



FPAGC

FAMILY PHYSICIAN AIRWAYS GROUP OF CANADA
l'Association canadienne des médecoms de famille contre l'asthme

Chairman's Report December 2002

Happy Holiday season to all. The FPAGC continues its mandates of physician education and representation regarding the respiratory issues of primary care physicians in Canada.

There are new guidelines coming; do we want them? Family Physicians have many sets of guidelines for multiple conditions; can we really expect to utilize them all? This has been a common criticism of all guidelines created until now. In addition, the dissemination of guidelines and their implementation have been criticized. Therefore these lessons must be learned! A prime focus of the new guidelines will be their dissemination and ease of application to the busy family physician.

COPD is common and a killer. It robs patients of their quality of life. It is a subject of interest in family physicians recurrently in surveys of CME interest. The Canadian COPD Guidelines group is being led by Dr. Paul Hernandez of Halifax. The FPAGC has now been invited to assist in their development and implementation.

CNAC (Canadian Network of Asthma Care) has agreed to be the umbrella organization supporting Pediatric consensus guidelines for Asthma. This will be chaired by Dr. Allan Becker, a pediatric immunologist in Winnipeg, Manitoba. Particular topics will be reviewed including, but not limited to, Primary Prevention of Asthma and Allergy, Asthma Education, Steroids and Effects on Growth and Bone, Steroids and Effects on Long Term Outcomes, Diagnosis in the Preschool Child, Secondary Prevention of Asthma, Intermittent Therapy with Steroids, Is It Safe and Effective?, LTRAs as Monotherapy, Add on Therapy vs. Higher Doses of ICS, Delivery Devices, Immunotherapy, and Adolescent Care.

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Chairmans Report *(Continued From Cover Page)*

The FPAGC continues to run Mainpro C courses on asthma and spirometry. We have also had input into a new Mainpro C program on COPD produced by one of our pharmaceutical company partners. In Ontario, the Ontario Lung Association has created a Mainpro C program on Asthma management based on the guidelines of 1999.

Dr. Stephen Coyle, of Winnipeg, has agreed to rejoin our Executive in the capacity of Research Co-ordinator. If any of you are active researchers, we would love to hear from you as this is one of our short term goals. We need help to get this started and your expertise would be appreciated.

CNAC, the Canadian Network for Asthma Care, meets twice yearly. There are a couple of CNAC initiatives which may affect future practice paradigms and review. The College of Physicians and Surgeons of Ontario is considering a pilot

program with CNAC to explore using the guidelines as part of the peer assessment process. This would also help to evaluate the implementation and dissemination of the guidelines efficacy in Family Practice. CNAC will also work with the CCHSA (Canadian Council on Health Services Accreditation) to incorporate asthma education resources (such as use of CAEs) as part of a benchmark or standard of care in the hospital accreditation process.

Please take a moment to check out the website www.fpagc.com and check out future events and meetings, review old newsletters, and please feel free to email us with questions, criticisms, or suggestions. We are here as Family Physicians FOR Family Physicians: we really do want to hear from you and I promise that your emails will be personally answered!

Alan Kaplan MD CCFP(EM)

Chair, FPAGC

What are the numbers?

I thought that I would relate to you some of the newest surveillance data regarding asthma as compiled by Health Canada and reviewed at the November 22, 2002 CNAC meeting.

In 1999 15.2% of patients 4-11 years old have asthma based on the question "Have you every been diagnosed with asthma?"

Only 8.8% of kids 4-11 said yes to "Do you currently have asthma?"

From 1994 to 1999

Asthma rates have risen from 13.7 –18.1 in 8-11 year olds

Asthma rates have risen from 7.0-9% (40%) in women

Asthma rates have risen from 5.5- 6.9 (20%) in men

Ratio of asthma related to sex is 1.4 men to women in kids, and 1.4 women to men in adults

Hospitalization data decreasing.

