



PRESENTS

WHAT IS THE HISTORY OF ARDSnet (EVIDENCE BASED MEDICINE) AS APPLIED TO PULMONARY VENTILATORS?

On May 28, 1976, Congressional US Medical Device Legislation was activated by the US FDA on all new medical devices that were not substantially the same as existing commercially available medical devices in clinical use on May 28, 1976.

All US marketed medical devices that were not being manufactured and in clinical service on May 28, 1976, were required to conform to an actual existing medical device by being substantially the same to meet FDA 510K marketing rights. If they were not the same, they were mandated to enter a very expensive FDA Investigational Medical Device Program for denial or approval.

Thus, if the FDA decided that the NEW production medical device presented to them was indeed MORE OF THE SAME, a 510K (grandfather) medical device exemption was issued to the manufacturer of the device to start commercial marketing without issuance of a new costly IDE/PMA medical device approval.

The only exception (BY FDA ACCLAMATION) was the application of electronic microprocessors to substitute for existing pneumatic and mechanical energy sources, for the presentational programming of functions available on Volume/Pressure limited pulmonary ventilators.

Manufacturers under FDA mandate began assembling 510K (grandfather) electronic volume oriented pressure limited ventilators employing state of the art May 28, 1978, technology with extensive re-labeling of traditional functional terms. Existing re-labeling such as "ASSISTED RESPIRATION" WAS CHANGED TO "PRESSURE SUPPORT," etc.

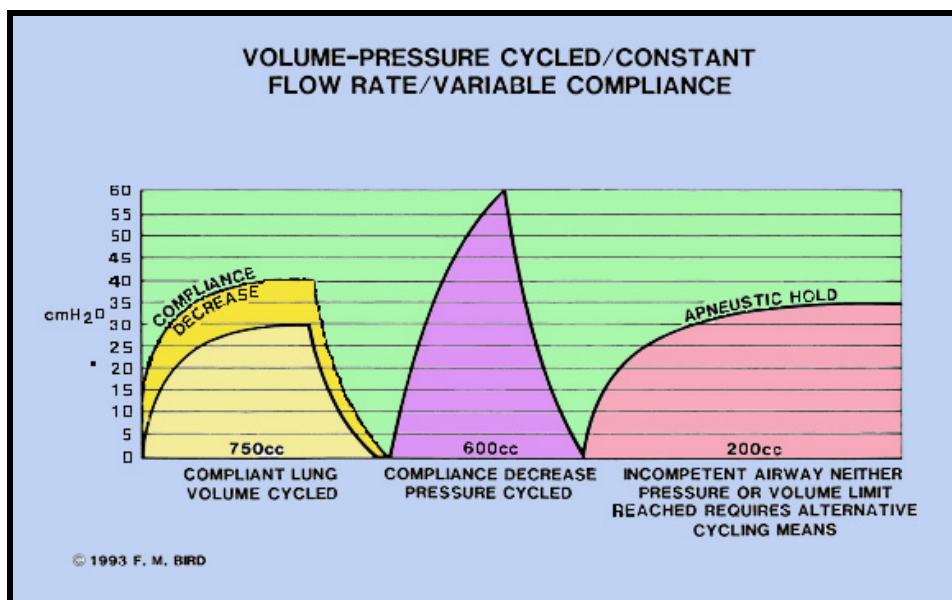
THESE SUPPOSEDLY NEW REPACKAGED VOLUME/PRESSURE PULMONARY VENTILATORS WERE FDA MANDATED TO BE FUNCTIONALLY MORE OF THE SAME CIRCA MAY 28, 1976, WITH “ALLOWED BY ACCLAIM” MICROPROCESSOR TECHNOLOGY.

The same US FDA 510K exemptions are still in effect in 2009. Over the years, five or more 510K (circa May 28, 1976) electronic pulmonary ventilators have been serially repackaged while remaining in compliance with the original FDA 510K (more of the same) mandate. Being ALL SUBSTANTIALLY THE SAME, they have been addressed categorically as CONVENTIONAL VENTILATORS.

AN EXPANDED HISTORICAL REVIEW OF VOLUME/PRESSURE VENTILATORY MANAGEMENT TO BE READ IN CONTEXT

The use of Volume/Pressure oriented ventilators with a selected tidal volume delivered under an arbitrary pressure limit, were designed for the maintenance of pulmonary functions in patients without adequate spontaneous ventilation.

This Volume/Pressure category of mechanical ventilators were originally conceived and designed to super-impose controlled ventilation upon an embarrassed spontaneously breathing patient by means of a Controlled Mechanical Ventilation (CMV) employed for ventilatory maintenance, with the use of analgesia, if necessary, to prevent the spontaneous breathing patient from fighting the established ventilator program.

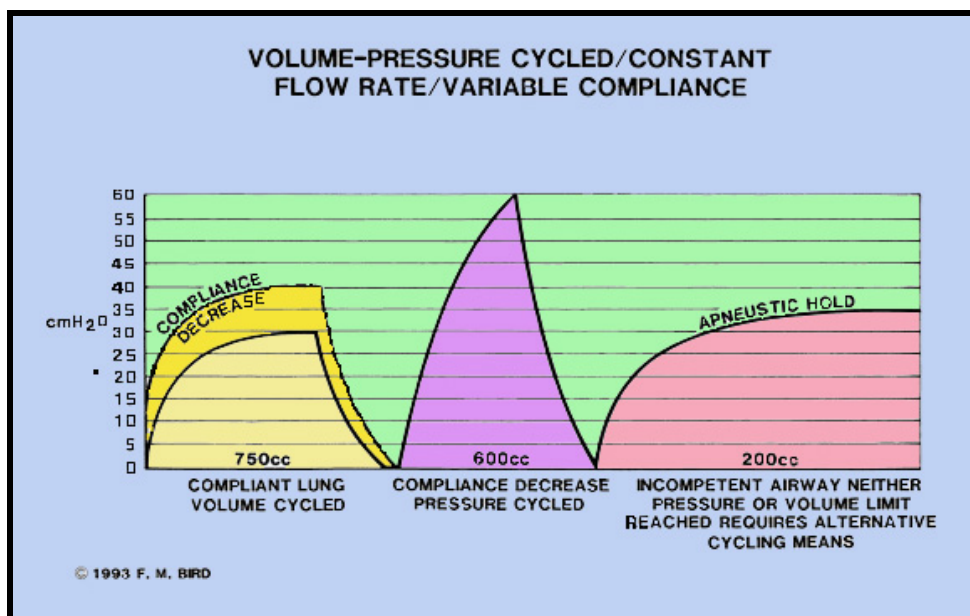


A Volume/Pressure cycled ventilator has limited primary options when ventilating a patient with low gross pulmonary compliance with diffuse peripheral airway obstruction and alveolar hyperinflation.

A “Volume/Pressure cycled” ventilator is primarily scheduled to repetitively deliver a selected volume of a respiratory gas into the lungs under an arbitrary programmed peak positive pressure (PIP).

Essentially, flow/pressure is used to force the lungs to expand. If the selected flow/pressure (PIP) is not sufficiently high to force the selected tidal volume into the lungs, thus failing to deliver the tidal volume selected, the ventilator becomes “pressure limited.”

