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Asthma. "The end of the beginning". How to prevent the onset of an attack rather than treating it after the event?

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Roger L Price
Respiratory Physiologist and Integrative Health Educator, USA

Correspondence: Roger L Price, Respiratory Physiologist and Integrative Health Educator, Breathing Well LLC, 1425 Broad St Suite B, Clifton NJ 07013, USA, Tel (973) 778-9225, (505)331-1051, Email rogerlprice@gmail.com

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NOTE: The complex physiological and biochemical processes have been deliberately simplified in order to allow lay people to grasp the concepts without being confused by the pure science. What is essential is for people to understand 'why' and not necessarily to have to know 'how'.

Author's Note:

I fully accept that asthma is a potentially life threatening condition and strongly support the use of appropriate medication when required. What I do not support however, is the reckless abandon with which 'puffers' are prescribed and used, for everything from a niggling cough to fundamentally dysfunctional breathing - which has nothing to do with asthma.

How does an 'asthma attack' begin?

The first signs are usually tightness in the chest and difficulty in breathing.

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.

Asthma and Allergy Foundation of America

what causes asthma

"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 50years 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:

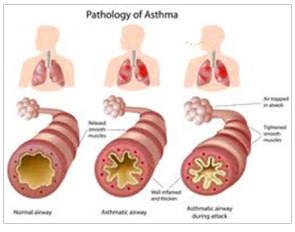
i. The bands of smooth muscle surrounding the bronchioles, are thicker, stronger and tighter than in people with no genetic tendency - and when these bands tighten - it is very difficult to get them to relax.

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 'heredity 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 provokes 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 24hour 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

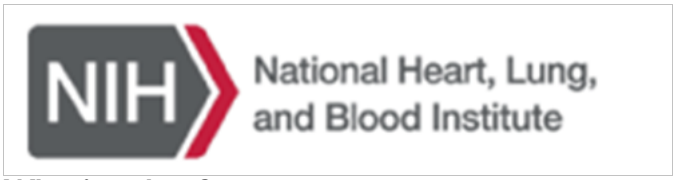
Everyone knows that cortisone is given to suppress the immune system so that it will not react to, or reject foreign objects. This has been the standby of the organ transplantation industry for decades. Remember however, that in the case of cortisone saturation to prevent rejection, the patient was so 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'.



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 CO_2 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/)

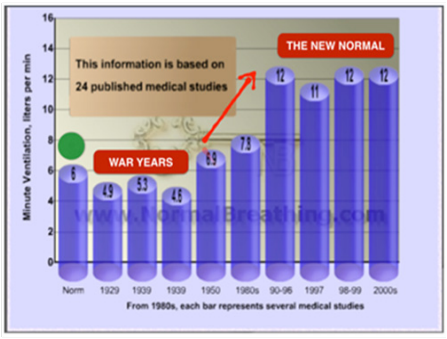
These corrected "norms" then become accepted as fact - and using the orthodontic profession as a prime example - end up drawing the conclusion that 3rd molars are no longer necessary in modern day life - and 28 teeth are now the "norm". The fact that 3rd molars simply cannot erupt because the Western 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.



Just remember, in 1930, before the start of WW2, the average minute volume was 4.5liters/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 12liters/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, drags 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 $38.2billion dollars generated by these drugs, just on 40% - $11.7billion – were for asthma and COPD drugs.

How much of this could be saved by reducing, or even eliminating the unnecessary usage – due to misdiagnosis?

