1986 YEAR BOOK OF

PULMONARY DISEASE

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1986

The Year Book of PULMONARY DISEASE

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Table of Contents

The material covered in this volume represents literature up to October 1985.

in FEV_1 that did not reach significance. There was no evidence to suggest any time effect over the 4-week treatment period for either PC_{20} or FEV_1. There was no evidence for treatment interaction and no evidence of a treatment-order effect caused by the crossover design. Compliance rates between active treatment (84.75%) and placebo treatment (87.6%) were not different. When individual compliance rates on beclomethasone were plotted against change in PC_{20}, a direct linear relationship was found ($P = .022$), indicating that improvement was largest in subjects with good compliance.

These results suggest that regular treatment with beclomethasone can reduce bronchial responsiveness to histamine by mechanisms other than improvement in airway caliber.

▶ Bronchial hyperresponsiveness appears to be important in the asthmatic diathesis. Will modification of this hyperresponsiveness result in a clinical benefit for asthmatics? The answer to this question is unknown. However, this study, as well as other studies using agents such as sodium cromoglycate,[1] suggest that bronchial responsiveness can be decreased slightly by pharmacologic manipulation. Whether the slight modification of reactivity reported in this article can be increased by other treatment regimes and whether this modification will have a clinical benefit are unclear. Efforts to modulate bronchial reactivity will require long-term therapy (i.e., months or years), and so studies designed to test this hypothesis will require well-designed and controlled studies with long follow-up.—Stephen P. Peters, M.D.

Reference

1. Lowhagen O., Rak S.: Modification of bronchial hyperreactivity after treatment with sodium cromoglycate during pollen season. *J. Allergy Clin. Immunol.* 75:460–467, 1985.

Tracheobronchial Mucociliary Clearance in Asthma: Impairment During Remission

D. Pavia, J. R. M. Bateman, N. F. Sheahan, J. E. Agnew, and S. W. Clarke (Royal Free Hosp. and School of Medicine, London)

Thorax 40:171–175, March 1985 2–18

Patients with stable asthma have impaired mucus transport in the tracheobronchial tree. To determine whether this function is reversed during remissions of the disease, tracheobronchial mucociliary clearance, as measured with an objective radioaerosol technique, was determined in eight nonsmoking patients with asthma in complete remission. The patients were symptom free and had taken no medication for 1 to 6 months before assessment. Mucociliary clearance was also measured on 2 occasions in eight nonsmoking healthy subjects with similar physical characteristics and pulmonary function. In the first assessment, the healthy subjects inhaled the tracer radioaerosol under laminar flow conditions similar to those used

for the asthmatics. In the second assessment, they inhaled the aerosol rapidly to simulate the alveolar deposition of the asthmatics.

Under conditions of similar inspiratory flow, the mean alveolar deposition for the asthmatic and healthy groups were 46% (SEM 4%) and 60% (3%) ($P < .01$), indicating a considerable deposition of particles proximally in the asthmatic patients. When the depth of radioaerosol lung penetration was similar in both groups, tracheobronchial clearance was poorer 6 hours after radioaerosol inhalation in the asthmatic group ($P < .01$).

These data raise doubt about whether asthma ever remits completely. Alterations in the architecture of the airways of asthmatics due to the presence of increased mucus, edema or spasm, increased tortuosity, or asynchronous ventilation in the airways may account for the reduced alveolar deposition during remission.

▶ This study (as well as the histologic study on epithelial damage in asthmatics abstracted above) suggests that mucociliary clearance (and ciliary morphology) is impaired in asthmatics, even in remission. However, such an "impairment" could only be demonstrated when the authors controlled for proximal or "alveolar" deposition of the aerosol. When inspiratory flow rates were matched and controlled at approximately 25 L/minute in both controls and asthmatics, no difference in whole lung or tracheobronchial clearance was noted (60% of the delivered dose was deposited proximally in the asthmatics, while only 46% was deposited proximally in controls). These studies raise questions not only about mucociliary clearance in asthmatics, but also about issues of aerosol deposition, as discussed above.—Stephen P. Peters, M.D.

