Chest diseases in Australia

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When the *Endeavour* under the command of Captain James Cook set sail from Plymouth on 25 August 1768, no one could have foreseen the momentous long-term results of this voyage of discovery (Moorehead, 1966). After spending 6 months charting the coastline of the two islands of New Zealand, Cook decided to strike westward until reaching the eastern seaboard of the country of New Holland, only the west coast of which was known in rough outline at the time, the east coast being then entirely unexplored. On 19 April 1770, after 18 days at sea, Lieutenant Zachary Hicks first sighted land which later proved to be the extreme south-eastern corner of the continent. Nine days later after sailing up the east coast, they entered a harbour later to be called Botany Bay, and claimed the land in the name of King George III.

It is thought that no chronic pulmonary diseases afflicted the Australian aboriginal before the arrival of Captain Cook. It is indeed likely that because of their nomadic habits, the smallness and sparsity of their communities, and the generally equable climate of Terra Australis, diseases of the lungs were a rarity amongst the indigenous population. The arrival of the white man soon put an end to this and it is of interest to note that the first European buried in Australia, one of Cook's seamen, died of consumption. Cook in his private log wrote—'Tuesday, May 1st. (1770) . . . Last night departed this life Forby Sutherland, Seaman, who died of a consumption (*sic*) and in the a.m. his body was entard (*sic*) ashore at the watering place. This circumstance occasioned my calling the south point of this bay Sutherland's Point' (Cleland, 1968). And so literally, the tubercle bacillus was implanted in Australian soil. Indeed until after World War II the study of diseases of the chest virtually centred around tuberculosis and physicians who specialized in 'chest diseases' were really phthisiologists. It is therefore appropriate that in this review tuberculosis should be in vanguard.

Tuberculosis

In the eighteenth century tuberculosis was a common disease in Britain. According to Brownlee

(Goldberg, 1946) consumption was responsible for 13% of all deaths in London in 1715, rising steadily until 1801 when it caused 30% of all mortality. It is therefore not surprising that the first Englishman to die in New Holland did so from tuberculosis.

In the early part of the nineteenth century medical literature was much concerned with the effect of climate on disease and in 1829 Sir James Clark laid down the principles upon which to base the climatic treatment of consumption; and so it was that the long sea voyage to Australia was frequently recommended as a form of treatment for this disease. Consequently many migrants assayed this treatment and an excellent account of such a trip aboard the *Sobraon* was given by Francis Workman in 1886 (Thomas & Gandevia, 1959). Of the 124 passengers over 11% died from pulmonary tuberculosis within 9 months of embarkation and it is recorded that on an earlier voyage by the same ship in 1879, 10% of the passengers were 'very far gone in consumption, many later dying of the disease'.

In 1875, the first year for which there are reliable statistics, the annual death rate from tuberculosis in the colony of New South Wales was 154 per 100,000 and after reaching a peak of 160 in 1882 (Holmes, 1937) the mortality steadily declined except for one or two temporary upward tendencies, to its present low level of 2 per 100,000. This satisfactory state of affairs can be attributed to a number of factors, but a great deal of the credit for the improvement in recent times must be given to Sir Harry Wunderley who was the first Commonwealth Director of Tuberculosis at the time when the Commonwealth Government passed the Tuberculosis Act of 1948. This Act provided for a case-finding programme based on routine chest X-ray examinations, compulsory notification and the maintenance of a case register, special allowance for sufferers from the disease and their dependents where necessary, and means for dealing with recalcitrant patients. Although the Act came into operation in July 1948, momentum was not gained for several years and the annual number of notifications of tuberculosis rose to a

peak of just under 5000 in 1953. Since then there has been a steady decline in both the incidence and mortality of tuberculosis, to the present level of 18 per 100,000 and 2 per 100,000 respectively. This is particularly creditable in view of the fact that since the end of World War II Australia has accepted over a million migrants from Europe and it has been found that the incidence of tuberculosis amongst this group is twice that of the indigenous population. To achieve these results the Commonwealth Government has expended approximately 200 million dollars in the last 20 years.

In recent years the problem of infection with atypical mycobacteria has received considerable attention in Queensland and Western Australia, the two states where this form of pulmonary disease has been most frequently found. Much of the work on the subject has been done by Ellis Abrahams, Director of Tuberculosis of Queensland, at whose instigation a unit devoted entirely to research into atypical mycobacteria has just been established in Brisbane. In 1958 in the Cairns area which lies in the tropical zone of Queensland, reactors to the tuberculin test in the 16 age group gave a remarkably high positive rate of 87.4%; contrary to expectation this was associated with a low incidence of clinical tuberculosis in the area. Abrahams & Silverstone (1961) found a remarkable pattern of tuberculin reaction in eastern Australia. In 1959 the percentages of positive reactors in the eastern capitals amongst children of school leaving age were as follows: Hobart 2.5, Melbourne 6.5, Sydney 7.6 and Brisbane 21.1. Investigation by the Queensland Institute of Medical Research showed that atypical mycobacteria could be recovered readily from many sites notably children's tonsils, swimming pools and beaches, soil and water, house dust and domestic animals. Many of these species appeared to be the same as those recovered from the secretions of the lungs of patients. In 1965 Abrahams reported the isolation of atypical mycobacteria from 129 patients, the majority of whom were over the age of 50. Of these forty-eight had significant pulmonary disease apparently due to mycobacteria, forty-seven had some other lung disease and in thirty-four the isolation was thought to be without significance. In Western Australia 460 patients were found to excrete atypical mycobacteria between 1959 and 1963 but only seventy-eight of these satisfied strict criteria for the diagnosis of 'pseudo-tuberculosis'. As in Queensland the large majority of the organisms belonged to the Group 3 (Battey) type. It is hoped that the Research Unit in Brisbane will shed more light on this troublesome form of mycobacterial infection which is resistant to the standard anti-tuberculous drugs though fortunately susceptible to ethambutol and capreomycin.