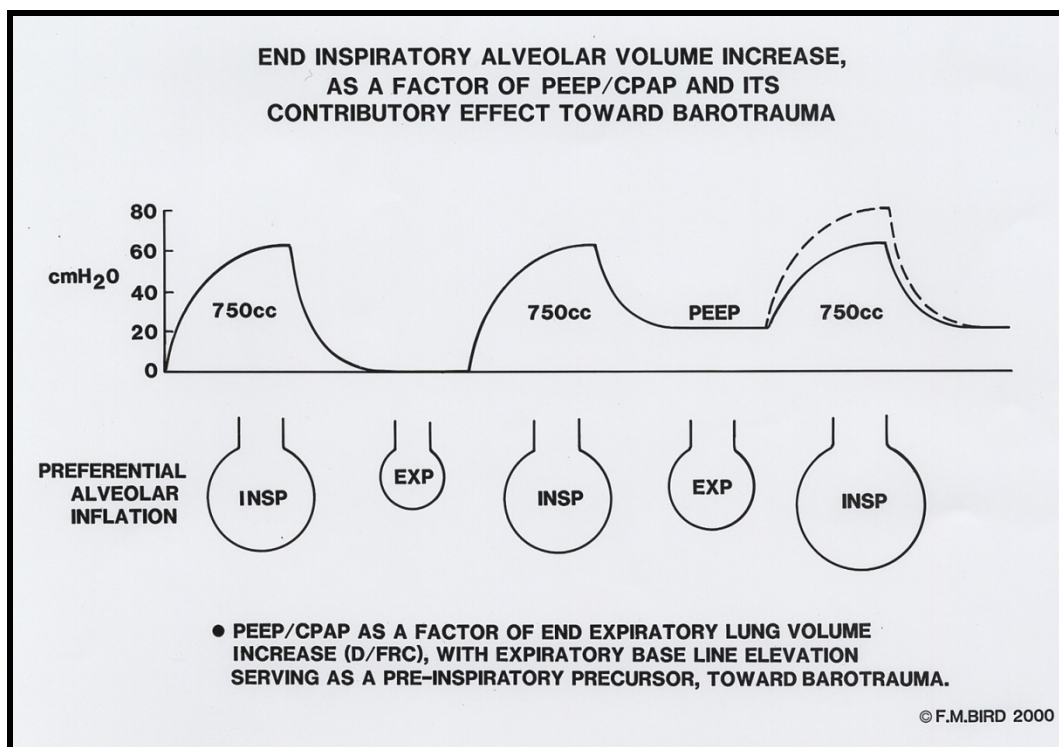
1. By mal-scheduling a lengthened inspiratory inflation time, a breath hold at end inspiration can be programmed. Under apneustic hold programming, the ventilator can be cycled upon time under a selected peak inspiratory pressure limit (PIP).
2. Thus the lungs could potentially be held inflated at pressures up to the selected peak delivery pressure (PIP) until mechanically time released.



The endobronchial delivery of a specific programmed tidal volume under a selected pressure cycled schedule is only possible with a “cuffed” endotracheal tube. The cuffed endotracheal tube is designed to prevent ambient leaks in the ventilator inspiratory circuit and the conducting physiological airway. A non-leaking ventilator/physiological airway interface must be maintained to allow accurate volume-pressure cycling.

3. The selection of a Positive End Expiratory Pressure (PEEP) serves to prevent the apneic patient from exhaling (emptying) the lungs to the normal end inspiratory resting position. The (PEEP) programming increases the Functional Residual Capacity (FRC) of the lungs (the amount of air left in the lungs by the next scheduled inspiratory phase).

4. When a positive end expiratory pressure (PEEP) is selected under a pre-selected tidal volume (with sufficient delivery pressure reserve), the selection of PEEP can cause an appropriate increase in the functional residual lung volume (FRC), which could lead to the hyper-inflation of certain Preferential Bronchiolar airways in patients with peripheral lung diseases.



5. Generally, the greater the lung injury the greater the mandated requirement for increased Oxygen concentrations in the respiratory gases used to ventilate the patient (FIO₂). The greater the clinical efficiency of the mechanical ventilator (all else being equal) the less the required (FIO₂) during the mechanical ventilation of the lung. It is well established that continuous inhaled Oxygen concentrations of over 40% become increasingly toxic with concentration (partial pressure) and increasing time.

A CMV pulmonary ventilator was not primarily designed with “lung protective strategies” generally employing an over pressure relief venting and alarming.

Patients with acute or chronic variable diffuse Bronchiolar and Alveolar hyperinflation with Preferential Bronchiolar Airways are prime candidates for Preferential Airway hyperinflationary Barotrauma when ventilated with Volume/Pressure oriented CMV ventilators.

By the 1990's the predominate electronic pulmonary ventilators were all based upon Volume/Pressure programming which were manufactured by major manufacturers with extensive Medical Lobbyists who influenced certain medical journal publications as well as medical convention interfaces. This led to an integrated consortium of Medical Clinicians, Biomedical Technologists, organizations with medical spokespersons, and PEER journal editors, creating a format for initiating a directive under the premise of "EVIDENCE BASED MEDICINE". In the Mechanical Ventilator field of influence this was called "ARDSnet".

Members of this basic clinical oriented consortium were predominately highly qualified clinicians with major experience using electronic Volume/Pressure ventilators, with limited clinical experience with other advanced means of mechanical pulmonary ventilation such as IPV® and VDR®.

WHAT IS ARDSnet?

Higher versus Lower Positive End-Expiratory Pressures in Patients with the Acute Respiratory Distress Syndrome

The National Heart, Lung, and Blood Institute ARDS Clinical Trials Network*

ABSTRACT

BACKGROUND

Most patients requiring mechanical ventilation for acute lung injury and the acute respiratory distress syndrome (ARDS) receive positive end-expiratory pressure (PEEP) of 5 to 12 cm of water. Higher PEEP levels may improve oxygenation and reduce ventilator-induced lung injury but may also cause circulatory depression and lung injury from overdistention. We conducted this trial to compare the effects of higher and lower PEEP levels on clinical outcomes in these patients.

METHODS

We randomly assigned 549 patients with acute lung injury and ARDS to receive mechanical ventilation with either lower or higher PEEP levels, which were set according to different tables of predetermined combinations of PEEP and fraction of inspired oxygen.

RESULTS

Mean (\pm SD) PEEP values on days 1 through 4 were 8.3 ± 3.2 cm of water in the lower-PEEP group and 13.2 ± 3.5 cm of water in the higher-PEEP group ($P < 0.001$). The rates of death before hospital discharge were 24.9 percent and 27.5 percent, respectively ($P = 0.48$; 95 percent confidence interval for the difference between groups, -10.0 to 4.7 percent). From day 1 to day 28, breathing was unassisted for a mean of 14.5 ± 10.4 days in the lower-PEEP group and 13.8 ± 10.6 days in the higher-PEEP group ($P = 0.50$).

CONCLUSIONS

These results suggest that in patients with acute lung injury and ARDS who receive mechanical ventilation with a tidal-volume goal of 6 ml per kilogram of predicted body weight and an end-inspiratory plateau-pressure limit of 30 cm of water, clinical outcomes are similar whether lower or higher PEEP levels are used.

The members of the Writing Committee (Roy G. Brower, M.D., Johns Hopkins University, Baltimore; Paul N. Lanken, M.D., University of Pennsylvania, Philadelphia; Neil MacIntyre, M.D., Duke University, Durham, N.C.; Michael A. Matthay, M.D., University of California, San Francisco, San Francisco; Alan Morris, M.D., LDS Hospital, Salt Lake City; and Marek Ancukiewicz, Ph.D., David Schoenfeld, Ph.D., and B. Taylor Thompson, M.D., Massachusetts General Hospital, Boston) of the National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network assume responsibility for the integrity of the article. Address reprint requests to Dr. Brower at Johns Hopkins University, 1830 East Monument St., Rm. 549, Baltimore, MD 21205.

