WHY does this happen?
No matter which definition you use, nor which set of data are used as a reference, the message is always the same. “Asthma is a chronic inflammatory disease of the airway causing the breathing tubes to narrow.”

Again - one must ask the question WHY? What is the cause of the inflammation, how does it happen, and what can be done to prevent this?

But firstly, let’s look at some facts about ‘asthma’.

The following statement has been taken directly from the Website of the American Asthma and Allergy Foundation.

“Since asthma has a genetic origin and is a disease you are born with, passed down from generation to generation, the question isn’t really “what causes asthma,” but rather “what causes asthma symptoms to appear?” People with asthma have inflamed airways which are super-sensitive to things which do not bother other people. These things are called “triggers.”

If this is true - where does the notion of “late onset asthma”, “hidden asthma” and “exercise induced asthma” come from?

And furthermore, why is it that in the vast majority of cases that I have come across in the 50 years plus, that I have been working in this field, very little - if any - family history is taken, relative to the incidence of asthma in parents, grandparents, siblings and children? The diagnosis usually relies on either spirometry, peak flow or provocation tests - and the outcome is predictable.

My understanding of the nature of heredity in asthma is confined to three specific areas:

i. The bands of smooth muscle surrounding the bronchioles, are thicker, stronger and tighter than in people with no genetic tendency - and when these bands tighten - it is very difficult to get them to relax.

ii. The mucus producing cells in those with genetic asthma are larger and more productive, so on stimulation, will produce copious amounts of mucus - causing the wheezing.

iii. People with ‘heritage asthma’ usually have a far wider range of allergic triggers, creating an enhanced environment for problems to occur.

BUT this does not adequately answer the question as to what causes the onset of an attack.

If one looks at the structure of a bronchiole it is easy to see what happens when the smooth muscle bands go into spasm.

The airway narrows and makes breathing difficult.
What is it that triggers the spasm in the smooth muscle bands?

Surprisingly enough the main trigger is the sudden drop in Alveolar or End Tidal CO2. The moment the brain detects that the PaCO2 pressure is dropping and the pH of the respiratory system is heading towards alkalosis, it immediately acts to restrict further loss, by narrowing the bronchioles.

If the person persists in gasping, over breathing and any other activity which continues to drop the ETCO2, then the mucus cells respond by producing copious amounts of mucus minimizing loss by further occluding the airway.

This is NOT a disease. It is a protective mechanism initiated by the body to prevent cell death from respiratory alkalosis brought about through hyperventilation/over breathing.

The simple answer to a complex question is that it is primarily mouth breathing, or overbreathing/hyperventilation, that causes low CO2 levels, or hypocapnia.

Could you imagine a person sitting quietly in a chair, breathing gently through their nose, suddenly having an ‘asthma attack’? Unlikely. Most ‘attacks’ come through a sudden change in breathing patterns - usually accompanied by a rapid drop in ETCO2. Exercising with open mouth, crying, laughing, coughing - all lower ETCO2 - provoking bronchospasm - ultimately leading to a full blown attack.

According to a study in the UK published in January 2015, more than one million people in the UK have been misdiagnosed as having asthma.¹

In my own practice, in which I have certainly handled more than 10,000 ‘asthmatics’ over the years, less than 10% have required ongoing management with bronchodilators and corticosteroids. The vast majority have been able to lead perfectly normal lives just by learning how to breathe functionally.

This has been borne out in numerous trials, papers and reports - published in the cream of respiratory journals such as Thorax, Chest, and the main medical journals such as the BMJ, AMJ, MJA and others. See the list of published articles and trials at the end of this article.

So this calls into question the accuracy and validity of the current method of diagnosing asthma

The first fundamental law of scientific measurement states that the measuring methodology should not alter the parameters of the function being measured.

Considering the rapid effect that a sudden drop in ETCO2 has on bronchioles, causing almost an instant response, does spirometry and peak flow not provoke bronchospasm?

If that bronchospasm is provoked, and the patient is then nebulized in order to break the spasm, and the next reading taken when the airway is artificially ‘propped open’, where is the validity in the ‘diagnosis’ that the person has ‘asthma’?

The gold standard for asthma management

I remember only too well the sacrosanct command that if a reliever was used more than 4 times a week, asthma was out of control, there was a danger of heart problems developing from the over-stimulation brought about by the adrenalin-type action of the salbutamol, and that it was then ‘mandatory’ to use a steroid preventer to reduce the amount of reliever.