↓ ▶ The following four articles discuss cellular events that may be important in the pathogenesis of bronchial asthma. The first two discuss cellular infiltration and activation during late phase asthmatic reactions induced by allergen. As introduced above, experimental asthma induced by several agents including small-molecular-weight chemicals, allergens, and exercise is associated not only with an immediate decrease in pulmonary function, but in many subjects with a second, often prolonged physiologic response that occurs hours after the initial challenge procedure. These delayed or "late" reactions have been considered to be more representative of the asthmatic state than the immediate response to bronchial challenge.[1–3] This is because many of these late reactions display kinetics (i.e., they are long-lasting) and pharmacology (i.e., they are difficult to treat, requiring more than simple inhaled bronchodilator) more characteristic of the asthmatic diathesis than immediate or early reactions. Therefore, considerable work is being performed to explore mechanisms involved in the production of late asthmatic reactions.—Stephen P. Peters, M.D.

References

1. Kaliner M.: Hypotheses on the contribution of late-phase allergic responses to the understanding and treatment of allergic diseases. *J. Allergy Clin. Immunol.* 73:311–315, 1984.

2. Cockcroft D.W.: The bronchial late response in the pathogenesis of asthma and its modulation by therapy. *Ann. Allergy* 55:857–862, 1985.
3. Schleimer R.P.: The mechanisms of antiinflammatory steroid action in allergic diseases. *Ann. Rev. Pharmacol. Toxicol.* 25:381–412, 1985.

Bronchoalveolar Eosinophilia During Allergen-Induced Late Asthmatic Reactions

Jan G. R. De Monchy, Henk F. Kauffman, Per Venge, Gerard H. Koëter, Henk M. Jansen, Henk J. Sluiter, and Klaas De Vries (State Univ. Hosp., Groningen, the Netherlands; Univ. Hosp. of Amsterdam; and Univ. Hosp., Uppsala, Sweden)

Am. Rev. Respir. Dis. 131:373–376, March 1985 2–19

The late asthmatic reaction (LAR) is characterized by slowly progressive bronchial obstruction starting 3–4 hours after allergen inhalation and usually peaking after 7–8 hours. Recurrent nocturnal attacks sometimes occur. The local inflammatory response associated with the LAR was investigated by performing bronchoalveolar lavage in 19 asthmatic patients and 5 controls. The patients were 15 males and 4 females with a mean age of 24 years. All had increased bronchial reactivity to histamine and acetylcholine. None had acute attacks in the previous 2 months. Sixteen patients and all of the controls underwent lavage 6–7 hours after inhaling increments of house dust mite extract.

Six patients had both early and late reactions to allergen inhalation, whereas 5 had an early asthmatic reaction only. No patient required steroid or antibiotic treatment. More eosinophils were obtained during LAR than in other settings, but no differences in percentages of neutrophils or lymphocytes were apparent. The ratio of eosinophil cationic protein to albumin was significantly elevated in the LAR group compared with the others, except for the early reaction group.

These bronchoalveolar lavage findings suggest that infiltration of eosinophils, rather than of neutrophils, accompanies the early stages of the late asthmatic reaction. Eosinophils and their mediators may be involved in development of the LAR after allergen inhalation. The eosinophils might be attracted by chemotactic substances released during mast cell degranulation in the early asthmatic reaction.

▶ This article suggests that eosinophils infiltrate the lung in individuals who experience late responses to inhaled allergen, but not in those subjects who have only early responses or in controls. While these results are from a small number of patients and must be confirmed, they suggest that eosinophils might play a role in the development of late asthmatic responses. The mechanism by which this could occur remains speculative; however, eosinophils have been shown to produce large amounts of the spasmogenic leukotrienes (leukotrienes C_4 and D_4 or SRS-A) discussed above, and eosinophil-derived proteins have been shown to be cytotoxic to bronchial epithelial cells.—Stephen P. Peters, M.D.