Deaths decreasing 1999 411 deaths (40 deaths under the age of 45)

Editorial:

Therefore, we are seeing more asthmatics, perhaps due to disease, perhaps due to diagnostic improvements. The rates of hospitalizations and deaths are falling, therefore there is reason for optimism, but 45 deaths under 45 years old is a large issue that needs remediation.

Should combination therapy with inhaled corticosteroids and long acting B2 agonist be prescribed as initial maintenance therapy for asthma?

CMAJ 29 OCT 2002; 167(9)

There are now two products which combine ICS with LABA. They include Advair (Fluticasone with Salmeterol) and Symbicort (Budesonide with Formoterol). These products have been clearly shown to be effective in patients whose asthma is not optimally controlled with a moderate dose of ICS. OPTIMA and FACET studies have shown some superiority of adding on a LAB2 to doubling ICS dose in multiple measured parameters. In addition, the combination of products in a single molecule may well improve their activity at a cellular level and studies have shown an improvement in endpoints with each combination product compared to the two products taken separately together.

There is a growing trend to use the combination products as initial maintenance therapy for asthma. It seems attractive to use them to improve compliance and convenience. The authors (a number of prominent respirologists who were prominently involved in the Guideline process) discussed this and felt that further evidence should be considered with large enough studies to look at exacerbations as an end point. They felt that initial therapy should be inhaled corticosteroids and followed from there.

AK's note: Asthma is a dynamic condition which is variable in presentation and level of disease. One size does not fit all, the treatment must be tailored to the individual. I feel that some patients with severe symptoms, especially nocturnal symptoms, may benefit from use of LABAs early, and individuals may benefit from combining the medications to improve compliance and maybe even its rapidity of activity.

However, we must not lose sight of the

fact that the treatment of asthma must be based on the treatment of the underlying inflammation, and LABAs do not do that.

ASED 6

November 27-28, 2003 is the date of ASED 6 in Montreal. The theme is asthma education, assessment, application, and evaluation; the cycle of success. There will be didactic lectures, workshops and debates. Many topics will be covered including occupational asthma, learning styles, spirometry, allergy evaluation, asthma masqueraders, update on pharmacotherapy, action plans, ER education, comorbidities, alternative medicine, therapy for MDs based on guidelines, and education of different age groups. Tools to evaluate programs will also be reviewed. Critical review of literature will also be done.

This is going to be a great meeting for those of you looking for an update in asthma management. It would also be great for any of you docs who are involved in asthma education in your community. If you are involved in your local asthma education clinic, this program is a MUST, as it will offer you the ability to interact with your peers.

Just for interest, ASED 7 will be held November 17, 2005 in Calgary, Alberta.

[Steroids in COPD, what is their role?](#)

Steroids have their place in the management of COPD, but not for everyone. There are a lot of misconceptions regarding the utility of steroids, especially inhaled steroids in the maintenance phase of patients with COPD. I will break this up into two articles. The management of acute exacerbations of COPD is very different, with different goals, than the treatment of stable COPD.

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asthma combination (Continued From Page 3)

I. Steroids in Acute Exacerbations of COPD.

a) Systemic steroids in Acute Exacerbations of COPD.

Niewoehner DE, Erbland ML, Deupree RH, Collins D, Gross NJ, Light RW, et al. Effects of systemic glucocorticoids on exacerbations of chronic obstructive pulmonary disease. Department of Veterans Affairs Cooperative Study Group. N Engl J Med 1999; 340(25):1941-47

This study randomized patients admitted to hospital with AECB and randomized them to receiving steroids for either two or eight weeks or placebo. Treatment failure, which was defined as death, intubation and mechanical ventilation, readmission for COPD, or change in therapy, was significantly lower in the steroid treatment group. The eight week course was no better than the two week course.

Other studies have shown that oral steroids will do the same thing. Steroids were given once daily at a dose of 30 mg per day for two weeks. These patients again showed shortened hospital stays and increased FEV₁. Shorter three day courses were not as effective as 10 day courses for COPD².

