

THE FIFTY YEAR HISTORY OF
the involvement of
Forrest M. Bird, M.D., PhD., ScD.
in
THE RESPIRATORY HOME CARE MANAGEMENT OF
COPD
LEADING TO THE DEVELOPMENT OF THE
HOME THERAPY (HT™) IMPULSATOR®

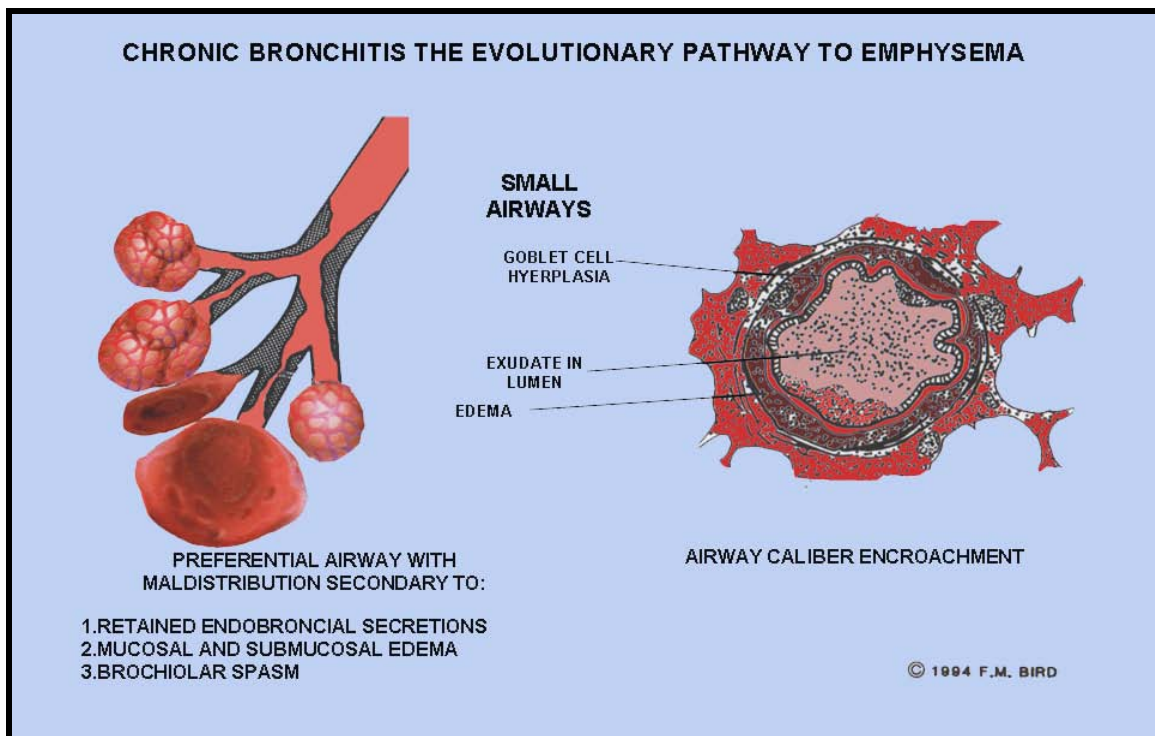
JUSTIFICATION

Peripheral lung disease often called COPD inflicts patients in all age groups, which have various degrees of narrowing within their small Bronchiolar airways that transport air in and out of the pulmonary Alveoli. Typically, within a cross section of the diseased Bronchiolar airways there are various degrees of Bronchiolar airway restrictions from minor to those that are totally obstructed. Additionally in the same cross section, there will be randomized Bronchioles that are normal with functioning Alveoli which are called PREFERENTIAL AIRWAYS. The Preferential airways transfer respiratory gases in and out of what are called the DEPENDENT ALVEOLI defined as the Alveoli serving to provide the sole source of pulmonary oxygenation.

Bronchitis, is a lung disease that can become increasingly chronic from childhood into middle age. Bronchitis patients are prone to have

a disproportionate number of chest colds with increasing involvement with aging.

Over time the patient's become increasingly short of breath after each pulmonary infection until they develop major end stage lung disease called Pulmonary Emphysema.



Bronchitis patients are prone to have a disproportionate number of chest colds, with increasing involvement, as they get older. Over time the patients become increasingly short of breath after each infection as they insidiously enter into the development of the end stage lung disease called Pulmonary Emphysema.

Any tissues in the body that do not maintain adequate blood supply are said to be ischemic.

A persistent ischemia (lack of circulation) will lead to a tissue necrosis (deterioration).

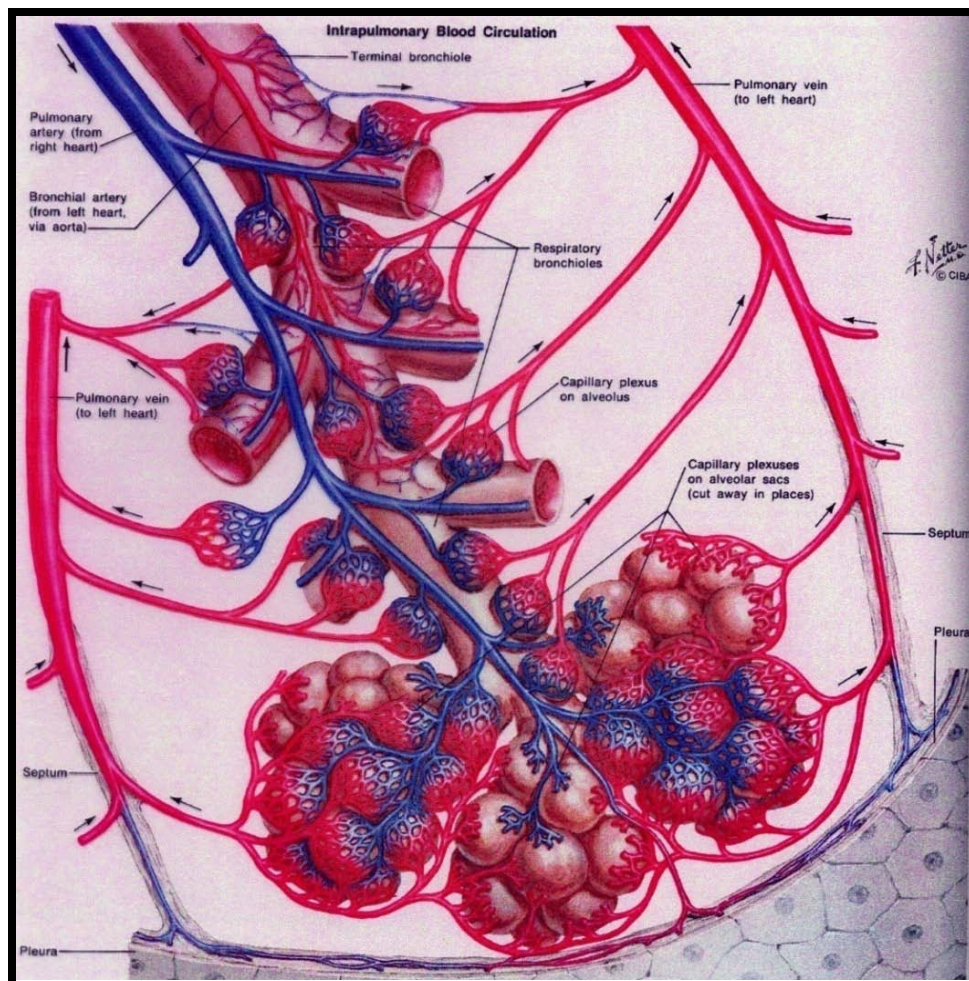
The pulmonary (breathing) structures within the chest have their own blood supply called the Bronchiolar circulation which normally receives about 2% of the arteriolized blood from the left ventricle of the heart to nourish the lung tissues.

