THE NZ JOURNAL OF RESPIRATORY HEALTH

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THE NZ JOURNAL OF RESPIRATORY HEALTH

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– breathe easy this summer

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DISTANCE LEARNING ASTHMA/COPD NURSING COURSE INFORMATION

Applications are now invited from registered nurses wanting to enrol in the Asthma New Zealand/Unitec Institute of Technology Asthma Nursing Course for February 2017 and COPD Nursing Course for April 2017. The programmes are offered by distance learning. Not everyone has the same pace of learning; some students pick up things fast, others need time to grasp a concept. One of the biggest advantages of distance learning is that you can study at a pace that is comfortable for you. The primary aim of the Asthma and COPD Nursing Courses is to provide nursing health professionals with a high level of evidence-based asthma and COPD knowledge that promotes best practice and is consistent with national policy.

Since the commencement of the Asthma and COPD Nursing Courses, 1,075 nurses have enrolled in these courses. Many applicants had not undertaken any additional study since completing their nursing training, which may have been years before. However, most find the courses to be challenging but a thoroughly enjoyable learning experience that is within the grasp of any competent Registered Nurse.

Asthma New Zealand in association with Unitec Institute of Technology offers these courses within the Bachelor of Nursing Programme. Both courses are at level 7 and attract 15 credits each. **A grant towards the cost is available for registered nurses from Asthma New Zealand.**

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The closing date for enrolment is 10th February 2017 for Asthma Nursing Course
18th April 2017 for COPD Nursing Course

Upcoming events and courses

ASTHMA NEAT COURSE – AUCKLAND

15 March 2017
20 June 2017

HALF DAY COPD COURSE – AUCKLAND

16 May 2017
26 July 2017

WORLD ASTHMA DAY

Tuesday 2 May 2017

Further enquiries for any of these events phone **09 630 2293** or **www.asthma.org.nz**

MESSAGE TO READERS

Asthma NZ welcomes two new branches.

I would like to personally welcome both Asthma Wellington and Asthma Rotorua as full branches of Asthma NZ. It's been a long time coming too – we first started down this route in 2009 for Wellington and a couple of years later for Rotorua. It's an exciting time for Asthma NZ as we continue to grow and adapt, remaining always motivated and responsive and relevant to our community needs. Our organization is confronting a time of many changes and to meet these changes we must remain relevant and fluid during a time of larger nationwide and global change. The world of asthma is an exciting area in which to work, and we'll continue to meet and bring inspired people together delivering best practice and to ensure our organization remains at the cutting edge.

We're transforming the way we operate to continuously improve our ability to educate and support people with respiratory disease. Our employees and partners have continued to meet the challenges of our field and to excel despite setbacks. We should all be very proud of where we are today and excited about where we are headed.

I'd like to thank our staff for bringing their expertise to our organization. You have the vision, the knowledge, the wherewithal and the experience to help us pave our way into the future. You are truly our greatest asset today and tomorrow, and we could not accomplish what we do without your support and leadership. I ask you to stay engaged, keep us proactive and help us shape the future of asthma. My personal respect and thanks goes out to all of you.

I wish our patients, staff and health professionals and their families throughout NZ a happy and safe holiday season, we look forward to working with you all in 2017.

Linda Thompson
Executive Director
Asthma New Zealand

FOOD ALLERGIES AND ASTHMA IN CHILDREN

By Ann Wheat RN BN

Many young children have both asthma and food allergies. Having both of these problems may put them more at risk of having severe asthma, and more likely to have near-fatal or fatal anaphylactic reactions to those foods that they are allergic to.[1] Food allergies are often found in young children and precedes the development of asthma, plus food allergies are a risk factor for persistent, problematic asthma in young children.[1] It is also possible for older children and adults to develop food allergies.[2]

Food allergy and food intolerance – is there a difference?

Yes, there is a difference between the two, and it is very important to be aware of this but it is also known that many of the signs and symptoms are similar.[2] The difference between the two is that with a food allergy, there is an immune response in the body, whereas in food intolerance there is no immune response.[3] Food intolerance can be caused by an enzyme deficiency which means that they cannot digest food properly whereas an allergy is not caused by an enzyme deficiency. Food intolerance symptoms often start sometime after eating whereas a food allergy appears soon after eating a food.[3] People with food intolerance can often eat a small amount of the food with no adverse reaction whereas a person with a food allergy cannot tolerate even a small amount of food.[3] Symptoms of food intolerance are intestinal gas, abdominal pain or diarrhoea whereas in food allergy the symptoms are often hives, itchiness and swelling. If there are skin problems, there are often respiratory symptoms as well.[3]

In 1970, in fact, food allergies were rare. But over the last twenty years or so, the number of children with food allergies has soared. In fact, in New Zealand the rate of food allergies has risen by 50% between 1997 and 2011.[4] As a consequence during that time, it was recommended that foods such as dairy products should be kept out of a child's diet until one year of age, eggs until two years of age, and peanuts, nuts and fish should be kept out of the diet of infants till three years of age.[5]

Egg sensitization is one of the commonest food allergies in childhood and becomes a risk fact, for aeroallergens and asthma later in life.[1] Many children who have an egg intolerance do eventually gain a tolerance to egg and go on to be able to eat them with no problems.[1] In fact, the latest research is now saying that it is important to start solids early including egg, nuts and other foods as it has been shown that starting them early may actually help to reduce allergies in children.[5]

What are the main foods that trigger allergies in children?

The foods that are most commonly associated with food allergy are:

- cow's milk
- soy
- egg
- wheat
- peanut
- tree nuts
- sesame
- fish
- seafood/shellfish[2]

How do we treat food allergies and asthma?

The most important factor when dealing with food allergies, asthma or both is that both are well-managed. This will help both morbidity and mortality.[1] First priority for both conditions is that they are diagnosed accurately.

It is really important to assess asthma symptoms, triggers and a person's response to bronchodilators to ensure that the correct diagnosis is made.[1] The next step is to ensure that asthma is treated correctly using inhaled corticosteroids (ICS) and reliever medication as needed. ICS medication must be taken twice daily even when well to maintain good control of asthma. If still not controlled, and reliever medications are used more than twice per week, then a long-acting beta agonist should be added to the management and further assessments made to again ensure good control. There are situations that food allergies may be considered as the cause of asthma and this is where there is an episode of life-threatening asthma without any due cause, or outside of the winter season where viral infections are one of the main triggers for asthma.[1] The other situation is in highly allergic children with severe persistent asthma that is not responding to medical treatment where food allergies may not be recognized due to fractured care where children are living between two homes.[1]

Food allergies are diagnosed using an appropriate skin test, blood test or food challenge.[1] If a food allergy has been diagnosed and an anaphylactic reaction has occurred then strict avoidance of the food in question has to be maintained.[1] It is imperative that these people have the appropriate medication on hand such as an Epipen (epinephrine auto-injector) to immediately treat an acute anaphylactic episode. Reading of food labels is also advised to ensure that accidental ingestion of a food is avoided. Some people may be so allergic that even the smell of an allergen such as peanuts can be sufficient to trigger an episode. It is therefore very important that all measures are taken to avoid what one is allergic to.

In conclusion, both asthma and food allergies can occur separately but they can also co-exist in the same person. When both conditions are present, they have to be managed extremely well to maintain good asthma control and to avoid an anaphylactic reaction to a food. Food allergies are often diagnosed in young children and the evidence now is pointing to the fact that foods have been kept out of the diets of our young children for too long. The foods need to be introduced early and if there is a family history then caution needs to be taken but again, they should be introduced at the same time as for those with no food allergy history.

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DEAR NURSE

Dear Nurse, why does taking an inhaled preventer cause oral thrush?

Dear Reader, thrush occurs when the corticosteroid inhalers that depress the immune system in the lungs to control your asthma have the same effect on the surface of the throat.