Drug money The top prescribed name-brand drugs in the U.S. that don't have direct generic competition have all enjoyed sales growth in the past five years, even in cases where prescriptions are declining

PRODUCT	ASSOCIATED WITH TREATING	MAKER	PRESCRIPTIONS FILLED IN 2015	% CHANGE IN PRESCRIPTIONS, 2011-15	SALES IN 2015	% CHANGE IN SALES, 2011-15
CRESTOR	High cholesterol	AstraZeneca	20,302,910	-21%	$6.3 billion	39%
VENTOLIN HFA	Asthma and chronic lung diseases	GlaxoSmithKline	19,626,994	25%	$0.9 billion	53%
ADVAIR DISKUS	Asthma and chronic lung diseases	GlaxoSmithKline	13,117,809	-28%	$4.7 billion	2%
LANTUS SOLOSTAR	Diabetes	Sanofi-Aventis	11,481,467	82%	$5.8 billion	241%
VYVANSE	Attention-deficit disorder	Shire	11,070,043	31%	$2.6 billion	97%
LYRICA	Fibromyalgia and nerve pain	Pfizer	10,239,133	8%	$3.8 billion	112%
JANUVIA	Diabetes	Merck	9,329,671	19%	$4.2 billion	90%
SYMBICORT	Asthma and chronic lung diseases	AstraZeneca	9,070,334	93%	$2.7 billion	159%
SPIRIVA HANDIHALER	Asthma and chronic lung diseases	Boehringer Ingelheim	9,003,319	-2%	$3.4 billion	40%
LANTUS	Diabetes	Sanofi-Aventis	8,597,291	-21%	$3.8 billion	83%

SOURCES: IMS Health; FDA NOTE: Sales reflect wholesale, nondiscounted revenues and do not account for discounts, promotions, coupons or rebates

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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, 24hour 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 'stress-symptom-stress-symptom' cycle. This then completes the ***idiopathic-iatrogenic*** loop of "I don't know what is causing it", and, "what I am doing is actually making it worse"

There is a better way to manage this 'pandemic'.

Acknowledgements

None.

Conflict of interest

The author declares no conflict of interest.

References

1. http://www.dailymail.co.uk/health/article–2929353/1m–asthma–cases–misdiagnosed–fear.html
2. Controlling Asthma by Training of Capnometry–Assisted Hypoventilation (CATCH)

Versus Slow Breathing:A Randomized Controlled Trial. CHEST 11/22/14 Thomas Ritz, PhD1*, David Rosenfield, PhD1, Ashton M. Steele, MA1, Mark. M. Millard, MD2, and Alicia E. Meuret, PhD1*

