CONFERENCES AND MEETINGS

Thoracic Medicine

[FROM A SPECIAL CORRESPONDENT]

A conference on various aspects of thoracic medicine was held at the Royal College of Physicians of London on 15 and 16 June. At the first session—under the chairmanship of Professor R. R. A. COOMBS, F.R.S. (University of Cambridge), and devoted to immunology—Professor J. G. SCADDING (Institute of Diseases of the Chest, London) reviewed the conditions in which antigen-antibody reactions were concerned. Classifying the four known types of hypersensitivity reaction, he said that only two of these were commonly associated with bronchopulmonary disease. Type I, the anaphylactic type with immediate weal and flare reactions, was a familial tendency seen in such conditions as hay-fever, perennial vasomotor rhinitis, and infantile eczema. Type III reactions were of the delayed, Arthus type present in farmer's lung. The other forms of hypersensitivity—the cytolytic or cytotoxic Type II—seen in haemolytic anaemia—and the tuberculin Type IV were of less importance in chest disease.

Professor Scadding went on to describe some of the features of aspergillosis—in which allergy to aspergillus infection caused asthma—and farmer's lung, which was an alveolitis due to inhalation of the spores of thermophilic actinomyces released from mouldy hay. There were similarities between the changes seen in the extrinsic asthma of aspergillosis and the alveolitis of farmer's lung which suggested a parallel with intrinsic asthma and cryptogenetic fibrosing alveolitis, and provided a line for future research, since some as yet unknown type of reaction might underlie the latter conditions.

Inhaled Organic Antigens

Concentrating on hypersensitivity to inhaled organic antigens, Dr. J. PEPYS (Brompton Hospital, London) said that Type I hypersensitivity reactions took place in atopic persons, who made up about 10% of the population. They had a constitutional predisposition to sensitization by ordinary, everyday substances, to which they produced reactions of rapid onset and short duration. Delayed hypersensitivity reactions of Type III were seen mainly among non-atopic individuals sensitized by intensive exposure. These reactions were slower in onset, more tissue-damaging, and more prolonged than those of Type I. Inhalation tests had shown that extrinsic asthma could be of either type alone, or mixed. Allergic alveolitis was of Type III and included a number of conditions resembling farmer's lung. These were all caused by fine organic dusts with particles small enough (about 1 μ diameter) to lodge in the alveoli—and included bagassosis, mushroom picker's lung, wheat weevil disease, bird fancier's lung, pituitary snuff-taker's lung, and suberosis caused by inhalation of cork or oak bark dust.

Dr. Pepys concluded by emphasizing that treatment for allergic conditions should always be directed to the patient as a whole and not confined to the immunological findings.

Describing recent work on autoallergic aspects of respiratory disease, Dr. MARGARET TURNER-WARWICK (Elizabeth Garrett Anderson Hospital, London) outlined studies which had demonstrated pulmonary autoimmune reactions of several distinct types. There was, as yet, no direct evidence of a causal connexion between autoantibodies and pulmonary disease.

Surveying the part played by immune reactions in coal-workers' pneumoconiosis, Dr. J. C. WAGNER (Llandough Hospital, Penarth) said that 1% of all coal miners still developed pneumoconiosis, while in Wales the figure was 4%. Evidence for an immunological factor in the aetiology was provided by the presence of a raised titre of rheumatoid factor in many patients with complicated pneumoconiosis. Thus the rheumatoid factor was found in the serum of only 7 out of 35 patients with simple pneumoconiosis, compared with 8 out of 10 with the Caplan type and 10 out of 15 with progressive massive fibrosis and vasculitis; there was often no joint disease in the patients with a raised titre. In the discussion Professor Coombs pointed out that nobody yet knew whether the rheumatoid factor played any part in the pathogenesis of rheumatoid arthritis.

Immunity-deficiency Syndromes

Dr. J. F. SOOTHILL (Hospital for Sick Children, London) discussed immunity-deficiency syndromes, which included functional and quantitative deficiencies in both humoral and cellular immunity. Thus circulating antibodies could be immunologically deficient, though present in normal quantities, or reduced in quantity—as in hypogammaglobulinaemia. Similarly, cellular immunity was deficient functionally in sarcoidosis and quantitatively in lymphopenia. Cellular immunity tended to be neglected because—compared with antibodies—it was hard to measure. Primary antibody deficiency occurred in several familial forms and in the "acquired congenital" hypogammaglobulinaemia of congenital rubella, while secondary deficiencies ranged from physiological low levels in infancy and childhood, to myelomatosis and toxic effects of drugs or neoplasia. As a method of clinical testing, electrophoresis was being superseded by direct immunological studies.