Systemic steroids are problematic for the patients and this must be kept in mind. The risk of hyperglycemia is not trivial. Davies study¹ showed that 20% of the steroid treated patients developed hyperglycemia. There have been concerns of dose related increased incidences of weakness, cataracts, candidiasis, and skin bruising. The specter of avascular necrosis is present. Concerns re osteoporosis abound.

I would consider using systemic steroids in all patients with AECB if they have moderate to severe COPD. A 10-14 day course is necessary, and use the minimum dose required; doses over 30 mg need not be used in non-hospitalized patients.

b) Inhaled Steroids in Acute Exacerbations of COPD

We just do not have the data to support the use of inhaled steroids for AECB. One trial³ of almost 200 randomized patients into nebulized budesonide, oral prednisolone and placebo. Other than similar improvements in FEV₁ at 72 hours, there was not the same benefits to the ICS group as the systemic steroids group. More research is needed into this area.

II. Steroids in Stable COPD

a) Systemic steroids in stable COPD

There is no supportive data for the routine use of systemic steroids in patients with COPD of any severity. Adverse effects potentially obviate any benefits in long term use, unless the benefits were extraordinary. Most of those patients who are on low dose oral steroids can be weaned off slowly with safety. Ensure nutritional and emotional support as the systemic steroids often give a (false) sense of well being.

There may well be a role for oral steroids in identifying the 20% of patients with stable COPD who are steroid responders. A steroid trial of 30 mg per day for two weeks in a stable, non-smoking, bronchodilated individual will separate the patients with an FEV₁ increase of 20% (positive) vs. all others (negative). If positive, the dose of steroid should be tapered to the lowest dose maintaining the level of FEV₁. The GOLD guidelines suggest that a steroid trial be done with three months of equivalent of 500 ug Fluticasone twice daily to observe a 20% increase in FEV₁ in the same type of individuals. This has the advantage of safety, but the disadvantage of longer time to the diagnosis and increased cost.

b) Inhaled steroids in stable COPD

Here is the tough one! There seems to be a lot of pressure to prescribe inhaled steroids to patients with COPD. We feel that on average, 20% of patients with COPD are steroid responders and will have reversibility in their lung function with improvement. Then why are 70% or so of our patients on inhaled steroids?

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Inhaled steroids cause oral candidiasis, bruising, the potential for decreased bone mineral density and possibly increased risk of cataracts.

Many studies, Renkena et al 1996, Weir et al, 1999, EUROSCOP 1999, Copenhagen 1999, Lung Health Study 2000, and ISOLDE 2000, have looked at this issue. Only ISOLDE with Fluticasone 500 ug BID in patients with moderate to severe ($FEV_1 < 50\%$) COPD showed any benefit. At that, it did not show benefit to lung function or mortality, although there was fewer exacerbations, decreased length of hospitalization and a slower decline in quality of life. Criticisms of the study include that it did not separate smokers and non-smokers, nor did it identify those with reversibility prior to therapy.

c) Combination with LABAs

Trials are currently underway to assess the potential for benefit from the combination agents. TRISTAN⁴ is one such trial wherein the combination product of salmeterol and fluticasone was compared to each of its individual parts separately and found to be better. In Asthma we see the beneficial effect of the single molecule in terms of compliance and rapidity of onset compared with the two agents separately. Hopefully these studies will clarify these issues.

Summary:

After all that, what to do?

COPD is increasing and disabling. We have known therapies that bronchodilate. We have new bronchodilators coming (see Spiriva). New therapies such as phosphodiesterase inhibitors are emerging. The role of steroids continues to be controversial. In our efforts to help our patients we must remember the dictum of "do no harm".

This author's recommendations are the following.

AECB in patients with anything other than the mildest COPD should be accompanied by 1-2 weeks of systemic steroids in the lowest effective dose.

