February 2005

Family Physician Airways Group of Canada

In this issue:

Report from the Chair

Fall has been wonderful with a late Indian summer and the usual Canadian change of seasons. It has allowed Ragweed to survive much later into the year for your asthmatics, however, and this has made some of their lives miserable! Winter is here, so feel free to curl up beside a warm fire and read the newsletter!

I had the occasion to participate in the COPD Alliance meeting in Montreal from November 26-28, 2004. It was a wonderful combination of new research, epidemiology, review of the CTS COPD guidelines, management debates and behavioral issues. There was a good turnout of Family Physicians from the executive of the FPAGC, but not many others. We plan on rectifying this with a Family Physician Stream in the next meeting in Calgary in 2006. I hope you will consider joining us there.

The Adult Asthma guidelines have been released in the Canadian Respiratory Journal May/June 2004, Volume 11, Supplement A. We will spend some time distilling these into quick informative messages for the next newsletter.

There are many exciting initiatives in respiratory medicine. Infections have been a huge issue this last year with SARS and the concern of the 'bird flu'. I have reviewed the new study on the higher risk of pneumonia in those on PPIs later on in the issue. What other risk factors may we find for infection and respiratory problems?

Sleep apnea is a devastating problem that one must think of for your patients. We are pretty good at thinking about it when the wife comes in to tell us that her husband snores and stops breathing, but what about the patient with fatigue, hypertension, or unexpected vascular events. Keep those antennae working, if you do not look, you cannot find.

There are lots of exciting new articles in this issue; remember,
Stay warm,

ALAN KAPLAN, MD CCFP(EM)
CHAIR, FPAGC

All articles and letters to the editor for the May issue should be sent to the FPAGC office no later than April 10, 2005

GOAL: Gaining Optimal Asthma controL

Bateman et al. and The GOAL Investigators Group. *Am J Respir Crit Care Med* 2004

We have learned in Canada from the Asthma in Canada Landmark Study[1] that we do not have optimal control of asthma in our patients. We overestimate the degree of control with specialists assuming 90% of patients are under control and general practitioners estimating 77%. When the patients were actually checked only 43% had controlled asthma. Similar studies in the UK, USA, and Australia show that this is a worldwide problem.

The new Canadian consensus guidelines define control the same as the 1999 guidelines as in table 1. Due to recent studies like Optima, we see that additional therapy should be added in at lower doses of inhaled corticosteroids, perhaps even at 200 µg per day. (Figure 1)

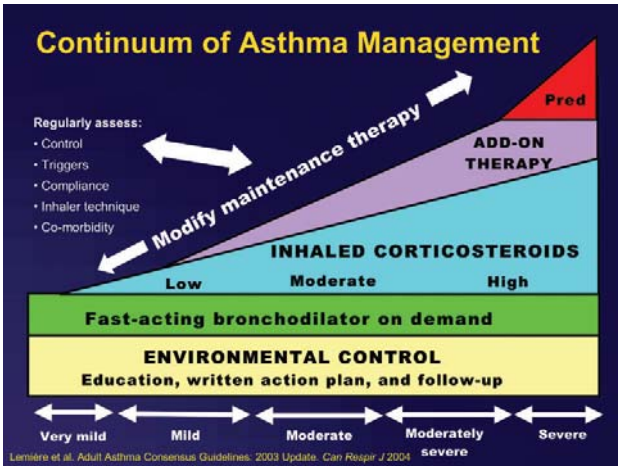

Figure 1: Canadian Guidelines 2003[3]

Canadian Asthma Control Criteria[2]

- Daytime symptoms < 4 days/week
- Night-time symptoms < 1 night/week
- Normal physical activity
- Mild, infrequent exacerbations
- No absenteeism due to asthma
- Fewer than four doses/week of SABA needed*
- FEV_1 or PEF >85% of personal best or greater (ideally 90%)
- Diurnal variability in PEF less than 15%

Table 1:

GOAL was the first prospective trial to assess whether guideline defined asthma control is clinically realistic. It was a truly global study, involving 326 centres, 3,416 patients, in 44 countries, including 16 sites in Canada. Single endpoints such as FEV_1, symptom scores, etc. are likely to overestimate the actual level of control achieved. Rather than relying on single parameters of asthma control, GOAL was conducted using a composite measure. This robust endpoint provided an indication of *overall* disease control by accurately reflecting treatment effectiveness and patient well-being[4,5,7]. So stringent was this composite measure, that, to be deemed Totally Controlled, patients needed to have had no symptoms whatsoever in at least seven of the eight weeks of each assessment period.

It also compared the ability of getting this control in patients on combination ICS/LABA therapy to ICS alone.

GOAL Objectives:

To determine:

- Proportion of patients achieving TOTAL CONTROL and WELL CONTROLLED asthma
- Dose at which control is achieved
- Time to achieve control

To determine the impact of aiming for TOTAL CONTROL on:

- QOL
- Exacerbation rates
- Traditional asthma outcomes, such as lung function
- Safety

Study design:

This was a 1 year, stratified, randomized, double-blind, parallel group study of adults and adolescents as young as 12 years with history of asthma for at least 6 months divided up into three strata based on ICS use in 6 months before randomization:

- Stratum 1: corticosteroid-naive/-free
- Stratum 2: ≤ 250 µg fluticasone or equivalent
- Stratum 3: > 250 to < 500 µg fluticasone or equivalent

These patients were not well controlled at baseline with uncontrolled asthma 2 out of 4 weeks, less than 10 pack years smoking and an FEV_1 reversibility of ≥ 15%.

(Phase 1) The patients were then titrated up until there was Total Control or the maximal dose of ADVAIR® was reached at 500/50 or Flovent at 500 µg. This was done in up to three steps depending on the starting dose of ICS. (Phase II) The patients were then maintained on the dose achieving Total Control or maximum steroid dose. In subjects not reaching total control at the end of the study, a 4 week open label phase was added where patients were given Prednisone (0.5 mg/kg up to 60 mg) in addition to the maximal dose of ADVAIR® (this group will be reviewed in a subsequent newsletter).