During inspiratory breathing the pulmonary airways become larger as the patient inhales to fill the pulmonary alveoli (air sacks) of the lungs.

The deeper the breath the more the airways expand). During exhalation, the "inspiratory lung inflationary muscles relax" and the elastomeric fibers (and surface tensions) within the walls of the pulmonary alveoli and airways normally contract, causing the alveoli and airway contraction to generate an "outflow of air from the lungs".

The Pulmonary and Bronchial circulatory blood supplying vessels within the lungs are attached to the outer walls of the pulmonary airways and the alveoli of the lungs. During inhalation the pulmonary airways increase in diameter (expand), this causes the "pulmonary blood vessels attached to their outer walls to be stretched and narrowed decreasing the amount of blood in the lungs". During exhalation as the pulmonary airways passively contract (get smaller) "the attached blood vessels get larger and refill with blood". This is a normal physiological occurrence.

Notes:



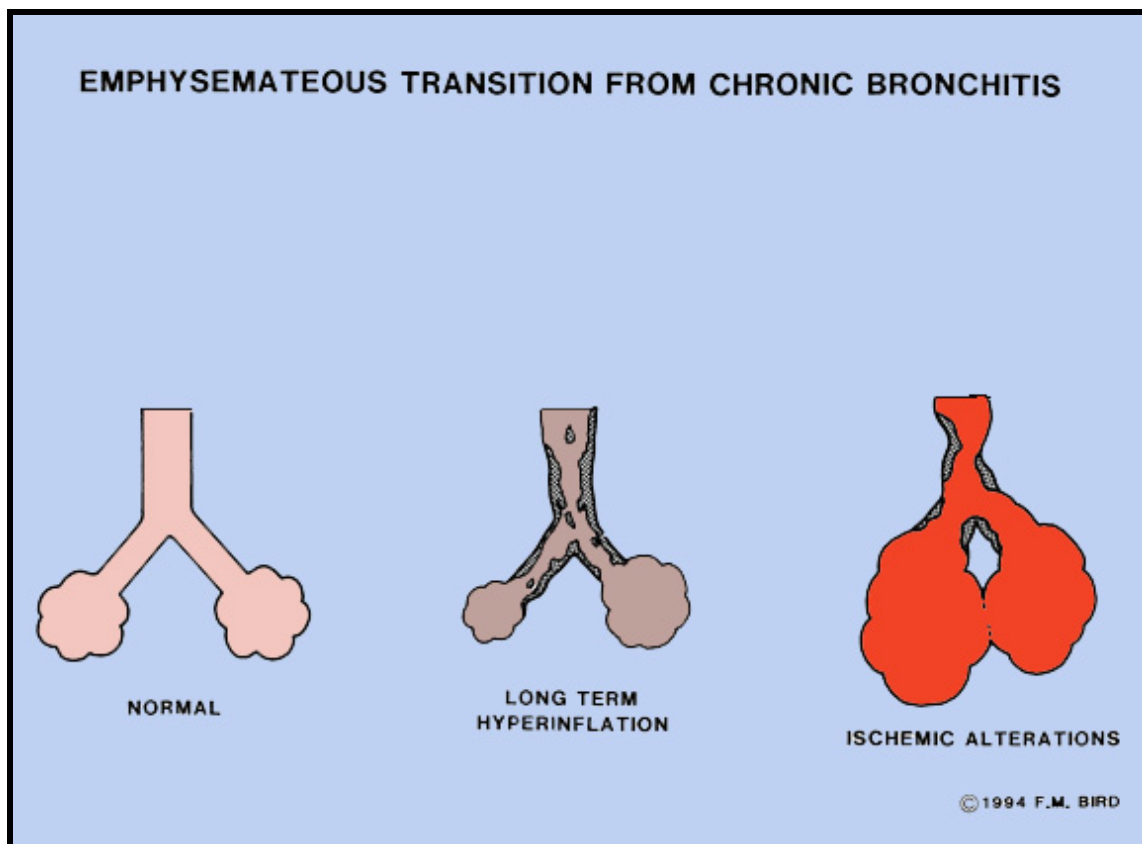
Pulmonary circulation by Netter courtesy of Ciba®

Patients with peripheral lung diseases like Bronchitis, Emphysema and others, have narrowing within their Bronchiolar airways causing the narrowed pulmonary airways to become obstructed during early exhalation, "before the normal amount of alveolar deflation has occurred". This causes the Bronchiolar airways and their Alveoli to remain partially inflated. Partial airway inflation increases resistances to blood flow through the Pulmonary and Bronchiolar blood vessels; which will be determined by the degree of airway stretching and narrowing caused by the obstructive pulmonary airway disease.

Over time, as this population of patient's age, they experience an advancing chronicity (worsening of their disease), which can be exacerbated (increased) by transient acute pulmonary infections.

Alveolar Air Trapping is a component of COPD increasing with pulmonary infections, which together can ultimately cause an insidious decrease in Bronchiolar Blood flow, creating a diffuse

pulmonary tissue ischemia. This is the classical textbook pathway toward the progressive end stage lung disease Pulmonary Emphysema.



Generally, when the historical treatments of a disease process has been studied; it has revealed "that in most cases" the initial treatment protocols were directed toward the amelioration (getting better) of the "symptoms and not at the cause of the disease".

Historically, in 1945 Dr. Bird converted a prototype positive pressure U S Army Air Corps, airman's breathing regulator to the first "manual intermittent positive pressure breathing respirator" for the lung treatment of a fellow pilots WW 1 Emphysemateous father.