Patients with stable COPD, once maximally bronchodilated and not smoking should be offered a steroid trial if there symptoms continue to be symptomatic. If positive, their dose of steroids should be maintained at the dose that keeps their lung function improved. If negative, there does seem to be a 100-150 ml single time improvement in patients with moderate to severe COPD and the ISOLDE trial shows that there may be decreased exacerbations in these patients placed on high dose ICS. The very real issues of adverse effects and cost must counterbalance the benefits to these patients. It should therefore not yet be routine to use ICS in non-steroid responders. Also, response to systemic steroids during an acute attack does NOT predict a successful steroid trial. Combination agents may well change this paradigm, and we await the results of the analyses of TRISTAN and other landmark trials.

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Pneumonia, How long to treat?

Pakistan Multicentre Amoxicillin Study Group. Clinical efficacy of 3 days vs. 5 days of oral amoxicillin for treatment of childhood pneumonia. Lancet 2002, Sept. 14; 360:835-41

2000 children with non-severe pneumonia from 2-59 months were randomized to receive 45 mg/kg of Amoxicillin for either 3 to 5 days. They were diagnosed by WHO diagnostic criteria for non-severe pneumonia, ie. they were not cyanotic, hypoxic, having chest wall retractions or change in mental status while having cough, difficulty breathing or increased respiratory rate. The children were reevaluated at 14 days and had similar rates of treatment failure as described by lack of improvement,

change of antibiotics, development of severe pneumonia or death (~20%).

A subgroup who had radiograph proven pneumonia had comparable results.

Wow, shorter duration of antibiotics at a time we are trying to decrease antibiotic use because of antibiotic resistance. This parallels the data seen with otitis media and even urinary tract infections. This study needs to be repeated in developed countries to ensure its reproducibility, but it is an exciting possible new treatment paradigm. I do not, however, think that a success rate of only 80% would wash in North America!

*Alan Kaplan MD CCFP(EM)
Chair, FPAGC*

Asthma, Are pets OK?

Ownby DR et al. Exposure to dogs and cats in the first year of life and risk of allergic sensitization at 6 to 7 years of age JAMA 2002 Aug 28; 288:963-72

Does early exposure to cat and dog antigen cause asthma or actually decrease the risk of asthma? 474 children in a Detroit HMO were evaluated to determine the relationship between having atopy at 6 to 7 years of age to exposure to dog and cat in the first year of life. Atopy was measured by serum IgE levels or skin prick sensitivity. Adjustment in the measurements were made for other allergic risk factors.

Skin test sensitivity was found in 34% of those kids with no pet exposure vs. 15% of those with exposure to at least 2 dogs or cats. IgE levels were also lower in the exposure group. Methacholine responsiveness was lower in boys with exposure to at least 2 dogs or cats. The presence of asthma at 6-7 years of age was not affected in either sex child. Pet exposure at the time of the testing (6-7

yr.) did not affect the presence of atopy. This study has some methodological flaws as it only used surrogate markers of asthma in terms of skin prick testing and IgE levels. This data is, however, supported by a couple of other studies.

Von Ehrenstein et al, Clin Exper Allergy 2000;30:187-93 showed a reduced risk of hay fever(rhinitis) and asthma among children of farmers. The reduction in risk was stronger for full-time farming families compared with part time. Farmer's children had lower prevalences of hay fever, asthma, and wheeze. An Austrian study showed that children living on a farm had less hayfever, asthma, and allergic sensitization. *Reider J, et al. Clin Exper Allergy 2000;30:194-200* studied children 8-10 years old. It showed that living on a farm significantly reduced prevalences of hayfever, asthma, and 1+ positive skin test.

This is difficult to explain, isn't it! One theory is the **Hygiene hypothesis**. This theory encompasses the theory of the

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increased prevalence of asthma in our Western society. The theory is that with our increasingly clean society, airtight homes and decreased exposure and infection, the Th0 cells get differentiated not into Th1 but into Th2 which causes increases in IL4 and IL5 and increased eosinophils; thus increased allergic disease.

