BRONCHIAL ASTHMA AND ENVIRONMENT**

M.S. AGNIHOTRI*

I am thankful to the Tuberculosis Association of India for giving me this opportunity to address you, the delegates of 39th National Conference on Tuberculosis and Chest Diseases. House of "Wanders" deserves appreciation, as it is because of their assistance that the oration is a permanent feature of the conference.

Environment includes everything around us. Therefore, the scientists from various other fields are also contributing a lot in understanding the mysteries of environment. We medical men are interested in health and long life of mankind and freedom from illness and physical impairment. So my talk will be limited to the effect of environment on human health. Environmental diseases are closely related to the lung. Therefore, they concern us - the Chest Physicians.

Food, water and air are the basic raw materials required for normal living of mankind which he utilises from his environment. Man can live without food for 5 weeks, without water for 5 days but cannot live without air for more than 5 minutes. In one day, man requires about 1.3 Kg. of food, about 2 Kg. of water but requirement of air is 13 Kg. per day. Air is most vital for normal human life. Community is advised not to eat or drink unhygienic food or water but one has to inhale polluted air if present in the environment. Most of the air pollutants are invisible and affect the health. The air pollutants enter the body through exposed organs, commonest being the lungs. Inhaled substances include inorganic and organic dust, spores and pollen, microorganisms, fumes, vapours, gases and radio-active particles. Depending on their physical & chemical properties, concentration, duration of exposure, host factor and site of deposition, they are capable of producing various types of reaction in the lung such as mucosal inflammation, alveolitis, asthma, fibrosis, granuloma, emphysema and neoplasm (Comming et al, 1980)

Further, I shall limit myself to asthmatic reaction of lungs caused by the various environmental factor.

Definition of Asthma

Asthma is defined as a disease characterised by wide variation over short period of time in resistance to flow in intrapulmonary airways (Scadding 1983). Increased resistance to airflow is due to environmental factors especially inhaled substances in concentrations that do not affect the majority of persons. Detectable factors include allergens, exercise, and physical and chemical stimuli. Diminution in increased resistance in response to bronchodilator drugs and steroids is usually demonstrable. Although asthma is characterised by paroxysms it may become continuous later on. In such instances diagnosis may be justified by the evidence that wide variability has been present in the past.

Epidemiology of Asthma

Epidemiological studies have thrown light on various aspects of bronchial asthma. Epidemiological studies in U.S.A. have revealed that 5% of adults and 10% of children suffer from asthma in U.S.A. (Gregg, 1977). Very few prevalence surveys of asthma have been conducted in India (I.C.M.R., 1961 and Viswanathan et al 1969).

A. Increase in Prevalence of Asthma

Various prevalence surveys confirm the clinical impression of increase in the frequency and severity of asthma in children and adults in the last 20 years. The interpretation and comparison of various prevalence surveys of asthma is difficult because of different definitions of asthma and type of prevalence (point/cumulative) studied (Tables I & II).

Cullen (1972) conducted prevalence surveys in Britain and Australia simultaneously with the same methodology and it was found that the prevalence of asthma was much higher in Australia as compared to Britain. The findings of this study disproved the notion that prevalence of respiratory diseases in general was higher in Britain because of its climate and also showed that asthma was less common in British children.

B. Importance of genetic and environmental factors

Morrison Smith (1971) compared prevalence

*Professor of Tuberculosis (Respiratory Allergy) K.G.'s Medical College, Lucknow.

**"Wander-TAI Oration" was delivered at the 39th National Conference on TB & Chest Diseases held in Cuttack in January 1985.
TABLE 1

Prevalence of Asthma in Children

<table>
<thead>
<tr>
<th>Author</th>
<th>Prevalence %</th>
<th>Author</th>
<th>Prevalence %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Britain</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Morrison Smith (1961)
                      | 1.8          | Cullen (1972)        | 6.8C         |
| Horn & Gregg (1973)  |              |                      | 5.1C         |
| U.S.A.               |              |                      |              |
| Collins (1935)       | 0.5C         | Peckham & Butler (1978) | 3.5C     |
| Broder et al (1962)  | 4.1C         | Arbeiter (W)         | 9.4C         |
| Australia            |              |                      |              |
| Patrick (1962)       | 5.4C         |                      |              |
|                      |              |                      |              |
| C=Cumulative Prevalence |            |                      |              |