The main purpose of the inhaled steroid - and of course the systemic prednisone, was to suppress the immune response and reduce the inflammation to such a degree that it was no longer necessary for the regular use of bronchodilators, avoiding the associated side effects.

Contradiction one

If the use of a short-acting bronchodilator more than 4times a week is deemed dangerous, where is the justification in giving someone 24 hour long-acting bronchodilator, which is the equivalent of 6-8 puffs of short acting beta 2 agonist, every day of their lives?

What has been suggested is that by altering the chain length of the beta-2 agonists, there is less of a “jolt stimulation” to the heart and a lower risk of an adverse effect. This is borne out by the warning that the long-acting beta-agonist (LABA) should not be used as a ’rescue’ to address an immediate attack, as it can take up to 40 minutes before the effect is felt. The reasoning further goes on to explain that by using the long-chain drug there is a smoother and more sustained bronchodilating effect which has a lower risk.

But what about the effect of a 24 hour bronchodilator? There are numerous papers and articles written about the remodeling of the airway as a result of long-term (lifetime) asthma medication – and it is no secret that in many segments of modern medicine ‘iatrogenesis’ or as it is more subtly put, ‘unintended consequences’, have the potential to cause additional comorbid diseases.

Propping the airway open, in direct conflict with nature’s response to shut it down, has the potential to cause inflammation of the mucous lining. Is this perhaps the reasoning behind adding the inhaled steroid to the combination drug, to address the inflammation that was caused by propping it open in the first place? A little like the boy who shot both parents and then asked the judge for clemency on the grounds that he was an orphan.

Contradiction two

Everyone knows that cortisone is given to suppress the immune system so that it will not react to, or reject foreign objects. This has been the standby of the organ transplantation industry for decades. Remember however, that in the case of cortisone saturation to prevent rejection, the patient was so at risk of bacterial or viral infection, that people had to be ‘hazmat suited’ before being allowed to visit.

If cortisone suppresses the immune system how in the name of any sensibility can it ‘protect the lungs’ during the cough and cold virus winter season, and even more bizarre, at a higher dosage?

Cortisone certainly helps to reduce the inflammation - but renders the user more susceptible to catching common infections - especially in the winter months.

Using the same general principle, in the same way that the ‘orthodontist’ accepts that the teeth are crooked and have to be straightened, that the ENT accepts that the tonsils are infected and have to be removed, the pulmonologist just accepts the fact that the airway becomes inflamed and has to be treated with steroids.

Just look at the definition of ‘asthma’ and it would appear that the inflammation is of an unknown etiology - usually an immune response to allergic triggers. That is VERY vague - just like saying that the ‘teeth are crooked’ or ‘the tonsils are enlarged’.

Citation: Price RL. Asthma. “The end of the beginning”. How to prevent the onset of an attack rather than treating it after the event? / Lung Plume Respir Res. 2016;3(2):65–72. DOI: 10.15406/jlprr.2016.03.00080
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What is asthma?

"Asthma (AZ-ma) is a chronic (long-term) lung disease that inflames and narrows the airways. Asthma causes recurring periods of wheezing (a whistling sound when you breathe), chest tightness, shortness of breath, and coughing. The coughing often occurs at night or early in the morning.

Asthma affects people of all ages, but it most often starts during childhood. In the United States, more than 25 million people are known to have asthma. About 7 million of these people are children."

AND–how does this definition then correlate with that of the Asthma and Allergy Foundation of America statement that “Asthma is a disease you are born with?” The contradictions are bizarre and, quite frankly, embarrassing to say the least. What we have here is a concept that appears to be fundamentally flawed. Well... If the concept is fundamentally flawed, and you specialize in it, all you become is a specialist in a flawed concept. It does not make that concept any more valid.

Nowhere does it explain HOW and WHY the inflammation occurs - it just accepts that it is there and medicates it.

Why does the airway become inflamed?

The answer is so simple it is embarrassing. The airway becomes inflamed largely because it is subjected to a large volume flow of inhospitable air, and is simply not designed to be able to cope with this onslaught.

The air entering the lungs needs to be:

i. The correct volume
ii. Filtered
iii. Sterilized
iv. Warmed/cooled to body temperature
v. Humidified so that the lungs are able to allow the gases to permeate (Henry’s and Fick’s Laws)

The NOSE is the perfect 4 stage filtration system, and in addition to the filtering process, nasal breathing stimulates the paranasal sinuses to produce and release Nitric Oxide, which is a potent antimicrobial as well as a vasodilator.

