and the lower lung into a better perfused unit (decreased $\dot{V}/\dot{Q}$). In fact, as much as 70% of ventilation is distributed to the nondependent lung, with zero PEEP in the lateral decubitus position.[24] Tidal volume and PEEP can then be independently adjusted and optimized as previously described to shift ventilation back toward the dependent lung, thereby achieving a better match of ventilation to perfusion. In study cases, a decreased shunt fraction and improved oxygenation were indeed obtained.[24]

Other reported uses of ILV include unilateral pulmonary hemorrhage where the double-lumen tube can isolate and protect the healthy lung from contamination by the opposite side. ILV has also been used to minimize barotrauma in the less compliant lung in cases of unilateral or asymmetric disease. The ability to decrease tidal volume and airway pressures in the injured lung helps to minimize the likelihood of injury. Finally, ILV has been used to treat traumatic lung cysts that are expanding on conventional ventilation. Here, too, the ability to decrease tidal volume, PEEP, and airway pressure selectively is the important property, and the use of ILV can arrest the expansion of these cysts.

Conclusion

ILV is a relatively new technique and its applicability is still being defined in the critically ill trauma patient. Clearly, in carefully selected patients with asymmetric lung disease and unequal physical properties causing $\dot{V}/\dot{Q}$ mismatch, ILV can yield significant improvements in gas exchange. Areas for further work include expanding the indications, defining optimal modes of ventilation, monitoring the results of therapy, and delineating the criteria for weaning. The need for synchronization of ventilators is still controversial. ILV, a technique of great flexibility, has been firmly established in the armamentarium of trauma critical care.

References

1. Geiger K, Bethke U, Lutz H. Differential lung ventilation in pulmonary contusion. Intensive Care Med (in press).
2. Canillese JM, Garrigosa F, Prieto C, Oncins JR. A selective ventilation distribution circuit (S.V. > D.C.). Intensive Care Med 1979;5:95.
3. Powner KJ, Eross B, Grenvik A. Differential lung ventilation with PEEP in the treatment of unilateral pneumonia. Crit Care Med 1977;5:170.

4. Venus B, Pratap KS, Op'Tholt T. Treatment of unilateral pulmonary insufficiency by selective administration of continuous positive airway pressure through a double-lumen tube. Anesthesiology 1980;52:74.
5. Glass DD, Tönnesen AS, Gabel JC, Arens JF. Therapy of unilateral pulmonary insufficiency with a double-lumen endotracheal tube. Crit Care Med 1976;4:323.
6. Sachdeva SP. Treatment of postoperative pulmonary atelectasis by active inflation of the atelectatic lobe(s) through an endobronchial tube. Acta Anaesthesiol Scand 1974;18:65.
7. Siegel JH, Stoklosa JC, Borg U, Wiles CE III, et al. Quantification of asymmetric lung pathophysiology as a guide to the use of simultaneous independent lung ventilation in posttraumatic and septic adult respiratory distress syndrome. Ann Surg 1985;202:425-39.
8. Asbaugh DG, Petty TL, Bigfelow DB, et al. Continuous positive pressure ventilation in acute respiratory failure. N Engl J Med 1970;24:1430-6.
9. Carlon GC, Kahn R, Howland WS, et al. Acute life-threatening ventilation-perfusion inequality: an indication for independent lung ventilation. Crit Care Med 1978;6:380-3.
10. Baehrendtz S, Hedenstierna G. Differential ventilation and selective positive end-expiratory pressure: effects on patients with acute bilateral lung disease. Anesthesiology 1984;61:511-7.
11. Geiger K. Differential lung ventilation. Int Anesthesiol Clin 1983;21:83-98.
12. Oppenheimer L, Craven KD, Forkert L, Woods LDH. Pathophysiology of pulmonary contusion in dogs. J Appl Physiol 1979;47:718-28.
13. Light RB, Mink SN, Wood LDH. The effect of unilateral PEEP on gas exchange and pulmonary perfusion in canine lobar pneumonia. Anesthesiology 1981;55:251-5.
14. Hurst JM, DeHaven CB Jr, Branson RD. Comparison of conventional mechanical ventilation and synchronous independent lung ventilation (SILV) in the treatment of unilateral lung injury. J Trauma 1985;25:766-70.
15. Lev A, Barzilay E, Geber D, Bishara H, Prego J. Differential lung ventilation: a review and 2 case reports. Resuscitation 1987;15:77-86.
16. Hillman JM, Barber JD. Asynchronous independent lung ventilation (AILV). Crit Care Med 1980;8:390-5.
17. East TD, Pace NL, Westenskow DR. Synchronous versus asynchronous differential lung ventilation with PEEP after unilateral acid aspiration in the dog. Crit Care Med 1983;11:441-4.
18. Trew F, Warren BR, Potter WA. Differential lung ventilation in man. Crit Care Med 1976;4:112-4.
19. Carlon GC, Ray C, Klein R, Goldiner PL, Miodownik S. Criteria for selective positive end expiratory pressure and independent synchronized ventilation of each lung. Chest 1978;74:501-7.
20. Brendenberg CE, Webb WR. Experimental pulmonary edema, the effect of unilateral PEEP on the accumulation of lung water. Ann Surg 1979;189:433-8.
21. Barber JD, Hillman KM. Asynchronous independent lung ventilation (AILV). Crit Care Med 1980;8:390-5.
22. Dodds CP, Hillman KM. Management of massive air leak with asynchronous independent lung ventilation. Intensive Care Med 1982;8:287-90.
23. West KJ, Zandstra DF, Miranda DR. Post lobectomy atelectasis: the use of a Servo 900B as a high-frequency ventilator. Intensive Care Med 1987;13:78-80.
24. Bochenek KJ, Brown M, Skupin A. Use of a double-lumen endotracheal tube with independent lung ventilation for the treatment of refractory atelectasis. Anesth Analg 1987;66:1014-7.
25. Hedenstierna G, Bindslev L, Santesson J, Norlander OP. Airway closure in each lung of anesthetized human subjects. J Appl Physiol 1981;50:55-64.
26. Katz JA, Ozanne GM, Zinne SE, Fairley HB. Time course and mechanism of lung-volume increase with PEEP in acute pulmonary failure. Anesthesiology 1981;54:9-16.