A type of yeast called Candida causes thrush. We all have this yeast in our bodies but it's kept in check by our immune system so that we don't know we have it. The most obvious sign of thrush are spotty white blotches on the tongue and throat. Always rinse out your mouth, and if old enough, gargle with water after using your inhaler.

Dear Nurse, can I have a nebuliser at home for saline to clear mucous when I have a chest infection with COPD?

Dear Reader, saline nebules need to be prescribed by a doctor, as in some cases, saline can actually cause narrowing in the airways of people living with COPD. Therefore, administration of saline nebules should be monitored by health care professionals. Other factors to be considered when the nebuliser is used infrequently are maintenance of the equipment and keeping the equipment sterile to avoid introducing bacteria into the lungs.

Best practice for clearing mucous from the lungs for people living with COPD is an Active Cycle of Breathing. This involves breathing control (gentle breathing) then 3 or 4 deep breaths in, breathe out gently; breathing control again followed by 1-2 huffs. To huff, take a medium breath (not a deep one) then squeeze the air out using tummy and chest muscles at the same time, then breathing control again. Repeat until the mucous comes up or rest as you need to. You can ask your asthma nurse or your GP to refer you to a physiotherapist to help you learn the technique.

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Dear Nurse, my child is 4 years old, is that too young to start on a preventer inhaler?

Dear Reader, if your 4 year-old has been diagnosed with asthma and is on a reliever, a preventer may be introduced according to the frequency of your child's symptoms. Most children from 1 to 5 years with an asthma wheeze only have symptoms during a viral illness but others will have symptoms between these times. Children who have increasing or high frequency of reliever use should be considered for a preventer.

Indications for introduction of a preventer includes: wheezing or breathing problems more than 2 days per week and/or during the night or when your child wakes; wheezing or breathlessness during exercise, vigorous play or laughing; and needing the reliever more than 2 days per week.

Your doctor will review your child's persistence of asthma symptoms during the day and night, around your child's activity and frequency of flare-ups of asthma symptoms.

According to symptom persistence, your child may be started on a tablet (Montelukast) or a preventer inhaler, and your GP will review your child's response after 4 weeks with a trial period of 3 months, as the medication takes this long to take full effect.

Remember the goal of asthma management is to achieve control of symptoms while minimising asthma symptoms which includes, reducing symptoms day and night, minimal use of reliever and no limitation to play or activity. Viral illnesses are frequent in under 5 year-olds so best practice is to aim for control of interval symptoms and management of illness.

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Dear Nurse, how often should my asthma be reviewed?

Dear Reader, according to the Global Initiative for Asthma Guidelines (GINA) 2016, patients should preferably be seen 1-3 months after starting treatment and every 3-4 months after that, except in pregnancy when they should be reviewed every 4-6 weeks. If you have had a hospital admission or an emergency treatment for an asthma exacerbation, you should be reviewed within a week.

Frequency of review depends on how well your asthma is controlled and your response to treatment. Remember, asthma is a variable condition and there may be times when treatment can be reduced if asthma is well controlled but on the other hand treatment may need to be stepped up if asthma is poorly controlled.

The Asthma Control Test (ACT) consisting of five quick questions about how well your asthma has been over the last 4 weeks, is a helpful tool to assess asthma control at each review. Also, your peak flow should be checked at the review. It is important for you to know what your best peak flow is when you are well so you need to have your own peak flow meter to use at home to monitor this.

The review is also a good time to have your inhaler technique checked and rechecked.

IF YOU HAVE A QUESTION PLEASE EMAIL OR POST TO:
editor@asthma.org.nz or Dear Nurse, Asthma New Zealand, PO Box 67066, Mt Eden, Auckland 1349.

PHARMAC

by Janet Delooze RN

This year saw the arrival of several new inhalers for asthma and COPD which is great news for the people out there with these conditions. Even better, is that they are all once daily medications, something novel for those with asthma. But how does the arrival of new medications work in the New Zealand health system? This article briefly explains the role of PHARMAC, the Pharmaceutical Management Agency and the decisions that are made about what medications will be available for the people of New Zealand.

PHARMAC is the government agency that decides which medicines and medical devices will be funded and included on the New Zealand Pharmaceutical Schedule. It was set up in 1993 following a massive increase in medication prices in the 1980s; PHARMAC decides which medications that the district health boards (DHBs) will fund through the Combined Pharmaceutical Budget (CPB).[1] Their main aim is to provide cost effective drug therapy to achieve the best outcomes within that budget. PHARMAC also have a Discretionary Pharmaceutical Fund (DPF) where individual applications can be made. They report directly to the Director-General of the Ministry of Health, and work closely with other agencies. PHARMAC's main functions are described in Table 1, and their roles in Table 2.

Table 1. PHARMAC's main functions.[2]

PHARMAC has four main functions:

1. **Managing the Pharmaceutical Schedule**
   Consisting of about 1900 Government-subsidised community pharmaceuticals, 2600 medicines used in public hospitals, and 14,000 hospital medical devices (September 2015).
2. **Promoting the responsible use of medicines**
3. **Managing the Named Patient Pharmaceutical Assesssment policy for patients in exceptional circumstances**
4. **Engaging in research as required**
   We are guided by a number of laws, regulations and Goverment guidelines, and Medicines New Zealand – the Government's strategy for the medicines system, as well as our own internal decision making framework and the Factors for Consideration.

Table 2. PHARMAC's role in New Zealand.[2]

PHARMAC's role within the New Zealand health system is to make decisions on which medicines and medical devices are funded in order to get the best health outcomes from within the available funding. Our effectiveness depends significantly on the work of others. Medicines New Zealand is the Government's strategy for the medicines system. It defines three main outcomes for the medicine system:

- **Access:** New Zealanders have access to the medicines they need, including equity of access to medicines
- **Optimal use:** medicines are used to their best effect
- **Quality:** medicines that are safe and effective.

PHARMAC's work focuses on access and optimal use of medicines. Quality is primarily the role of other organisations, in particular Medsafe.

Medicines New Zealand strategy
Medicines New Zealand provides an overall framework for PHARMAC's operations. The strategy, published in 2007, intends to inform decision making over the long term and to deliver a world-class medicines system for New Zealanders.

PHARMAC works closely with other government agencies, (see Table 3) including Medsafe – the New Zealand Medicines and Medical Devices Safety Authority.[3] They are responsible for the regulation of medicines and medical devices in New Zealand. They ensure that medicines and medical devices are acceptably safe.

In 2012, PHARMAC took over the management of the National Immunisation Schedule from the Ministry of Health (MoH) including purchasing all vaccines after their success in the purchase of influenza vaccines from 2004.[5]

Since PHARMAC was established, it claims to have negotiated better contracts for medications and is able to supply NZ with three times the amount of medications for the same sum of money that it did in 1993.[1] It has been able to do this by negotiating multi-drug contracts and introducing competitively priced generic brands. PHARMAC makes its purchasing decisions based on feedback from the DHBs, Medsafe, the Pharmacology and Therapeutics Advisory Committee (PTAC) and the Consumer Advisory Committee (CAC).1 Since its establishment, PHARMAC's purchasing role has expanded to include medical devices, vaccines, cancer medicines and haemophilia treatments. It works within the framework of the Medicines New Zealand strategy whose guiding principles are equity, effectiveness, confidence, value for money, affordability and transparency[4]

So, although PHARMAC is often reported in the news for the controversial decisions it has made regarding unapproved medicines, 2016 has been a fruitful one for new inhaled medications.