The adenoids and tonsils are the final stage of micro-filtration to ensure the quality of the air entering the lungs.

Surely it does not take a great leap of imagination to see that bypassing this sophisticated system, and breathing large volumes of untreated outside air, straight into the delicate lung tissue, could be the major cause of inflammation and infection?

The bypassing of the Nitric Oxide production/release removes a very powerful vaso/bronchodilator from the system, and the rapid loss of CO₂ from the large volume of mouth breathing, is the main trigger for the protection provided by bronchospasm.

Does it make sense?

That if the bronchioles are shutting down in self defense, in order to protect the body, that propping them open twenty-four hours a day is self-defeating, and can only aggravate the condition further?

It is not possible that it is this very action that perpetuates the inflammation, and that is why ICS is added to the LABA to counteract the inflammation caused?

If the Gold Standard calls for the use of steroids to offset the overuse of bronchodilators, does it not make sense that reducing the need for bronchodilators will reduce the need for steroids?

In the face of all this ‘sense’ how can it be justified to increase bronchodilator use and thereby consign the patient to a lifetime of steroid medication - with zero chance of the ‘disease’ being ‘cured’?

Rewriting the rules

It is no secret that ‘standards’ change under the observation and reporting of data which is collected on a routine basis. The SRG is a group of clinical pathologists who constantly review collected data and adjust the “norms” to reflect what is being noticed in pathology reports coming in from participating countries. (http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1769782/)

These corrected “norms” then become accepted as fact - and using the orthodontic profession as a prime example - end up drawing the conclusion that 3rd molars are no longer necessary in modern day life - and 28 teeth are now the “norm”. The fact that 3rd molars simply cannot erupt because the Western diet has largely removed the requirement of ‘chewing’ and resulted in under-developed jaws - is conveniently ignored, and wisdom teeth are ‘expected’ to be impacted and therefore require surgical removal.

Commonplace but not natural/normal

Under the deluge of propaganda, advertising, TV promotion and pressure from representatives of drug and medical equipment companies, society has accepted that commonplace equals normal, and is unavoidable. In other words, crooked teeth are commonplace and have to be straightened. Inflamed tonsils are commonplace and have to be removed. High blood pressure is commonplace and has to be medicated.

The reality is that these things are commonplace, but are NOT normal or natural, and CAN largely be avoided by early detection and remediation.

As will be seen from the accompanying chart - Minute Volume - the amount of air inhaled and exhaled per minute - has always been accepted as approximately 6 liters per minute. Simple mathematical calculation, based on lung volume and respiratory rate, then shows that functional breathing at rest should be approximately 8-10 breaths per minute - breathing between 4-6 liters of air per minute.
Asthma. "The end of the beginning". How to prevent the onset of an attack rather than treating it after the event?

Just remember, in 1930, before the start of WW2, the average minute volume was 4.5 liters/minute and at an average breath rate of 6-8 per minute.

With the advent of fast foods in the 1950s and onwards, minute volume and breathing rate steadily escalated to the present ‘accepted norm’ of 12 liters/minute and 18-20 breaths per minute.

That is Hyperventilation, and is not normal. Just because it is commonplace it means nothing other than there are a lot of dysfunctionally breathing people out there - contributing in no small measure to the epidemic of the awful Western disease called Average Health.

The physical structure of the nose, the airway and the lungs can happily accommodate this rate and volume, and functional breathing is silent - with no irritation to, or vibration of the tissues of the nose, mouth and airway.

This can ONLY be achieved through nasal breathing, driven by the diaphragm, and this is the way the body was designed to function.

Mouth breathing, with its accompanying hyperventilation, draws more than double the volume of air, unfiltered and non-sterilized, at more than twice the rate that the airway structure is designed to handle - causing localized and systemic inflammation - as well as severely disrupted biochemistry. This in turn leads to compromised and compensatory physical and postural behavior which in turn aggravates other functions of the body.

Why not prevent the attack from happening?

Why not teach people how to avoid an attack by not creating the conditions that cause one?

Numerous double-blinded, randomized, placebo controlled trials have proved - conclusively - that by changing breathing from high-volume, chest/mouth, to low volume nose/diaphragm, bronchodilator usage can be reduced by 86% and ICS usage by 50% - with absolutely zero side effects.

These trials have always been criticized on the grounds that they have not shown any improvement in FEV1. That is a total red herring - and maintains the resistance to clinically trialed and proven facts.