*The participating institutions are listed in the Appendix.

N Engl J Med 2004;351:327-36.

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After reviewing the above brief ARDSnet abstract, typical questions are:

How was the acute lung injury created? Was the ARDS trauma created by the conventional electronic Volume/Pressure ventilator and FiO₂ programming?

The following ARDSnet patient **EXCLUSION** list appears to create **PATIENT SELECTION** denying the use of Volume/Pressure programming on a vast patient population. How did the ARDSnet program accommodate patients requiring mechanical pulmonary ventilation listed under their extensive exclusion criteria?

ARDSnet

Patients were excluded:

- 📁👉 If 36 hours had elapsed since they met the first three criteria;
- 📄👉 They were younger than 18 years of age;
- 📄👉 They had participated in other trials within 30 days before the first three criteria were met;
- 👤👉 They were pregnant;
- 📊👉 They had increased intracranial pressure,
- ⌚👉 Neuromuscular disease that could impair spontaneous breathing,
- 🏠👉 Sickle cell disease,
- 👤👉 Severe chronic respiratory disease;
- 📏👉 They weighed more than 1 kg per centimeter of height;
- 📁📁👉 They had burns over more than 30 percent of their body-surface area;
- 📁📁👉 They had other conditions with an estimated 6-month mortality rate of more than 50 percent;
- 📁📄👉 They had undergone bone marrow or lung transplantation;
- 📁📄👉 They had chronic liver disease (as defined by Child-Pugh class C)
- 📁📄👉 Their attending physician refused or was unwilling to agree to the use of full life support.

DESIGN FACTORS OF THE THREE GENERATIONS OF VOLUME/PRESSURE RESPIRATOR/VENTILATORS

1. The first generation of Volume/Pressure respirator ventilators were assisters delivering a selected volume and associated inspiratory flow rate under specific peak delivery pressures. This included the mechanical piston respirators.
2. The second generation Volume/Pressure ventilators were assister-controllers with pre-measured volumes generally delivered from elastomeric displacement reservoirs (bellows) under a selected integrated flow rate and peak pressure, through elastomeric breathing circuits with a moderate compliance factor.

Note: As the ventilator delivery pressures increased, the compliance of the bellows reservoir, breathing circuit and pulmonary structures all yielded to the imposed stretching pressures. When an inspiratory inflow (flowrate) into a distensible reservoir (the lungs) is obstructed, the delivery pressure is generally caused to rise proportionate to inflow velocity.

The second generation of Volume/Pressure ventilators possessed three compliance (amount of give for the amount of pressure) sources. They were the distensible bellows type tidal reservoir, the breathing circuit and the Pulmonary structures.

As lung volumes were increased, the compliance “stretch factors” were distributed three ways, serving as a buffer against acute intrapulmonary pressure rises, as intrapulmonary airway resistances to mechanically created inspiratory inflows varied.

Thus a multiple compliance design factor served to protect the lung from being the only (total) compliance (stretch) factor during lung volume increases. This design provided for a limited lung protective strategy during Radcliff scaled, tidal volume deliveries. Thus hyperinflation barotraumatic incidences were minimal and/or not reported.

3. The third generation of Volume/Pressure ventilators are assister controllers with Tidal Volumes measured by $\text{Flow} \times \text{Time} = \text{Tidal Volume}$. The tidal reservoir (bellows) compliance was eliminated while the breathing circuit compliances were substantially decreased. Thus the pulmonary structures became the near total means of providing compliance (stretch) during the inspiratory Tidal Volume delivery.

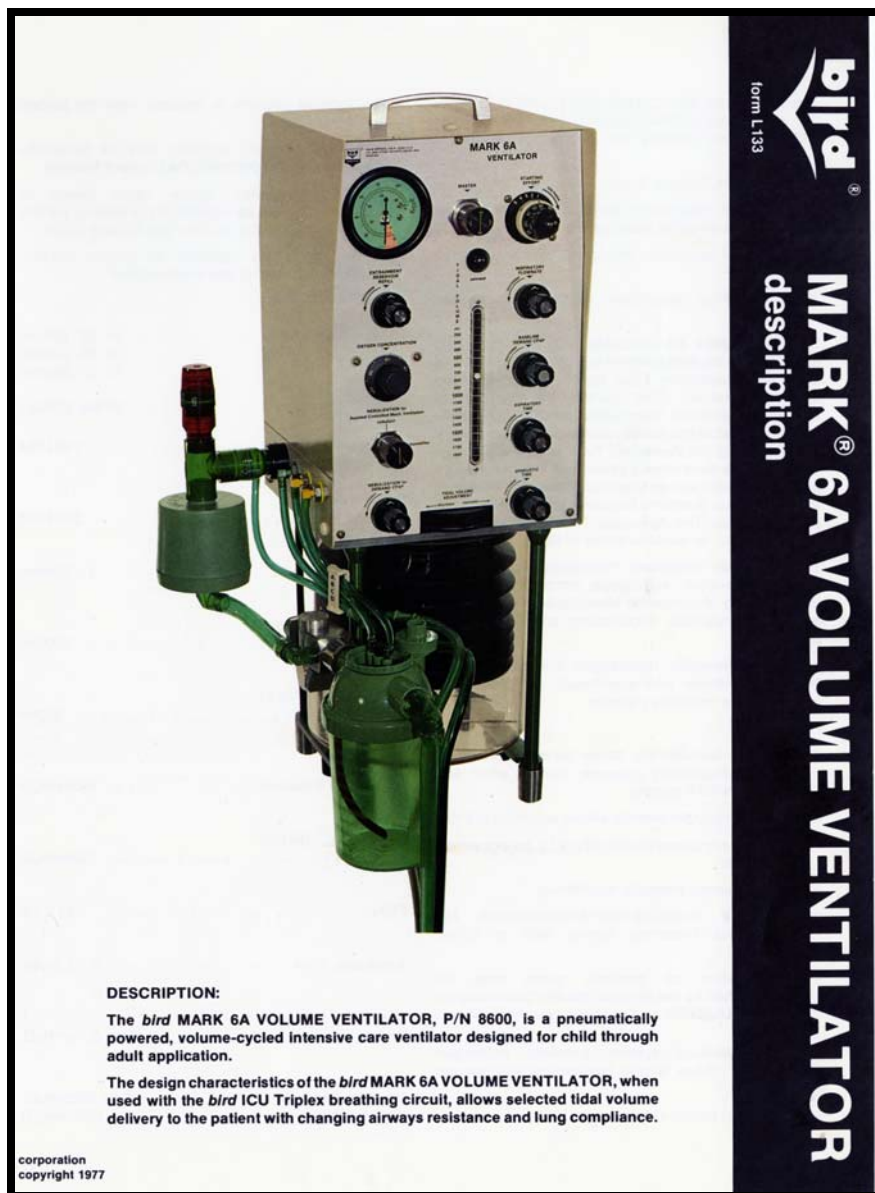
When patients with diffuse bronchiolar-alveolar obstructive diseases are ventilated with Tidal Volumes delivered endo-bronchially under a positive pressure, the variably obstructed peripheral Bronchiolar Airways without obstructions (PREFERENTIAL AIRWAYS) are first inflated, creating alveolar hyper-inflation with potential barotraumas.