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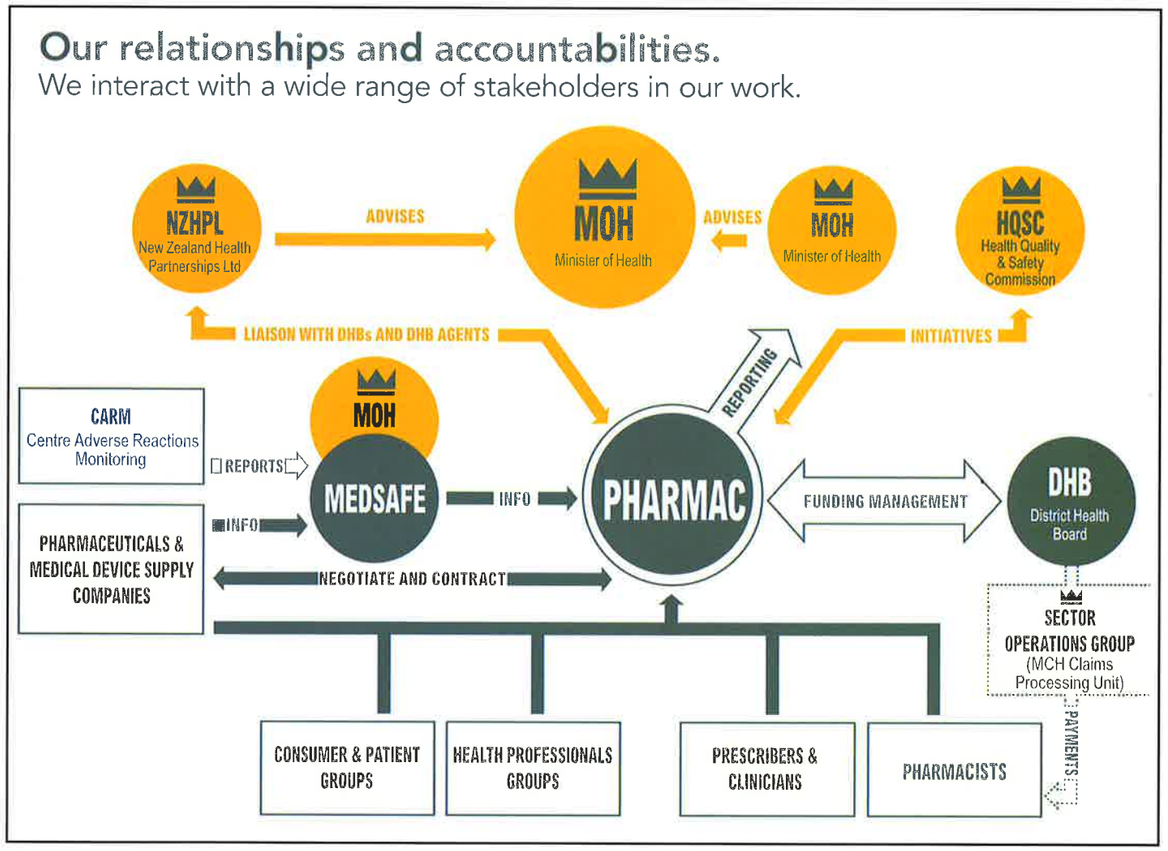
Table 3. PHARMAC's relationship and accountabilities.[2]



IRRITANTS CAN TRIGGER ASTHMA

By Elaine Murray RN, Asthma Nurse Educator

Asthma is a common and potentially serious chronic disease that imposes a substantial burden on patients, their families and the community. It causes respiratory symptoms, limitation of activity, and flare-ups (attacks) that may be life threatening. Fortunately, asthma can be effectively treated, and most patients can achieve good control of their asthma.

Asthma causes symptoms such as wheezing, shortness of breath, chest tightness and cough that may vary over time in their occurrence, frequency and intensity.[1] There is an inappropriate inflammatory response in the airways which makes them hyper-responsive to a wide range of triggers, so that they narrow excessively. This inflammatory process can cause permanent changes in the airways.[2] The symptoms of asthma vary widely from person to person. The absence of typical symptoms does not exclude asthma.[2]

When detecting possible asthma, it is important to get a history of current symptoms, usually over the last four weeks; what is the pattern of symptoms, e.g. worse in winter or summer; what are the triggers; does it impact on work and life style; what is the home and work environment like; any past history of eczema, hay fever or atopy in the family? Do the symptoms respond to the reliever inhaler?[2]

What are asthma triggers?

Triggers are factors that make your asthma worse. Trigger factors may be allergic (e.g. allergy to pollens, cat or dog, house dust mite) or non-allergic (e.g. respiratory infections, exercise, cold air, temperature changes or chemicals and fumes).

Could it be an irritant?

There are a variety of irritants that can trigger asthma.[3] Could it be the fragranced products you are using in the home? Fragrances are pervasive. They are in hundreds of different products that we use, such as soaps, candles, laundry detergents, air fresheners and insect repellents, deodorants perfumes and aftershave. The common reactions to fragranced products are:

Respiratory problems such as coughing, difficulty breathing and shortness of breath

- Nasal congestion, sneezing, red itchy eyes
- Headaches
- Hives, skin rash or dermatitis
- Asthma attacks

There are other irritants that can trigger asthma such as cigarette smoke, fireplace smoke, bonfires, wood burning stoves, unvented gas heaters or stoves, car exhaust or diesel fumes.

There may be chemicals in the work place such as paint fumes, glues and cleaning chemicals.

Irritants are different from allergies. People who don't have allergies or asthma can be affected by irritants, though this may not cause serious problems. For those with asthma, irritants can lead to airway inflammation and flare-ups requiring medical attention.[4]

As an asthma nurse educator, I am often told that perfumes, chemicals and fumes are triggers for asthma. A high school student told me that she always hated getting on the bus in the morning as the fumes from the perfumes and after-shaves that everyone had used before leaving for work or school, triggered her asthma. A young boy told me that he had an asthma attack after his brother sprayed a body deodorant all around his bedroom. A young girl told me that she could not walk down the cleaning product aisle at the supermarket as it made her feel really tight in the chest and short of breath.

I visited a client at home to provide her with information and asthma education. She had moved to Auckland from Tauranga and her asthma had suddenly got worse. She could not understand why, as she was still using her preventer every day as prescribed but was also needing to use her reliever every day. We finally worked out that the wall-mounted automatic insect repellent was the trigger and once it was removed she had no further problems. A young girl experienced an asthma episode that required emergency treatment at a hospital after inhaling smoke from a bonfire and the fire crackers while celebrating Guy Fawkes.

The first step in managing your asthma is to know what your triggers are, whether they are allergic allergens or irritants so that you can try to avoid them or minimise your exposure to them. Avoid using chemical cleaners in your home. There are many eco-friendly products available. Baking soda, white vinegar and lemon are very good for cleaning. Remember to ventilate your home to get rid of stale odours and smells, to avoid having to use commercial air fresheners. Chlorine used in swimming pools can be a trigger, especially if it is an indoor pool, so it is best if you swim in an outdoor pool or at the beach. Air pollution, car exhaust, and gas fumes should be avoided. If you live by a motorway or busy intersection, there will be a high concentration of fumes in the air, so it may be best to move. Wood burning stoves, fire places and unvented gas stoves or heaters can worsen asthma symptoms, therefore, electrical appliances would be a better option. A person with asthma should never smoke; smoking should not be allowed in the person's home, and second hand smoke should be avoided whenever possible.

Industrial or occupational exposure to chemicals is responsible for a lot of cases of asthma.

If you have asthma, and have experienced worsening symptoms following an exposure to an irritant, you need to consider trying to avoid that irritant in the future, whether at home, at work or during leisure time activities.

Always use your preventer inhaler as prescribed so your asthma is well controlled and see your doctor regularly for a review.

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ASTHMA MANAGEMENT AT SCHOOL

By Alice Paul RN, Asthma Nurse Educator

One in four children in New Zealand will have asthma at some time during childhood. Asthma is a chronic childhood illness which sadly leads to absenteeism from school.