Dr. Bird's manual operated positive pressure breathing device delivered a Racemic Epinephrine vasoconstrictor-bronchodilator aerosol deep into the pulmonary airways. This served to deliver a therapeutic aerosol into bronchiolar airways (that could be recruited) providing the patient with a more effective aerosol delivery than the available hand bulb nebulizers. This concept was also effective in mobilizing and raising retained endobronchial secretions.

By 1957 "with a medical education" Dr. Bird had developed an advanced automatic assister controller respirator for the treatment of patients with lung injuries and/or failures, including cardio-respiratory diseases.



BIRD MARK 7 RESPIRATOR 1957

The Bird Mark 7 Respiator became the world standard for in hospital acute or chronic COPD management.

In the 1960's Dr. Bird's wife Mary was diagnosed with advanced chronic bronchitis and diffuse associated pulmonary Emphysema agravated by an Alpha 1 indication. Dr. Bird felt his manual IPPB device was the most effective lung secretion mobilizing device for COPD patients when in skilled hands. However, the automatic Bird® Mark® 7 universal respirator when programmed for pulmonary airway secretion mobilization and lung ventilation might be eqally effective.

Dr. Bird's Emphysemateous wife served to cause Dr. Bird to become dedicated toward the study of the disease and the development of a home care IPPB type respirator; providing for an advanced airway secretion clearance, with the ability to ventilatorily "wash out CO2". This led to the conception of a number of home care respirators for the mobilization and raising of retained endobronchial secretions.



By 1965 Dr. Bird's PORTABIRD® had become a standard for the home care of COPD patients.

During the next twenty some odd years "Bird Corporation" manufactured tens of thousands of IPPB devices for world wide hospital and home patient care. In fact IPPB was grossly over used in many non COPD hospital environments.

In 1978 Dr. Bird merged his expanding Bird Corporation into the 3M Company, returning to full time aeromedical research and development.

By 1978 Dr. Bird's wife Mary while being provided comfort from the symptoms (of her advancing pulmonary Emphysema) with IPPB home therapy, was becoming increasingly chronic (worse) with advancing diffuse Pulmonary Emphysema. Each acute pulmonary infection was a constant threat to her life.

By 1978 Pulmonary Emphysema had been clinically determined to be an ischemic endobronchial end stage lung disease. There was little doubt that the perfusion of the pulmonary (lung) tissues by the Bronchial circulation was selectively being encroached upon by Bronchiolar airway and Alveolar air trapping (not being able to exhale adequately) "causing secondary compressional narrowing and associated resistance to blood flow within the involved Bronchial vessels" of the lungs.

It is well known that patients with typical Asthma rarely if ever became emphysemateous, while patients with Bronchitis almost always (over time) become Emphysemateous. Peripheral obstructive bronchiolar lung diseases are associated with alveolar air trapping. However, the basic clinical difference between Asthma and Bronchitis is; the Asthmatic patient has periodic (once in a while) alveolar air trapping; while the Chronic Bronchitis patient has unrelenting (all the time) alveolar air trapping, which over time will encroach (limit) upon diffuse Bronchial circulatory, blood flow through the lungs.

Therefore, the treatment of the patient with chronic bronchitis should be directed toward a daily routine therapeutically created remission from the constant alveolar air tapping.

In other words, therapeutically make a typical asthmatic out of the patient with chronic bronchitis to prevent the progressive impairment of the Bronchial circulation, terminating in end stage lung disease (Pulmonary Emphysema).

Historically, Back in 1921 Dr. Waters et al at the University of Wisconsin proved that when a patient received rapid bursts of air into the lungs, it provided a measurable pulsatile surge in systemic arterial pressures (like the systolic beat of the heart).

After the 1978 merger of his (totally owned debt free) Bird Corporation into the 3M Company Dr. Bird was left personally unencumbered with his extensive available aeromedical R & D facilities. Dr. Bird's wife Mary provided him with a major stimulus to immediately discover a method to recruit the Bronchioles and Alveoli of COPD patients. Additionally, the therapeutic means to support the three intrathoracic circulations, namely the Pulmonary, Bronchial and Lymph was of vital importance. The primary therapeutic direction had to be directed toward the maintenance of the Bronchial circulation within the lungs.

Dr. Bird's goal was to discover a means to deliver continuous bursts of gas into the lungs with such a velocity they would reach the Bronchiolar level with a reserve of energy without causing hyperinflation barotraumas. This logic had been previously approached by Dr. Bird and others however; it was well known that high percussive small bursts of air into the lungs cause a Hering Breuer stretch reflex reaction, causing the patient to enter into a suppressed cough reaction.

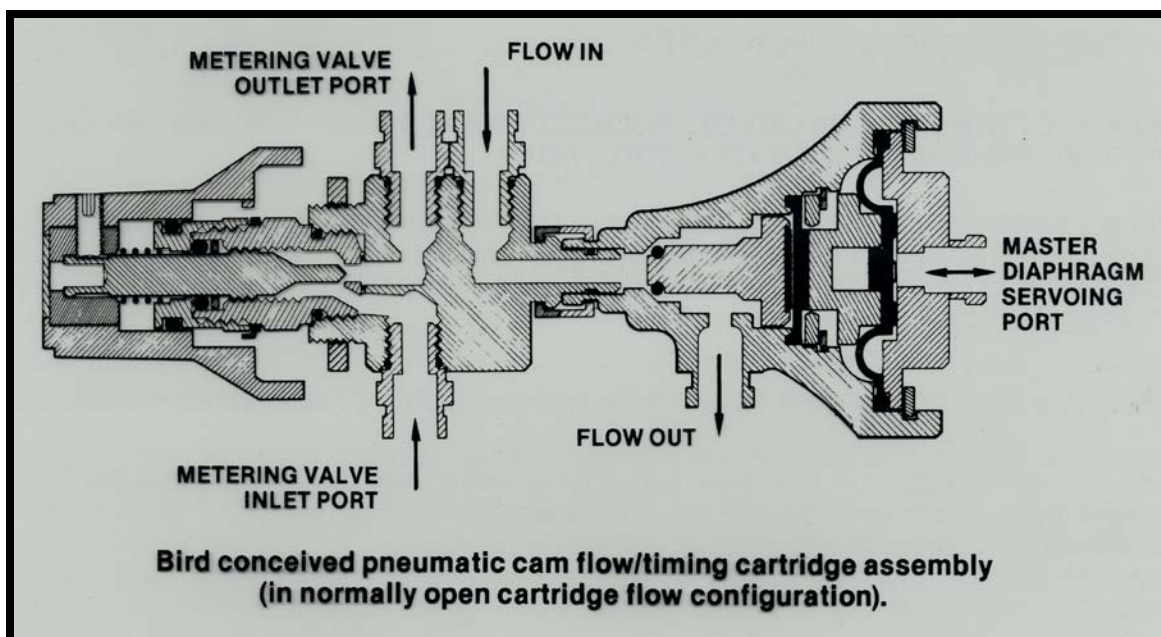
Dr. Bird was well aware that the odds of overcoming the patients intrapulmonary stretch receptor re-action as well as, creating such a percussive apparatus was a very great challenge with little if any promise of a monetary reward. Dr. Bird's wife was reaching an end stage Emphysema fighting for her eighteen thousand daily breaths. Mary's medical condition eliminated all other rationals for not persuing a method of Peripheral Lung Recruitment.