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Leukocyte Activation in Allergen-Induced Late-Phase Asthmatic Reactions
S. R. Durham, Mary Carroll, G. M. Walsh, and A. B. Kay (Brompton Hosp., London)
N. Engl. J. Med. 311:1398–1402, Nov. 29, 1984 2–20

Specific and nonspecific stimuli may activate mast cells and possibly other mediator cells in both the early- and late-phase reactions of asthmatics to allergen inhalation or exercise. The role of neutrophil activation in late-phase reactions induced by a specific allergen was examined in 17 nonsmoking, atopic asthmatics who were taken off bronchodilator therapy for 12 hours before evaluation. Eleven patients with dual asthmatic reactions were studied on two separate days after challenges with methacholine and allergen. The 6 subjects with single, early asthmatic reactions were challenged with allergen by using a tidal-breathing method. The modified Boyden method for assessing neutrophil chemotactic activity was utilized.

A time-dependent increase in neutrophil complement rosettes was noted during the early reaction, accompanied by an increase in neutrophil chemo-

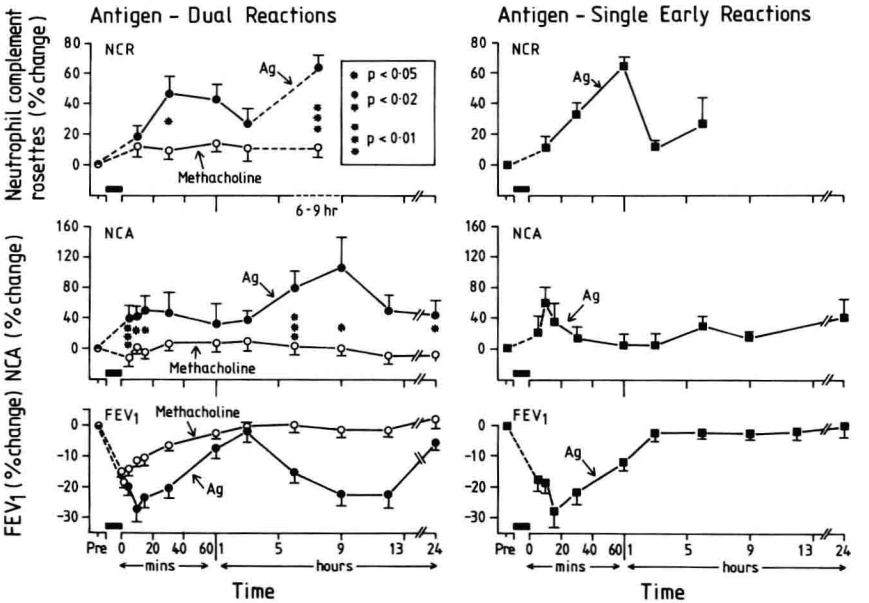

Fig 2–7.—Neutrophil complement rosettes, NCR; serum neutrophil chemotactic activity, NCA; and forced expiratory volume in 1 second, FEV_1; expressed as the percentage of change (mean ±SEM) in 11 subjects with dual asthmatic reactions after allergen (Ag) and methacholine, and in 6 subjects with single, early reactions after allergen. The solid lines represent the period of challenge. For subjects with dual responses, the baseline values were as follows: FEV_1, 3.45 ± 0.28 and 3.40 ± 0.25; NCA, 92.7 ± 12.6 and 93.6 ± 15.5; and NCR, 27.8 ± 1.8 and 26.7 ± 2.0 after methacholine and allergen, respectively. For subjects with a single, early response, the baseline values were as follows: FEV_1, 3.33 ± 0.18; NCA, 94.9 ± 15.2; and NCR, 30.7 ± 3.1. In the dual response group the late-reaction NCR values were measured at 6 hours (six subjects), 7 hours (two subjects), or 9 hours (three subjects). (Courtesy of Durham, S.R., et al.: N. Engl. J. Med 311:1398–1042, Nov. 29, 1984. Reprinted by permission of The New England Journal of Medicine.)

tactic activity (Fig 2–7). These changes were not significantly different in the early reactions of asthmatics with dual responses and those of subjects with a single early response to allergen challenge. The increases during late reactions, however, were greater than in the subjects with a single response. Increases in monocyte complement rosettes in the late reaction also exceeded the values seen in subjects with a single early response. Most subjects had comparable peak early reductions in FEV_1 on the 2 challenge days.