The GOAL definitions of Total Control and Well-Controlled were derived from the treatment goals of the GINA/NIH guidelines[4,5]. Equal weighting was given to each criterion, and failure to achieve any one of these resulted in failure to achieve control for that week. For a patient to be classified as Totally Controlled, this level of control had to be achieved for each day of the week and sustained for at least 7 out of the 8 consecutive weeks. (Table 2)

TOTAL CONTROL: The complete absence of all measured parameters of asthma for at least 7 out of 8 weeks. Total Control meant no daytime or night-time symptoms, no exacerbations or emergency visits, no PRN $ß_2$-agonist use, no adverse effects and ≥ 80% predicted AMPEF every day in at least seven of the eight weeks of each assessment period.

WELL-CONTROLLED: No more than 2 days per week with symptoms for at least 7 out of 8 weeks. No night-time symptoms, no exacerbations. By aiming for Total Control, more patients achieved Well-Controlled.

Table 2[6]:

GOAL Clinical Endpoints[4,5]

Based on GINA/NIH Guidelines

(National Institutes of Health)

Composite Measure Parameters *(Equally Weighted)*	Well-Controlled Asthma# *(2 or more of parameters 1-3 and ALL of parameters 4-7)*	Totally Controlled Asthma# *(ALL of parameters 1-7)*
1. Daytime Symptoms*	≤ 2 days / week with a symptom score > 1	None
2. PRN β_2-agonist Use	≤ 2 days / week; max. 4 occasions (8 puffs) / week	
3. Days at ≤ 80% predicted am PEF**	≥ 80% predicted every day	
4. Night-time Awakening	None	
5. Exacerbations†	None	
6. Emergency Visits	None	
7. Adverse Events	No treatment-related adverse effects enforcing a change in asthma therapy	

Table Footnotes:

\# Maintained for at least 7 of 8 weeks during an 8 week assessment period.
* Symptom score: 1 was defined as "symptoms for one short period during the day". Overall scale: 0 (none) – 5 (severe).
** Predicted PEF was calculated based on the European Community for Steel and Coal standards for patients aged 18 years and older and on the Polgar standards for patients aged 12-17 years.
† Exacerbations were defined as deterioration in asthma requiring treatment with an oral corticosteroid or an emergency department visit or hospitalization

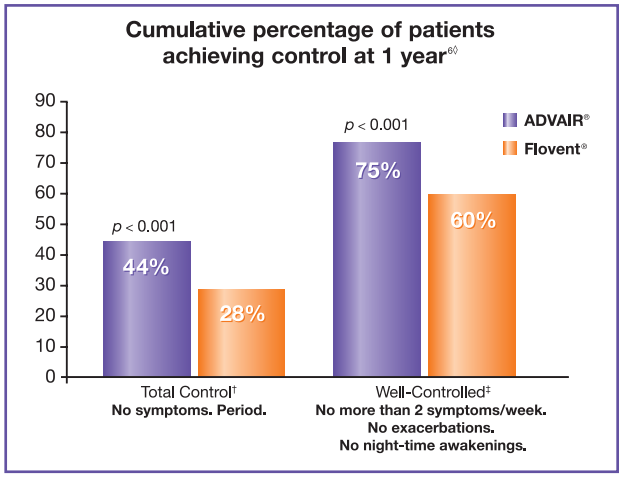

Figure 2[6]

Adapted from Bateman E *et al.* 2004.

◊ Stratum 2 subset of patients from a randomized, multi-centre, stratified, double-blind, parallel-group, 1 year step-up comparison of asthma control achieved with ADVAIR® vs. fluticasone. Patients uncontrolled on ICS (≤ 250 mcg fluticasone propionate or equivalent daily at baseline) n = 1,163. **Phase 1 Result: Total Control; ADVAIR® 32% vs. FP 20% (p < 0.001), Well-Controlled; ADVAIR® 69% vs. FP 52% (p < 0.001). Phase 2 (endpoint results) and p-values as per chart.**

† Total Control: The complete absence of signs and symptoms on all measured parameters of asthma (daytime symptoms, night-time awakenings, exacerbations [need for oral steroids, and/or hospitalization or emergency visits], PRN β_2-agonist use, ≥ 80% predicted am PEF, emergency visits and AEs leading to treatment change) for at least 7 out of 8 weeks.

‡ Well-Controlled: Two or more parameters of either daytime symptoms (score of 1 on ≤ 2 days/week), rescue medication use (max. 4 occasions/week) and ≥ 80% predicted am PEF every day plus no night-time awakenings, no exacerbations, no emergency visits and no treatment-related adverse events. Maintained for at least 7 out of 8 weeks during an 8 week assessment period.

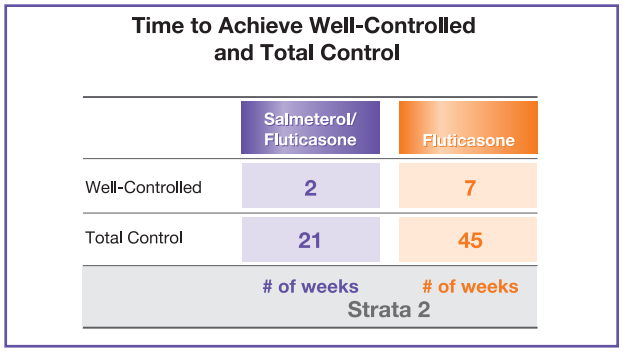

Figure 3

Results:

First of all, it showed that well controlled asthma was achievable in the majority of patients with asthma using regular medication (Figure 2). Total control was achieved overall less often, but more frequently in patients with ADVAIR® than Flovent alone. When doses of the inhaled steroid were compared, the dose to get control was lower with ADVAIR® than with Flovent alone. In addition, control was achieved earlier with the combined medication than with ICS alone (figure 3).

In addition, with sustained treatment, more patients can achieve control, and the majority of patients can maintain control. This occurred in both the total and well controlled groups.