During an asthma exacerbation, the child experiences breathing problems, coughing, wheezing, tight chest and shortness of breath. Unfortunately, asthma cannot be cured but with a good management plan it can be well controlled, and children can run and play as normal.

New Zealand, along with countries like Australia and Great Britain, has one of the highest rates of asthma in the world. It disproportionately affects Maori and Pacific Island people, and those from lower socio-economic groups.[1]

So what is the best way to manage asthma at school?

- Education
- School wide asthma plan/policy
- Keep the environment free of asthma triggers
- Use of inhalers in school

Education

Ensuring staff get the opportunity to participate in an asthma education session which is available from the Asthma NZ nurse educators. It is also important for teaching staff to recognize students whose asthma is poorly controlled and communicate that with the parents/carers and be able to give them information about how to access asthma education.

Many young people have exercise induced asthma and it is important for PE teachers/coaches to be familiar with the nature of the disease, signs and symptoms, first aid and correct method for administering a reliever.

School wide asthma plan

Schools should consider developing a school asthma policy which reflects that the school is looking after students with asthma. It also ensures that staff have the knowledge and skill to help a student with asthma if the need arises.

What is a health policy?

A health policy is a set of broad goals that outline the approach an ECE (early childhood education) service or school will take to support children and young people with health conditions. Policy goals are commonly expressed in detail in ECE service or school procedures. Procedures should be practical, easy to implement, and ensure all children with health conditions receive the care and support that they need to attend regularly and engage in learning. Talk to families and whanau, seek the advice of health professionals and refer to the legal framework and requirements relevant to your sector as you develop, review or adapt your policy and procedures.[2]

Keep the environment free of asthma triggers

Asthma is an inflammatory condition of the airways, which causes breathing to become difficult. When children and adolescents are exposed to things in the environment, such as dust mites, moulds, pollen, strong fumes (chemicals, perfume, cleaning liquids), an asthma attack can occur. These are called asthma triggers.[3]

We generally concentrate on the child's home environment but must remember that the child spends 6 hours a day in the classroom. The school environment may be an important site of exposure to indoor allergens, including cockroach, cat, dog, mouse, dust mite, and moulds, known to be important in the home environment.[4]

Use of inhalers in school

Use of medications is different for every individual. Some children take a reliever only, and only for exercise-induced asthma; others take a variety of daily medication. However, everyone with a diagnosis of asthma should have a bronchodilator or quick-relief (reliever) medication to quickly reverse the narrowing of the airways that happens during an asthma flare-up. The only medication the school should concern itself with is the bronchodilator reliever inhaler (usually blue). Schools are allowed to hold emergency reliever inhalers. This is to ensure that if the student has forgotten to take their own reliever to school there is one available in an emergency.

Conclusion

Managing asthma at school works well if there is good communication between staff, parents and the students themselves. We, as educators, are working hard in the school arena and would like to see an asthma policy in all schools to ensure the safety of the students.

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O2 JOURNAL - YOUR OPINIONS MATTER!

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https://www.surveymonkey.com/r/3WCF6BW

MANAGING RESPIRATORY DISEASE IN A NATURAL DISASTER

By Adie Riddell RN

A natural disaster is a catastrophic event defined as a disruption of human ecology that exceeds the community's capacity to adjust, so that outside assistance is needed. This has the potential to increase the risk of acute respiratory diseases plus other infectious disease transmission or outbreaks within days, weeks or even months after the event.[1]

Chronic illnesses, such as asthma and chronic obstructive pulmonary disease (COPD) are at high risk of being exacerbated by the conditions caused by a disaster (e.g., lack of food, lack of clean water, extremes of cold or heat, physical and mental stress, injury, exposure to infection).

There is also a high risk of medication, medical equipment, and medical supplies being difficult to access for a period of time after the event. This makes the older population (who often have multiple chronic conditions and comorbidities with multiple medications) particularly at risk. This can be complicated further by people with chronic respiratory disease often not accessing routine health care, therefore, not having medication available at the time of a disaster occurring.[2]

What better topic to write about this month than how a natural disaster can impact on respiratory disease management, having experienced both an earthquake with a tsunami warning thrown in to the mix, followed 24 hours later by major flooding in much of the affected area. I live and work in Wellington and can only be thankful that I live here and not Kaikoura (thoughts are with you Kaikoura). I wonder how many people in the community who are reliant on medication to manage their respiratory conditions, have an easily accessible emergency supply available to them?

Where populations are displaced, creating reduced access to health services and to antimicrobial agents for treatment, acute respiratory infections (ARI) are known to be a major cause of illness and even death, especially in young children.[3] Access to health services is likely to be disrupted which can lead to suboptimal or delayed treatment for ARIs. With natural disasters comes the high likelihood of displacement from ones normal place of abode. This may include lengthy stays in emergency shelters which are likely to be crowded and uncomfortable with disrupted sleep/rest. Nutrition could be potentially altered and the potential for fumes such as cooking with open stoves, may lead to an exacerbation of respiratory illness. The reported incidence of ARI increased four-fold in Nicaragua in the 30 days after Hurricane Mitch in 1998, and ARI accounted for the highest number of cases and deaths among those displaced by the tsunami in Aceh in 2004 and by the 2005 earthquake in Pakistan. Risk factors among displaced persons include crowding, exposure to indoor cooking fumes using open flame, and poor nutrition.

Physical inactivity has also been identified as a risk factor for symptom aggravation and mortality in COPD.[3] There is the potential for people to be restricted to either emergency shelters or their homes for an extended time, and public recreation facilities such as pools and walking tracks may be inaccessible.

But it is not only people with known respiratory illness that can be affected by these natural disasters. Acute symptoms like bronchospasm and haemoptysis and infectious diseases, both common ones like influenza and rare ones like Nocardia, have been known to occur with increasing frequency.[4]

Following the 2011 Great East Japan Earthquakes in 2011, research revealed a notable increase in patients with COPD being hospitalized compared with pre-earthquake, with patients on oxygen therapy at home needing to access the same care at a hospital.[4] The problem with this, however, was that hospital beds were full with casualties from the disaster, requiring special centres to be set up to deal with these clients. Their care was complicated by some patients having been drenched by the tsunami, others being deprived of oxygen supplies, and some also deprived of their prescribed drugs, and this interruption of regular treatment may have partly contributed to the worsening of symptoms. There was a notable decline in their condition in the subacute phase. The increase in exacerbations was attributed to multiple contributing factors as well as lack of medication.

An increase in tracheobronchial infections impacts on COPD. Unfavourable conditions such as temperature change, water and food shortages, damaged dwellings, stress, and an inability to maintain the appropriate level of personal hygiene are likely to result in the occurrence of respiratory infections. Air pollutants such as dust and toxic fumes, chemicals and biological materials were all found to have increased worsening respiratory symptoms among COPD patients.

Planning for an event where you may be displaced for a period of time is essential. Ensuring your emergency kit containing appropriate spare emergency medication (you can discuss your needs with your family doctor) to manage your respiratory condition.

It is wise to always ensure you have picked up your new prescription for your medications well in advance of them running out. Don't leave it until that last day! Write down any specific needs you or your child might have. Make a list and put it in your purse or wallet. Photos of medication may help. Ensure your child has a medication kit at their school or early childhood centre

Find someone, a spouse, roommate, friend, neighbour, relative, or co-worker, to help you in case of an emergency. Give them the list. You may wish to provide a spare key to your home, or let them know where they can find one in an emergency. Have at least 3 days medication in your emergency kit. For health professionals planning for assisting populations with chronic diseases, especially vulnerable older adults, during a disaster is essential to meeting their special needs.

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COMMUNITY EXPOS

The nurses at Asthma Auckland continue to collaborate with other health providers in the community to raise awareness of asthma and chronic obstructive pulmonary disease (COPD). At these open days, it is great to network with other organisations and talk to families that we would not be able to reach in any other way.