Early antigen exposure in sufficient amounts may well prevent sensitization.. In the meantime, animal avoidance for

asthmatics with documented allergy to pets should be encouraged. This data is not yet strong enough for us to tell all of our asthmatic parents to go get two cats or dogs as soon as they are pregnant to prevent their kids from getting asthma, but the paradigm may well be changing. Maybe we encourage these parents to get a couple of cats, and then we have to get rid of the animals if they get symptoms! (My tongue is a little in my cheek.)

SPIRIVA, A new more specific anticholinergic bronchodilator for COPD.

From GOLD guidelines to the upcoming new Canadian COPD Guidelines the first treatment after smoking cessation for COPD is bronchodilators. Vagally mediated bronchoconstriction is thought to be the major reversible component of airway obstruction in these patients. Bronchodilators, with anticholinergics and B2 Agonists, alone or in combination, are recommended in all stages of COPD treatment.

Muscarinic receptors in the airways control a significant amount of smooth muscle function in human airways. There have been three types of muscarinic receptors identified. M1 receptors facilitate cholinergic neurotransmission through the parasympathetic ganglia. M2 receptors are located on the postganglionic cholinergic nerves and provide negative feedback modulation of acetylcholine release. Therefore, inhibition of M2 receptors results in increased release of acetylcholine and bronchoconstriction. M3 receptors are located on bronchial smooth muscle and mucous glands. They mediate the airway smooth muscle contractile response and mucus secretion which occurs as a response to acetylcholine.

Tiotropium bromide is the newest member of the quaternary ammonium class of the anticholinergic bronchodila-

tors, which dilate bronchial smooth muscle through antagonism of muscarinic receptors located in the airway smooth muscle. It binds equally to the M1, M2, and M3 receptors, but dissociates much more slowly from the M1 and M3 receptors than from the M2 receptors. Therefore, the negative feedback from the M2 receptor is decreased which allows a long duration of action and therefore once daily administration in patients with COPD.

Twenty percent of an orally inhaled dose of tiotropium is deposited in the lung. It is rapidly absorbed into the systemic circulation, with peak plasma levels at 5 minutes declining to low levels in less than one hour. There was no evidence of drug accumulation once steady state was achieved. Seven percent of the delivered dose is excreted unchanged in the urine. At steady state, the mean plasma elimination half life was 5 to 6 days. 18ug has been decided to be the optimal dose. Inspiratory flow rates necessary for the HandiHaler device of 15 L/min are sufficient for lung deposition. This is possible even in patients with severe COPD (FEV₁ 65% to <27% of predicted¹)

Lung function as measured by trough FEV₁, trough FVC, and am and pm PFR. These were all improved by Tiotropium

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Spiriva (Continued From Page 7)

vs. placebo in one year studies^{2,3}.

Lung function as measured by trough FEV₁ and FVC, am/pm PFR, and FEV₁ at one year were improved in all severities of COPD compared with Ipratropium⁴.

Lung function measured by trough FEV₁, FVC, and PFR showed an improved response with tiotropium to Salmeterol 50ug BID in a placebo controlled six month strategy⁵.

Health related quality of life, COPD exacerbations and dyspnea were also superior^{2,3,5}.

Dyspnea, measured by the TDI (Transition Dyspnea Index) was improved vs. placebo, Ipratropium, and Salmeterol^{1,2,3,5}.

Similarly, time to first exacerbation was lengthened, the number of exacerbations were reduced, there were fewer hospitalizations for COPD, and fewer days spent in hospital due to exacerbations vs. Placebo³. There were reduced incidents of COPD exacerbations, the number of COPD exacerbations, time to first exacerbation and time to first exacerbation vs. Ipratropium². There was a tendency for fewer exacerbations with COPD in the tiotropium vs. Salmeterol⁵. Tiotropium patients took fewer rescue Salbutamol in all the trials (both groups reduced vs. Salmeterol). Tiotropium may cause reduced salivation; therefore the commonest side effect is that of dry mouth. It is generally of mild intensity and resolves with continued treatment and did not necessitate treatment cessation².

Tiotropium is delivered once daily, morning or evening but at the same time every day, via dry powder by the HandiHaler. A capsule, which must be kept in its sealed pouch until just before use, is punctured in the device and the dry powder is inhaled. The capsule should not be swallowed. The capsules should be stored in a cool dry place, and not be frozen or heated.