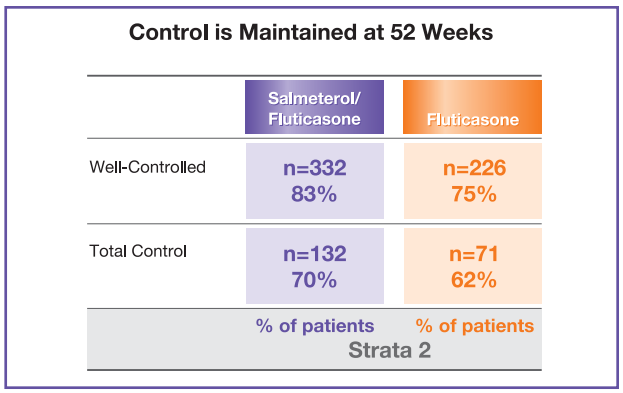

Figure 4

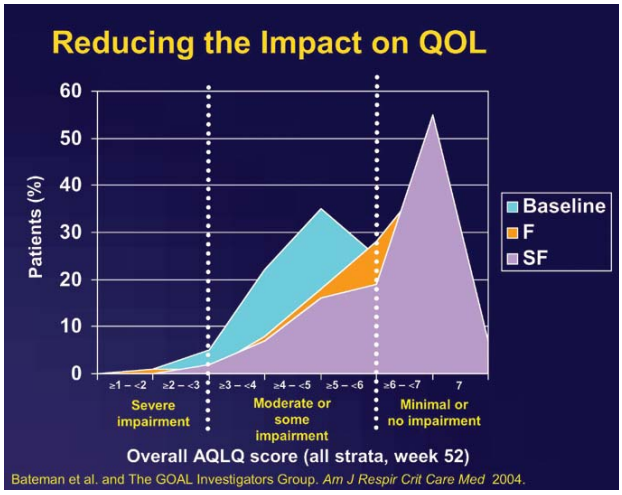

Figure 5

The study also reviewed exacerbations that were significantly reduced by aiming for total control, and were reduced more by the combination therapy than ICS alone. QOL[7] was also significantly improved by both treatments, but more in the combination therapy group (Figure 5).

GOAL has taught us that:

- Over a third of patients can achieve TOTAL CONTROL
- Up to 80% of patients achieve guideline-defined WELL-CONTROLLED asthma
- With sustained treatment (i.e. Over time with continued therapy)
  - More patients can achieve control
  - Majority of patients can maintain control

It has taught us that compared with ICS alone, control is achieved with combination treatment in **more** patients, **earlier**, and at a **lower** ICS dose. By aiming for TOTAL control there are the added benefits of having **exacerbations being virtually eliminated** and **near maximal QOL** can be achieved. There were **no safety implications** when aiming for TOTAL control identified in this one year study, implying that aiming for total control can be done safely.

What GOAL means for your patients.

Canadian and global studies show that the majority of asthma patients suffer from a high rate of symptoms and disruption of daily life due to their disease.

Aiming for Total Control with ADVAIR® may offer many uncontrolled asthma patients the possibility to achieve symptom-free asthma. In addition, the strategy of aiming for Total Control may provide the possibility of substantial benefit to patients even if they fail to achieve this rigorous level of control. The use of combination ICS/LABA therapy allows control earlier and at a lower dose of inhaled steroid.

Author's comment:

This study goes a long way in teaching us some valuable points.

- First of all, we learn that by aiming high, we get good results; something we have not been doing so far. This has pointed us in the direction of aggressive therapy, but not limited us in how we are to be aggressive.
- Second, we can do this faster and at a lower dose by using combination therapy in adults and adolescents over 12. This reiterates what we learned in the OPTIMA studies. This data has not been proved conclusively in children.
- Third, we learn that sustained therapy continues to increase the rate of improvement over time. This perhaps begins to answer what I feel is an important question; how long to keep your patient on ICS before tapering the dose down?
- Fourth, our patients want to be symptom free and free of exacerbations. We have to be aggressive in our management and comfortable in being aggressive to reach these currently unreached milestones.

References:

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Patient Goals in Asthma Care

Alan Kaplan, MD CCFP(EM)

What are your goals for your asthma patient? Decreased symptoms and utilization of SABAs? Better lung function? Natarsha Kruithof is a researcher in Aberdeen who looked at patient set Treatment Goals, a more sensitive measure of change than standard symptoms questions. Ninety five percent of patients were able to identify one goal and 91% could identify three goals. There were six consistent themes including exacerbation, lung function, medication, symptoms, activity, trigger and psychological. The four themes of activity, medication, symptoms and exacerbations represented 92% of all primary goals. Not surprisingly, improvement in lung function was only a primary goal in 6% of the patients. 59% identified a desire to "reduce activity limitation due to asthma". Thirty two percent of those who set goals identified a goal within this theme as their primary or only goal. Twenty four percent of patients chose as their primary goal the desire of "reducing/avoiding medication".

It may just be that patient goals are a more sensitive measure of change than our traditional questionnaires and lung function measurements. I will try this with my next asthmatic patients, and then see whether the treatment I have prescribed has helped them reach their goals!

System Issues: Doctors Need Help to Control Asthma, What Is Out There?

Alan Kaplan, MD CCFP(EM)

In many areas of Canada, we suffer from a shortage of physicians; some residents are unable to even find a family physician of their own. In other areas, there are sufficient primary care physicians, but even those doctors often feel overwhelmed by the patient numbers, escalating needs as they age and survive multiple ills, and that greatest curse, paperwork (I won't even go there!)

At the same time we have quite significant goals to reach in many areas. Our target blood pressures and sugars keep falling. Our control criteria for asthma are quite stringent. We strive for no exacerbations, no night symptoms, minimal to no symptoms in the day, no job or school loss due to the asthma and optimization of our patients' lung function. After seeing the Asthma in Canada Landmark study, which is actually quite similar to the results in Australia, UK, and the USA, we know that this is just not happening. Part of the reason is that physicians are too busy, and dealing with multiple chronic diseases and their guidelines, can be overwhelming. Where do we get help? Our consultants have such long waiting lists that they are often not of help. Besides, more and more care is being sent to the family physician by the super-specializing consultant. I would like to review some projects that were presented from other countries; hopefully there are lessons there for us.