Papakura Seniors Expo 3rd September 2016

Karen and Janet attended this celebratory event for Papakura Seniors and the launch of the new Legends-active living leaders programme. Statistics NZ projects that by the year 2038, there will be 1.285 million people over the age of 65 which is 23.4% of the population. By the same year, the number of people 90 and older is expected to reach 81,900!

Over 22 stallholders provided information and activities to the general public who attended the event. Asthma Auckland performed free spirometry testing on a number of people who had either been diagnosed with COPD, or who were worried about a cough or shortness of breath. We provided stop smoking advice and written information. Many extended families attended so we also provided asthma education to over 30 people.

Manurewa Community Expo 6th October 2016

Karen and Janet again, were involved in this annual event. This Expo is hosted by the Manurewa Community Network, Manurewa Business Association, and the Southmall Shopping Centre. The Expo aims to provide a platform for community social, health, wellbeing, educational, local and central government agencies to showcase their services and programmes. Over 80 stallholders participated. As we were situated in the middle of Southmall, we had many people passing by after completing their shopping as well as the public who attended after seeing the event advertised. We spoke to over 100 people about asthma, allergies, stopping smoking and COPD.

Asthma nurse educator Janet Delooze, engaging with the community.

Kelston Community Health Expo

Ann and Elaine attended the Kelston Community Health Expo held on the 6th October 2016 at the Kelston Community Centre. This was held during the school holidays, encouraging the entire whanau/family to enjoy a range of health related information, events and services.

It was well attended and there were over 30 exhibitors in the main hall for people to visit to get information. There was also Tai Chi, Pacific Zumba, Dance Pacifica, Bollyrobics and Up and Active activities to join in. Once you had worked up an appetite, there were fruit smoothies, veggie platters and easy dips to enjoy.

Despite the weather it was well attended.

ASTHMA WELLINGTON REGIONAL REPORT

Over the past three months Alice, Kim and Adie have been busy in the community.

At Wellington Hospital, we have attended several meetings with both adult and paediatric medical staff which continues to reaffirm the relationship between hospital and community. We now have systems in place to allow for easy referral by hospital staff to our service. Our goal is to visit all children at home once they have been discharged from hospital with a respiratory condition.

We have been approached by Otago University who are doing the ' He Kura' study, and Massey University who are doing the 'Asthma' study in 8-18 year olds. They have requested our input and collaboration, and we see this as a very positive involvement, and an opportunity to help in promoting these worthwhile and important asthma studies.

Throughout the year, we have offered a 'road show' to primary care practices providing a short education session for doctors and nurses about the asthma/COPD medications and devices. We have also provided education sessions for the nursing staff at aged care facilities to update them on the new medications and devices for COPD/Asthma.

Our 'schools' (Asthma Education for Staff) programme continues to keep the nurses busy with great interest from kindergartens and primary schools.

This year, we had the pleasure of working with Wellington City Council Housing and were invited to run a series of 'healthy breathing' seminars at their larger housing developments. It was a great success and we are all very keen to repeat this next year.

From August, Glenn White has run several 'Buteyko' breathing courses using our premises in Johnsonville. It feels good to be able to offer our community some options in optimising their respiratory health.

In August, both of the Wellington nurses attended the Thoracic Society of Australia and New Zealand (TSANZ) conference in Queenstown which had an emphasis on bronchiectasis. This provides a great opportunity to network and keep up with current respiratory research.

Kid's Page

1 Fill in the blanks! Use words from the list below.

When I'm in my green zone, I feel ..... .1. I still have to use my .....2. I have to watch out for .....3 that may set-off my asthma. When I'm in my yellow zone, I'm starting to have a .....4. I might wheeze or have other .....5. Then I have to use my .....6 inhaler. When I'm in my red zone, breathing is very .....7. I need to get .....8 right away.

hard
help
good
preventer
triggers
flare-up
symptoms
reliever

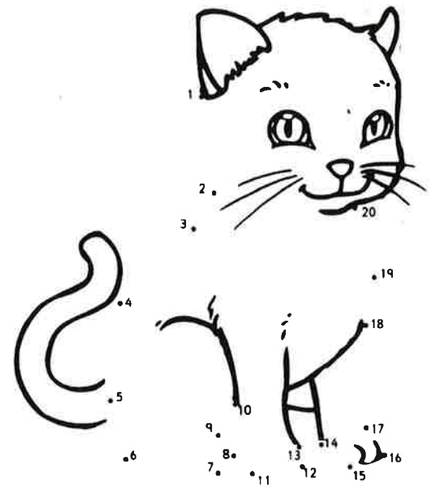
2 Pick the correct spelling:

Astma	Ashtma	Asthma
Vacuum	Vaccuum	Vacumm
Leisure	Liesure	Leasure
Hygiene	Hygiend	Higyine
Vegetarian	Vegatarian	Vegeterian
Size	Seize	sieze
Caucasion	Coucasian	Caucasian
Tounge	Toung	Tongue
Harras	Harrass	Harass
Rapsberry	Raspberry	Rasberry
Redicilus	Rediculous	Ridiculous
Privelige	Privilege	Previlege
Grammar	Gremmer	Gremmar
Recomend	Recommend	Reccomend
Misspell	Misspel	Mispell

3 What do I feel like when I have asthma? Unscramble these words...

Ads
Itred
Pygurm
Espedrdse
Tgrfednehi
Gnrya

4 Cat dot to dot



2) Pick the correct spelling

		Misspell
	Recommend	Grammar
	Raspberry	Ridiculous
		Harass
		Tongue
		Caucasian
	Seize	
		Vegetarian
		Hygiene
		Leisure
		Vacuum
Asthma		

Answers

1) 1. good, 2. preventer, 3. triggers, 4. flare-up, 5. symptoms, 6. reliever, 7. hard, 8. help