1Southern Methodist University, Dallas, Texas, USA

2Baylor University Medical Center, Dallas, Texas, USA

3. American Thoracic Society Committee on Diagnostic Standards. Definitions and classification of chronic bronchitis, asthma, and pulmonary emphysema. *Am Rev Respir Dis*. 1962;85:762.
4. Slader HK, Reddel LM, Spencer EG, et al. ASTHMA Double blind randomised controlled trial of two different breathing techniques in the management of asthma CA. *Thorax*. 2006.
5. Sandberg S, Järvenpää S, Penttinen A, et al. Asthma exacerbations in children immediately following stressful life events:a Cox's hierarchical regression. *Thorax*. 2004;59(12):1046–1051.
6. Atherton M. Outcome measures of efficacy associated with a web–enabled self management programme: findings from a quasi-experiment. *Disease Management and Health Outcomes*. 2000;8(4):233–242.
7. Barnes G, Partridge MR. Community asthma clinics:1993 survey of primary care by the national Asthma Task Force. *Qual Health Care*. 1994;3(3):133–136.
8. Barraclough R, Devereux G, Hendrick DJ, et al. Apparent but not real increase in asthma prevalence during the 1990s. *Eur Respir J*. 2002;20(4):826–833.
9. Beasley R, Cushley M, Holgate ST. A self help management plan in the treatment of adult asthma. *Thorax*. 1989;44:200–204.
10. Beilby JJ, Wakefield MA, Ruffin RE. Reported use of asthma management plans South Australia. *Med J Aust*. 1997;166(6):298–301.
11. Villiger PM, Hess CW, Reinhart WH. Beneficial effect of inhaled CO_2 in a patient with non–obstructive sleep apnoea. *J Neurol*. 1993;241(1):45–48.
12. Hancox RJ, Subbarao P, Kamada D, et al. Beta2-agonist tolerance and exercise–induced bronchospasm. *Am J Respir Crit Care Med*. 2002;165(8):1068–1070.
13. http://www.nhibi.nih.gov.
14. International consensus report on the diagnosis and treatment of asthma .National Heart, Lung and Blood Institute, National Institute of Health. Bethseda, Maryland 20892 publication no.92– 3091. *EUR Resp Journal*. 1992;5(5):601–641.
15. Bousquet J, Jeffery PK, Busse WW, et al. Asthma. From bronchoconstriction to airways inflammation and remodeling. *Am J Respir Crit Care Med*. 2000;161(5):1720–1745.
16. Bowler S, Green A, Mitchell C. Buteyko breathing techniques in asthma: a blinded randomised controlled trial. *Med J Aust*. 1998;169(11–12):575–578.
17. Tobin MJ, Chadha TS, Jenouri G, et al. Breathing patterns. 1. *Normal subjects*. 1983;84(2):202–205.
18. Brewster CE, Howarth PH, Djukanovic R, et al. Myofibroblasts and subepithelial fibrosis in bronchial asthma. *Am J Respir Cell Mol Biol*. 1990;3(5):507–511.
19. Bryce FP, Neville RG, Crombie IK, et al. Controlled trial of an audit facilitator in diagnosis and treatment of childhood asthma in general practice. *BMJ*. 1995;310(6983):838–842.
20. Bucknall CE, Robertson C, Moran F, et al. Management of asthma in hospital: a prospective audit. *Br Med J (Clin Res Ed)*. 1988;296(6637):1637–1639.
21. Bucknall CE, Slack R, Goddley CC, et al. Scottish Confidential Inquiry into Asthma Deaths (SCIAD), 1994–6. *Thorax*. 1999;54:978–984.
22. Burney P. The changing prevalence of asthma? *Thorax*. 2002;57(Suppl II):ii36–ii39.
23. Burr ML, VerrallC, Kaur B. Social deprivation and asthma. *Respir Med*. 1997;91(10):603–608.
24. McHugh P, Aitcheson F, Duncan B, et al. Buteyko Breathing Technique for asthma: an effective intervention. *N Z Med*. 2003;116(1187):U710.

25. Simon D Bowler, Amanda Green, Charles A Mitchell. Buteyko breathing techniques in asthma: a blinded randomised trial. *Medical Journal of Australia*. 1998;169:575–578.

26. Buteyko K, Odintsora M, Nasonkina N. [The Ventilation Test in Patients with Bronchial Asthma]. *Vrach Delo*. 1968;4:33–36.

27. Cambach W, Wagenaar RC, Koelman TW, et al. The long term effects of pulmonary rehabilitation in patients with asthma and chronic obstructive pulmonary disease:a research synthesis. *Arch Phys Med Rehabil*. 1999;80(1):103–111.

28. Burggraaf J, Westendorp RG, in't Veen JC, et al. Cardiovascular side effects of inhaled salbutamol in hypoxic asthmatic patients. *Thorax*. 2001;56(7):567–569.

29. Carey OJ, Cookson JB, Britton J, et al. The effect of a lifestyle on wheeze, atopy and bronchial hyperreactivity in Asian and white children. *Am J Respir Crit Care Med*. 1996;154(2 Pt 1):537–540.

30. Carswell F, Robinson EJ, Hek G, et al. Bristol Experience:Benefits and cost of an 'asthma nurse' visiting the home of asthmatic children. *Bristol Med Chir J*. 1989;104(1):11–12.

31. Sears MR. Changing patterns in asthma morbidity and mortality. *J Investig Allergol Clin Immunol*. 1995;5(2):66–72.

32. Charlton I, Charlton G, Broomfield J, et al. Audit of the effect of a nurse run asthma clinic on workload and patient morbidity in a general practice. *Br J Gen Pract*. 1991;41(347):227–231.