Asthma Outreach Programs provided by rural community pharmacists, a feasibility perspective. In this Australian study, whose primary author was V. Kritikos, community rural pharmacists were educated in the Adolescent Asthma Action program and a community forum was provided to the community. The pharmacist knowledge base was increased according to questionnaires. Visits to the pharmacy for asthma information increased over four different time points. This feasibility study shows that even in rural areas partnership with community pharmacists can be helpful.

A similar study was carried out in Sydney called The Pharmacy Asthma Action Plan Project. A community pharmacy based pilot aimed at optimizing asthma management, with principal author B. Saini, showed that a pharmacist intervention of reviewing device technique, medication expectations and a written action plan revealed a 63% positive response from involved patients in that they have achieved or worked towards the goals that they had set for themselves with their asthma.

The community can also be involved as with *A collaborative approach to rural asthma management, Acute and community sectors working together,* principal author J. Moore. This was a multifaceted study which also included a project in Australia called EAM or Emergency Asthma Management. This is an educational program to tell lay people such as teachers, coaches, parents and employers about asthma and how to deal with exacerbations.

Another public relations project, albeit a local one in Australia (I. Charlton) is called Asthma Watch. This encouraged patients to go to their physicians to have their asthma properly assessed and to receive an Asthma Action Plan. Hospitalizations in the area were monitored and showed a 10% reduction. This is impressive as the area had a large increase in population in this same timeframe.

Asthma Education clinics are now reasonably commonplace in Canada, although underutilized. They are available at the local GP in the UK in the form of practice asthma nurses. In the US they work as part of respiratory departments. In Australia they are also part of the primary care physician practice in the 3+ program. This program is a government paid program to the family physicians to complete a three-visit asthma educational program. They do not have a counseling code, so this is new for them. In Ontario, we do have a K013, which can be billed once annually per diagnosis. Thus we do have some capacity to do this—at least in Ontario.

How can physicians help each other? Dr. Ryan from Leicestershire, UK, reviewed the UK system. Physicians in their primary care Airways group (GPIAG) have been identified as being primary care specialists and perform some consultations to bridge the gap between primary and secondary care. I think that we all realize that we have individual strengths in our training and interest. Can we not partner up with other primary care docs and help each other out; ultimately teaching each other and improving patient care? This does happen in some of our communities. Jacques Bouchard in Quebec City runs an asthma and allergy clinic. Tony D'Urzo runs a primary care Asthma Clinic in Toronto. I do respiratory consultations in my community. Rob Hauptman runs a couple of Asthma clinics in Alberta. We do have the people in Canada, let's look at this here! I can see a consult a lot sooner than the local consultants can!

Dr. Hilary Pinnock did a workshop on the use of telephone consultations in primary care. She showed data that phone consults were actually faster than office visits and had similar outcomes in selected issues. In the fee for service model, this clearly is not logistical. However, the new reforms to capitation may encourage this type of treatment/assessment and it may not be all bad! With the computer age here, email is becoming another medium to discuss health care directly to the patient.

All of this requires four things. First is interest by the physician who needs the help. Secondly, the government needs to recognize the need and thus find the resources to fund studies on the programs and subsequently their implementation. Third are the resources. Fourth is consistent education. This is actually the easy part. The 3+ program is established in Australia. Certified Asthma Educators all have to pass a standardized examination in Canada for their certification. Guidelines are reasonably clear and consistent. There are good educational programs also available in New Zealand the UK, and the Netherlands.

The last program I wanted to review is a fascinating program in Australia called the Home Medication Review Program. This is a government funded project in which pharmacists are trained to go into the patients' homes to interview them. They look at medications, interactions, metabolism, device techniques, side effects, barriers and identify areas of concern. This is compiled into a written report that goes back to the Family Physician who then reviews this with the patient, and GETS PAID AN EXTRA FEE (which is around $150) to do so. The patient can only get this service at the recommendation of the Family Physician (Specialists are NOT allowed!). There are some criterion, but they are very inclusive. I must admit to being impressed with the government of Australia and the physicians in creating such a great program.

ER Management of Asthma: Discharge Delivery of Corticosteroids

Alan Kaplan, MD CCFP(EM)

I have given many dissertations over time of the need for steroids in the discharge plan once your patients are to be discharged after an acute exacerbation. The standard teaching in pediatrics is 2 mg/kg day one and 1 mg/kg day 2-5 in addition to inhaled steroids and bronchodilators. I have written on the use of IM steroids in those patients whose compliance is questionable. The authors studied the use of inhaled steroids, an attractive choice with all of the concerns of the safety of systemic steroids.

Nakanishi and colleagues compared the use of oral vs inhaled steroids in children presenting with acute exacerbations of their asthma. Patients were all evaluated with standard measurements that included oximetry and spirometry. They were treated with albuterol +/- ipratropium until PFR was greater than 70% predicted and the patient felt able to be discharged. The child was then randomized into one of two groups.

Group one received oral prednisone at 2 mg/kg/day for seven days along with a placebo inhaler dosed at 4 puff BID (8/day) Group two received Flunisolide with a valved holding chamber in a dose of two puffs qid and a placebo pill. All patients were given Albuterol to use if PFR was less than 80% predicted and a Peak Flow meter and diary card. Spirometry was repeated at day three and seven.

There was no difference between symptom scores, morning PFR, repeat ER visits or side effects. The FEV_1 was higher at day three and seven in the oral steroid group. The authors concluded that Inhaled steroids could potentially be used on discharge for pediatric patients instead of oral.

Editor's Note:

This study goes against our current dogma of almost all acute exacerbations needing systemic steroids. We must interpret this with CAUTION. In a study situation, patients may well comply with a qid dosing; not often in real life. All patients were well instructed in the use of their MDI; technique is a real barrier in the real world. Oral steroids are cheap—puffers and spacers are expensive. Flunisolide is no longer available in Canada, but I suspect this is not drug specific. Also, the improved spirometry shows that there is a more rapid resolution of the asthma in those treated with oral steroids.