Neutrophil activation accompanies both exercise-induced asthma and allergen-induced early and late asthmatic responses. Monocyte activation also is noted during dual reactions. The leukocyte changes cannot be attributed to bronchoconstriction, since mediator release and leukocyte activation are not associated with methacholine-induced immediate reactions. Mast-cell degranulation and activation of secondary inflammatory cells in the late response may have an important role in peribronchial inflammation, which in turn may contribute to the development of nonspecific bronchial hyperreactivity and to ongoing asthma.

▶ This group has been at the forefront in defining cellular events associated with early and late asthmatic responses to allergen exposure and exercise in humans. In an elegant series of articles that originated from several different laboratories (reviewed in the index article), it has been shown that antigen and exercise bronchoprovocation are associated with the release of a high-molecular-weight neutrophil chemotactic activity in the blood of volunteers who experience early and late phase reactions. The abstracted article suggests that both neutrophils and monocytes are activated by allergen inhalation, as shown by an increase in complement rosettes (an index of complement receptors on these cells). The authors of both this and the preceding article suggest that mast cell activation triggered by allergen exposure is responsible for initiating events that eventually result in eosinophil infiltration of the lung and in the activation of neutrophils and monocytes. While it is tempting to speculate that these events are causally associated with the resulting clinical syndromes (i.e., late asthmatic responses), such a causal relationship has not been shown. However, it is being clearly demonstrated that inflammatory events such as cellular recruitment and activation appear to be important in the pathogenesis of bronchial asthma.—Stephen P. Peters, M.D.

Secretion of a Chemotactic Factor for Neutrophils and Eosinophils by Alveolar Macrophages From Asthmatic Patients

Philippe Gosset, André B. Tonnel, Michel Joseph, Lionel Prin, Anne Mallart, Jacques Charon, and André Capron (Pasteur Inst., Lille, France)

J. Allergy Clin. Immunol. 74:827–834, December 1984 2–21

To investigate the possible production of a neutrophil chemotactic factor (NCF) or eosinophil chemotactic factor (ECF) by alveolar macrophages (AM), the authors tested the in vitro chemotactic activity of AM supernatants after stimulation by anti-IgE. Fluid specimens from 15 asthmatic

patients and 15 nonatopic patients (control subjects) were obtained by bronchoalveolar lavage.

Incubation of normal AMs previously sensitized by 20% nonheated allergic sera for 60 minutes with anti-human IgE antibody or the related allergen induced the release of chemotactic activity for polymorphonuclear neutrophils (PMN) and eosinophils in culture supernatants. Direct incubation of AMs from asthmatic patients with anti-IgE or the related allergen induced the same chemotactic activity, though incubation with an unrelated allergen failed to produce chemotactic activity. Neutrophil chemotactic activity after addition of anti-IgE was 22.5 ± 3.5 cells per high-power field, after addition of related allergen was 15.8 ± 3.6 cells, and using an unrelated allergen was 0.7 ± 1.8 cells ($P < .0001$). Studies to partially characterize the NCF showed that enzymatic treatment by trypsin or carboxypeptidase or by heating at 56 C for 3 hours failed to abolish the neutrophil chemotactic activity. After gel filtration, 80% of the neutrophil chemotactic activity was recovered among low molecular weight components (300 to 1,300 daltons). Preliminary deactivation of PMN using leukotriene B_4 suppressed the chemotactic activity of the AM supernatants.

The results demonstrate that IgE-dependent stimulation of alveolar macrophages produces neutrophilic and eosinophilic chemotactic activity that is present in a low-molecular-weight fraction possibly related to leukotrienes. Although additional studies are needed to clarify the in vivo relevance of NCF release, the findings emphasize the role of alveolar macrophages in inflammatory lung processes during allergic asthma. In addition to enzyme release and platelet-activating factor production, the generation of NCF by alveolar macrophages following allergic or anti-IgE stimulation suggests an alternative mechanism for initiation of the influx of circulating inflammatory cells into alveoli and amplification of the inflammatory response in the adjacent lung parenchyma.