The logic for selecting time cycled electronic Volume/Pressure ventilators as the ARDSnet choice must have been based upon the wide distribution of the third generation of electronic Volume/Pressure ventilators. The design limits of this type of a Volume/Pressure mechanical ventilator excluded a vast number of patients requiring mechanical ventilation of the lungs, creating a major patient selection process. THIS IS CALLED PATIENT SELECTION.

ARDSnet advanced the use of the term “EVIDENCED BASED MEDICINE” without alternatives for patients requiring mechanical pulmonary ventilation listed in the ARDSnet EXCLUSION LIST.

All ARDSnet Volume/Pressure ventilator design types are mandated by their FDA 510K marketing releases to be substantially equivalent to available functions circa May 28, 1976. Thus, "EVIDENCED BASED MEDICINE" as applied to ARDSnet, limits Volume/Pressure ventilator clinical technology to May 28, 1978, by FDA 510K mandate.

WHAT WERE THE KEY AVAILABLE TECHNICAL SOURCES OF
EXISTING TECHNOLOGY FOR FDA 510K marketing rights
For Volume/Pressure PULMONARY VENTILATORS
circa May 28, 1978



THE BIRD MARK 6A VOLUME/PRESSURE PULMONARY VENTILATOR

The **bird MARK 6A** VOLUME VENTILATOR features the **bird** Demand CPAP system. The **bird** Demand CPAP system permits the following ventilatory protocols:

1. Continuous Positive Airway Pressure (CPAP).
2. Intermittent Mandatory Ventilation (IMV) with or without an elevated baseline (PEEP).
3. Controlled ventilation with or without an elevated baseline.
4. Assist/Control ventilation without an elevated baseline.

The **bird MARK 6A** VOLUME VENTILATOR also incorporates the **bird** systems for Apneustic Flow and Time with Inspiratory Flow Acceleration and slope control. Apneustic Time permits the clinician to impose an adjustable, time-cycled, mechanical breath-hold at the end of the preset volume-cycled inspiratory phase. During the Apneustic Time phase, a plateau-type pressure wave form is generated. This inspiratory pressure plateau serves to enhance the distribution of inspired gases, therefore increasing the ventilation to perfusion ratio. The Apneustic Time interval may be either dynamic or static in terms of flow.

The adjustable modulated Inspiratory Flowrate with Flow Acceleration and slope control permit the programming of numerous flow/pressure wave forms including sinusoidal, decelerating and square flow patterns.

bird has essentially repackaged the existing **bird MARK 6** ventilator into a compact, comprehensive intensive care ventilatory device.

FEATURES:

- **MARK 1®** Sequencing Servo permits control of maximum inspiratory pressure, starting effort and manual ON/OFF cycling
- Integral air/oxygen blender allows selection of FIO₂
- Pneumatically powered with 50 p.s.i.g. sources of air and oxygen
- Proximal airway pressure monitoring
- Adjustable nebulization/humidification for spontaneous breathing during IMV or CPAP modalities
- Humidification of inspired gases may be accomplished by use of either the **bird** 500ml dual jet **microNEBULIZER®** or a cascade-type humidifier
- Failsafe lockout system prevents prolonged inspiratory phase should mechanical malfunction occur
- Mainstream bacterial filter

- The internal circuitry is isolated from the patient breathing circuit
- The patient and/or ventilator may be optionally monitored by the **birdwatcher** Patient Monitor
- Failsafe emergency intake valve opens to accommodate the spontaneously breathing patient should mechanical or source gas failures occur
- Single circuit (as opposed to double circuit), therefore minimizing gas consumption

SPECIFICATIONS:

Dimensions	H 25" (64cm) W 14" (36cm) D 18" (46cm)
Weight	30 lbs. (14Kg.)
Mandated Flow	110 LPM
Spontaneous Demand Flow	200 LPM
Cycling Rate	1 - 30/min.
Maximum Tidal Volume	1800ml
Minimum Tidal Volume	200ml
Maximum Inspiratory Pressure	100cmH ₂ O
Pressure Relief Valve	110cmH ₂ O
FIO ₂21 - 1.0
Apneustic Time	0 - 2.5 sec.
Starting Effort Range	-5 to -5.0cmH ₂ O
Elevated Baseline (Optional)	0 to 35cmH ₂ O (50cmH ₂ O)

Dr. Bird's third generation of novel volume/pressure limited patented respirators were in clinical use and commercially available on May 28, 1976.

The Bird Mark 6A respirator with advanced functional characteristics was available to the FDA for 510K for comparison on May 28, 1976.

How many of the above described Bird conceived functions for Volume/Pressure programs (mechanical ventilation of the lungs) were conceived by the progressive technological developments of Dr. Bird over some forty years, forming the basic programming of current electronic Volume/Pressure ventilators?

In 1958, Dr. Bird's initial programming for his Mark 7 respirator with Inspiratory Flowrate suggested using a reduced inspiratory flowrate and peak delivery pressures of less than 30 cm H₂O to increase alveolar distribution (called PULMONARY CONFORMANCE). He did not use PEEP/CPAP to minimize mean intrathoracic pressures. This early flow-pressure programming essentially eliminated hyperinflation Barotrauma. Is certain Volume/Pressure ARDSnet programming of today similar?

In 1976, Dr. Bird's earlier conceived "state of the art" Bird Mark 6A Volume/Pressure respirator essentially possessed the programming capacities of today's 2009 electronic Volume/Pressure ventilators with the same Preferential Airway Barotraumatic limitations.

Inherent "design created" potential Preferential Airway barotraumas, caused Dr. Bird to advance his clinical design concepts beyond his 1976 existing Volume/Pressure cycled pulmonary ventilation techniques.

By 1990, Dr. Bird's Intrapulmonary Percussive Ventilation (IPV®) and Volumetric Diffusive Respiration (VDR®) techniques had been investigated by the US FDA under IDE/PMA as well as 510K protocols. IPV® and VDR® scheduling, which have been in world wide clinical routines for over twenty five years, have proven to have far greater clinical efficacies when compared with Volume/Pressure programming in the mechanical ventilation of critical patients with Preferential Bronchiolar airways.

The clinical significance of IPV® and VDR® over Volume/Pressure ventilatory programming is the inherent designed "LUNG PROTECTIVE STRATEGY" while providing for a peripheral lung recruitment without the potential for Preferential Airway Barotrauma.

WHAT IS THERAPEUTIC "INTRAPULMONARY PERCUSSIVE VENTILATION (IPV®)" AND THE CRITICAL CARE VERSION "VOLUMETRIC DIFFUSIVE RESPIRATION (VDR®)"?

Dr. Bird started his fourth generation of pulmonary respirators in 1978. This generation was directed toward further increasing LUNG PROTECTIVE STRATEGIES during critical care ventilation.

IPV® was directed toward the long-term therapeutic preservation of the intrapulmonary Bronchial Circulation, (secondary to persistent Alveolar Hyperinflation as in Chronic Bronchitis etc.). Long term sustained Bronchial airway hyper-inflation serves to inversely decrease pulmonary Bronchial circulation, potentially culminating in necrotic end stage Pulmonary Emphysema.

Dr. Bird's fourth generation of conceptual medical respirators (ventilators) use percussive higher frequency sub tidal volumes delivered intrapulminarily by time cycled scheduling in milliseconds. This advanced designing was directed toward decreasing the existing hyperinflational bronchiolar-alveolar barotraumatic potentials related to PREFERENTIAL AIRWAY HYPERINFLATION caused by the mechanical ventilation of lungs with diffuse differential bronchial and alveolar airway obstructions.