In summary, Tiotropium is a long acting anticholinergic bronchodilator that is administered once daily by dry powder inhalation for the treatment of COPD. Studies show improvement in lung function and decreased exacerbations of COPD. There has been no evidence of tachyphylaxis and the studies hint that disease progression, as measured by FEV₁ at one year, will be blunted in patients receiving tiotropium. There will be benefit to our patients with this product, but also to the health care system with reduced exacerbations. We do not know yet if this medication will modify the natural course of COPD after one year (ie. slow down the progressive decline in lung function in COPD). We also will see if combining tiotropium with long acting B2s will further improve the situation.

Alan Kaplan MD CCFP(EM)

Chair, FPAGC

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Emergency Rounds

Food-dependent exercise-induced anaphylaxis (FDEIA)

FDEIA is a specific variant of exercise-induced anaphylaxis that requires both vigorous physical activity and the ingestion of specific foods within the preceding several hours. Urticaria, angioedema, upper respiratory obstruction and cardiovascular collapse are the potential symptoms that occur with anaphylaxis. This syndrome is unusual in that it requires both of the factors to occur, i.e., neither the food allergen alone nor exercise alone will trigger the reaction. This can occur in both adults and children.

It is an IgE mediated reaction with mast cell degranulation. It is felt that the food allergen and the subsequent IgE response lowers the mast cell release threshold. Exercise will then trigger the degranulation of the mast cells and the anaphylaxis occurs. There are two similar syndromes, which I will distinguish.

Cholinergic urticaria¹ is a syndrome wherein an urticarial rash occurs as a response to elevation of body temperature; either due to active heat generation (exercise) or passive heating. It is generally not associated with angioedema, bronchospasm, or hypotension.

Exercise-induced anaphylaxis² results from exercise-related temperature elevation independent of food intake. It is associated with other atopic disorders. It is episodic and tends to stabilize or decrease over time. Exercise will not always be problematic and a particular degree of exercise will not reproduce the anaphylaxis predictably.

Food-dependent exercise-induced anaphylaxis requires both the ingestion of the specific foods and vigorous exercise within several hours after ingestion. The most common food triggers implicated are shellfish, alcohol, tomatoes, cheese, celery, strawberries, milk, wheat products, and peaches. The diagnosis is made

clinically, with skin-prick food allergy testing³ and possibly with an exercise test after food ingestion (depending on the degree of risk).

Epinephrine is the mainstay for the treatment of anaphylaxis. H₁ antagonists and salbutamol are secondary agents. Occasionally combined H₁-H₂ antagonist therapy will improve symptom relief⁴. Corticosteroids will help prevent the biphasic reactions and may help especially the severe reactions such as those requiring resuscitation and intubation¹⁻³.

Please ask your patients about vigorous exercise within 6 hours of eating in those patients that present with severe allergic reactions. While the allergist appointment is pending, advise your patients to not exercise within six hours after eating, and to carry injectable epinephrine and antihistamine with them while exercising. This does NOT mean that they must stop exercising. Patients should likely not exercise alone.

Correct diagnosis of FDEIA will allow patients to take control of their lifestyle and hopefully prevent future acute events and ER visits.

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Risk of Acute Coronary Syndrome related to *B2* agonists.

Oral *B*-Blockers have been proven to lower the risk for repeat MI and all cause mortality in patients who have had an MI. It has been shown that *B2* agonists used for obstructive lung disease have been shown to increase the risk of cardiovascular mortality in these patients. A previous study by the same authors showed that the new use of inhaled *B* agonists resulted in a 7-fold increased risk of MI in patients with cardiovascular disease. This study assesses the association between the use of *B2* agonists as MDIs and hospital admissions.