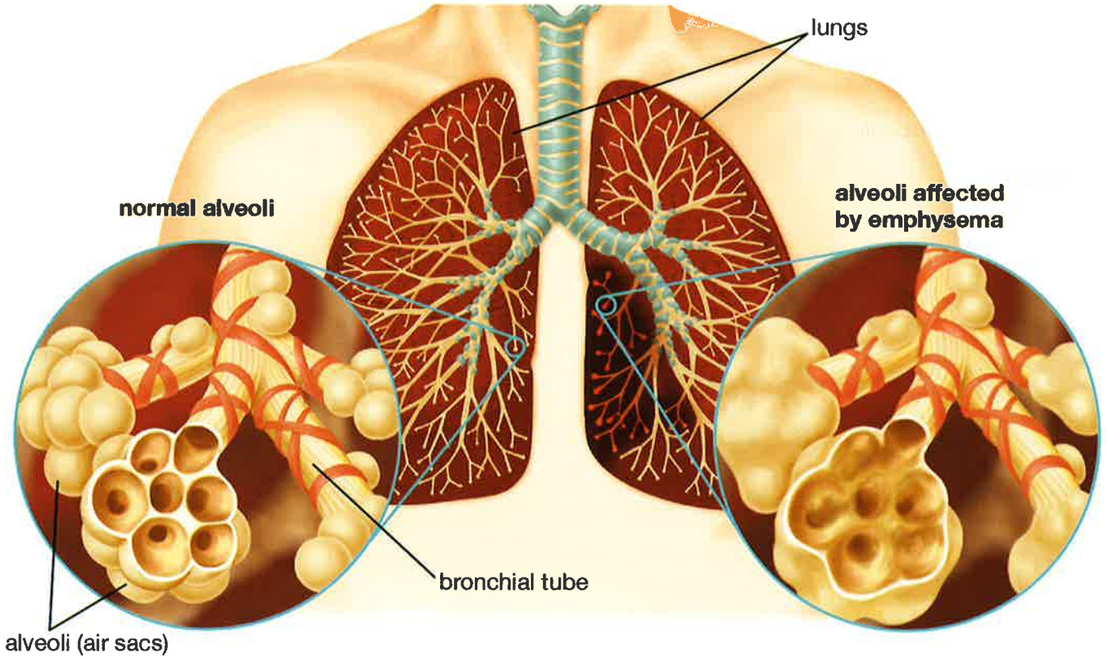
3) Unscrambled words

Sad, Tired, Grumpy, Depressed, Frightened, Angry

EMPHYSEMA

By Karen Little, Asthma Nurse Educator

It is often necessary to explain to clients what emphysema is, and why it is a condition included in the broad title of chronic obstructive pulmonary disease (COPD). COPD covers the spectrum of disease such as chronic bronchitis, emphysema and poorly controlled asthma. When offering stop smoking advice, it is also an advantage to be able to explain how smoking can destroy alveolar walls. The airway inflammation in COPD, represents an exaggeration of the normal inflammatory response to irritants such as cigarette smoke. You can have emphysema for many years without noticing any signs or symptoms. The main symptom of emphysema is shortness of breath, which usually begins gradually. You may start avoiding activities that cause you to be short of breath, so the symptom doesn't become a problem until it starts interfering with daily tasks. Emphysema eventually causes shortness of breath even while you're at rest.



The destruction of alveolar (air sac) walls results in abnormal and permanent enlargement of airspaces, and loss of lung elasticity resulting in reduced surface area for gas exchange. It is unclear how this process occurs although the findings of large numbers of inflammatory cells in emphysema suggest that the process is an abnormal response to injury. Preservation of the blood supply to alveoli is essential and occlusion of this blood supply precedes parenchymal dissolution – "dissolving lung". Parenchyma is a term that refers to the parts of the lungs involved in gas transfer; these include the alveoli, interstitium, blood vessels, bronchi and bronchioles.

The lung is very much like a very finely-pored sponge, containing about 300 million alveoli.[1] Only 1% of emphysema is caused by alpha-1 antitrypsin deficiency. (This topic was well covered by Ann Wheat in the August issue of this magazine). Cigarette smoking is, by far, the most common cause of COPD, accounting for more than 95% of cases in industrialized countries. The fact that only 10-20% of smokers develop COPD may be largely determined by, as yet, unidentified genetic factors. It is very likely that multiple genes determine a smoker's susceptibility to developing COPD.[2] Occupational, inhalational and environmental exposures including biomass fuel cooking are also risk factors. Throughout the world COPD is a disease of occupation and environmental pollutants.[3]

Emphysema is due to an imbalance between proteases (that digest elastin and other structural proteins in the alveolar wall) and antiproteases that prevent against this attack. Matrix metalloproteinases (MMP) are a group of over 20 closely related endopeptidases that are capable of degrading lung parenchyma including elastin, collagen and fibronectin. MMPs are produced by neutrophils, alveolar macrophages and airway epithelial cells. The inflammation is further amplified by oxidative stress. Oxidants are produced from cigarette smoke and are released from inflammatory cells. Oxidants may contribute to emphysema in several ways including damage of protease inhibitors, potentiation (enhancement of one agent by another so that the combined effect is greater than the sum of the effects of each one alone) of elastase activity and increased mucous secretion.[4] Loss of individual alveoli with connecting wall destruction leads to airflow limitation in two ways. Firstly, loss of alveolar wall results in a decrease in elastic recoil which limits airflow. Secondly, the loss of alveolar supporting structures is indirectly responsible for airway narrowing, again limiting airflow.[2] When you exhale, the damaged alveoli don't work properly and old air becomes trapped, leaving no room for fresh, oxygen-rich air to enter. Treatment may slow the progression of emphysema, but it can't reverse the damage.

The pathology of region-specific emphysema is divided into three patterns:
- Centriacinar (centrilobular)
- Panacinar (panlobular)
- Paraseptal

Centriacinar emphysema is the most common type mainly localised to the proximal respiratory bronchioles with focal destruction and mainly found in the upper lung zones. The surrounding lung parenchyma is usually normal with untouched distal alveolar ducts and sacs. Panacinar emphysema destroys the entire alveolus uniformly and is predominant in the lower half of the lungs. Paraseptal emphysema involves the distal airway structures, alveolar ducts and alveolar sacs.[6]

Stopping smoking is the single most beneficial management strategy and is the only intervention that reduces the accelerated decline in lung function. One of the most important developments in the future will be the detection of COPD at an earlier stage before symptoms appear. This will depend on screening of cigarette smokers in the community and instituting preventative measures such as smoking cessation and possible drug therapy. To prevent emphysema, don't smoke, and avoid breathing secondhand smoke. Wear a mask to protect your lungs if you work with chemicals fumes or dust.

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ASTHMA–COPD OVERLAP SYNDROME (ACOS)

by Janet Delooze RN, MHPrac

Asthma and chronic obstructive pulmonary disease (COPD) are long-term conditions that are common in New Zealand. Some people, however, have overlapping diagnoses where they have components of both conditions, known as overlap syndrome (ACOS).

Incidence

Currently, there does not appear to be statistics available of New Zealanders with overlap syndrome although we know that it is more prevalent in older adults.[1] In a small study in Australia of 44 adults, 65% were found to have overlap syndrome.[2] A larger study carried out in Finland in 2011 found that 15% of the 1,546 participants had clinical features of both asthma and COPD.[3] Data from three pulmonary clinics in the US found between 15.8% and 24.3% with overlap syndrome.[1]

Characteristics of obstructive airways diseases

Asthma is characterized by episodic airflow obstruction that is reversible. It tends to start from an early age however it can occur at any age. It is often triggered by allergens but exercise, irritants and viral infections can also trigger symptoms.

COPD tends to occur in older age groups and is mostly caused by smoking (90%),[4] although occupational irritants, biofuels and genetic factors can also be causative. COPD is a disease state that is characterized by airflow limitation that is progressive and not fully reversible.

COPD is an umbrella term for chronic bronchitis and emphysema, though for most patients there is usually an element of both conditions.

Chronic bronchitis is defined as the presence of a cough productive of sputum on most days for at least three months of two successive years. Emphysema is characterised by abnormal airspace enlargement and destruction of lung parenchyma.

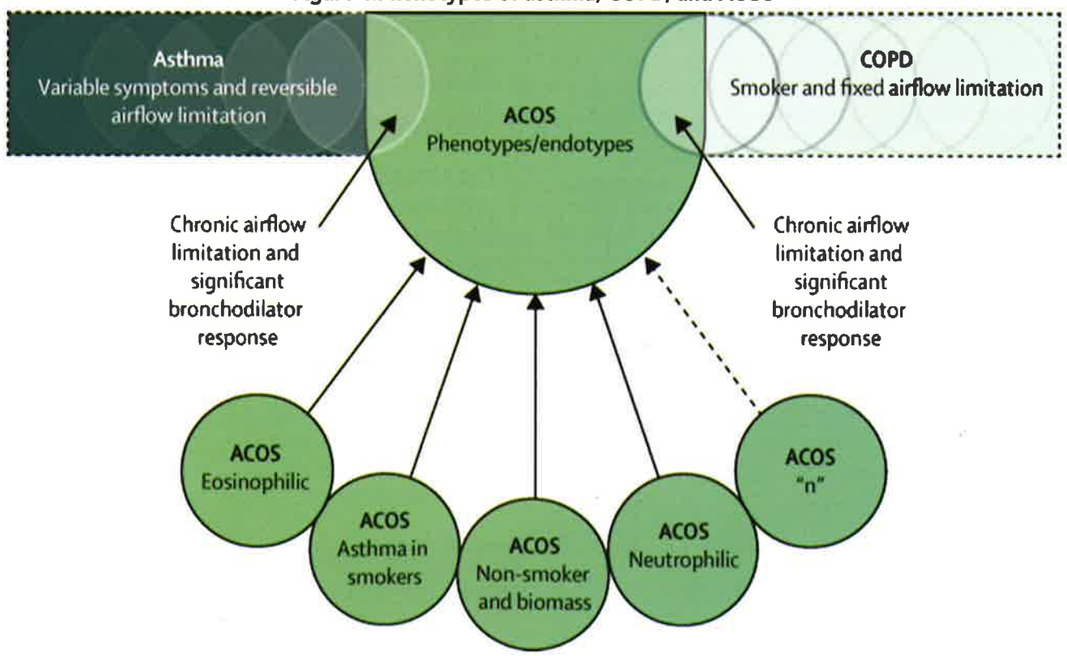
The mechanisms of inflammation are different in asthma and COPD: asthma is primarily characterised by an eosinophilic inflammation whereas COPD is associated with a neutrophil and macrophage-induced inflammation (Figure 1).[5]

With ACOS, there are variable clinical features, such as patients with COPD where there is reversible airflow obstruction, others with asthma where there is incomplete reversibility and airway remodelling, and non-smokers who develop COPD.