To elucidate the differential between existing Volume/Pressure lung maintenance ventilation and therapeutic Intrapulmonary Percussive Ventilation (IPV®) as well as critical care Volumetric Diffusive Respiration (VDR®); the ventilation of a patient with typical obstructive chronic Bronchitis will be examined starting on next page.

Notes:



PRESENTS

**THE RATIONALE FOR THE MECHANICAL VENTILATION OF THE
LUNG WITH BRONCHITIS
OFTEN ASSOCIATED WITH PULMONARY DISEASES.**

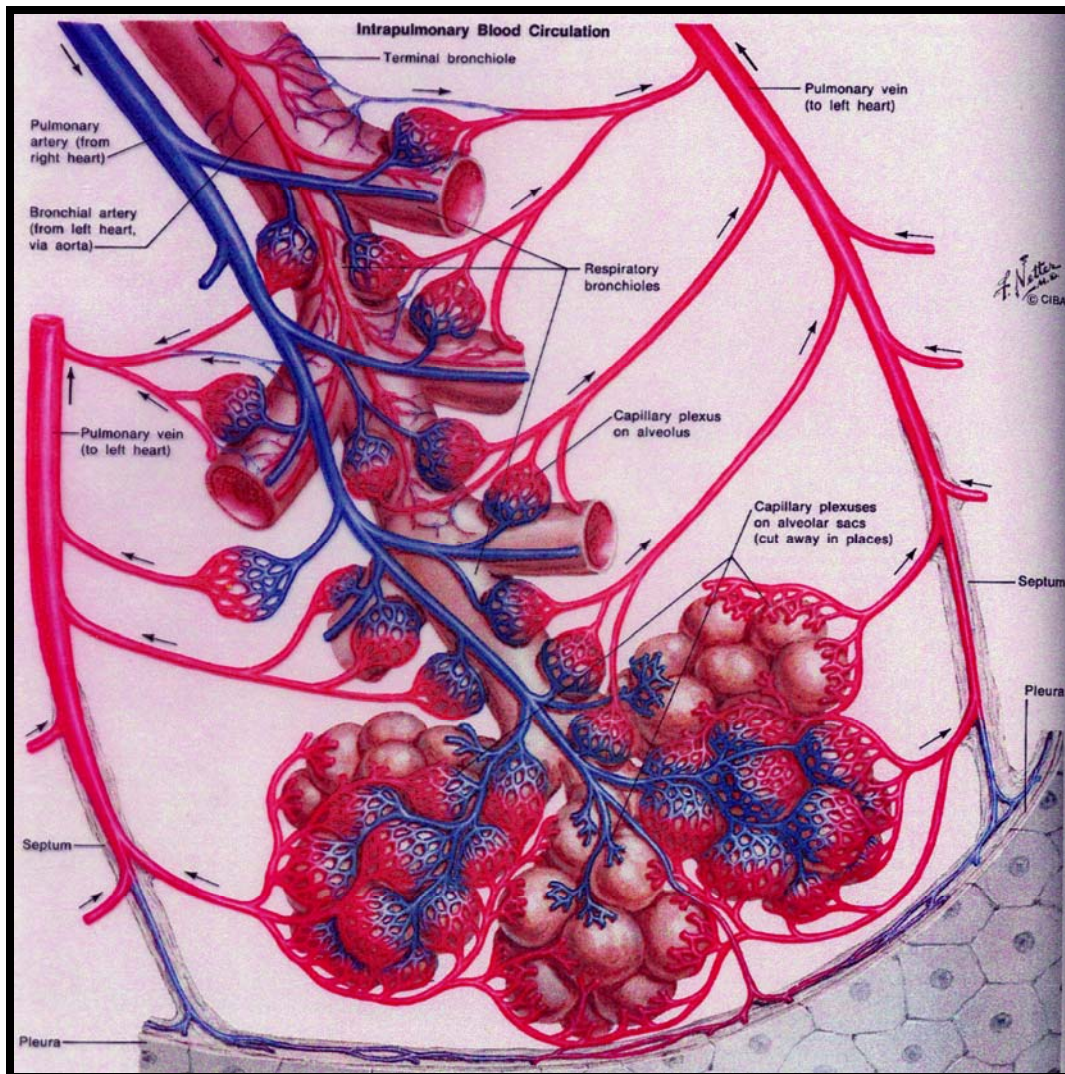
This data conforms to standard TEXTBOOK rationale for involved pathophysiology, pharmacology, physics and meteorology. Primary direct pathophysiology and physical principals and descriptions are employed to describe the many associated disciplines involved.

THE OVERSIMPLIFIED PATHOPHYSIOLOGY OF BRONCHITIS-

- A. The millions of very small bronchioles (airways) serve to transport the respired intrapulmonary air in and out of the pulmonary alveoli.
- B. Continuous effective air exchange within the pulmonary airways serves to deliver the “down flow” of airway Oxygen molecules from inside the alveoli, across the alveolar capillary membranes to re-arterialize the circulating venous blood as well as to create the disassociation “up flow” of Carbon Dioxide out of the alveoli into the pulmonary airways for exhalation from the lungs.
- C. Bronchitis creates an uneven narrowing of the millions of small Pulmonary Bronchiole (hairlike) lumens (calibers) from none and/or little obstructions to their near total obstruction.
- D. It is the diffuse uneven levels of obstruction from unobstructed to total obstruction of the bronchiolar airways that create the uneven alveolar air exchange (alveolar gas exchange).

When the bronchiolar obstruction is sufficient to prevent alveolar gas exchange for the patients activity (metabolic) levels with spontaneous breathing, artificial ventilation may become life saving.

- E. Initially Bronchitis is reversible; however, over time it becomes **CHRONIC Bronchitis**, which ultimately reduces the effectiveness of the Bronchial Circulation in providing an adequate generalized arterialized blood supply to the tissues of the lungs.



F. Netter Courtesy Ciba 1976

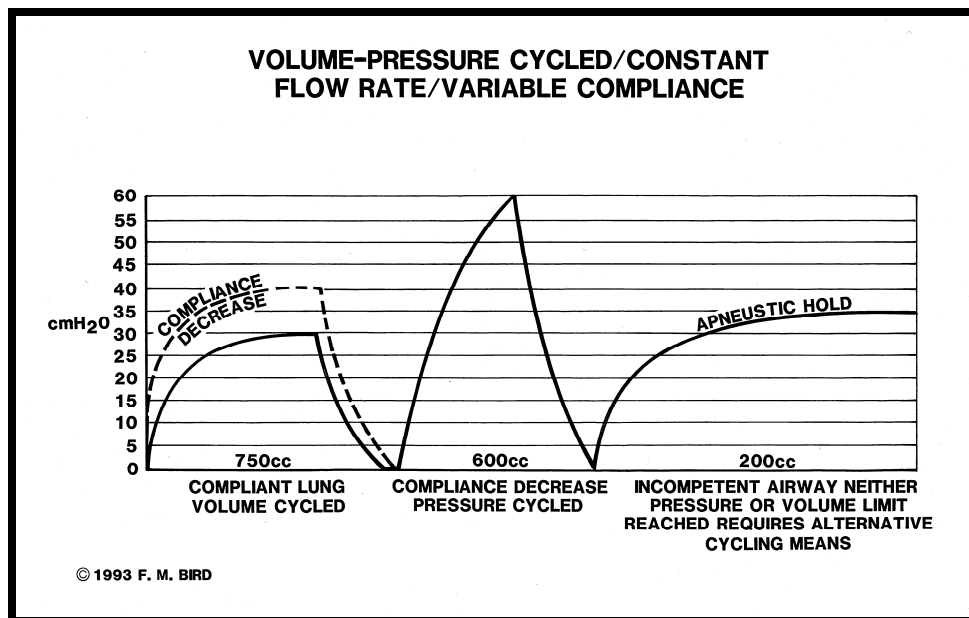
- F. In the preceding, bronchial circulation delivers arterialized blood within the pulmonary structures to the most distal (furthest) alveolar areas to be supplied with arterialized blood.
- G. At this point within the alveolar walls, the arterialized bronchial blood flow becomes venous blood (desaturated).
- H. The bronchial circulation vessels are attached to the exterior walls of the bronchial airways, similar to the way the pulmonary vessels are attached.