In the early 1980's "high frequency mechanical respiration" was literally promising the total resolution of all respiratory challenges. Dr. Bird had earlier looked at high frequency ventilation with a special variation of his Mark 7 Respirator and found that the high frequency diffusive ventilatory concept could not "wash out" the patients normal Carbon Dioxide from the lungs, ultimately causing a CO₂ intoxication. The majority of the medical community was led to believe that Dr. Bird was investigating a typical diffuse high frequency means of pulmonary ventilation.

Dr. Bird had one of the finest aeromedical related machine and mold making facilities available, plus he had several highly experienced tool and die makers as well as himself, to conceive and develop the

required components necessary to create an apparatus capable of percussively delivering Sub Tidal volumes of air into the pulmonary airways.

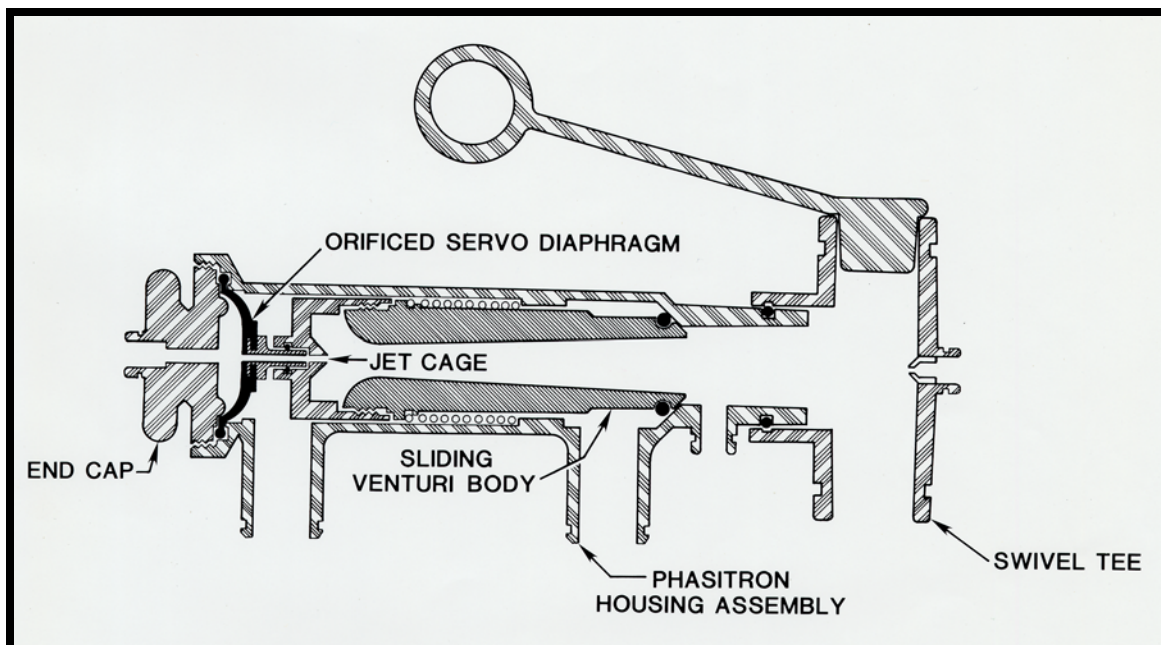
The first component that had to be created was that of developing a reliable pneumatic valve that “would cycle full stroke open and full stroke closed” which could cycle for billions of strokes without self destructing, (which had earlier eliminated electronic solenoid actuated valves). Dr. Bird’s precise pneumatic valve and molecular metering device was successfully prototyped and refined.



The next major challenge was creating a device that would serve as a respirator at the patient's proximal airway (mouth).

This device would have to inject Sub Tidal volumes (small air bursts) of air in milliseconds at high velocities down the patient's pulmonary airways with enough velocity to reach the patient's pulmonary alveoli.

Notes:



The PHASITRON® allowed Intrapulmonary Percussive Ventilation

The Phasitron took almost four years to reach a point of clinical and operational perfection. This enabled what Dr. Bird would call Intrapulmonary Percussive Ventilation (IPV®).



Dr. Bird's wife Mary was the first patient on the first prototype of the IPV® apparatus. At that critical time, Mary was near terminal with an

acute infection, when she started on the prototype IPV® device. Within several days, Mary was dramatically improved. Mary continued to improve including again driving her car. Later she received the first IPV®-1 Percussionator® above. Dr. Bird started several other long term patients on the initiating prototype IPV®-1 percussionators® to which they responded similar to Mary.

Beyond IPV® Dr. Bird continued to complete his critical care Percussionator® devices and protocols which he would name Volumetric Diffusive Respiration (VDR®). This concept rapidly distinguished itself in patients failing conventional volume-pressure oriented CMV ventilators. The VDR® Percussionator was able to recruit the lungs of new born babies to large adult patients when all other available means of mechanical lung ventilation had failed.



An immature newborn baby with heart-lung problems, may spend weeks on VDR® cardiopulmonary support with expected survival.



A severely burned baby on VDR who would go on to survive.



Dr. Bird created and improved the self contained heavy Impulsator at the request of his wife Mary. This enabled Mary to travel by car from her Palm Springs, California home to Idaho, for several summers.

Mary took at least four or more IPV® treatments daily which enabled her to live a near normal quality life.

After about three years Mary was requiring an IPV® treatment about every three hours, to allow her to maintain the same activity levels that she had required, two year or so earlier.

Finally, on Labor Day in 1986, Mary succumbed to a major acute atypical pulmonary influenza type infection; about four years beyond the critical state she was in when she started IPV®.