33. Charlton I, Charlton G, Broomfield J, et al. Evaluation of peak flow and symptoms only self management plans for control of asthma in general practice. *BMJ*. 1990;301(6765):1355–1359.

34. Clark NM, Nothtwehr F. Self–management of asthma by adult patients. *Patient Educ Couns*. 1997;32(1 suppl):S5–20.

35. D'Souza W, Burgess C, Ayson M, et al. Trial of 'credit card' asthma self management plan in a high risk group of patients with asthma. *J Allergy Clin Immunal*. 1996;97(5):1085–1092.

36. Demeter S, Cordasco EM. Hyperventilation Syndrome & Asthma. *The American Journal of Medicine*. 1996;81:989–994.

37. Donnelly PM. Exercise Induced Asthma:The Protective Role of CO2 during Swimming. *Lancet*. 1991;337(8734):179–180.

38. Robert Cowie. Resident Respirologist of Foothills Hospital in Calgary and head researcher on the Buteyko Breathing Technique Medical Trial; 2005.

39. Droogan J, Brannigan K. Organisation of asthma care:what difference does it make? *Nurs Times*. 1997;93(34):45–46.

40. Xie A, Rankin F, Rutherford R, et al. Effects of inhaled CO_2 and added dead space on idiopathic central sleep apnea. *J Appl Physiol*. 1985;82(3):918–926.

41. Egbagbe E, Pavord ID, Wilding P, et al. Adenosine monophosphate and histamine induced bronchoconstriction: repeatability and protection by terbutaline. *Thorax*. 1997;52(3):239–243.

42. Ahrens T, Schallom L, Bettorf K, et al. End–tidal carbon dioxide measurements as a prognostic indicator of outcome in cardiac arrest. *Am J Crit Care*. 2001;10(6):391–398.

43. Ernst E. Complimentary therapies for asthma:what patients use. *J Asthma*. 1998;35(8):667–671.

44. Feder G, Griffiths C, Highton C, et al. Do clinical guidelines introduced with practice based education improve care of asthmatic and diabetic patients? A randomised controlled trial in general practitioners in east London. *BMJ*. 1995;311(7018):1473–1478.

45. Gallefoss F, Bakke PS. Impact of patient education and self management on morbidity in asthmatics and patients with chronic obstructive pulmonary disease affect. *Respir Med*. 2000;94(3):279–287.

46. Gern JE, Lemanske RF Jr, Busse WW. Early life origins of asthma. *J Clin Invest*. 1999;104(7):837–843.

47. sru@soc.surrey. ac.uk

48. Gibson PG, Wilson AJ. The use of continuous quality improvement methods to implement practice guidelines in asthma. *J Qual Clin Pract*. 1996;16(2):87–102.

49. Goss JD, Leinbach TR. Focus groups as alternative research practice. *Area*. 1996;28(2):115–123.

50. Griffiths C, Naish J, Sturrdy P, et al. Prescribing and hospital admission for asthma in east London. *BMJ*. 1996;312(7029):481–482.

51. Guba EG, Lincoln YS. Competing paradigms in qualitative research 1994. p. 105–117.

52. Hansler DF, Cooper C. Focus groups n:New dimensions in feasibility study. *Fund Raising Manage*. 1986;17(5):78–82.

53. Harrison TW, Oborne GB, Wilding PJ. Sahaja yoga in the management of beta–agonist reduction in asthma. *Thorax*. 1999;54:98.

54. Jill McGowan. Health Education:Does the Buteyko Institute Method make a difference?, Education and training consultant in Asthma Management. *Thorax*. 2003;58(suppl III):28.

55. Heard AR, Richards IJ, Alpers JH, et al. Randomised controlled trial of general practice based asthma clinics. Med J Aust 171(2):68–71.

56. Hensley, MJ, Gibson PG (1998) Promoting evidence–based alternative medicine. Med J Aust 169:573–574.

57. Higgins BG, Britton JR (1995) Geographical and social class effects on asthma mortality in England and Wales. *Respir Med*. 1999;89(5):341–346.