- I. The degree of chronic Bronchitis (obstruction) within the bronchioles will determine how much the Bronchiolar circulatory vessels are stretched and narrowed, reducing (obstructing) blood flow through the alveolar Bronchial circulations.
- J. Chronic Bronchitis resulting in long term Bronchiolar-Alveolar hyperinflation which serves to reduce Bronchiolar blood flow reaching a diffuse Ischemic level, causing a non-reversible necrotic breakdown of the associated alveolar re-Oxygenation processes. This insidious progressive chronic Bronchitis can become the end stage lung disease called "Pulmonary Emphysema" (a well defined circulatory ischemic pulmonary disease).

THE MECHANICAL VENTILATION OF THE LUNGS OF PATIENT'S WITH ACUTE OR CHRONIC BRONCHITIS, WITH THEIR DIFUSE BRONCHIOLAR LUMENS; UNOBSTRUCTED AND/OR PARTIALY OR TOTALLY OBSTRUCTED SECONDARY TO BRONCHITIS INDUCED PERIPHERAL AIRWAY ENCROACHMENTS SUCH AS EDEMA AND RETAINED ENDOBRONCHIAL SECRETIONS.

There are two basic forms of mechanical pulmonary ventilation. They are:

1. PRESSURE- VOLUME (CMV), lung maintenance ventilation.
2. LUNG RECRUITMENT AND MAINTENANCE (IPV®-VDR®) ventilation.



Pressure-volume limited continuous mechanical ventilation (CMV) is a form of lung maintenance (intrapulmonary gas exchange) using an arbitrary positive pressure to deliver a selected constant tidal volume (with a scheduled inspiratory flow rate) until reaching the selected tidal volume and/or arbitrary peak positive pressure selection (PIP).

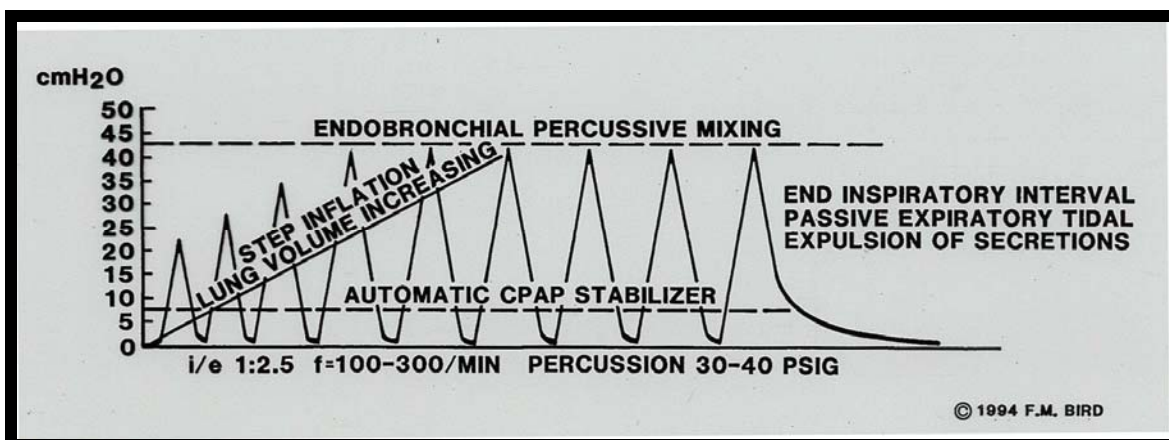
Thus, if the scheduled Tidal Volume is not delivered in the “time allowed” under the established arbitrary peak positive pressure, the ventilator becomes primary pressure limited as opposed to tidal volume limited. The positive Inspiratory to Expiratory I/E ratio is programmed in seconds.

The pressure differential behind the tidal volume delivery is inter-dependent upon the gross intrapulmonary resistances. Thus an accelerating flow/pressure gradient is held against the entire pulmonary airway until the required positive delivery pressure is reached and/or upon failure to deliver the Tidal Volume. The PIP is then held against the pulmonary airways (apneustic plateau) until the selected Inspiratory Time is mechanically terminated.

THE BASIC CONVENANTS OF INTRAPULMONARY PERCUSSIVE VENTILATION (IPV®-VDR®) AS A LUNG RECRUITMENT AND MAINTENANCE VENTILATION PROTOCOL WITH A CONCEPTUAL LUNG PROTECTIVE STRATEGY.

The Percussive ventilation of the lungs is directed toward increasing Alveolar ventilation and provides for a powerful means of lung mobilizing through Bronchiolar “Lung Recruitment” and the stabilizing of the recruited peripheral pulmonary airways.

Concomitant with Percussive pulmonary gas exchange, a dense therapeutic aerosol is topically delivered throughout the endobronchial structures. The percussive higher rate (frequency) delivery of sub tidal volumes of respiratory gases into the pulmonary structures serves to provide for a “Newtonian” pumping action within the elastomeric endobronchial airways to mobilize and raise endobronchial secretions. Unique IPV® programming can accommodate all patient populations from neonates through pediatrics to large adults.



As the patient breathes through a mouthpiece, mask or endotracheal tube, the IPV® Percussionator delivers mini-bursts of respiratory gases into the lungs at selected rates of from 100 to 300 cycles per minute.

Generally, the patient only takes a breath when physiologically mandated and/or desired, allowing the Percussionator® to do the patient's "work of breathing". During the percussive bursts of gas delivered into the pulmonary airways, a continuous endobronchial pressure wedge is maintained.

While pulsatile intra-airway oscillatory pressure changes occur, between baseline and the oscillatory plateau, the Bronchial airways are maintained in a semi-dilated state as cyclic pressure changes occur.

