

## Bronchial asthma. A scientific and critical preliminary essay

Bronchial asthma is a complex functional respiratory pathology; the study of which must be focused now from the same scientific perspective I have used to interpret the other pulmonary functional pathologies referred to above, since Nature is consequent with its Principles and Living Beings are dynamically integrated units, under common functional structures and common factors.

Let me first refresh a few pertinent fundamental concepts, developed by me, in order to ease the understanding of my contributions to the important theme of Asthma,

1. It is well known that the Vagus-Sympathetic autonomic System innervates the Lung and extrapulmonary airways

2. I have proved that the Lung's well-defined structure: *lobes* and *lobules* perform complementary and integrated specific mechanical functions, under Vagus and Sympathetic nervous control, leading to balanced gas exchange with the blood.

3. The *lobar bronchial trees* are innervated by the Vagus, causing their muscles cyclic contraction, being mainly concerned with atmospheric air intake, its displacement, acclimatization and balance to *conditions required by the lobular bronchioles*, simultaneously *balancing blood circulation*.

4. The normal effects produced by the muscles of the airways contraction-relaxation result in required balance of the intrapulmonary air masses in

their different sectors, and this balance is relative to the atmospheric air mass per volume unit, which in turn is relative to altitude and the tiny volume-mass required by the alveolar units.

5. The Vagus nerve and its impulse mediator Acetylcholine, is a bronchi-constrictor, as also are synergic drugs like *Histamine and Methacholine among others*, thus being these latter potential causes of Asthma

6. The *lobular bronchioles* are concerned with the rhythmic final balance of air and blood for gas exchange, under Sympathetic-Adrenaline control. Artificial stimulation of this sub-system, as well as artificial supply of Adrenaline or adrenergic drugs, erroneously considered as broncho-dilators, cause an increase in the rhythm and force of contraction, with pathological results, asphyxiation even death (Iatrogenia).

Some well-known Sympathetic-Adrenaline-synergic drugs are Beta 2 agonists, Theophylline, among others.

Consequently, when talking of agonists or antagonists it is necessary to make clear *agonist to which subsystem?*. Since confusion could be and has been fatal.

I would like to refer here to some conclusions of the Conference "Advances in the Understanding and Treatment of Asthma", published on Annals of the New York Academy of Sciences. Volume 629. Part 1.

## Control of Asthma with emphasis on Prevention of Fatalities.

Page 3. Paragraph 1. “The basic abnormality of Asthma is *unknown* and the only common physiological-pharmacological abnormality is *bronchial hyperactivity* as clinically shown by excessive response to low doses of agonists such as histamine or methacholine” .

These drugs are agonists with Vagus-Acetylcholine sub-system.

“Table 1. Shows that the *common method of asthma’s control is the use of inhaled beta agonist or cromolyn*” and in a note below express “*Bronchodilators: inhaled or oral beta agonists*”.

It is of utmost importance to make clear now the traditionally transmitted error in relation with bronchi-constriction and **constrictors** on one hand, and bronchi-**dilatation** and bronchi-**dilators** on the other.

The traditional confusion derives from the fact that the mechanical role of the lobular bronchioles, and their innervations by Sympathetic nerves have been ignored and clinical observations have also been wrongly interpreted; therefore its contribution to Iatrogenia.

In another paragraph it reads, “Iatrogenic causes of death are not rare” ...attributing it to” toxic effects to Beta 2-agonists and Theophylline”

I must clarify in this context that the above named products are *agonists to Sympathetic adrenergic System*, whose action and effects on the Lung I have unveiled and analysed apart in several chapters.

I have synthesized above some important concepts, which clarify that *these products are constrictors and rhythm accelerators of the fine lobular structures, as far as the respiratory bronchioles*. Therefore, their use is contrary to the nature of the problem they are supposed to treat and is greatly iatrogenic.

## My working hypothesis

Bronchial asthma has been defined as *bronchial hyper reactivity*. Nevertheless, as the role of the tiny lobular structure, under command of the Sympathetic-Adrenaline subsystem control has been ignored on this context, it is necessary to define the structure subject to hyperactivity, whether only the lobar bronchi, extra pulmonary airways and or direct or indirectly on the *bronchiolar-alveolar structures*.

Among the generally accepted concepts are:

1. The basic abnormality of asthma is *unknown*.
2. The only common physiological-pharmacological abnormality is that of the *bronchi*, clinically shown as excessive response to low doses of *agonists such as Histamine and metacholine*.

I have experimentally proven that injections even of tiny doses of Histamine or Acetylcholine produce similar effects, which are: exaggerated lobar broncho constriction<sup>3</sup> exaggerated vagal-like response. It is clear that Histamine is agonistic to acetylcholine.

I consider that here is the key to understanding the initial response to the unknown initial stimulus, only lacking an analysis of the possible presence of other substances working on different types of Asthma, as well as other parallel effects in order to determine the magnitude of their effects on the lobular sector, innervated by the Sympathetic nerves.

As the Lung is a working integrated unit, any alteration in the lobar bronchi alters the lobular function.

I would like to emphasize the above named pathologies derived from the ignored dynamics of the Lung in its balanced integration with the Atmosphere.

1. **Pulmonary Emphysema** and Chronic Pulmonary Cor are consequences of hyper-activity of the **Lobular Structure**, which is innervated by Sym-

pathetic nerves. The agent today generating this hyperactivity is Tobacco Nicotine, which is a potent Sympathetic-Adrenergic drug, consumed in a steady manner over many years, due to the smoking habit.

2. *Acute High Altitude Sickness* is the consequence of the environmental extra-organic dynamic factor, which is low *mass of air per volume unit at high altitudes*. The Lung exhausts its potential working capacity to balance this physical factor,

hence fails, with all its consequences.

3. *Acute Pulmonary and Cerebral Oedema at high Altitude* (Really Visceral general Oedema) which is caused by great imbalance of blood circulation as a result of the Lung's dynamic failure, in itself as in the reflex working System of floodgates and presses.

4. Bronchial Asthma, the study of which is now in progress.

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The Diaphragm is a striated flat muscle dividing the capacity of the Trunk into two functional cavities: the abdominal and the thoracic, leading to their functional balanced mechanical integration, via reflex.

Contrary to traditional belief, the Diaphragm is not the motor of the Lung; on the contrary, the Lung is an autonomic-automatic working organ, physically attached to the Diaphragm. This latter receives a cyclic mechanical traction from the Lung, to stimulate the Phrenic nerve for a contraction of the Diaphragm's central part, via reflex.

The peripheral part of the Diaphragm, inserted on the Thorax-Walls at the four or five lower intercostals spaces, jointly with the correspondent intercostals muscles and the upper part of the ante-lateral abdominal flat muscles, integrating these somatic structures with press and floodgate functions to balance the circulation of fluids throughout the Lung.