TABLE 2

Prevalence of Asthma in Adults

<table>
<thead>
<tr>
<th>Author</th>
<th>Prevalence %</th>
<th>Author</th>
<th>Prevalence %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Britain</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stocks (1949)</td>
<td>0.9</td>
<td>Buir et al (1975)</td>
<td>3.4 C</td>
</tr>
<tr>
<td>Williams (1951)</td>
<td>1.7</td>
<td>Buir et al (1979)</td>
<td>6.5 C</td>
</tr>
<tr>
<td>U.S.A.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Collins (1935)</td>
<td>0.1-I C</td>
<td>Dodge &amp; Burrows (1980)</td>
<td>6.2</td>
</tr>
<tr>
<td>Broder (1962)</td>
<td>4.1 C</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

C=Cumulative Prevalence

rate of asthma and wheeze in English and immigrant school children in U.K. Prevalence among Asian and West Indies children who had been born in England was similar to that of English children. Much lower prevalence rate was found in immigrant children who had been born in their native country.

The prevalence of Asthma in Xhosa children living in African township of Capetown was determined by exercise challenge test and was 3.20% (Van Nieberk et al 1979). This is much higher than in the case of Xhosa children in Transkei using identical methods.

Cumulative prevalence of Asthma in Tokelauan children in Newzealand was found to be 25.3% (Waite et al 1980). This is greatly in excess of that found in Tokelau. These studies have shown that environmental factors are more important than genetic factors in pathogenesis of Asthma.
C. Identification of environmental factors

In 1971 the Americans in Japan suffered from asthma due to industrial chemical pollution and this epidemic of asthma is known as Tokyo Yokohama asthma (Salvaggio et al 1971). On the other hand in 1954 asthma epidemic was due to exposure of Neo Orleans to organic allergens in environment (Huber et al 1954). Prevalence rate of asthma with rhinitis found in a study in Sudan clearly demonstrates the importance of environmental agent—Green Nimmitti—as provocative factor (Kel et al, 1983). Thus, the epidemiological studies have proved that various environmental factors are responsible for precipitating attacks of asthma.

Various types of asthma caused by environmental factors may be:

1. Extrinsic asthma due to various detectable allergens present in the atmosphere.
2. Asthma due to various occupational agents present in the environment is called occupational asthma.
3. Asthma caused by food allergens is known as Alimentary Asthma.
4. Drug induced asthma is cased by injection, infusion and ingestion of drugs.
5. Asthma after exercise is known as Exercise induced Asthma.

Allergic bronchial asthma is characterised by early onset of symptoms, family history of allergic diseases and association of other allergic diseases. Patients are sometimes able to identify offending allergens and symptoms have a seasonal variation. There is no toxaemia but eosinophils are increased.

Allergens are classified as inhalants, ingestants and contactants according to their route of entry. The allergen responsible for symptoms in a particular patient can be recognised by history, skin tests (Shivpuri 1962) and bronchial provocation test (B.P.T.) (Hargreave et al 1972.)

Our experience in allergy/asthma clinic has shown that patients of asthma have a specific pattern of attacks. Patients having attacks at change of season think that symptoms are due to change in temperature, but it is not so. During change of season, e.g. Sept./Oct., and Feb./March the amount of pollens in the environment is maximum. Therefore the patient allergic to pollens gets into attack during the change of season. Ours was the first allergy clinic in the State of U.P. So patients of asthma from the hills of U.P. also reported at Lucknow. It was observed that patients from hills used to feel better as they came to plains because the environment of hills is different from that of plains. Another group of patients had attacks of asthma during winter nights. Some patients reported increase of symptoms in home and relief in outdoor environment, while others had symptoms during the day. For them occupational exposure is an important factor because patients are exposed to occupational agents during the day. So by taking careful history it is possible to find an offending allergen for a particular patient which is to be confirmed by skin test and B.P.T.

Skin is a mirror of immunological reactions of individual (Pepys et al 1975). Immediate, late and delayed reactions can be elicited in the skin by the use of proper antigenic extracts. Immediate reaction develops within minutes and resolves in one to two hours, and is mediated by skin sensitising anti-body. Tissue reaction is reversible and is characterised by wheal. Erythema response is produced by vasoactive amines released due to degranulation of mast cells. Cortisone has no effect on type one reaction. Late or Aarthus type of reaction develops slowly within five to seven hours and resolves by 26 to 36 hours. Late reactions are mediated by precipitating type of anti-body. Tissue reaction is characterised by ill defined, extensive, soft swelling produced by toxic immune complexes which is tissue damaging. Delayed reaction develops after 72 hours and is mediated by T-lymphocytes due to release of lymphokines. Tissue reaction is characterised by a damaging
and firm induration. Bronchial provocation tests arc performed by nebulizing the antigen in the bronchial tree and assessing the degree of spasm produced. Bronchial Provocation Tests are more specific than skin tests in identifying offending allergens. Dominant aero-allergens of Lucknow were reported for the first time in 1971 (Agnihotri et al 1971). In 1982 the allergenicity of pollens of various trees, grasses and weeds present in the atmosphere of Lucknow were reported (Verma 1982). 971 cases of respiratory allergy were skin tested and analysed. It was observed that Holoptelia, Prosopis, Lantana, Ricinus, Brassica, Moringa, Azadirachta Phoenix, Albizia, Bombax, Eucalyptus, Althaea, Citrus, Ehretia, Melia and Morus Pollens were important in the etiology of respiratory allergic diseases. Amongst weeds and grasses pollens of cynodon, chenopodium, cannabis, amaranthus, ageratum, penisetum, sorghum, saccharum, panicum, dichanthium, triticum, anthemis and aregmone were found to be important allergens. It was further reported that majority of these plants pollinate during change of season, i.e. March, April, September, October. Thus this study demonstrated that maximum pollens are present in atmosphere during change of season and are important aero-allergens.