Dr. Bird's critical clinical appraisal of IPV® at the passing of Mary was:

1. IPV® does recruit and substantially maintain the bronchiolar airways and their alveoli that have not become Emphysemateous, providing for a much improved life style.
2. IPV® does not re-activate any advanced ischemic components of the Bronchiolar circulation. However, it may retard the rate of Bronchiolar vessel ischemia by initially recruiting certain long obstructed Bronchiolar airways.
3. IPV® can not after a certain point of emphysemateous lung decompensation, recruit a lung with a major advanced debilitating pulmonary infection.
4. IPV® must be started before a patient with typical Bronchitis has sufficient loss of their Bronchiolar circulation to have reached a critical point of NO RETURN concerning the perfusion of their three Intrathoracic circulations, namely, Pulmonary, Bronchiolar and Lymph.

Following and studying the work of "Waters in terms of a Pneumatic Systole" which Dr. Bird et al named "Intrapulmonary Vesicular Peristalsis" which was created by the directional Peristaltic Compressional Waves, associated with each PERCUSSIVE Sub Tidal volume delivery. This higher frequency systolic type, circulatory flow boost is accomplished by a mechanically generated

percussive pulsed enhancement to the “physiological Venous Pump” technologically called (intrapulmonary vesicular peristalsis).

Now some twenty five years later with thousands of IPV® home care Impulsators® serving COPD patients world wide; a vast amount of clinical home care has been subjectively and objectively collected and summarized.

First- Some fifty percent of home care patients do not maintain their personal health programs as prescribed, such as only using their IPV® devices when they do not feel well.

Typically this group of COPD patients take IPV® treatments when a passing storm drops the barometric pressure, making them short of breath and/or when they have infections such as colds etc.

1. Patient's who do take personal care of themselves and use their IPV® therapy as prescribed:
 - a.) Report they feel near immediately better; then within weeks, are able to live a much improved quality of life.
 - b.) Report that they previously had to be hospitalized with acute infections at least once or more a year. IPV® has eliminated their expected acute care hospitalizations.
 - c.) With home care IPV® regimes COPD patients are able to increase their rate of their daily IPV® home treatments when needed, with no threat of repeated acute care hospitalizations.

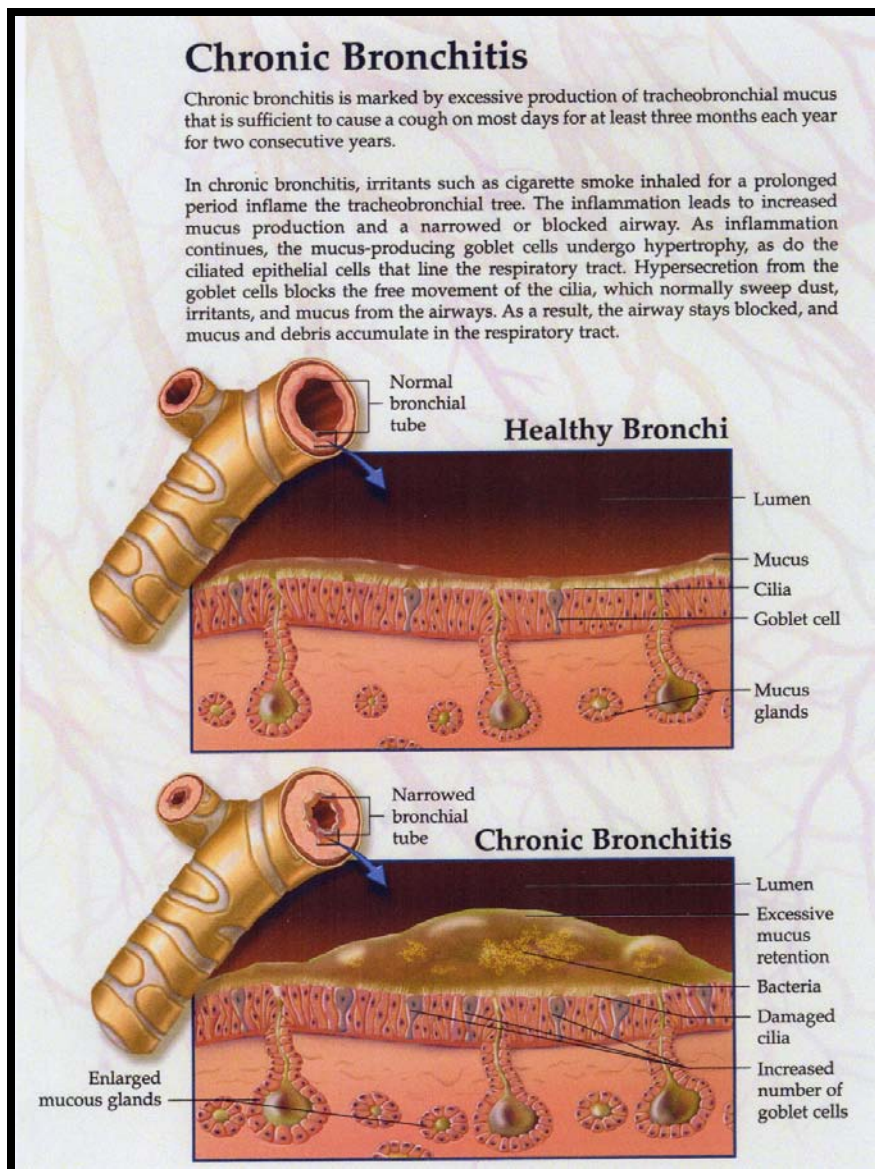
Most important, a number of early bronchitis patient's who started taking IPV® treatments prophylactically while maintaining personal health maintenance programs; have remained a-symptomatic. They had not been hospitalized by any acute pulmonary infections, which were resolved by increasing their daily routine IPV® schedules, until they were over their acute infections. “This data would serve to

indicate it is the insidious ischemic break down of the Bronchiolar circulation that enhances the insidious progression of peripheral obstructive lung diseases.