The Tiffeneau-Pinelli Index - better known as the FEV1/FVC index was designed for restrictive and obstructive lung disease - where there is a degradation in lung tissue - such as related to pathological and degenerative diseases.

This is yet another example where a metric – designed to be used as a comparator, or indication of progress or regression, has become perverted and is used as an empirical diagnostic tool in a totally different environment. The FEV1 has no correlation with the triggers that cause overbreathing. All it attempts to do is measure the consequences of congestion/dysfunction brought about by provocation – and is therefore of dubious value in the context in which it is used, namely to discredit the numerous trials. I become very frustrated when I hear ‘holier-than-thou’ specialists dismiss these valid, clinically based, published and peer reviewed trials on the grounds that “it did not improve FEV1 – therefore it is of no value.”

Asthma is not a disease. It is a condition which only manifests itself when provoked. Remove the provocation and the condition is controlled.

There is no doubt that asthma can be life threatening - but this is in a very small number of cases across the spectrum. Brittle Asthma is a reality - and people need hospitalization and extreme care when this happens.

What I am talking about is the extremely high percentage of misdiagnosed cases - due to a flawed diagnostic process - where the diagnostics provoke the condition. It is this very significant percentage - estimated at approaching 90% of those diagnosed, who are being over medicated, spending billions of unnecessary dollars on medication which they do not need, and potentially causing iatrogenic issues later on in life.

The solution

In the same way that a person can be coached in a sport so that they do not hurt or harm themselves, it is possible to coach people in how to breathe functionally.

Our diet and lifestyle are working against us - and the vast majority of people are in a state of constant stress.

This sympathetic dominance has them in a state of alertness all the time, and as a result of this Fight/Flight response, they are hyperventilating or overbreathing.

There are simple and effective ways of addressing this situation and teaching people how to return to normal breathing.

Massive costs in terms of medication

Given that the diagnosis of asthma is so often incorrect, but the patients are still placed on ‘puffer therapy’, it is no wonder that the costs to individuals, as well as the system, are as high as they are.

In a recently published survey, by IMS Health: FDA, the top 10 most frequently prescribed drugs were listed by wholesale prices. This means what was earned by the manufacturers, not what the consumer pays, which could be between 50% and 100% more.

Of the $38.2 billion dollars generated by these drugs, just on 40% - $11.7 billion – were for asthma and COPD drugs.

How much of this could be saved by reducing, or even eliminating the unnecessary usage – due to misdiagnosis?
There is no shortage of people available to teach, train and guide ‘asthmatics’ in how to prevent the onset of an ‘attack’. The ideal people are Occupational and Physical Therapists, and other trained, licensed and registered Respiratory and Manual therapists, as they have the correct training, understand the human body as a whole, and will readily learn the specialized skills required to teach people how to normalize their breathing.

‘Idiopathic-iatrogenic’- the final indignity

There is no doubt that stress is one of the major drivers of breathing disorders. The Fight/Flight response instantly changes breathing rate, depth, dynamics and mechanics, as well as major physiological and biochemical responses, resulting in a multitude of changes throughout the body. Being in a state of constant stress, as a large percentage of “asthmatics” are, maintains a level of sympathetic, or cortisol dominance, and a reduction in the time spent in parasympathetic recovery.

Inhalation drives the sympathetic and exhalation drives parasympathetic responses, and the ratio should be roughly 40% to 60%, thereby allowing the person more time in recovery mode than in excitation mode. Most people’s breathing patterns are reversed - with longer inhalations and shorter exhalations - due to the brainstem response initiating the next inhalation before the full exhalation has been completed. This is as a result of many years of dysfunctional breathing causing the medullary trigger to ‘kick in’ earlier than it should.

To take someone who is in a constant state of adrenalin/cortisol dominance, and place them on permanent long-term, 24-hour medication, with a combination drug whose components are long-term beta-2 agonists and inhaled corticosteroids, can only aggravate this condition and consign the sufferer to a lifetime of the iatrogenic term beta-2 agonists and inhaled corticosteroids, can only aggravate this condition and consign the sufferer to a lifetime of the iatrogenic ‘asthmatics’ are, maintains a level of sympathetic, or cortisol dominance, and a reduction in the time spent in parasympathetic recovery.

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There is a better way to manage this ‘pandemic’.

Acknowledgements

None.

Conflict of interest

The author declares no conflict of interest.

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