Physical inspection of the houses of patients of asthma giving history of attack at home revealed growth of fungi on the damp walls. We identified th fungi in the bedrooms of asthmatics and aspergillus niger, A fumigatus and A. flavus gave higher colony count in the bed-rooms as compared to the atmosphere (Agnihotri et al 1979). In the second study of 25 patients of allergic bronchial asthma having symptoms in their homes, the etiological significance of various species of aspergillus was studied by skin test, bronchial provocation tests and precipitation tests (Sharma 1980). It was observed that extracts of Aspergillus niger and Fumigatus gave 68% clinically significant reactions whereas 90% and 85.7% bronchial provocation tests were positive in these patients. Precipitin tests revealed precipitin line in 39.1% and 52% tests done by Aspergillus niger and Aspergillus fumigatus respectively. These two studies have clearly demonstrated that the amount of fungal spores is higher in damp rooms and Aspergillus spores present in the damp rooms cause bronchial asthma. Thus pollution of indoor environment by fungal spores is a health hazard.

Lichens are composite plants having algal and fungal elements and they grow on barks of trees, rocks and ground in hills. In a study at Allergy/Asthma Clinic it was observed that the patients of various respiratory allergy coming from hills gave positive skin reactions in 27% cases, whereas 3.5% non-hilly patients of respiratory allergy gave positive reactions to Lichen extract. (Agnihotri et al 1978).

Asthma is often seen in patients using pillows filled with various type of organic material such as cotton, semal cotton clothes, furs, foam, maize husk and flowers. We know that inhalation of organic dust can cause allergic reaction in lung. This observation prompted us to study the allergenicity of semal cotton. Bombex ceiba flox becomes powdery after use and dust which comes out of the pillows is inhaled by the patients. In this study patient of respiratory allergy having symptoms during winter night were studied. It was observed that 52.2% patients demonstrated positive skin reaction to extract of semal cotton and 54.5% patients were using semal cotton filled pillow? (Agnihotri et al 1976). This study has demonstrated that patients having symptoms in winter night should avoid using pillows stuffed with cotton specially semal cotton.

House Dust is a well-recognised cause of asthma. The allergenic component of house dust is 'Mite—an' organism which grows on mattresses and thrives on human scales. Mite allergy is not so common in India because of our habit of putting bedding in the sun. Mite on the mattress cannot survive in the Indian sun. Animal allergy is not very important in India because we do not live with animals as in the west. Animal allergy is often seen in patients involved in occupations dealing with animals.

Occupational Asthma

Occupational asthma is due to specific well-defined exposure of occupational agents and these patients get relieved by avoiding exposure. Asthmatic symptoms are produced either by reversible airway obstruction due to spasm of the bronchial muscles, swelling of the mucus membrane lining the bronchi and presence of secretions in the bronchial lumen, or due to reversible parenchymal injury leading to pulmonary oedema, inflammatory alveolitis and bronchiolitis. Occupational agents cause various types of reaction in the lung (Fish 1982 Lessof et al 1981 and Harrison et al 1976).

Toxic Reactions

Gases and fumes can cause direct tissue injury. The site of tissue, reaction depends on their solubility and concentration of gases. Highly soluble gases get dissowed in the bronchial secretions easily and cannot reach parenchyma and, therefore, symptoms of tracheobronchitis are produced whereas less soluble gases and fumes reach upto the parenchyma
and produce bronchiolitis or alveolitis. Gases in low concentration increase non-specific sensitivity of the bronchial mucosa due to epithelial injury. In high concentrations fumes cause parenchymal injury.

