the problem of what it is that is being modified when we give our patients steroids. In addition to this immediate reaction, we get a later one, coming on after several hours and which we believe to be immune complex mediated—a type III sort of reaction. This reaction is very well inhibited by corticosteroids and I have a strong feeling that when our patients are benefited by corticosteroids, what we are in fact modifying is this immune complex reaction. Now, if this is so, why are they no longer reacting in an immediate sense? This is a problem which we now come to appreciate and face. Such findings make it necessary to reassess our views on the immunologic mechanisms underlying asthma.

Dr. Farr: I think we certainly have to incorporate the heterogeneity among asthmatics into our way of thinking and use the term reversible obstructive airway disease, and to try to put an etiologic diagnosis on it—reagin mediated, looked for, not found or found—or type III disease, looked for, found or not found—that infection is playing an important role, frequent or infrequent. I would really like to make this pitch. We are doing this on our charts now and it is very comfortable. People picking up the charts can tell what we are thinking; we may be wrong, but they can at least tell what we are thinking.

Dr. Schwartz: I certainly have no particular quarrel with the comments. I would just like to re-emphasize that when we looked at the patients, both groups contained what we would have referred to as intrinsic asthmatics, as well as extrinsic asthmatics. The role of infection, cigarette smoking and so on seemed by our analysis to be prevalent in both groups to a similar degree. The groups were not separable by clinical criteria. In other words, we did not challenge them by aerosol, did not study them with regard to an early and late pulmonary response, which I suppose would have been the final way to settle this question.

Dr. Nadel: Concerning the autonomic regulation, if we leave the alpha adrenergic system out, we know that the vagus nerves innervate the lungs; when we stimulate the vagus nerves we get contraction of the muscle. If we then stimulate the sympathetic nerves to the airways, we can partially inhibit that contraction. So, we have a potent cholinergic system contracting the muscle and a sympathetic system that inhibits contraction. When the nerves are intact some tone normally exists in the airways. Now under that circumstance, if you give atropine, the airways will dilate. However, since these airways now have little or no tone, one would expect no further effect when the sympathetic nerves are stimulated. If propranolol is administered when the system has a great deal of cholinergic tone, any sympathetic dilator tone that is present will be abolished and further bronchoconstriction will occur. The studies of propranolol suggest that beta blockade unmasks cholinergic activity (bronchospasm) that can be abolished by cholinergic blockade (atropine). Alpha adrenergic receptors need not be implicated to explain the findings!

SESSION III: EPIDEMIOLOGY AND ADVERSE AIRWAY RESPONSES

New Orleans Epidemic Asthma: Semiquantitative Aerometric Sampling, Epidemiologic and Immunologic Studies*

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Periodic outbreaks of reversible obstructive pulmonary disease have been noted in the city of New Orleans for years, but a plethora of studies have incriminated no point source industrial pollutant. The episodes are primarily nocturnal and often involve more than 100 “asthma” admissions to the Charity Hospital emergency room in a single 24-hour period. We demonstrated that atopic individuals with broad patterns of type 1 skin reactivity to a battery of common inhalant allergens were involved in “epidemics.” Individuals involved in epidemics did not differ quantitatively from nonin-
volved asthmatics in degree of bronchial sensitivity to mecholyl and histamine. Outbreaks were seasonal in nature, occurring in greater frequency and magnitude during the late summer and fall months.

A two-year study of the relationship between climatologic variables, hospital asthma emergency room admission rates and semiquantitative particulate pollen, and fungal spore counts was performed to establish interrelationships between these variables. Spore and pollen aerometric sampling was performed with automatic intermittent rotoslide samplers which periodically rotated x 1 minute at 1650 rpm every 12 minutes. Suspended particulates (plus 1 percent sulfates and benzene soluble organics) were quantitated using a standard high-volume air sampler and total dustfall collected in plastic buckets. In an attempt to simplify spore groupings for estimations of seasonal fluctuations they were grouped into three general categories: 1) small round to oblong spores 1-6 x 1-12μ (primarily small deuteromycete spores, basidiospores and likely ascospores); 2) large spores easily recognizable as deuteromycetes (fungi imperfecti) approximately 10-130μ; 3) spores resembling large basidiospores and myxomycete spores. Airborne pollen collected during “epidemic” seasons was almost exclusively of the ragweed tribe (Compositae-Ambrosiaceae).