58. Holgate S. Mediator and cytokine mechanisms in asthma. *Thorax*. 1993;48(2):103–109.

59. Holgate ST, Davies DE, Lackie PM, et al. Epithelial-mesenchymal interactions in the pathogenesis of asthma. *J Allergy Clin Immunol*. 2000;105(2 pt 1):193–204.

60. G Hoskins, C McCowan, RG Neville, et al. Risk factors and costs associated with an asthma attack. *Thorax*. 2000;55:19–24.

61. Hoskins G, Neville RG, Smith B. The link between nurse training and asthma outcomes. *Br J Comm Nursing*. 1999;4:222–228.

62. House of Lords Select Committee on Science and Technology. Complementary and alternative medicine. 6th report 1999–2000 [HL123]. London.

63. Osborne CA, O'Connor BJ, Lewis A, et al. Hyperventilation and asymptomatic chronic asthma. *Thorax*. 2000;55(12):1016–1022.

64. Integrated care for asthma: a clinical, social, and economic evaluation. Grampian asthma Study of Integrated Care (GRASSIC) *BMJ*. 1994;308:559–564.

65. Israel E, Fischer AR, Rosenberg MA, et al. The pivotal role of 5lipoxygenase products in the reaction of aspirin–sensitive asthmatics to aspirin. *Am Rev Respir Dis*. 1993;148(6 pt 1):1447–1451.

66. Jadad AR, Moher M, Browman GP, et al. Systematic reviews and meta–analyses on treatment of asthma: critical evaluation. *BMJ*. 2000;320:537–540.

67. Jones A, Pill R, Adams S. Qualitative study of views of health professionals and patients on guided self management plans for asthma. *BMJ*. 2000;321:1507–1510.

68. Jones K, Cleary R, Hyland M. Predictive value of a simple asthma morbidity index in a general practice population. *Br J Gen Pract*. 1999;49(438):23–26.

69. Juniper EF, Guyatt GH, Ferrie PJ, et al. Measuring quality of life in asthma. *Am Rev Respir Dis*. 1993;147(4):832–838.

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

70. Keeley D, Osman L. Dysfunctional breathing and asthma. *BMJ*. 2001;322:1075–1076.

71. Kemmis S, Grundy S. Educational action research in Australia: The state of the art. Australian Educational Researcher; 1981.

72. Kitzinger J. Introducing Focus groups. 1995.

73. Knafl K, Howard M. Interpreting and reporting qualitative research. *Res Nurs Health*. 1984;7(1):17–24.

74. Kumar P, Clark M. Clinical Medicine, Fourth Edition. Saunders; 1996.

75. Laffey J, Kavanagh B. New England Journal of Medicine. 2004.

76. Levy ML, Robb M, Allen J, et al. A randomized controlled evaluation of specialist nurse education following accident and emergency department attendance for acute asthma. *Respir Med*. 2000;94(9):900–908.

77. Lieu TA, Capra AM, Quesenberry CP, et al. Computer–based models to identify high–risk adults with asthma: is the glass half empty of half full? *J Asthma*. 1999;36(4):359–370.

78. Littlejohns P, Ebrahim S, Anderson R. Prevalence and diagnosis of chronic respiratory system in adults. *British Medical Journal*. 1989;298:1560.

79. Lozano P, Finkelstein JA, Carey VJ, et al. A multisite randomized trial of the effects of physician education and organizational change in chronic–asthma care:health outcomes of the Paediatric Asthma Care Patient Outcomes Research Team II Study. *Arch Pediatr Adolesc Med*. 2004;158(9):875–883.

80. Manoccha R, Marks GB, Kenchington P, et al. Sahaj yoga in the management of moderate to severe asthma: a randomised controlled trial. *Thorax*. 2002;57:110–115.

81. Martinez FD, Wright AL, Taussig LM, et al. Asthma and wheezing in the first six years of life. The Group Health Medical Associates. *N Engl J Med*. 1995;332(3):133–138.