▶ An article abstracted above observed the infiltration of eosinophils into the lung in allergen-induced late asthmatic reactions. We speculated that mast cell activation was important in such a process. This article suggests another possible mechanism. This article demonstrates that the antigen equivalent anti-IgE can directly cause the release of a factor chemotactic for neutrophils and eosinophils from alveolar macrophages from asthmatic subjects. While these authors have indirect evidence to suggest that the chemotactic agent might be leukotriene B_4, Fels et al.[1] demonstrated that nonspecific activation of human alveolar macrophages (with the ionophore A23187) results in the release of leukotriene B_4 in vitro. The conclusion that one can draw from these studies is that the lung contains immunocompetent cells that can produce the inflammatory response that we are observing in these models of asthma.—Stephen P. Peters, M.D.

Reference

1. Fels A.O.S., Pawlowski N.A., et al.: Human alveolar macrophages produce leukotriene B_4. *Proc. Natl. Acad. Sci. USA* 79:7866–7870, 1982.

Anaphylatoxin C3a Enhances Mucous Glycoprotein Release From Human Airways in Vitro

Z. Marom, J. Shelhamer, M. Berger, M. Frank, and M. Kaliner (Mt. Sinai Med. Ctr., New York; Natl. Inst. of Health, Bethesda, Md.; and Case Western Reserve Univ.)

J. Exp. Med. 161:657–668, April 1985 2–22

During the course of complement activation, anaphylatoxins are generated capable of causing mast cell and basophil degradation. Anaphylatoxins may be generated during immunologic pulmonary inflammatory reactions. Thus, the ability of C3a to affect mucous glycoprotein (MGP) secretion in vitro was investigated in human airways tissue culture. Its mechanism of action was determined through effects of several pharmacologic manipulations on the release of radiolabelled MGP.

C3a, but not C3a des Arg, caused a dose-related increase in MGP release, with as low as 5 μg/ml stimulating a 25% increase. The activity of C3a was rapid, causing MGP release within 30 minutes, and specific, as reflected in the ability of C3a antisera to prevent or absorb C3a activity.

Several experiments were undertaken to determine whether the secretion-stimulating activity of C3a was related to the generation of such putative secretagogues as neurohormones, mast cell-derived mediators, and a macrophage-derived mucus secretagogue (MMS). There was no evidence of histamine release accompanying C3a-induced MGP release. Moreover, cAMP inhibited both the MGP and histamine release induced by reverse anaphylaxis while having no effect on C3a actions. Eicosatetraynoic acid or specific cyclooxygenase inhibitors did not influence the C3a-induced MGP release, nor were leukotrienes detectable on the supernatants of C3a-stimulated airways, indicating that oxidative products derived from arachidonic acid had no effect on C3a-induced MGP release. Cycloheximide failed to affect C3a secretion-stimulating actions. Thus, it was unlikely that C3a stimulated MGP release by activating MMS generation.

C3a is a potent mucus secretagogue. Although its mechanism of action remains unclear, it is possible that C3a may act directly as a glandular stimulant. It seems likely that C3a generated during pulmonary inflammation may contribute to the mucus secretion associated with pulmonary infections.

▶ The role of complement in the pathogenesis of bronchial asthma is a topic for more complete discussion in another volume of the YEAR BOOK. However, this article demonstrates that the anaphylatoxin C3a is a potent stimulus of mucous secretion from human airways in vitro and suggests that complement components may be important in mucous hypersecretion seen in several different pathologic conditions such as pulmonary infections and perhaps in asthma as well.—Stephen P. Peters, M.D.

Treatment

Direct and Indirect Costs Associated With the Management of Childhood Asthma

Richard J. Marion, Thomas L. Creer, and Russ V. C. Reynolds (Ohio Univ.)
Ann. Allergy 54:31–34, January 1985 2–23

Currently escalating health care costs have a particular impact on families with chronic disease such as asthma. Childhood asthma creates many physical and emotional problems for the child and imposes an emotional and financial burden on the entire family. Indirect costs may ultimately be a greater expense than direct health care costs. The direct and indirect costs of childhood asthma were estimated in 50 families that included a child with a diagnosis of chronic intractable asthma and that participated in a self-management project between 1977 and 1980. Thirty-six families completed the assessment procedures, and 25 included in the analysis reported data for an average of 10 months.