The clinical picture of toxic reactions appears with the first exposure to all exposed persons. There will be simultaneous evidence of irritation of nasal and conjunctival mucosa. Pulmonary function tests (PFT) may be normal or abnormal. Chest X-rays show infiltrates or evidence of pulmonary edema.

B. Immediate hypersensitivity reactions

Occupational agents have immunological potential to increase the production of specific IgE, and to trigger an attack of bronchial asthma similar to that of non-occupational asthma. The etiological significance of occupational agents can be demonstrated by direct skin test, passive cutaneous anaphylaxis and by demonstration of specific IgE. The clinical picture of immediate hypersensitivity reactions are wheeze and dyspnoea, chest-tightness, with normal X-ray and abnormal PFT during the attack.

The toxic and hypersensitivity reactions to occupational agents can be differentiated on the basis of history and routine examinations as shown in the following table:

<table>
<thead>
<tr>
<th>Toxic Reactions</th>
<th>Hypersensitivity Reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms</td>
<td>First exposure</td>
</tr>
<tr>
<td></td>
<td>Subsequent exposure after a long period.</td>
</tr>
<tr>
<td></td>
<td>All are affected.</td>
</tr>
<tr>
<td></td>
<td>Only a few are affected.</td>
</tr>
<tr>
<td></td>
<td>Irritation in eyes &amp; nose.</td>
</tr>
<tr>
<td></td>
<td>Associated rhinitis and conjunctivitis.</td>
</tr>
<tr>
<td>Threshold Limit Values</td>
<td>Effective</td>
</tr>
<tr>
<td></td>
<td>Non-effective</td>
</tr>
<tr>
<td>PFT</td>
<td>Normal/abnormal</td>
</tr>
<tr>
<td></td>
<td>Abnormal</td>
</tr>
<tr>
<td>Chest X-ray</td>
<td>Patchy infiltrates.</td>
</tr>
<tr>
<td></td>
<td>Pulmonary edema.</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
</tr>
</tbody>
</table>

Same occupational agents can cause hypersensitivity reaction of ill-defined mechanism and other irritant reaction in lung. Late and delayed hypersensitivity reaction due to exposure of various avian and mammalian proteins, varieties of moulds and chemical agents is observed in Extrinsic allergic alveolitis.

Various types of dusty occupations which cause asthma in our society are Kirana shop workers, grain merchants, godown workers, rics, dal and timber mill workers. The worker is exposed to occupational agents during the day and therefore, one must consider occupational cause in patients having asthma during the day.

At the Allergy Asthma Clinic it was observed that various food articles can precipitate attack of asthma in an individual. 37.5% cases of asthma reported precipitation of attack by dal, wheat, nuts, milk, meat, fish, egg, banana, tomato and orange in 1979 (Agmhotri et al 1979). Number of factors both genetic and environmental predispose development of food allergy. Infants fed on breast milk and not given "formula" foods are less likely to develop milk allergy whether there is genetic predisposition or not (Lessof et al 1981). Gastroenteritis increases the permeability of gastrointestinal mucosa and thus macromolecules of food allergy are absorbed from intestines which are capable of producing specific anti-bodies to foods (Harrison et al 1976).

Food allergy can produce remote manifestations such as asthma, eczema or rhinitis possibly due to transmitted effect of mediators which are first liberated in gastrointestinal tract (Buisseret et al 1978). Recently we have observed that amongst our patients, milk was the most offending food. Therefore, we studied milk allergy in patients of bronchial asthma attending Allergy-Asthma Clinic. Correlation between skin test and elimination and oral challenge test revealed that in patients with positive skin reaction, elimination test were positive in 35 patients. Out of these 35 patients, 31 (88.57%) had positive oral challenge also. Thus 88.5% skin positive patients were also positive for elimination and oral challenge tests indicating that skin test by milk antigen is a good indication for diagnosis of milk allergy.
allergy in allergic patients (Arya 1983). It was further observed that chances of development of milk allergy were more in asthmatics who had milk other than the mother’s milk in the first 3 months of life, thus emphasising that breastfeeding prevents development of milk allergy.

**Drug Induced Asthma**

Drug induced bronchospasm is a common problem. It may occur as pulmonary manifestation of anaphylaxis or after inhalation, ingestion or infusion of drugs. The drugs most commonly associated with induction of acute episodes of asthma are aspirin and tartarazine. (Fadden 1984).

**Aspirin Asthma** : It affects adults; starts with perennial vasomotor rhinitis followed by hyperplastic rhino sinusitis with nasal polyp and then progressive asthma. Symptoms appear 1-3 hrs. after administration of the drug. 10% of asthmatics have aspirin induced asthma. There is cross sensitivity with other non-steroidal anti-inflammatory drugs and desensitisation to aspirin is possible. The mechanism involved in aspirin asthma is unknown.