**A REVIEW OF MEDICAL TEXTBOOK PATHO-PHYSIOLOGY MAY REVEAL
HOW BRONCHITIS BECOMES INCREASINGLY CHRONIC.**

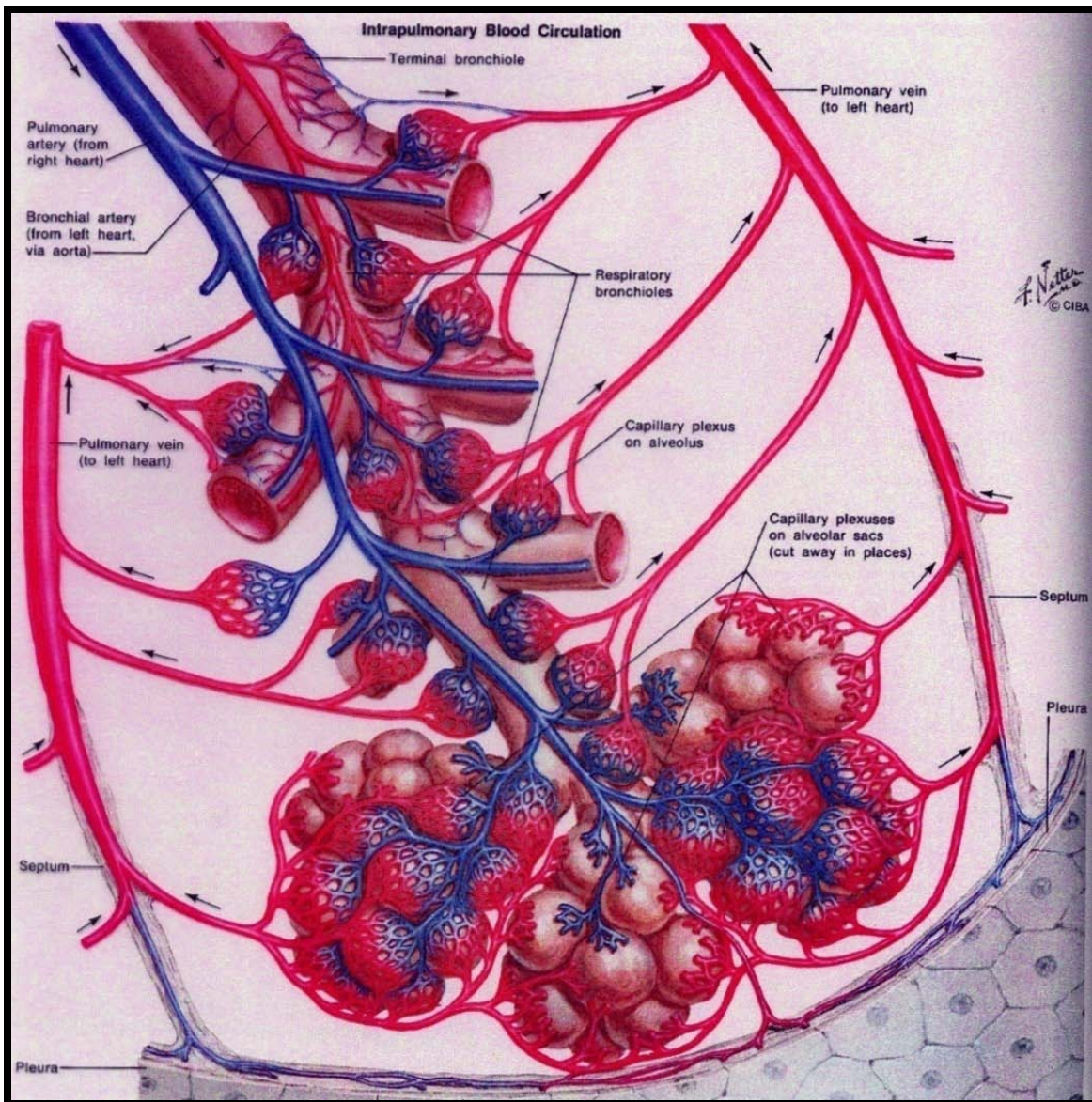
An insidiously increasing component of Chronic Bronchitis is hyperinflated (partially inflated after exhalation) Bronchiolar airways as well as hyperinflation within their attached alveoli; which cause their externally attached Bronchial and Alveolar blood vessels to be narrowed and constricted, creating diffuse Bronchial blood flow restrictions (perfusion), thus reducing normal perfusive blood flow to the tissues of the lungs.

Notes:



While the average clinician may understand perfusion (blood flow) within the major intrathoracic pulmonary circulation, the minor intrathoracic Bronchial Circulations are of lesser interest to certain clinicians.

Notes:



THE PULMONARY CIRCULATION BY NETTER COURTESY OF CIBA®

When the bronchial airways and their attached alveoli are expanded, their attached circulatory vessels are stretched and narrowed.

Guyton, in his textbook on "Medical Physiology" describes the bronchial and lymph circulation as follows:

THE BRONCHIAL VESSELS: A minor accessory arterial blood supply to the lungs exits directly from the aorta through usually one bronchial artery to the right lung and two bronchial arteries to the left lung.

The blood flowing in the bronchial arteries is oxygenated by "arterial" blood, and it supplies the supporting tissues of the lungs, including the connective tissue, the septa, and the large and small bronchi. After

this bronchial arterial blood has passed through the supporting tissues, it empties into the pulmonary arteries and veins and enters the left atrium rather than passing back to the right atrium. Therefore an average of about 1 percent (but on rare occasions as high as 10 to 50 per cent) more blood flows through the left side of the heart than through the right side.

THE LYMPHATICS:

Lymphatics extend from all the supportive tissues of the lung to the hilus pulmonis and thence into the thoracic duct. Particulate matter entering the alveoli is usually removed very rapidly via these channels, and protein is also removed from the lung tissues, thereby preventing extra alveolar edema.

With a primary understanding of the transition from bronchitis to chronic bronchitis to end stage emphysema, does it not make more sense to prophylactically start a therapeutic regime directed toward limiting the progression of the disease process? This must be accomplished by an educational alliance between the Patient and the Clinician.

By applying oversimplified logic, would it not be advisable to project a therapeutic regime on a daily basis that therapeutically recruits the peripheral lung, by reducing the processes that create mucosal and submucosal edema within the terminal bronchial airways "without masking the cause of the disease?"

The average patient with COPD is usually seen by their physician during their acute care hospitalization, which is generally resultant to an acute pulmonary infection "exacerbating their existing bronchitis. Thus, the patient may believe that his outpatient symptoms can be totally pharmacologically managed while they insidiously becomes increasingly chronic secondary to an ischemic Bronchial circulation.

INNOVATION FROM EXPERIENCE

By employing clinical and technical feed back information from thousands of patient's with various forms of heart-lung diseases over a

twenty-five year period, the data was carefully screened. The correlation of the data from hundreds of the thousands of long term HOME CARE COPD patients provided the clinical and technical data to innovate an advanced HOME THERAPY INTRAPULMONARY PERCUSSIVE VENTILATION (IPV®) system, from the existing clinically proven IPV® IMPULSATOR® concepts.

Additionally, in hospital and transport data using Intrapulmonary Percussive Ventilation (IPV®) derivatives have been carefully integrated into home therapy requirements. Medical ground and air evacuation (logistics) have revealed the importance of universal operational scheduling ease as well as, the weight and durability of the transportable therapy respirators.