Surgical and medical aspects of scoliosis were the subject of the first morning session, at which Sir HERBERT SEDDON (Royal National Orthopaedic Hospital, London) took the chair. Mr. C. W. S. F. MANNING (Royal National Orthopaedic Hospital) classified scoliosis as postural—which tended to straighten on bending forward—and structural—in which there was fixed rotation and little alteration on bending. Progressive scoliosis was of the structural type and became more severe during growth, especially in early adolescence. Though vertebral, neuromuscular, and thoracic causes accounted for 44% of cases of structural scoliosis, the aetiology was unknown in 56% of 2,000 patients included in a survey, and over two-thirds of these "idiopathic" cases had been adolescents of 11 or over. Treatment consisted of support in a plaster cast or Milwaukee brace, designed to extend the spine without restricting the chest, or operation for spinal fusion.

Effects of Scoliosis

Turning to the medical effects of scoliosis, Dr. P. A. ZORAB (Brompton Hospital) said that breathlessness was associated with peripheral lung deficiency in addition to mechanical causes. Reduction in pulmonary vascular marking noted radiographically was most severe in congenital cases, and post-mortem pulmonary angiograms had demonstrated a definite reduction in peripheral vascularity. There were wide variations in exercise potential. Severe scoliosis could be compatible with full exertion, while mild scoliosis might be associated with severe dyspnoea and disability. On the other hand, pregnant patients generally had normal deliveries even if respiration was impaired.

Continuing, Dr. Zorab said that scoliosis did not cause heart murmurs and had little effect on the electrocardiograph, since the heart's electrical axis was not much changed. Cardiorespiratory failure in middle-aged scoliosis was best treated in much the same way as emphysema—with bronchodilators, antibiotics, and drugs which acted on the heart.

Virus Infections

At the second morning session the chairman, Sir CHRISTOPHER ANDREWES, F.R.S., introduced Dr. D. A. J. TYRRELL (Common Cold Research Unit, Salisbury), who outlined the types of virus known to cause respiratory infection in man. Many of these were very selective in their choice of host cells, and their study had been simplified by the introduction of respiratory epithelium tissue cultures obtained from man or apes. Dr. Tyrrell went on to classify respiratory viruses. The myxovirus group included the viruses of influenza, causing mainly influenza, bronchitis, and bronchopneumonia; parainfluenza 1 and 2, causing bronchiolitis; and parainfluenza 3 and the respiratory syncytial virus, which were both responsible for infections at each end of the age spectrum—bronchiolitis and bronchopneumonia—and also for colds.

Among other types, the rhinovirus caused upper respiratory tract infections, and the Coxsackie and adenoviruses mainly sore throats or feverish colds. By contrast, croup, tracheitis, and sore throats were often due to streptococcal infection. Respiratory viruses could live independently for short periods only and spread most commonly by coughing and sneezing, but hardly at all during speech or normal breathing.

Dr. N. C. OSWALD (St. Bartholomew's Hospital, London), describing virus infection from the clinician's standpoint, regretted that clinical virology was virtually non-existent. Delays in obtaining a virological diagnosis by culture or immunological methods often meant that the treatment was settled and the patient out of hospital before a result was received from the virus laboratory. Electron microscopy, which could give very rapid results, was useful only for identification of the smallpox virus and the assignment of other viruses to particular groups.

Continuing, Dr. Oswald said that single complement-fixation tests had been performed on 100 consecutive patients admitted to the Brompton Hospital to try to obtain earlier diagnosis of virus infections, but this was only a beginning. More bridges would be needed between hospitals and the laboratory strongholds of the virologists if clinicians were to make intelligent use of antiviral agents when these became available.

Sir CHRISTOPHER ANDREWES concluded the session by questioning whether sneezes were necessarily the only means by which respiratory viruses spread. Simultaneous, widespread outbreaks suggested the likelihood of long-term virus latency.

Surface-active Agents

The afternoon session—under the chairmanship of Dr. K. ROBSON (St. George's Hospital, London)—was devoted to surface-active agents in the lungs, and Dr. R. E. PATTLE (Chemical Defence Experimental Establishment, Porton) described some of the properties of normal alveolar lining fluid, which had a very low surface tension. He demonstrated that lungs in which the surface tension was increased by damage to the alveolar lining collapsed into an airless, liver-like state when deflated.

The importance of low alveolar surface tension in the newborn was emphasized by Dr. L. B. STRANG (University College Hospital, London). Normal babies needed a pressure of about 18 cm. water for initial inflation of the lungs, followed by much lower pressures for subsequent breaths, whereas the lungs of immature infants required repeated high inflation pressures for each successive breath—because the alveolar surface tension was high and the lungs became airless on expiration. The ability to retain alveolar air and clear alveolar fluid via the lymphatics after birth depended on low surface tension, which probably developed in the human foetus at about 32 weeks.