Nebulizers versus Inhalers with Spacers for Acute Asthma in Pediatrics

Osmond M, Diner B.
Ann Emerg Med 2004;43(March): 413-415

I have worked as a physician for a summer camp. About ten years ago, I remember being woken up at 5 am by the nurse for a young man with an acute attack of asthma. They were very worried as the nebulizer was broken and they did not know what to do. The young man was actually quite proficient with MDI technique and he was treated with 16 puffs of Salbutamol and an IV dose of Solucortef, and he did great. While we did get the nebulizer fixed, it showed me quite clearly that alternatives to nebulization did exist for Acute Asthma. This is what the author studied in this paper.

This author did a Cochrane comprehensive review of the literature to evaluate the use of MDIs with chambers versus nebulizer treatments. Chosen were studies in adults and children over two years old with acute asthma treated with chambers vs. nebulization.

The relative risk of admission was 0.65 for the MDI and chamber vs.nebulization. ED length of stay was significantly shorter for the inhaler with chamber group also. These suggest that MDIs with chamber performed at least as well as nebulizer for children presenting with acute Asthma to the ER.

Comments:

MDIs with chamber are cheaper and faster than nebulization. They also have the advantage of being more portable and can be given in a less emergent area or in the prehospital setting. This strategy could be used on mild to moderate cases where no hypoxia is present. If hypoxic, oxygen therapy is needed to keep oxygen saturation over 94%.

SARS also taught us the dangers of nebulization. SARS molecules were dispersed throughout the room when patients were treated with nebulizers. This created new protocols for the treatment of acute brochospasm in febrile patients in most ERs. Many ERs as well as a new protocol for ER Asthma therapy being developed across Ontario, suggest treatment with MDI and chamber preferentially. Certainly this could be provided in your office with an initial dose of 8 puffs of Salbutamol being quite reasonable. Remember to wait at least 30 seconds between doses.

ALAN KAPLAN, MD CCFP(EM)

Steroids and Growth in Children

Alan Kaplan, MD CCFP(EM)

Once again Dr. Soren Peterson of Denmark has created a landmark study on the issue of steroids and growth in children. The study is called START (inhaled Steroids Treatment as Regular Therapy in early asthma) and is a five year, 24 nation study which followed nearly 3000 boys and girls for five years. The study had two phases. The first was a three year double blind randomized phase in which the children were randomized to once daily placebo or budesonide. This was followed by a two year open label phase in which all children received budesonide. In both of these phases the children continued to take their usual asthma medication as well. The dose of budesonide was 200 mcg/day if under 11 years and 400 mcg if older.

At three years the study clearly showed that the increase in height as an average of 1.29 cm LESS in the treated arm than in the placebo arm. Most of this difference occurred in the FIRST year. By the end of the five years, however, there was NO statistical difference in height between the two groups.

Editor's Note:
This study reaffirms and confirms what we know. There is an initial decrease in growth velocity in children taking inhaled steroids in the first year. This does NOT seem to affect long term growth results. I believe inhaled steroids are safe for children's growth, BUT, it behooves us to carefully watch these children for those who seem to be extra susceptible to growth retardation and look for alternatives if needed (LTRAs?).

Breast-feeding Reduces the Risk of Asthma During the First 4 Years of Life

Kull I, et al, J Allergy Clin Immunol 2004; 114:755.

Background:
The evidence for a preventative effect of breast-feeding on asthma and other allergic diseases in childhood is inconclusive.

Objective:
The aim of this study was to investigate the effect of breast-feeding on asthma and sensitization to airborne allergens among children up to four years of age.

Methods
A birth cohort of 4,089 children was followed. Exposure data was collected at two months and one year of age. The total dose of breast milk was estimated by combining periods of exclusive and partial breast-feeding. Outcomes data were collected at 1, 2, and 4 years of age. The response rate at four years was 90%, and 73% participated in a clinical investigation, including blood sampling for analysis of specific IgE and lung function testing. Children with onset of wheeze during lactation (n=217) were excluded in some of the analyzes to avoid disease related modification of exposure.

Results:
Exclusive breast-feeding for four months or more reduced the risk of asthma at the age of four years (odds ratio [OR], 0.72; 95% CI, 0.53-0.97), irrespective of sensitization to common airborne allergens (p=.72). Excluding children with wheeze during lactation tended to strengthen the risk estimate (OR, 0.64; 95% CI, 0.46-0.88). A duration of 3 months or more of partial breast-feeding seemed to offer additional protection; exclusive breast-feeding for three to four months combined with partial breast-feeding for three months or more resulted in an OR of 0.44 (95% CI, 0.21-0.87). The effects tended to be stronger in children without heredity for allergy.

Conclusion:
This is a nice study to reaffirm what makes really good sense to us already; but the data had not been conclusive up to now. Breast-feeding reduces the risk of asthma during the first four years of life. Throw out those bottles….

ALAN KAPLAN, MD CCFP(EM)

Allergy Shots – How Dangerous Are They?

Alan Kaplan, MD CCFP(EM)

Dr. Isobel Martin of New Zealand presented on allergy and reviewed a Cochrane analysis which showed that there was benefit, but the cost of immunotherapy may be greater than the benefit.

MS Ostergaard analyzed one years' reported cases of serious side effects of anaphylaxis following systemic immunotherapy to grass pollen in Denmark. Thirty nine cases were reported and included 10 with anaphylactic shock. Twenty nine of the 39 required admission and most of these occurred in a primary care setting.

This study screams at me! Allergy shots are a routine part of our practice. Are you prepared? Do you have Adrenaline, Benadryl, injectable steroids, and Oxygen? If you don't, DO NOT give allergy shots! Also, make sure the medications have not expired!

Sorry to preach, but I have seen this scenario occur; early aggressive treatment of the patient makes a large difference.

Intermittent Asthma – How Do We Treat This?

Alan Kaplan, MD CCFP(EM)

One of the great questions we currently have that is really not answered is what to do with the mild asthmatic, who truly has only intermittent symptoms. Usually this person is perfectly well outside viral infections and has normal lung function when not infected. We are not sure how long to treat, which drug and how much of it, and for how long.