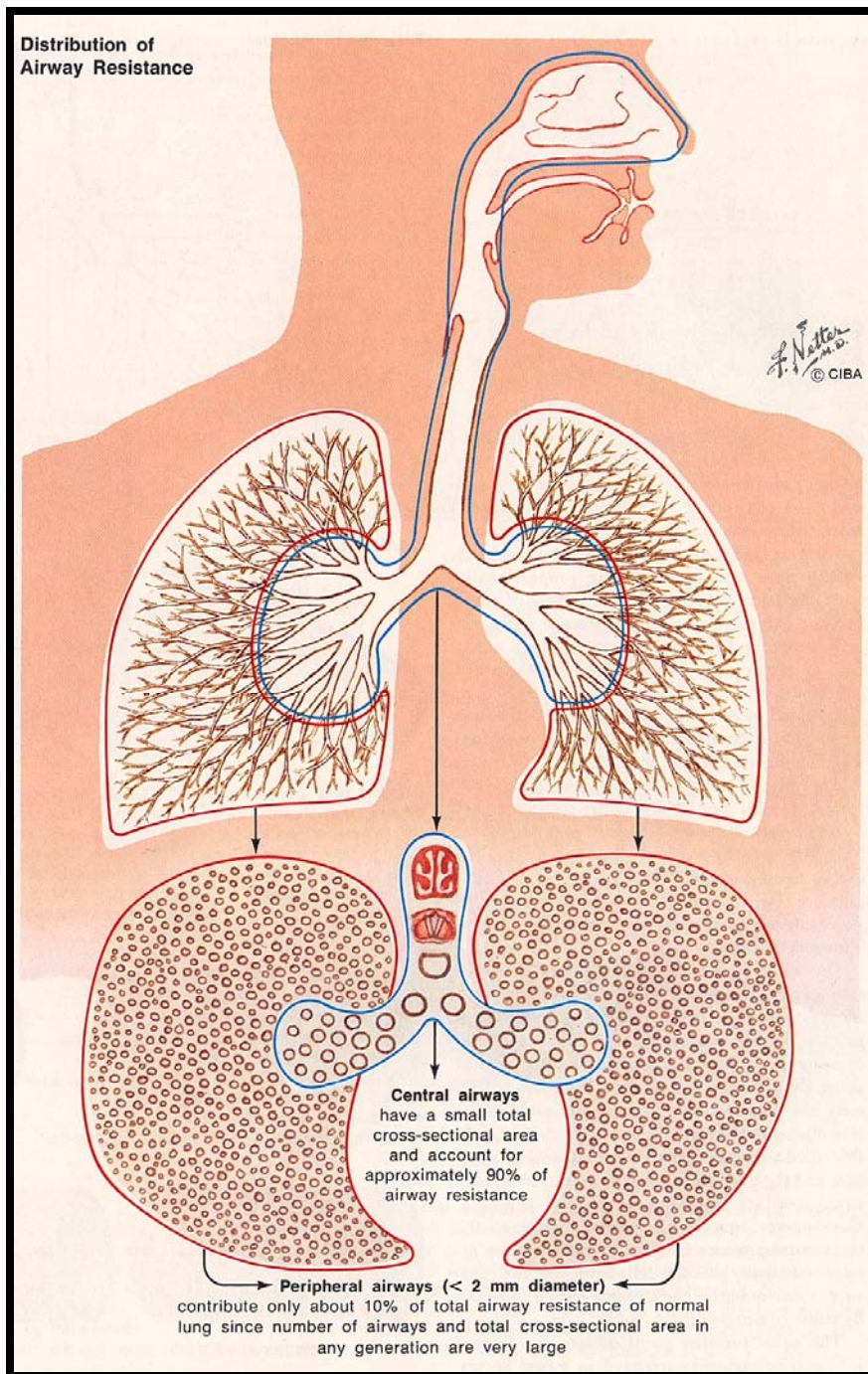
During therapeutic percussion an aerosol mist is delivered throughout the lungs by the high output aerosol generator of the Intrapulmonary Percussionator® breathing head assembly.

Aerosol misting within the endobronchial airways reduces the adhesive and cohesive forces of the retained secretions, decreases swelling within their walls, and relaxes potential spasm of the terminal bronchioles (small airways) of the lungs. The pulsatile intrapulmonary exchange of well-mixed respiratory gases serves to convectively flush out Carbon Dioxide and diffusively renew Oxygen.

Uniquely, millisecond IPV® sub tidal intrapulmonary injections do not hold a sustaining CMV Tidal Exchange positive pressure against the pulmonary airways in seconds. This is a conceptual means of maintaining a LUNG PROTECTIVE STRATEGY with IPV®-VDR® scheduling to prevent hyperinflation barotrauma.

Notes:

PULMONARY HYPERINFLATIONAL BAROTRAUMA CAUSE AND EFFECT.



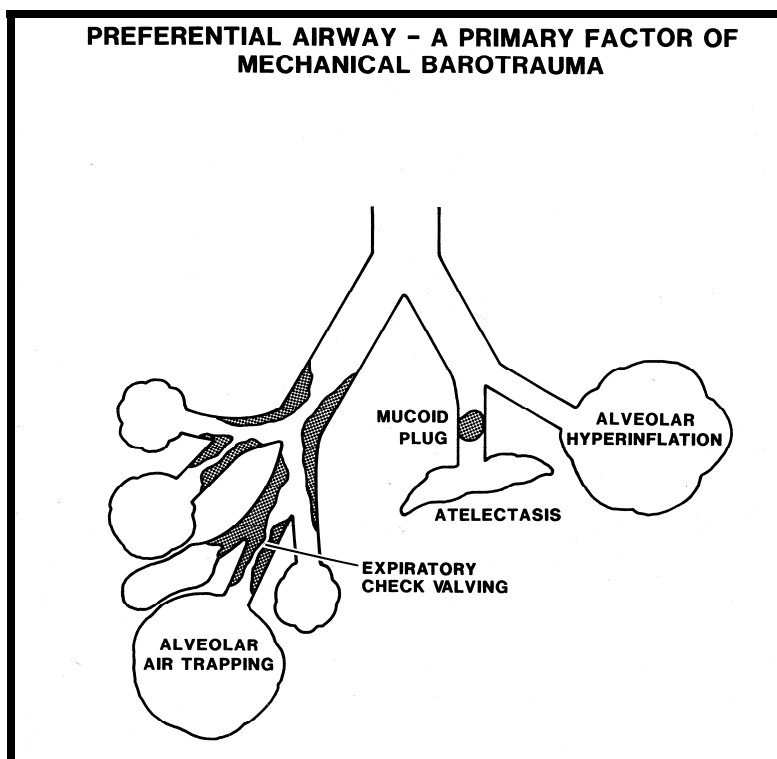
Pulmonary Airways by Netter courtesy of Ciba®

A brief study of a general cross section of the pulmonary airways reveals the potential millions of small peripheral bronchiolar airways.