82. McCarney RW, Lasserson TJ, Linde K, et al. An overview of two Cochrane systematic reviews of complementary treatments for chronic asthma:acupuncture and homeopathy. *Respir Med*. 1998;98(8):687–696.

83. McCarthy M. US panel calls for more support of alternative medicine. *Lancet*. 2002;359(9313):1213.

84. McDermott MF, Murphy DG, Zalenski RJ, et al. A comparison between emergency diagnostic and treatment unit and inpatient care in the management of acute asthma. *Arch Intern Med*. 1997;157(18):2055–2062.

85. Mielck A, Reitmeir P, Wjst M. Severity of childhood asthma by socioeconomic status. *Int J Epidemiol*. 1996;25(2):388–393.

86. Millar B, Maggs C, Warner V, et al. Creating consensus about nursing outcomes 1. An exploration of focus group methodology. *J Clin Nurs*. 1996;5(3):193–197.

87. Montefort S, Roberts JA, Beasley R, et al. The site of disruption of the bronchial epithelium in asthmatic and non–asthmatic subjects. *Thorax*. 1992;47(7):499–503.

88. Mowat DHR, McCowan C, Neville RG. Socio–economic status and childhood asthma. *Asthma Gen Pract*. 1998;6:9–11.

89. Mundinger MO, Kane RL, Lenz ER, et al. Primary care outcomes in patients treated by nurse practitioners or physicians: a randomized trial. *JAMA*. 2000;283(1):59–68.

90. Neville R. Two approaches to effective asthma audit. *Practitioner*. 1995;239(1548):203–205.

91. Neville RG, Hoskins G, Smith B, et al. Observations on the structure, process and clinical outcomes of asthma care in general practice. *Br J Gen Pract*. 1996;46(411):583–587.

92. Ng TP. Validity of symptom and clinical measures of asthma severity for primary outpatient assessment of adult asthma. *Br J Gen Pract*. 2000;50(450):7–12.

93. Nyamathi A, Shuler P. Focus group interview: a research technique for informed nursing practice. *J Adv Nurs*. 1990;15(11):1281–1288.

94. Birch M. Obstructive Sleep Apnoea and breathing retraining. *Aust Nurs J*. 2004;12(2):27–29.

95. Opat AJ, Cohen MM, Bailey MJ, et al. A clinical trial of the Buteyko breathing technique in asthma as taught by video. *J Asthma*. 2000;37(7):557–564.

96. Patterson C, Britten N. Organising primary health care for people with asthma: the patient's perspective. *Br J Gen Pract*. 2000;50(453):299–303.

97. Pauwels R, Joos G, Van der Straeten M. Bronchial hyper–responsiveness is not bronchial hyper–responsiveness is not bronchial asthma. *Clin Allergy*. 1988;18(4):317–321.

98. Pearson MG, Bucknall CE. *Measuring clinical outcome in asthma: a patient–focused approach*. London: Royal College of Physicians; 1999.

99. Powell RA, Single HM. Focus Groups. *International Journal of Quality in Health Care*. 1996;8:(5).

100. Premaratne UN, Sterne JA, Marks GB, et al. Clustered randomised trial of an intervention to improve the management of asthma:Greenwich asthma study. *BMJ*. 1999;318:1251–1255.

101. Thomas M, McKinley RK, Freeman E, et al. Prevalence of dysfunctional breathing in patients treated for asthma in primary care:a cross sectional survey. *BMJ*. 2001;322:1098–1100.

102. Roger L. Proceedings of the American Thoracic Society. 2006;3:A530.

103. Race KEH, DF Parker T. Rehabilitation program evaluation: use of focus groups to empower clients. *ERIC*. 1994;18(6):730–740.

104. Lavie P. Rediscovering the importance of nasal breathing in sleep or, shut your mouth and save your sleep. *J Laryngol Otol*. 1987;101(6):558–563.