The extrapolation of integrated data derived from the twenty-five year review of patient care, using Intrapulmonary Percussive Ventilation (IPV®) and Volumetric Diffusive Respiration (VDR®) have served to establish the following innovative clinical and functional design criteria for an innovative light weight Impulsator®. Typical of the suggestions were:

1. Reduce the weight and size while maintaining the clinical efficacy of the IPV® Impulsator.
2. Clinically conceive a means to allow the patient to easily create a manual Bi-phasic™ lung recruitment program; like the automated hospital intensive care VDR® sinusoidal scheduling.
3. Automate the air compressor energy generation. No gauges or knobs.
4. Quiet the air compressor, it currently makes too much noise.
5. The sterile hospital apparatus appearing housing of the current heavy Impulsator® should be replaced with a modern attractive light weigh box, which is easy to conceal within an attractive camera type traveling case.
6. The transport (carrying) case should have pockets to carry the needed accessories and medications.

7. Re-configure the current heavy Impulsator, patient thumb control over the selection of NEBULIZATION ONLY and/or INTRAPULMONARY PERCUSSION WITH NEBULIZATION to use the same control to manually schedule Bi-phasic™ VDR® (sinusoidal) lung recruitment.
8. Modify the patient treatment schedule to a very intuitive protocol enabling a patient with average intellect to schedule an effective IPV® clinical protocol. This could be done with simple written instructions, DVD programming, which can be supported by an Internet program.
9. Certain potential home care patients who are not bound by third party pay such as medical insurance and/or governmental social service programming etc. might have direct access to the manufacturer. For example; why couldn't a credit card purchase of an advanced Impulsator® device be negotiated over the Internet etc. circumventing established third party pricing structures through a hospital type medical device distribution system.

Dr. Bird, after carefully studying the twenty-five year critiquing of the heavy Impulsator® commenced a study program to comply with the general comments. While the Impulsator® had long been in routine world wide home care and hospital services it was the continuing "state of the art" for a self contained IPV® Percussionator. The limiting factor was an air compressor, which was capable of creating sufficient air-flow volume and pressure to provide for an adequate endobronchial percussion and concomitant aerosol delivery to recruit the obstructed bronchioles and their associated alveoli.

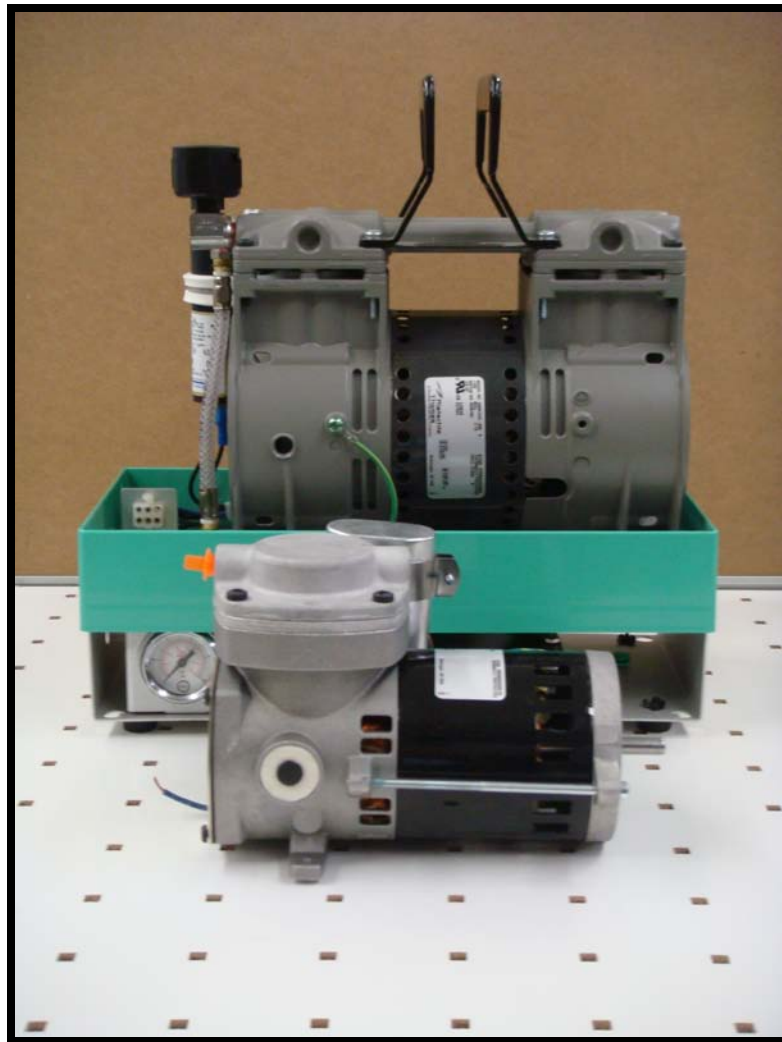
Dr. Bird had earlier attempted to use an aviation related air compressor with a much reduced air volume and pressure. However it required two of the smaller air compressors to produce a marginal flow pressure volume to maintain the percussive quality of IPV® therapy. This was not a viable option.

After extensive study Dr. Bird has “through innovation” created a prototype IPV® apparatus that could provide the clinical efficacy of the heavy IPV® Impulsator®, while utilizing a much smaller air compressor, with the reliability of the heavy Impulsator®. The following comparative data may explain the general logic behind Dr. Bird’s innovative developments leading to the creation of the light-weight Home Therapy (HT™) Impulsator®.

Technological conceptual developmental challenges, which were overcome to reach “a final design criteria”, for a productional HT™ Impulsator®:

1. The first requirement was, developing a light weight air compressor providing for an effective repetitive oscillatory higher velocity percussive Sub Tidal volume injection with a major decrease in required compressed air volume. This novel transformation was accomplished by:
 - A. Using a much smaller compressional piston area with an adjusted stroke, requiring much less motivational motor force during the compressional upstroke, to generate operational demand pressures with a lesser stroke volume.

Notes:



The heavy Impulsator® Air Compressor compared to the small mass of the light-weight HT™ Impulsator® air compressor.

Comparative advantages of the manual Bi-phasic™ IPV® versus the mono IPV® therapy system

1. In comparison, the low RPM (large piston) HIGH MASS WEIGHT (about 18 pound) compressor versus the high RPM (small piston) with a LOW MASS WEIGHT (about 6.5 pound) compressor.
2. By counting the rate of endobronchial Sub Tidal volume deliveries (for example), 250 injections per minute, then dividing the Sub Tidal deliveries 250 into the number of air compression strokes 3500 per minute, thus there would be about seven (7) air compression strokes for each Sub Tidal Volume injected into the patient's pulmonary airways.