Describing conditions in which alveolar surface tension was raised, Dr. C. M. OGILVIE (Liverpool Royal Infirmary) said that these were of two types: firstly, those in which the mean alveolar size was restricted by shallow respiration of any cause, pulmonary congestion, asthma, or emphysema ; and, secondly, those in which the lining film was abnormal, such as hyaline membrane disease, pulmonary oedema, postoperative atelectasis, respirator syndrome, pulmonary artery occlusion, bilateral vagotomy, and following oxygen or detergent inhalations.

Professor C. H. STUART-HARRIS (United Sheffield Hospitals) was in the chair at the second afternoon session and introduced Dr. LYNNE REID (Brompton Hospital), who emphasized that hypersecretion from the bronchial tree was the fundamental disability in chronic bronchitis. Studies in the rat had shown that it was possible by inhalation of sulphur dioxide under sterile conditions to produce progressive changes closely resembling those of chronic bronchitis, but in which infection played no part.

Further, Dr. Reid had found it possible to compare in the rat carcinogenic with the chronic-bronchitis-inducing effects of inhalation of sulphur dioxide, cigar, or cigarette smoke. All three agents increased the flow of mucus, while cigarette smoke induced numbers of mitoses many times greater than those following inhalation of cigar smoke or sulphur dioxide.

Studies on Chronic Bronchitis

Describing the results obtained so far in prospective studies of chronic bronchitis, Dr. C. M. FLETCHER (Royal Postgraduate Medical School, London) said that there was now considerable doubt whether infection governed the course of chronic bronchitis, except in the later stages. The most highly significant factors found were the initial level of the forced expiratory volume (F.E.V.)—which was related to the rate of decline—and cigarette smoking. No significant relationship had been found between the course of chronic bronchitis and such factors as episodes of infection, purulence of sputum, or sputum volume.

Though these studies had shown what appeared to be susceptible groups—with low initial F.E.V.—they had left us almost wholly ignorant of the pathogenesis of chronic obstructive bronchitis.

Dr. Fletcher also suggested that in future the histamine-inhalation test might help to distinguish asthmatic from bronchitic patients.

Advances in assisted respiration were the subject of the final session under the chairmanship of Professor Sir MAX ROSENHEIM, P.R.C.P. (University College Hospital). The use of controlled oxygen supplements was described by Professor A. CRAMPTON-SMITH (Radcliffe Infirmary, Oxford) with special reference to intermittent positive-pressure respiration via endotracheal or tracheostomy tubes in the treatment of 52 patients with obstructive lung disease. A small group of patients in status asthmaticus had responded well without tracheostomy and with no deaths ; twenty-four hours of intermittent positive-pressure respiration was usually sufficient if treatment was started early enough.

Among patients with exacerbations of bronchitis, tracheostomy had been performed at twenty-four hours if continued positive-pressure respiration was necessary. There were twelve deaths—all in this group. Dr. J. M. K. SPALDING (Radcliffe Infirmary, Oxford) concluded the meeting by describing the changes in circulation, renal function, and acid-base balance resulting from increased intrathoracic pressure.

TOMORROW'S BUILDINGS

New Teaching Hospital at Oxford

At a press conference in Oxford on 14 June details were announced of the new regional hospital planned for Oxford. The hospital will be built on a 69-acre site south-east of the city centre, part of which is occupied by the Osler and Sunnyside Hospitals. It is intended to serve two main functions—firstly, that of a community hospital, and, secondly, that of a major centre for medical research providing comprehensive teaching facilities at both undergraduate and postgraduate level.

Building on Phase I of the hospital is planned to start early next year. This will comprise a 167-bed maternity unit, the Nuffield Institute (a research laboratory), and residential accommodation for 147 staff. The maternity unit will be on seven floors, with an acute floor (including admission and delivery suites ; operating-theatres ; special nursing, medical care, and baby care units ; and the University Department of Neonatal Medicine) at ground level. The third floor will house the University Department of Obstetrics, while on the fourth, fifth, and sixth floors there will be wards containing a total of 141 beds. The seventh floor will house a 10-bed isolation unit with its delivery room together with a 10-bed private patients' unit.

It is hoped that building on the next stage of the development—Phase 2A—will start immediately after Phase I has been completed, possibly in 1971. This will contain a new accident and emergency department, beds for 450 patients, a new children's ward, and further residential accommodation. The University Grants Committee has approved the provision of a large research building to support the various clinical departments. In addition, there will also be a new headquarters for the undergraduate medical school with a central medical library, lecture theatres and seminar rooms, and a demonstration hall. In this way it is intended to create an academic centre open to students, teachers, general practitioners, and local-health-authority staff alike.

The consultant architects for the new hospital are Yorke Rosenberg and Mardall.