Optima A study did give us some insight into these patients. As you recall, this study looked at mild asthmatics who were steroid naïve and felt to be mild by their doctors. They were randomized into control and treatment and low dose ICS Budesonide 200 µg/day was effective at decreasing exacerbations. Addition of LABA did not do much for this group. What I found interesting is the fact that there was a rate of 0.76 exacerbations per year in the placebo group! Thus, what is mild? Again the definition is difficult. The current answer is to treat with ICS at a dose to optimize lung function and prevent exacerbations. Once someone has three exacerbations in a year, they should be considered for prophylactic treatment (my opinion). Maybe that should be at two, this is something that needs exploring and will likely be one of the themes of research for the IPCRG and the FPAGC, who better to study this group than the Family Physician?

D. Price et al reviewed an abstract at the IPCRG meeting called *Montelukast for intermittent asthma in children reduces health resource use and parent reported work and school loss, result of the PRE-EMPT study.* The theory was that since we know the onset of effect of Montelukast is within 24 hours of treatment, a short course may effect these outcomes. Children 2-14 with intermittent asthma were randomized in a double blind placebo controlled study of 4-5 mg of Montelukast, depending on age, which was started at the first signs of a viral URTI and continued for a minimum of seven days or until symptoms had resolved for 48 hours.

There were a total of 680 episodes treated (345 Montelukast and 335 placebo). Emergency attendance was decreased by 45% and health care utilization decreased by 27%. The duration of the exacerbation was not affected. Night awakenings were decreased by 9%. Time off school and parents missing work decreased by >33%. Overall symptom scores within the episodes were also decreased ($p< 0.05$).

This study gives some initial evidence that Montelukast can be used for the short term therapy of viral infections in intermittent asthmatic children.This is in addition to the recent paper showing the benefit of Montelukast in bronchiolitis; improved post-RSV clinical symptoms post RSV in children age 3-36 months.by Bisgaard (2003) Am *J Respir Crit Care Med 167: 379-83* and perhaps gives us a slightly different picture of this medicine that seems to be mostly used now as an add-on therapy for mild asthma with allergy and especially rhinitis.

COPD Exacerbations

Classifying COPD Exacerbations, A patient perspective

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This is a review of an IPCRG study that looks at how patients view their exacerbations of COPD and I feel it is valuable for the primary care doctor. We tend to look at AECOPD as those times when patients get infected and need antibiotics and steroids. We also know that exacerbations are a cause of deterioration of the COPD in the long term. Patients don't view them that way; this is of course not a surprise as our view and the patient's views/goals are often quite different (I will review this in a study on asthma that is similar). Patients were interviewed to explore the meaning of exacerbations for them.

This yielded four categories. The frightening changes were represented by acute severe dyspnea or hemoptysis. Changes in sputum colour were next. Gradual deterioration wherein the condition gradually changed over weeks was more difficult for the patient to delineate from their normal functions. Lastly were the diagnoses made 'opportunistically' at a doctor appointment for another issue where the physician noticed worsened dyspnea.

This data should allow us to understand our patients better; why do they choose to consult us early vs. late in the course of their illnesses?

ALAN KAPLAN, MD CCFP(EM)

2004 Canadian Thoracic Society COPD Guidelines Dissemination and Implementation Committee Meeting

Friday, November 26, 2004 Report for FPAGC

Gordon Dyck MD

It was a pleasure to sit in for Alan Kaplan in the widely represented group, from specialists to nurses, to respiratory therapists, family doctors, and industry representatives.

The target for 2005 is to expand awareness of the COPD guidelines beyond physicians to respiratory care professionals. Assessment of strategy implementation was defined as a parallel goal.

All publications addressing the COPD guidelines were requested to be forwarded to the project coordinator, Laura Monette (lauramonette@sympatico.ca), to ensure the reflection of the desired message. A Newsletter is currently sent out by the chair of the D&I Committee and contact information for designated recipients is being sought to expand the readership.

The Guidelines have been distributed to over 18,000 respirologist and physician groups, but the impact on behavior has not yet been assessed. A direct mail package is being considered for 2005.

The current marketing tools include a logo, tagline, and portable conference banner (completed), a slide kit, and pocket card (near completion), and an exam room poster, website and mouse pad (under development).

The fifteen key messages were defined by the three themes of treatable, preventable, and under diagnosed. The tag line of treatable, preventable was kept positive for marketing.

A Needs Assessment was reviewed in defining a global message tailored for each target audience group, including a public awareness campaign. Organizational support at all levels was acknowledged as imperative for the success of any implementation plan. CHE: a slide kit will soon be available at www.copdguidelines.ca.

Breathworks was reported on with video clips of media presentations on the rise of COPD in women, noting that it was the highest amount of awareness coverage generated 'ever'.

A lot of brainstorming took place around how to raise the diagnosis of COPD on the index of suspicion, and what immediate response (spirometry) should take place. Evaluating success of implementation is an ongoing challenge, and discussions took place regarding research to achieve such an evaluation.

There is a level of excitement in a group with a unified purpose. The FPAGC is aware of and is committed to the goals of the D&I Committee in communicating the needs of patients with COPD to health care professionals. It is easy to get caught up in the task of getting a message out. We need to think of those patients who have not been identified of having COPD early enough and suffered unnecessarily. The patients who need not suffer the same fate are the ones for whom we toil. I look forward to the participation of the FPAGC in the process of COPD guideline dissemination.

The Public's Response to Severe Acute Respiratory Syndrome in Toronto and the United States

Blendon EJ, Benson JM, et al. Clin Infect dis 2004;38(April):925-931

Personally, I had a unique perspective in dealing with SARS. I work at York Central Hospital in Richmond Hill, Ontario where the second SARS outbreak occurred. It has received much less attention than the first (Scarborough) and third (North York General) mostly due to the efficient infection control measures rapidly put into place by the head nurse of our Emergency Department and subsequently the entire hospital. In addition, I was named the Family Practice representative to the SARS Clinical Working group for the creation of treatment guidelines. I had the opportunity, as an ER physician at York Central to diagnose and treat SARS patients. I had to spend some time in quarantine and learned that people were quite fearful of what could happen to their families when friends of my children were not allowed to have them to their homes nor come to mine. Lastly, I have met with physicians who had SARS, including one whose partner succumbed to the illness.