During each compressor stroke there is an "energy spike" which is transported into the lungs by the modulation of each Sub Tidal Volume delivery. This would mean there would be about "seven (7) micro energy spike impactions against the endobronchial walls during each Sub Tidal volume delivery". This serves to decrease the elastometric permeability of the expanding pulmonary airways during inflation.

3. The compressed air volume produced by each compressional stroke of the compressor produces a micro impulse (energy burst), which impacts upon the endobronchial walls, during each Sub Tidal volume delivery. This "micro energy spike" serves to increase the dynamic elastance of the pulmonary airways.
4. The current (state of the art) mono IPV® IMPULSATOR® air compressor uses a substantial piston area creating a higher stroke compression volume than necessary for the required air generating demand thus mandating higher power demand and mass than necessary for the application.
5. The nominal air compression demand can be satisfied by an air compressor with considerably less compressional piston area (reducing stroke volumes) by increasing the rate of compressional stroke volume deliveries required to satisfy operational demands for a novel designed Bi-phasic™ IPV®.
6. Therapeutic peripheral lung mobilization and lung recruitment, (with ambient bleed off as currently done) to control the operational pressure denies the creation of an energy spike impulse to increase the dynamic elastance of the pulmonary airways.
7. Each percussive endobronchial Sub Tidal Volume delivery serves as an intrapulmonary transmission (carrying) vehicle to be modulated by the compressional energy spike (shock waves), created by each compressive piston upstroke. Thus the greater the rate of compressive stroke volume injections the greater the potential for endobronchial wall energy spike agitation.

8. The high temperature/pressure generated by air compression serves to near sterilize the compressed air. As the compressed air cools beyond the outlet of the Oscillator Cartridge, the near sterile water condensation is vented predominantly through the venturi orifice, partially humidifying the endobronchial Sub Tidal volume deliveries.
9. Thus, the Thermodynamic control over the internal heat dissipation (compressor cooling), is designed to prevent water condensation within the Percussion Cartridge and associated flow/time Cartridge components.
10. Percussive Sub Tidal volumes are further augmented by modifying the effective Phasitron® venturi flow/pressure entrainment ratios by increasing the velocity and volume of air flow from the venturi jet through the venturi aspirational throat geometry.
11. By computing the maximum required compressed air volume requirements within a Functional Oscillatory Band Width, of from about 80 to 500 intra-airway Sub Tidal injection per minute (with i/e ratios of about 1:2.5) optimal operational pressures of 30 to 35 psi, are required with a novel Impulsator® design.
12. The continuous mandated systemic air-volume generation for repetitive endobronchial percussive Sub Tidal volume flow/pressure injection can be controlled by balancing any excess compressor generated air volume, by purging the excess systemic air volumes into the nebulizer aerosol generating jet.
13. The nebulizer is capable of generating an aerosol far beyond that required for peripheral lung particulate deposition. Thus, the variation in excessive delivery of aerosol beyond optimal endobronchial airway deposition, is used to balance the systemic operational pressures which is of little, if any clinical consequence.
14. By the novel balancing and the augmentation of a lesser air compressor output, a light weight miniaturized mobile

Intrapulmonary Percussive Ventilation (IPV®) therapy system is enabled, reducing the logistical (carrying) stress imposed upon semi invalid patient's with chronic and acute cardiopulmonary diseases.

15. Patient operational ease while providing increased clinical efficacy is increased by:

A. The elimination of a Pressure Reduction Regulator and pressure Monitoring Gauge.

B. Operational flow and pressures do not have to be repeatedly regulated by the patient, to mobilize and then recruit endobronchial secretions, thus increasing operational ease.

C. Clinical Nebulization pressures are automatically regulated by the patient's Bi-phasic™ thumb control over the percussive amplitude during Sub Tidal volume deliveries, during both airway recruitment and airway secretion clearance.

16. Manometric monitoring of the patient's proximal airway pressures are eliminated, serving no therapeutic usefulness.

17. The selection of PERCUSSIVE frequency band width by the rotation of the only (patient access) control knob enables the patient or clinician to generally conform to the initiating size and severity of the patient's lung disease.

18. When the remote patient thumb activated Bi-phasic™ switch is at the lower Sub Tidal amplitude position before the patient selects an increased Sub Tidal impaction, there is a gradual amplitude increase. This is important when higher Sub tidal amplitude injection are programmed.

19. All of the above considerations allow unitized packaging with the following benefits over current "state of the art" containment:

A. Unitized box packaging can encapsulate the air compressor and oscillatory components as well as control access

within a semi closed sound reduction compartment, internally cooled by filtered convective air exchange.

- B. The top of the encapsulated compartment supports the electrical ON-OFF Switch, providing for terminated grounding with dual fused circuit power demand protection.
20. Access to PERCUSSIVE programming configuration and Breathing Circuit interfacing is provided by:
- A. Two independent "service sockets which provide for Bi-phasic™ percussion as well as nebulization.
 - B. A two position switch is labeled AEROSOL ONLY and Bi-phasic™ IPV® can be employed to produce a dense therapeutic aerosol alone and/or Sub Tidal volume delivery with Nebulization by means of a toggle switch labeled Bi-phasic™ IPV® and NEBULIZATION ONLY.
21. The REMOTE and GAUGE Service Sockets are removed in the HT™ Percussionator®, greatly simplifying the Phasitron® interfacing harness. Thus a two-channel web can be used to interface the patient Phasitron®- Nebulizer Breathing head assembly with the PHASITRON® and NEBULIZATION Service Sockets of the HT™ Impulsator® design.
22. The containing unitized container (box) is inserted within a typical weather resistant, padded, soft airline type travel pack to provide additional compressor noise obtunding, shock resistance if dropped, travel ease and obscuration of medical device transport. Accessory features of the Travel pack are:
- A. The encapsulating travel pack has pockets on three sides with an inclusive cover.
 - B. The left side pocket contains the electrical power cord, which is connected to the Power Switch through a grommet penetrating the inside wall of the fabric case and the wall of the device container with cord pull out protection.

- C. The front facing compartment accommodates the breathing circuit interfacing harnesses and medications.
 - D. A semi transparent pocket within the “flap cover” of the soft travel pack maintains the printed operational manuals.
 - E. The right facing end compartment accommodates the Phasitron® and Nebulizer breathing head components.
23. A Phasitron® Duo™ Breathing Head is interchangeable with the standard Phasitron Breathing Head.
24. The encapsulated IPV® Therapy System can be used within the institution, home or during vehicular travel when powered by direct ac 50 or 60 cycle and/or dc to ac power conversion; by a patient owner, professional institutional clinician administering to institutional patients, EMT etc. for mass cardiopulmonary casualty or localized respiratory care treatments.



THE Bi-phasic Home Therapy HT™ IMPULSATOR® travel pack.