Analysis of these findings established the following: a) high summer asthma admission rates were significantly associated with high “total” spore and pollen particulate counts, “basidiospore-like” counts and “small spore” counts, plus hot stagnant weather conditions characterized by high mean temperature, and low resultant wind velocity; b) peak ragweed (Ambrosia) season fall asthma admissions (September-October) were significantly associated with high mean ragweed counts, but large asthma “epidemics” were not necessarily related to sudden influxes of ragweed pollen. The association of low relative humidity, wind velocity and temperature with high daily asthma admission rates at this time plus the high total spore and pollen counts suggested a synergistic effect between ragweed pollen, airborne spores and climatologic variables; c) “post-ragweed season”—these “epidemics” were of considerable magnitude and occurred regularly in late October, November, and early December. They were significantly associated with sharp decreases in relative humidity, temperature, and wind velocity, and with rising barometric pressure, (stable, dry, climatologic conditions similar to those reported during low-level thermal inversions). “Total” spore and pollen counts and “basidiospore-like” counts were significantly elevated during these epidemics together with heavy deposits of amorphous, chitinaceous debris, which could have represented plant debris, arthropod remnants or hyphal fragments, and could not be accurately quantitated.

Little is known about the role of serum precipitins in pathogenesis of chronic obstructive airway disease. The technique of counterimmunoelectrophoresis (CIE) was used to detect precipitins in groups of “epidemic” asthmatics, nonatopic controls, atopic subjects who had not received immunotherapy, and atopics who had received allergenic extract immunotherapy. CIE was performed on 4” x 3½” Kodak slides coated with 15.0 ml ionagar No. 2 in veronal buffer pH 8.3 with 5.4 v/linear cm of agar x 60 minutes. Antigen concentrations were 20 mg/ml (7 μl quantities in 2.0 mm wells). Antigens employed included several commercial dusts, human dander, human dandruff, Candida, Alternaria, and Fusarium sp, bagasse, the Thermophilic actinomycetes, T sacchari, and M faeni and giant ragweed. All had a high net negative charge (anodal mobility) necessary for the procedure. IgG precipitins against human dander, house dust, selected organic dusts, fungal, thermophilic actinomyces, and pollen antigens were detected with equal frequency in epidemic asthma patients, non-epidemic asthmatics and “normal” nonatopic individuals. The highest incidence of precipitins was against organic dust, commercial house dust, human dander, human dandruff, and bagasse organic dust preparations and the lowest incidence against giant ragweed. Precipitins were specifically absorbable, and present in IgG fractions on DEAE cellulose and Sephadex G-200 column chromatography.

Our overall findings to date suggest that in New Orleans, epidemic asthma results from sensitization of the local atopic population by diverse “natural” particulate inhalants acting in seasonal patterns as commonly noted in such individuals. Ambrosia pollen, small deuteromycete and ascomycete spores, basidiospores and possibly common antigens in plant remnants constitute likely offending particulates. Certain climatologic conditions appear necessary for precipitation of epidemics, especially large fall outbreaks, and these factors may be directly responsible for increased atmospheric particulate matter concentrations.

Serum precipitins as detected by CIE against house dust, organic dust, human dander, and some fungal antigens appear to be present in the population at large and likely merely reflect continuous environmental exposure to these inhalants. If our findings are correct, New Orleans epidemic asthma may merely reflect the natural history of bronchial asthma as it affects the atopic population of a large metropolitan community.