105. Davis MS, Freed AN. Repeated Hyperventilation Causes Peripheral Airways Inflammation, Hyperreactivity, and Impaired Bronchodilation in Dogs. *Am J Respir Crit Care Med*. 2001;164(5):785–789.

106. Robinson N. *Journal of Advanced Nursing*. 1999;29(41):905–913.

107. Rona RJ. Asthma and poverty. *Thorax*. 2000;55:239–244.

108. Ross, Wilson. Anatomy and Physiology for Nurses Lippincot publishers; 1982.

109. Scott D, Usher R. Researching education data methods and theory in scottish intercollegiat guidelines network (1998). Guidelines for asthma management. HMSO; 2000.

110. Sicker J, Wimbush E, Watson J, et al. Qualitative methods in health promotion research :some criteria for quality. *Health Education Journal*. 1995;54:74–78.

111. Singh V, Wisniewski A, Britton J, et al. Effect of Yoga breathing exercise (prayanama) on airway activity in subjects with asthma. *Lancet*. 1990;335(8702):1381–1383.

112. Smith E, Alexander V, Booker C, et al. Effect of hospital asthma nurse appointment on inpatient asthma care. *Respir Med*. 200;94(1):82–86.

113. Sommaruga M, Spanevello A, Migliori GB, et al. The effects of a cognitive behavioural intervention in asthmatic patients. *Monaldi Arch Chest Dis*. 1995;50(5):398–340.

114. Szczeklik A, Nizankowska E, Sanak M, et al. Aspirin–induced rhinitis and asthma. *Curr Opin Allergy Clin Immunol*. 2001;1(1):27–33.

115. Szczeklik A. The cyclooxygenase theory of aspirin-induced asthma. *Eur Respir J*. 1990;3(5):588–593.

116. The British Thoracic Society, the British Guidelines on Asthma Management, Thorax, the Journal of the British Thoracic Society; 1997. 52 p.

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

117. www.astthmaabd.org/guideline/section1/definition.hhtm

118. The Role of Breathing Theory. National Asthma Campaign/Australian Association of Asthma Foundation, Annual Conference.

119. Thomas M, McKinley, RK, Freeman E, et al. Prevalence of dysfunctional breathing in patients treated for asthma in primary care; cross sectional survey. *BMJ*. 2001;322(7294):1098–1100.

120. Buteiko KP, Odintsova MP, Nasonkina NS, et al. *Ventilation Test In Patients With Bronchial Asthma*. 1968;:33–36.

121. William MV, Mark O'Hollaren, Kenneth ME, et al. Speciality differences in the management of asthma. A cross–sectional assessment of allergists' patients and generalists' patients in a large cross section HMO. *Archives of Internal Medicine*. 1997;157(11):1201–1208.

122. Ware JE, Sherbourne CD. The MOS 36–item short–form health survey (SF–36).I. Conceptual framework and item selection. *Med Care*. 1992;30:473–483.

123. Watanabe T, Oothta M, Murata M, et al. Decrease in emergency room or urgent care visits due to management of bronchial asthma inpatients and outpatients with pharmaceutical services. *J Clin Pharm Ther*. 1998;23(4):303–309.

124. Wesseldine LJ, McCarthy P, Silverman M. Structured discharge procedure for children admitted to hospital with acute asthma: a randomized controlled trial of nursing practice. *Arch Dis Child*. 1999;80(2):110–114.

125. White PT, Pharoah CA, Anderson HR, et al. Randomized controlled trial of small group education on the outcome of chronic asthma in general practice. *J R Coll Gen Pract*. 1989;39(322):182–186.

126. Woolcock AJ, Salome CM, Yan K. The shape of the dose–response curve to histamine in asthmatic and normal subjects. *Am Rev Respir Dis*. 1984;130:71–75.

127. Worral G, Chaulk P, Freake D. The effects of clinical practice guidelines on patient outcomes in primary care: a systematic review. *Can Med Assoc J*. 1997;156(12):1705–1712.

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