In the Wellington Respiratory Survey, five clinical phenotypes were identified as follows:

Cluster 1: severe and markedly variable airflow obstruction with features of atopic asthma, chronic bronchitis and emphysema
Cluster 2: features of emphysema alone
Cluster 3: atopic asthma with eosinophilic airways inflammation
Cluster 4: mild airflow obstruction without other dominant phenotypic features
Cluster 5: chronic bronchitis in nonsmokers.[6]

Figure 1.Phenotypes of asthma, COPD, and ACOS



"The relation between asthma, chronic obstructive pulmonary disease (COPD), and asthma–COPD overlap syndrome (ACOS) and possible phenotypes of these disorders that might be identified by comprehensive phenotyping. Both asthma and COPD are heterogeneous diseases and comprise several phenotypes. Some patients (those with chronic airflow limitation and a substantial bronchodilator response) can be viewed as having ACOS, as might patients with several features of both asthma and COPD, which, in time, might be identified as specific phenotypes of ACOS. Some suggested phenotypes of ACOS are presented as clear circles. ACOS "n" are hypothesised additional phenotypes of ACOS that might be identified in the future. Dotted arrow represents that ACOS "n" is not a single phenotype but consists of an unknown number of phenotypes."[7]

Why is it important?

Patients who have both asthma and COPD have more frequent exacerbations, more rapid disease progression, increased co-morbidities and worse health-related quality of life than if they had one condition.[7]

Whilst the Global Initiative for Asthma (GINA) guidelines advocate a step-wise approach in the treatment of asthma,[9] the Global Initiative for Obstructive Lung Disease (GOLD) advises on the management of COPD.[4] Overlap syndrome patients are often excluded from clinical trials of asthma and COPD and therefore, there is limited evidence to support guidelines for management. However, some guidance has been given via a joint project between GINA and GOLD.[10]

Stepwise approach to diagnosis

- Diagnosis is based on a detailed medical history, physical examination, and other investigations
- Syndromic diagnosis by comparing the features of asthma or of COPD, and assessing the level of certainty of diagnosis
- Spirometry to confirm airflow limitation
- Start treatment according to differential diagnosis following GOLD (COPD) or GINA (asthma) guidelines. In cases with an equal balance of features of both conditions, treatment should default to commencement of asthma treatment
- Referral for specialized investigations if necessary.[10]

Treatment

At the moment, the treatment tends to be drawn from the existing guidelines of GINA or GOLD depending upon the most prevalent clinical features[1]. Establishing whether the inflammation is eosinophilic or neutrophilic may be helpful, either by sputum or exhaled FeNO testing; there is poor response to inhaled corticosteroids (ICS) in non-eosinophilic inflammation.[11] In a recent study it was found that most patients were easily classified as either asthma or COPD but up to 19.8% of patients were difficult to diagnose based on clinical presentation and spirometry.[12]

In conclusion, overlap syndrome is present in a proportion of patients with obstructive airways disease and several classifications or phenotypes have been suggested. However, with few clinical trials being carried out with ACOS, management can be difficult . Hopefully, more work will be carried on this important area of respiratory disease.

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NEWSTREAM

Source: **Respirology**

Infant lung function predicts asthma persistence and remission in young adults; Owens L, Laing I, Zhang G, Le Souëf P; Respirology (Sep 2016)

BACKGROUND AND OBJECTIVE: Asthma in adults is associated with a persistent reduction in lung function from childhood, but this link has not been assessed back to infancy. Reduced infant lung function (ILF), a measure of antenatal and infant lung growth, is associated with asthma into adolescence. Our aim was to assess whether this link persists into adulthood and whether ILF can predict the remission of asthma symptoms in young adults.

METHODS: The study cohort was an unselected full-term birth cohort of 253 subjects enrolled antenatally with lung function assessments at 1, 6 and 12 months (maximum expiratory flow at functional residual capacity, V'maxFRC), and 6, 11, 18 and 24 years (spirometry) of age.

RESULTS: Infants with V'maxFRC in the lowest quartile at 1 month had an OR of 5.1 (95% CI: 2-13, P = 0.001) for asthma at 24 years. Subjects with asthma at 24 years had a mean V'maxFRC at 1 month of 69% predicted (95% CI: 48-90%) versus 110% (95% CI: 101-119%) in non-asthmatic patients (P = 0.001). Subjects with current versus resolved asthma symptoms at 24 years had a mean V'maxFRC at 1 month of 69% predicted (95% CI: 53-84%) versus 105% (88-123%), respectively (P = 0.003). Subjects with current asthma at 24 years had persistently lower lung function from infancy with a mean reduction of 16.2% (95% CI: 8.1-24.3%, P < 0.0001).

CONCLUSION: Reduced lung function in early infancy is predictive of persistent asthma in young adults and a persistent reduction in lung function, suggesting abnormal lung development and growth in utero or very early in life.

Source: **Am J Respir Crit Care Med**

Preeclampsia Associates with Asthma, Allergy and Eczema in Childhood; Stokholm J, Sevelsted A, Anderson U, Bisgaard H; American Journal of Respiratory and Critical Care Medicine (Sep 2016)

INTRODUCTION: Preeclampsia reflects an unusual increase in systemic inflammation during pregnancy. We studied associations between preeclampsia and asthma, allergy and eczema in Copenhagen Prospective Studies on Asthma in Childhood2000 (COPSAC2000) and in national registries.

METHODS: COPSAC2000 is a high-risk birth cohort of 411 Danish children. Asthma, allergy and eczema were diagnosed prospectively, and lung function measured at age 1mo and 7yrs. Sensitization was evaluated at age 6mo, 18mo, 4yrs and 6yrs by skin prick tests and IgE measurements. The register-based cohort included 1.7 million children from Danish national registries. Children born to mothers with preeclampsia were analyzed regarding risk of asthma, allergy and eczema in the 35-year-period 1977-2012.

RESULTS: COPSAC2000: 5.6% (23) was diagnosed with preeclampsia. Preeclampsia was associated with increased risk of treatment with inhaled corticosteroids at age 7yrs; adjusted Odds Ratio (aOR) 4.01 (1.11 to 14.43), p=0.0337, increased bronchial responsiveness to methacholine; adjusted β-Coefficient log-μmol -0.80 (-1.55 to -0.06), p=0.0348, and allergic rhinitis; aOR 4.83 (1.58 to 14.78), p=0.0057 in the 7-year-old children. Furthermore, the children had an increased risk of sensitization to both aero- and food-allergens, and increased amount of total-IgE during childhood. Registry-based cohort: 3.7% (62,728) were born to mothers with preeclampsia. Preeclampsia was associated with increased risk of asthma, eczema, and aeroallergen and food allergy, especially pronounced after a duration of preeclampsia of>14 days. Maternal asthma increased the risk of preeclampsia.