All of these led me to be quite interested in the above article. This article's objective was to review the public reaction to SARS in Toronto, the rest of Canada, and the United States. Surveys were sent to 501 adults in Toronto (one survey) and 4-9,000 adults in the US (8 surveys)The surveys looked at concerns about contracting SARS, precautions against SARS, attitudes about quarantine, and general information about the disease.

The results were fascinating. Sixty-nine percent of Toronto respondents were concerned about contracting the disease. The range was from 26-32% in the US, despite the far smaller number of cases and no deaths occurring in the US. Precautions taken in Toronto included (47%) disinfectant use, (27%) website research, (19%) discussion with doctor, (19%) avoidance of Asian restaurants, (16%) avoidance of public places, (14%) purchase of facemasks, (96%) avoidance of international travel. Most respondents said that they would agree to being quarantined if exposed to SARS or isolated if they contracted SARS (84-97%)

Ninety percent of respondents knew that SARS was infectious, but only 50% knew that there was no treatment or vaccine.

Personal Comment:

Fear of the unknown is a key determinant in panic and our jobs are to educate the communities in which we live. The Canadian College of Family Physicians actually did attempt to dispense education to family physicians, but when we do not really know the answers, this will fail. Physicians are the ones on the front lines of illnesses and we must protect ourselves. We also must educate the public and help control the panic that occurs when people do not understand the dangers of a new situation.

This hit very close to home when my children's friend's families refused to allow contact between our children. As someone working with the illness, I recognized early that this was a highly infectious agent in high risk people who dealt with extremely sick patients with high viral loads, but was much less of a community risk. It allowed me to use sensible measures for my office and allowed me to function with protective measures to protect my patients from me, as well as myself from them. I only hope that this experience with a less contagious infectious respiratory disease helps us prepare for some future more malicious bug.

Alan Kaplan, MD CCFP(EM)

Bronchitis, Acute

John Rea, MD

Diagnosis

Acute bronchitis is an acute inflammation of the lower respiratory tract. It is the most common respiratory infection presenting in the primary care setting and takes one of two forms. The first affects a previously healthy patient and is usually viral in origin. The second is an acute exacerbation of COPD and may be viral or bacterial in origin.[1] This section will deal with the first form only.

Bronchitis is usually self limiting and lasts from a few days to a few weeks.

Signs and symptoms include; a prodrome of URTI symptoms consisting of mild coryza (sore throat, cough, fever, runny eyes and nose), followed by cough (either productive or non productive) and often signs of airway obstruction including nocturnal cough and wheezing.

The cough occurs in 85% of patients within two days of the illness. The cough is usually gone in two weeks but lasts longer in 26% and may continue for 6-8 weeks.[2] Sputum colour and thickness, a useful sign in AECOPD, is irrelevant in management of acute bronchitis.

Etiology

The vast majority of cases are caused by viruses. The common viruses are; in patients under one year of age- respiratory syncytial virus, parainfluenza virus, and coronavirus, in patients 1-10 years of age—parainfluenza virus, enterovirus, RSV, and rhinovirus, in patients greater than 10 years of age– influenza virus, RSV, and adenovirus.[3]

Rarer causes include bacteria, yeast/ fungi, and environmental triggers.

Management

Antibiotics are used in 65 to 85% of patients with acute bronchitis.[4,5] There have been several studies looking at this practice with mixed results. A metaanalysis of eight randomized trials found that duration of cough and sputum production was decreased by half a day. This although statistically significant was not clinically significant.[1] In all studies patients consistently show improvement when not treated with antibiotics. Most guidelines do not suggest treatment with antibiotics unless there is strong suspicion of a bacterial superinfection or pneumonia. When this is the case treatment is as per pneumonia guidelines.[1]

Of note no studies have been completed with newer macrolides or quinolones. Such studies are ongoing.[1]

Symptomatic treatment may help patients feel better. Studies support the use of antitussives and short acting bronchodilators but not antihistamines.[1]

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Pneumonia

John Rea, MD

Diagnosis

Pneumonia is an acute infection of the lung parenchyma caused by a variety of pathogens including bacteria, atypical organisms, and viruses.[1] The common causes of community acquired pneumonia (CAP) are bacteria (S. pnuemoniae 23-50%, H. Influenza 3-10%, S. aureus 3.5%, M. catarrhalis 1-3%), atypical organisms (M. pnuemoniae 2-37%, C. pneumoniae 5-17%, Legionella pneumoniae), and viruses (Influenza A and B, Parainfluenza 1,2, and 3, Respiratory syncytial virus, and Epstein-Bar virus).[1] Treatment is based on this pathogen profile as there are no accurate means to differentiate between these organisms clinically.[2,3]

Diagnosis is based on clinical suspicion in the setting of two or more cardinal symptoms (Temp > 37.8, Pulse > 100, Decreased breath sounds, Rales, Respiratory rate > 20) and should be confirmed by chest x ray (demonstrating consolidation).[4]

Treatment may be started with negative CXR findings if the clinical suspicion is high. A negative CXR, however, usually suggests an alternate diagnosis.[1] Treatment may be warranted in patients with few clinical signs and a negative CXR if they have COPD, asthma, are smokers, are immunosuppressed or are elderly. Other investigations including white blood cell count, arterial blood gases, sputum gram stain, and sputum/blood cultures are not useful in making the diagnosis or

choosing therapy.[3] As a result the treatment of CAP is essentially empirical.[1,5] Sputum cultures are obtainable in 66 % of patients however 25% are infected with organisms not easily cultured and false positive and negative rates are high.[5] Sputum cultures may be useful to diagnose rare infections such as Histoplasmosis, Pneumocystis carinii, and M. tuberculosis. Gram stains have been shown to be useful in patients admitted to hospital.[6]