Au DH, et al. Association between inhaled *B* agonists and the risk of unstable angina and myocardial infarction. Chest 2002 Mar;121:846-51

Patients with an acute coronary event were assessed as to whether they had filled a prescription for a *B* agonist in the 90 days preceding the event. Those that did fill this prescription had an increased risk of event, which was independent of other risk factors or age. The risk increased depending on the number of canisters. Those patients on oral or systemic *B* agonists and inhaled *B2* agonists did not have an increased risks unless they used six or more canisters.

This is by no means proof of cause and effect. But it does encourage caution in the new use of *B2* agonists in patients with increased risk of cardiovascular ischemia

Use of FEF₂₅₋₇₅

After having done countless workshops and lectures on spirometry, a recurrent question that arises is the use of the FEF₂₅₋₇₅. This reading is felt to be the least effort dependent portion of the expiratory maneuver. Obstruction seems to begin in the small airways in COPD. The FEF₂₅₋₇₅ is seen as the value which represents the condition of the small airways and may be a better predictor of hypoxemia than the FEV₁.

A Turkish paper, 'Spirometric predictors for the exclusion of severe hypoxemia in COPD' (Can Respir J 2001;8(4);245-249) looked into the need of for arterial blood gases in patients with severe COPD. The objective was to investigate the correlation between

severe hypoxemia and multiple spirometric parameters in patients with COPD and FEV₁ 50% of predicted or greater. Patients with Hb less than 100 or with cardiovascular disease were excluded.

The study found that one in five patients with COPD and an FEV₁ of 50% or greater was hypoxemic (PaO₂ less than 60 mmHg). FEV₁, FEV₁/FVC, PEF parameters all failed to predict or exclude severe hypoxemia. The negative predictive value of the FEF₂₅₋₇₅ being greater than 50% of predicted value was 92%. Therefore it would be reasonable to withhold ABG analysis in the clinical analysis of patients with FEV₁ equal or greater than 50% and FEF₂₅₋₇₅ of greater than 50%.

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THE COMMITTEE

Dr. A. Kaplan
(Chairperson)
17 Bedford Park Avenue,
Richmond Hill, ON,
L4C 2N9
(905) 883-1100
E-mail: FOR4KIDS@aol.com

Dr. Robert Hauptman
(Secretary/Treasurer)
The Associate Clinic,
25 St. Michael Street,
St. Albert, AB,
T8N 1C7
(403) 458-1234

Dr. Josiah Lowry
333 Mary Street
Orillia, ON
L3V 3E9

Dr. Gord Dyck
Box 21427
Steinbeach, MB
ROA 2T3
(204) 326-6111

Dr. Alain Couet
181 Principale, Suite C-12,
Aylmer, PQ,
J9H 6A6
(819) 685-9110

Dr. John Rea
104 - 348 Muskoka Rd. 3
North
Huntsville, ON
P1H 1H8
(705) 789-3255

Dr. John Li
102 - 798 Mountain Road
Moncton, NB
E1C 2R4
(506) 859-8696

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MISSION STATEMENT

The Family Physicians Airways Group of Canada is committed to helping those with airway diseases lead a full life. The group is dedicated to helping all family physicians maintain and increase their skill in assisting those with asthma and COPD. The strategy of the Group is to maintain a speaker bank, a data base, and practical tools to help physicians attain these skills.

*"A group of family physicians
with a special interest in asthma."*

DÉCLARATION DE PRINCIPES.

L'Association canadienne des médecins de famille contre l'asthme. Un groupe de médecins de famille ayant un intérêt particulier pour le traitement de l'asthme. Les membres de l'Association canadienne des médecins de famille contre l'asthme s'engagent à aider les personnes atteintes d'asthme à jour pleinement de leur vie. L'Association veut aider tous les médecins de famille à entretenir et améliorer leurs connaissances dans le traitement de l'asthme. L'Association se propose de maintenir une liste de conférenciers et une banque de références, et colliger des informations pratiques pour permettre aux médecins d'acquérir ces connaissances.

The opinions expressed in this newsletter are those of the authors, and not necessarily those of the Family Physician Airway Group of Canada.

Website www.fpagc.com