CONCLUSIONS: Preeclampsia is a shared prenatal risk factor for asthma, eczema and allergy in childhood pointing towards in-utero immune programming of the child.

Source: **Respirology**

Clinical characteristics of eosinophilic asthma exacerbations; Bjerregaard A, Laing I, Backer V, Fally M, Khoo S, Chidlow G, Sikazwe C, Smith D, Le Souëf P, Porsbjerg C; Respirology (Sep 2016)

BACKGROUND AND OBJECTIVE: Airway eosinophilia is associated with an increased risk of asthma exacerbations; however, the impact on the severity of exacerbations is largely unknown. We describe the sputum inflammatory phenotype during asthma exacerbation and correlate it with severity and treatment response.

METHODS: Patients presenting to hospital with an asthma exacerbation were recruited during a 12-month period and followed up after 4 weeks. Induced sputum was collected at both visits. Patients underwent spirometry, arterial blood gas analysis, fractional exhaled nitric oxide analysis, white blood cell counts and a screening for common respiratory viruses and bacteria. An eosinophilic exacerbation (EE) was defined as having sputum eosinophils ≥ 3% and a non-eosinophilic exacerbation as < 3% (NEE).

RESULTS: A total of 47 patients were enrolled; 37 (79%) had successful sputum induction at baseline, of whom 43% had sputum eosinophils ≥3% (EE). Patients with EE had a significantly lower forced expiratory volume in 1 s (FEV1) % predicted (70.8%, P = 0.03) than patients with NEE (83.6%). Furthermore, EE patients were more likely to require supplemental oxygen during admission (63% vs 14%, P = 0.002). The prevalence of respiratory viruses was the same in EE and NEE patients (44% vs 52%, P = 0.60), as was bacterial infection (6% vs 14%, P = 0.44). Fractional expiratory nitric oxide (FeNO) correlated with sputum %-eosinophils (ρ = 0.57, P < 0.001), and predicted airway eosinophilia with a sensitivity of 86% and a specificity of 70%.

CONCLUSION: Our findings suggest that eosinophilic asthma exacerbations may be clinically more severe than NEEs, supporting the identification of these higher risk patients for specific interventions.

Source: **Respir Med**

Socioeconomic inequalities in adherence to inhaled maintenance medications and clinical prognosis of COPD; Tøttenborg S, Lange P, Johnsen S, Nielsen H, Ingebrigtsen T, Thomsen R; Respiratory Medicine 119 160-167 (Oct 2016)

BACKGROUND: Low socioeconomic status has been associated with adverse outcomes in chronic obstructive pulmonary disease (COPD), but population-based data are sparse. We examined the impact of education, employment, income, ethnicity, and cohabitation on the risk of suboptimal adherence to inhaled medication, exacerbations, acute admissions, and mortality among COPD patients.

METHODS: Using nationwide healthcare registry data we identified 13,369 incident hospital clinic outpatients with COPD during 2008-2012. We estimated medication adherence as proportion of days covered (PDC) one year from first contact. With Poisson regression we computed adjusted relative risks (aRR) of poor adherence and non-use. With Cox regression we calculated adjusted hazard ratios (aHR) of clinical outcomes.

RESULTS: 32% were poor adherers (PDC<0.8) and 5% non-users (PDC = 0). Analyses showed a higher risk of poor adherence among unemployed (aRR1.36, 95% CI 1.20-1.54), low income patients (aRR = 1.07, 95% CI 1.00-1.16), immigrants (aRR = 1.29, 95% CI 1.17-1.44), and patients living alone (aRR = 1.17, 95% CI 1.11-1.24). Similarly, non-use was associated with unemployment (aRR = 2.75, 95% CI 2.09-3.62), low income (aRR = 1.37, 95% CI 1.10-1.70), immigrant status (aRR = 1.56, 95% CI 1.17-2.08), and living alone (aRR = 1.53, 95% CI 1.30-1.81). Low education was associated with exacerbations (aHR = 1.21, 95% CI 1.10-1.35) and admissions (aHR = 1.22, 95% CI 1.07-1.38). Low income was associated with admissions (aHR = 1.20, 95% CI 1.09-1.32), and death (aHR = 1.11, 95% CI 0.99-1.25). The unemployed and those living alone had lower exacerbation-risk but higher mortality-risk.
CONCLUSIONS: In Denmark, health equity is a stated priority in a public health care system. Nevertheless, there are substantial socioeconomic inequalities in COPD treatment and outcomes.
Source: **Chest**

Eosinophils in chronic obstructive pulmonary disease exacerbations are associated with increased readmissions; Couillard S, Larivée P, Courteau J, Vanasse A; Chest (Oct 2016)

RATIONALE: A subset of patients with chronic obstructive pulmonary disease (COPD) demonstrates eosinophilic inflammation either in their sputum or blood. Previous studies regarding the association between increased blood eosinophils and poor readmission outcomes are conflicting.
OBJECTIVE: Investigate outcomes following severe COPD exacerbations in patients with higher blood eosinophils.
METHODS: With an observational study design, hospitalizations for severe COPD exacerbation were retrospectively gathered. Patient health data previous to and up to one year following the index hospitalization were included. Patients were stratified into the eosinophilic group if the blood eosinophil level on admission was ≥200 cells/μL and/or ≥2% of the total white blood cell count. Clinical outcomes were 12-month COPD-related readmission, 12-month all-cause readmission, length of stay, and time to COPD-related readmission. These outcomes were analysed using logistic, negative binomial, and Cox regression models.
RESULTS: A total of 167 patients were included: 55 with eosinophilia. Eosinophilia was associated with an increased risk of 12-month COPD-related readmission (OR 3.59 [1.65-7.82], p=0.0013), an increased risk of 12-month all-cause readmission (2.32 [1.10-4.92], p=0.0277), and a shorter time to first COPD-related readmission (HR 2.74 [1.56-4.83], p=0.0005). The length of stay was not statistically different between eosinophilic and non-eosinophilic patients. Sensitivity analyses using different eosinophilia definitions reveal a proportional increase in effect size with increasing eosinophil cell count definitions for predicting 12-month readmissions.
CONCLUSION: Blood eosinophils can be used as a biomarker in severe COPD exacerbations for predicting higher readmission rates.
Source: **COPD**

Inhaled Long-acting Anticholinergics and Urinary Tract Infection in Individuals with COPD; Gershon A, Newman A, Fischer H, Austin P, Daneman N, Bell C, Stephenson A, Gill S, Vozoris N, Rochon P; COPD 1-8 (Oct 2016)

Inhaled, long-acting anticholinergic medication (LAA), commonly used for moderate-to-severe chronic obstructive pulmonary disease (COPD), has been shown to decrease COPD hospitalizations, emergency department visits, and acute exacerbations but has also been associated with urinary tract infection (UTI) in a prior meta-analysis. The objective of this study was to verify if there was an association between LAA and UTI in older individuals with COPD. A population-based, real-world cohort study using health administrative data from Ontario, Canada was conducted. Incidence of UTI was compared between older people with physician-diagnosed COPD, who were new users of inhaled long-acting anticholinergics and new users of inhaled corticosteroids-a reference medication used in similar clinical settings that has no known association with UTI. Propensity score matching was used to minimize the effects of confounding. An overall association between LAA and various measures of UTI in older individuals was not found. However, in a priori defined stratified analyses, men newly initiated on LAA were 75% more likely to develop a UTI than men newly started on an inhaled corticosteroid (hazard ratio 1.75; 95% confidence interval 1.05-2.92). No significant association was seen in women. In conclusion, older men with COPD newly started on LAA are at increased risk of UTI. Men considering an inhaled LAA should be informed of this risk and, if they decide to take it, be provided with appropriate monitoring.

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