Prognosis

Management of the patient with pneumonia including drug choice and inpatient versus outpatient management can be aided by use of a clinical prediction rule.[1,7] Such a scoring system determines a Risk Class Level based on age, co morbidities, physical and lab findings. One such score, the Pneumonia Severity Index (PSI), has been proposed by Fine et al. using 19 independent risk factors.[7]

Demographic Factors		
	Age: Males	age in years
	Females	age in years
	Nursing Home Residents	+ 10
Co morbid Illnesses		
	Neoplastic disease	+ 30
	Liver disease	+ 20
	Heart Failure	+ 10
	Cerebrovascular disease	+ 10
	Renal disease	+ 10
Physical Examination		
	Altered mental status	+ 20
	Resp rate > 30/min	+ 20
	Sys BP < 90	+ 20
	Temp < 35 or > 40	+ 15
	Pulse > 125/ min	+ 10
Lab findings		
	pH < 7.35	+ 30
	BUN > 11 mmol/L	+ 20
	Sodium < 130 mEq/ L	+ 20
	Glucose > 14 mmol/ L	+ 10
	Hgb < 90	+ 10
	pO2 < 60 (O2 sat < 90%)	+ 10
	Pleural effusion	+ 10

Patients with 71-90 points generally can be treated as out patients. Exceptions include patients with impaired cognitive function, those unable to maintain hydration, those unable to perform ADLs, and those that are hypoxic. Thoracentesis should be considered in those with a pleural effusion. Patients with PSI scores greater than 91 are at significant increased risk and hospitalization should be considered.[1,7]

Management

Antibiotic choice depends on patient characteristics and the decision to treat as an in patient or out patient.[1,4]

Macrolides, specifically the newer generation ones (azithromycin, clarithromycin, and telithromycin), are first line for most young healthy patients.[1,4] Erythromycin use is limited by tolerability issues. This antibiotic class covers S. Pnuemoniae, H. Influenza, and the atypical organisms. Macrolide (except telithromycin) resistance is lower but parallel to penicillin resistance for S. Pnuemoniae and H. Influenza. Even for most species of penicillin resistant S. Pnuemoniae the MICs are low enough that penicillin should be effective.[1,8] Tetracyclines and/or trimethoprim-sulfamethoxazole are also acceptable alternatives.[1]

Broader spectrum antibiotics may be indicated for patients in older age groups (> 65) or those with co morbid illnesses. These patients are at risk for infections due to oral anaerobes, gram negative rods, S. aureus and Legionella.[1,4,8]

Respiratory quinolones (levofloxacin, moxifloxacin, gatifloxacin) are first choice for patients who have recently been on an antibiotics, on steroids or who have COPD of moderate severity.[1] Alternative choices for such patients include a combination of amoxicillin/clavulanate with a macrolide or a second generation cephalosporin (cefaclor, cefuroxime axetil, cefprozil) with a macrolide.[1,4,8]

Ciprofloxacin is used in patients with severe COPD or others at risk for Psudomonas infection.[1]

In the case of suspected macro aspiration (alcoholics) the use of amoxicillin/clavulanate is preferred.[1]

For patients intolerant of macrolides or other first line drugs, respiratory quinolones are recommended.[1]

Patient Education

In addition to antibiotics, patient education is important.

Patients should finish the entire course of antibiotics. Symptomatic relief can be achieved with oral hydration, acetaminophen or NSAIDS as well as other over the counter preparations.[1]

Criteria for follow up include difficulty breathing, worsening cough, worsening or onset of rigors, persistent fever (> 48 hours) or side effects to medication.[1]

Improvement generally occurs in about 48 hours. Return to work is generally reasonable 48 hours after resolution of fever and improvement in cough.[1]

Repeat CXR should be obtained at 6 to 8 weeks post treatment in smokers and patients older than 40.[1]

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Risk of Community-Acquired Pneumonia and Use of Gastric Acid–Suppressive Drugs

Alan Kaplan, MD CCFP(EM)

Context

Reduction of gastric acid secretion by acid-suppressive therapy allows pathogen colonization from the upper gastrointestinal tract. The bacteria and viruses in the contaminated stomach have been identified as species from the oral cavity.

Objective

To examine the association between the use of acid-suppressive drugs and occurrence of community-acquired pneumonia.

Design, Setting, and Participants

Incident acid-suppressive drug users with at least one year of valid database history were identified from the Integrated Primary Care Information database between January 1, 1995, and December 31, 2002. Incidence rates for pneumonia were calculated for unexposed and exposed individuals. To reduce confounding by indication, a case-control analysis was conducted nested in a cohort of incident users of acid-suppressive drugs. Cases were all individuals with incident pneumonia during or after stopping use of acid-suppressive drugs. Up to 10 controls were matched to each case for practice, year of birth, sex, and index date. Conditional logistic regression was used to compare the risk of community-acquired pneumonia between use of proton pump inhibitors (PPIs) and H_2-receptor antagonists.

Main Outcome Measure

Community-acquired pneumonia defined as certain (proven by radiography or sputum culture) or probable (clinical symptoms consistent with pneumonia).

Results

The study population comprised 364,683 individuals who developed 5,551 first occurrences of pneumonia during follow-up. The incidence rates of pneumonia in non–acid-suppressive drug users and acid-suppressive drug users were 0.6 and 2.45 per 100 person-years, respectively. The adjusted relative risk for pneumonia among persons currently using PPIs compared with those who stopped using PPIs was 1.89 (95% confidence interval, 1.36-2.62). Current users of H_2-receptor antagonists had a 1.63-fold increased risk of pneumonia (95% confidence interval, 1.07-2.48) compared with those who stopped use. For current PPI users, a significant positive dose-response relationship was observed. For H_2-receptor antagonist users, the variation in dose was restricted.

Conclusion

Current use of gastric acid–suppressive therapy was associated with an increased risk of community-acquired pneumonia.

Editors Note:

This is an interesting study that seems to indicate that current treatment with PPI increases the risk of acquiring pneumonia. People who had been treated remotely did not have the increased risk. This is interesting and perhaps can be added to our diagnostic algorithm for pneumonia. It is NOT enough data for me to stop using PPIs however; they are still remarkably effective for the management of GERD and Dyspepsia.

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