The direct and indirect asthma expenditures for all families are given in the table. The 25 evaluable families spent an average of 5.5% of their annual incomes for direct asthma costs. The average proportion of annual income consumed by indirect asthma costs was about 0.9%. Total asthma-related expenditures averaged 6.4% of annual income, ranging from 0.35% to 33%. Lower-income families lost about 12% of their total yearly income on average to childhood asthma, compared with 4% for higher-income families.

Physician fees were the largest direct expense, followed by pharmacy fees and hospital-related expenses. Indirect costs had a particular impact on low-income families. This, with evidence that the poor have greater medical needs, emphasizes the importance of finding ways to minimize

Direct and Indirect Asthma Expenditures for All Families and Costs by Percentage of Total Income

	Average	Range	
		Low	High
n = 25			
Income	$24,744.40	$5,500.00	$85,000.00
Direct costs	$940.72	$52.25	$3,935.55
Physician	$358.80	$0.00	$1,902.00
Pharmacy	$233.90	$0.00	$1,534.00
Hospital	$204.86	$0.00	$1,872.31
Indirect costs	$146.48	$0.00	$383.25
Miles	418 miles	0 miles	1,392 miles
Income loss	$50.00	$0.00	$280.00
Miscellaneous	$33.10	$0.00	$300.00
Total costs	$1,087.19	$88.86	$3,965.25
Percent of total income:			
Direct costs	5.50%	.10%	32.8%
Indirect costs	.87%	0%	6.3%
Total costs	6.40%	.35%	33.0%

(Courtesy of Marion, R.J., et al.: Ann. Allergy 54:31–34, January 1985.)

asthma-related expenditures. Teaching self-management skills to children and parents may be an effective means of lessening the financial impact of asthma on families. Low-income families without health insurance are most likely to benefit economically from such training.

▶ What is the cost of caring for an asthmatic child? According to this small but well-done study, about 6% of a family's income or $1,100 in 1979 dollars with approximately one third going to pay doctors' fees and 20% to 25% for hospital and pharmacy, respectively. For lower income families, the proportion of family income devoted to treatment increased to an average of 12% of total yearly income. The following article suggests one strategy for controlling such costs.—Stephen P. Peters, M.D.

A Randomized Trial of A.C.T. (Asthma Care Training) for Kids

Charles E. Lewis, Gary Rachelefsky, Mary Ann Lewis, Ann de la Sota, and Michael Kaplan (Univ. of California at Los Angeles and Southern California Permanente Med. Group, Los Angeles)

Pediatrics 74:478–486, October 1984 2–24

The authors conducted a randomized, controlled, clinical trial of the Asthma Care Training (ACT) for Kids self-management program. Seventy-six children, aged 8 to 12 years, who had asthma requiring treatment with medications at least 25% of the days of the month, were randomly assigned to control and experimental groups. The control group received three 1½-hour lectures on asthma and its management. Subjects in the experimental groups, each consisting of 5 to 7 children and their parents, received five 1-hour sessions. The children and their parents were interviewed before the sessions and 3, 6, and 12 months after "treatment" had been completed. The use of emergency rooms and hospitals was established by review of the records of the patients for the year before and the year after treatment.

There was no difference in knowledge gained regarding the symptoms of asthma and medications needed between the control and experimental groups. Both groups showed significant reductions in the perceived severity of their asthma attacks. However, parents in the experimental group, but not the control group, showed a significant shift in their perception of the severity of their child's asthmatic episodes. Children in the experimental group only showed significant improvement toward self-care behavior and taking extra medication when needed. Significant reductions in the number of emergency room visits and the number of days of hospitalization were documented in the experimental group only. It is estimated that the perceptual and behavioral modifications brought about through the ACT for Kids program represent a savings of about $180 per child annually.

▶ In any program that tries to achieve better (and cheaper) control of a chronic disease, understanding of disease pathogenesis and the rationale for treatment are, of course, prerequisites. Although patients vary markedly in the understanding of their disease, a recent report from Great Britain suggests

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