**Exercise Induced Asthma (EIA)**

Asthma can be induced or made worse by physical exertion. EIA is a feature of childhood asthma. Asthmatic attacks begin a short while after stopping the exercise and there is decreased response to further exercise after attack of EIA. This is known as refractory period. There is significant interaction between climatic environment in which exercise is performed and magnitude of post exertional airways obstruction. Inhalation of cold air during physical exertion markedly enhances the response whereas hot humid air can blunt or abolish it. (Welliver 1983 and Anderson et al 1982) Consequently, activities such as ice hockey, ice skating are more provocative than swimming in indoor heated, pools.

The principles of management of allergic bronchial asthma include avoiding offending allergens as shown by history, skin test or bronchial provocation test. But if it is not possible, prophylactic treatment is advocated. Prophylactic treatment includes specific prophylaxis by desensitisation treatment whereas non-specific prophylaxis is carried out with drugs like disodium chromoglycate. The desensitisation treatment aims at introducing a concentration of allergenic extract which the patient can tolerate. It produces a level of resistance which can cope with general concentration of allergens around the patients. This aim is achieved by production of blocking antibodies which interpose themselves between the allergen and the mast cell. It has also been shown that by the process of desensitisation, IgA anti-bodies are produced which protect the mucus membrane.

**Results of Immunotherapy**

The results of immunotherapy of 468 patients followed for a period of 2 years were reported in 1984 (Mishra, 1984). It was observed that about 60% of patients who continued immunotherapy for 6 months showed marked improvement and after 1 year 87% of them showed improvement. The percentage of improvement increased with passage of time. The results of immunotherapy depend upon proper selection of patients and allergens used in immunotherapy. Out of the various groups of allergens which can cause Respiratory allergy, immunotherapy is proved to be effective in pollen allergy, because standardised extracts of pollen are available and the active antigens have been recognised. In our country, pollens are very important aero-allergens and are present throughout the year. Therefore, immunotherapy has definite role in management of bronchial asthma in India. Immunotherapy is a prolonged treatment involving repeated injections. Immunotherapy is indicated only when the allergen is not avoidable and in severe asthma which requires intensive medication. Immunotherapy should always be used along with other medications.

In conclusion, most of the activities of man today are indoors, either during working or living. With urbanisation almost all the activities of mankind are indoors. Most of the hours of the day and most of the days of the years are spent in the same room of the same building in which he or she lives or works. Exposure to indoor air pollutants is prolonged and repeated. Indoor pollutants remain more concentrated as they have less chance of getting diluted as compared to outdoor air pollutants. These include virus and bacteria. All of us know that tuberculosis is more common amongst household contacts because of the presence of M. tuberculosis in indoor atmosphere. If a child suffers from measles, the other children living in same household have a possibility of suffering from the same illness because of indoor environmental pollution. Allergens like pollen, spores, dust, mites, insects, fumes and animal danders cause respiratory diseases in sensitive individuals. Combustion products and smoke produce indoor pollution. Consumer products such as paints, insecticides etc. can affect the inhabitants of that particular house. Thus, it is clear that so many things present in indoor environment can cause asthma. Out-door environmental factors of asthma can be checked by legislation on industries, but for indoor pollution, community is to be educated and it
is our duty. Every allergologist has to face the question—’Dr. I am allergic to what?’ It is only the avoidable allergens that should be told to the patients and not the unavoidable one.

We must know about our environment not only for the sake of knowledge but also for healthy living, because environmental pollution causes asthma.

REFERENCES


Broder, I. Berlow, P.P. and Horton, R.J.M: The epidemiology of asthma and hay fever in a total community, Tecumseh, Michigan, F. Allergy, 1962, 33, 513-23.


Fish, E, James: Occupational Asthma —A spectrum of Acute Respiratory Disorders, J. Occupational Medicine, 1982, 24, 397.


Horn, M.F.C and Gregg, I.: The role of viral infection and host factors in asthma and chronic bronchitis. Chest, 1973, 32 (Suppl. 4) 44-8.

Huber T.E., Joseph S.W., Knoblock E. et al A.M.A. Arch. Ind. Hyg. 1954, 10, 399.

Indian Council of Medical Research, New Delhi; Morbidity survey of contributory health scheme beneficiaries; 1961.

Kay, A.B., Maclean, C.M.U., Walkinson, A.H. and Gad Rab, M.O.: The prevalence of asthma and rhinitis in a Sudanese community seasonally exposed to a potent airborne allergen (the green nimitti midge, cladotanytarsus lewisi) 1983.