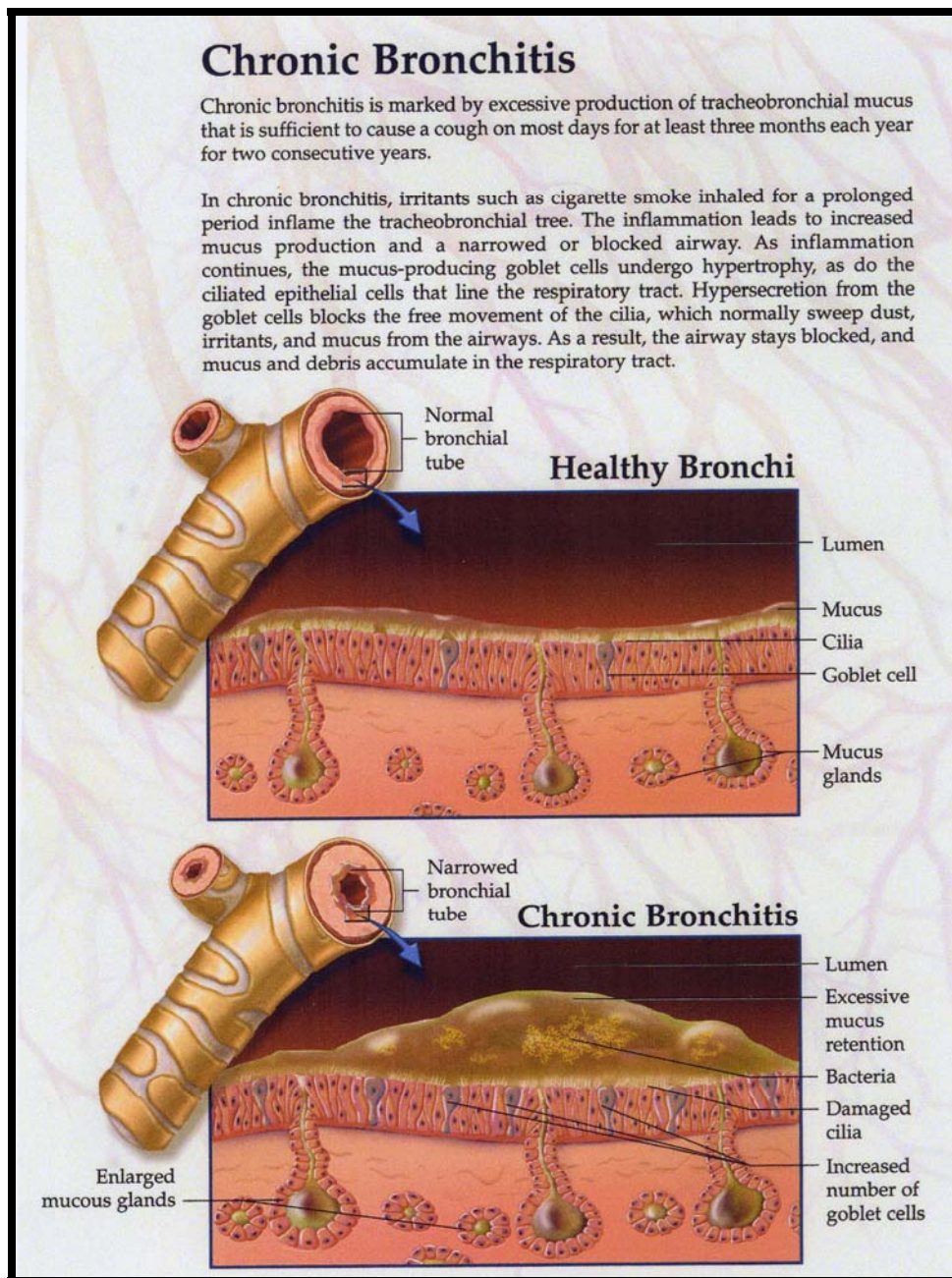
Patients with Bronchitis associated with the various types of peripheral lung diseases have a diffuse mix of Bronchiolar airway lumens (caliber). Certain bronchioles have little or no airway narrowing while others have airways with minor to severe obstructions caused by endobronchial edema and retained airway secretions as well as other factions.

If a mechanically induced timed positive distending pressure during volume-pressure CMV lung ventilation is held against the cross section of diseased Bronchial airways with various patencies (degrees of obstructions) and intermixed “non-obstructed Bronchiolar airways”, the PREFERENTIAL (non obstructed airways will be first to be mechanically inflated to the peak positive distending pressure.

During a mechanical tidal volume delivery, the intermingled non-obstructed Bronchial airways will allow a PREFERENTIAL inflow into their alveoli in the presence of other more obstructed Bronchioles. These unobstructed Bronchiolar airways are called PREFERENTIAL Bronchiolar airways. When scheduled intrapulmonary tidal volumes are delivered under moderate or high peak pressure limits secondary to gross endobronchial resistances, the open (non-obstructed) Bronchiolar airways are initially PREFERENTIALY hyper-inflated. The hyperinflation creates alveolar barotraumas leading to pneumothoricies. Thus the dependent (functioning) alveoli are the first to be damaged by hyperinflation encroaching upon the patient’s disease limited life support pulmonary reserves. Thus, volume-pressure CMV ventilation of patients with peripheral obstructive lung diseases can cause critical injuries.



THE ABOVE SCHEMATIC DEMONSTRATES THE VARIOUS DEGREES OF PERIPHERAL PULMONARY AIRWAY OBSTRUCTIONS AS WELL AS THE NON-OBSTRUCTED PREFERENTIAL AIRWAY. NOTE THE POTENTIAL FOR MECHANICALLY AGGRAVATED "ALVEOLAR AIRWAY GAS TRAPPING".



The above drawing reveals the lumen (patency) of a non-obstructed PREFERENTIAL Bronchial airway and the Bronchiolar airway narrowing by obstructive edema and retained airway secretions. Additionally, the Goblet cells secreting thick tenacious mucous secretions, which, if retained, provide for bacterial culturing.

ROUTES OF PHARMACEUTICAL DRUG ADMINISTRATION-

- A. Can be taken orally for systemic absorption, in pill form, and/or injected or infused intravenously into the systemic blood circulation.**
- B. Can be delivered into the pulmonary airways of the lungs as a topical aerosol (mist) for absorption by the circulatory blood vessels within the walls of the pulmonary airways as well as transported by the Lymphatic circulation from the lungs centrally into the systemic venous blood circulation.**

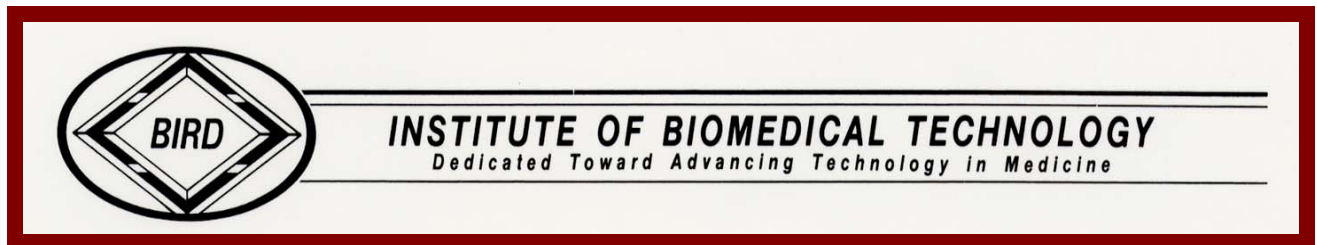
The clinical effectiveness of the aerosolized drugs is related to the “site delivery” of topical aerosol particles, which is in part, dependent upon the depth of aerosol particulate delivery within the pulmonary airways. Typical nebulizers (aerosol generators) “rain out” the aerosol particles before they reach the peripheral bronchiolar airways. Therefore, the main effect from the aerosolized drugs is created by the systemic absorption of the aerosolized pharmaceuticals into the systemic circulation and not the desired endobronchiolar topical delivery effect.

THE NATURE AND ACTION OF TYPICAL COPD MEDICATIONS-

- 1. Anti-histaminics- Primary side effect (predominately in males) is difficult urination.**
- 2. Steroids including synthetics- defined as anti-inflammatory agents, serve to mask Bronchiolar inflammatory processes.**
- 3. Beta Bronchodilators- Serve to relax Bronchiolar smooth muscle contractions associated with asthma.**
- 4. Alpha-Beta Agents such as Racemic Epinephrine-**
 - A. As an Alpha serves as a vasoconstrictor (with minimal rebound effect) to reduce bronchiolar mucosal and sub mucosal edema.**
 - B. As a combined Beta serves to act as a Bronchodilator to relax Bronchiolar smooth muscle spasm in Asthmatic patients.**
 - C. Using water as a diluent creates an osmotic endobronchial pressure, causing a more rapid endobronchial Alpha absorption into the bronchiolar basement membranes to reduce the causes of Bronchiolar airway edema.**

Note: Many years of experience with aerosolized aqueous Alpha Beta Racemic Epinephrine solutions delivered during Intrapulmonary Percussive Ventilation (IPV®) have demonstrated a lengthening of the time the pulmonary bronchioles remain recruited after routine IPV® therapy.

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Document ID: F-110909 FMB