Hydatid disease

Until recent years it has been stated with considerable justification that 'Australia rides to prosperity on the sheep's back'. Although our dependence on wool has diminished in the last decade it is still a very important factor in Australia's economy and hydatid disease in humans has been inseparable from the wool industry. Consequently its highest incidence is in New South Wales, Victoria and Tasmania. Following on the work of S. D. Bird, J. Davies Thomas and James Graham, Sir Harold Dew (1928) became the foremost Australian authority on the disease and obtained worldwide recognition for his contributions to this field. Despite the fact that the life cycle of *Tinea echinococcus* has been known for many years and methods of preventing the disease well established, there has been no reduction in its incidence in the sheep-growing districts of Australia. Dew reviewed the decade 1941-50 and found an incidence of hydatid disease on 1.3 per 100,000 population. Howkins (1966) conducted a similar review for the decade 1954-63 and found an incidence of 1.22 per 100,000. In the southern tablelands of New South Wales, surveys of infestation rates in sheep and dogs conducted in 1926 and again in 1958, showed that one third of the animals were infested and there had been no reduction in the infestation rate in the period of 30 years.

Carcinoma of the lung

This disease has achieved almost epidemic proportions in Australia as it has in Great Britain, U.S.A. and other countries where cigarette smoking is a common habit. In incidence Australia lies between Great Britain and U.S.A. and shows the same rapid rise in mortality rate. Whereas in the decade 1931-40 the average annual death rate from bronchial carcinoma was 35 per million, this had risen to 280 per million in 1968, the disease now being the commonest form of carcinoma in the male. Alastair Campbell (1961) drawing upon the resources of the Repatriation Department, carried out an extensive investigation of the relationship between pulmonary tuberculosis and lung cancer and concluded that the incidence of the latter was twice as great as in a comparable group who had not suffered from pulmonary tuberculosis.

Here as in Great Britain and U.S.A. it is generally considered there is a causal relationship between cigarette smoking and lung cancer and an organization known as the Australian Council on Smoking and Health was established in 1965 under the chairmanship of Dr Cotter Harvey, to bring to the public an increasing awareness of the dangers of smoking and to initiate action against this public health hazard.

Occupational lung disease

Although no nineteenth century description of lung diseases in miners has yet been located, subsequent Australian work in this field has been of considerable significance both locally and internationally. After local practitioners in the lay press had expressed disquiet at the prevalence of lung disease in Bendigo goldminers in 1903, Walter Summons was appointed to conduct a special investigation of the problem (Summons, 1907). His investigations included a review of mortality data, clinical examination of cases including working miners and those invalided, sputum examinations for tubercle bacilli, examination of autopsy material and chemical and physical analyses of lungs and of mine air. He concluded that silicosis was a specific disease solely attributable to dust and that while associated chronic bronchitis would produce disability, death was usually due to tuberculosis. He also analysed the problems of mine ventilation and produced a separate paper incorporating recommendations on this subject. In 1927 Robertson carried out a better survey epidemiologically in that over 90% of the underground workers were examined but the survey was less comprehensive from other points of view. Chest radiographs were not taken and the obvious arrest in the rising death rate amongst miners as a result of action on Summons's report was not given due emphasis. This survey revealed that 65% of contacts of advanced tuberculosis cases showed positive tuberculin reactions contrasted with 21% with no known history of exposure. Other important investigations of goldminers were those of J. H. L. Cumpston in 1911 in Western Australia, which included some animal experiments and concluded that the low rate of tuberculous complications was due to a relatively low overall prevalence of infectious tuberculosis in the community, and that of Nelson in 1926 which revealed a critical epidemiological approach in advance of its time and included spirometric and radiographic studies.

A high prevalence of pneumonia in Broken Hill attracted attention in 1912 and led to a Royal Commission in 1914 which without radiographic aid, concluded that pneumoconiosis not tuberculosis in this area was a rarity (Cumpston, 1968).

Between 1919 and 1922 comprehensive surveys systematically employing chest radiographs for the first time in Australia in occupational investigations, established the presence of pneumoconiosis in Broken Hill. W. E. George who played a prominent role in the organization of the Bureau of Medical Inspection at Broken Hill, subsequently joined the Joint Coal Board and did valuable work on coalworkers' pneumoconiosis (George, 1953). The Sydney investigator who achieved an international reputation on this subject was Charles Badham, a

grandson of the Charles Badham who had first described chronic bronchitis a little over a century before. Badham demonstrated that coalworkers' pneumoconiosis could be attributed specifically to coaldust and differed from silicosis, a view not generally accepted in the 1930s. Less well known is his view that the associated disabilities were due to chronic bronchitis and emphysema. R. K. Outhred and his colleagues of the Joint Coal Board have recently produced further evidence to support this contention. Badham also did notable work on the silicosis associated with metalliferous mining, quarrying and sandstone tunnelling. Much of his work was summarized in a rare monograph published in 1938 (Badham & Taylor, 1938).

If the present extremely low prevalence of coalworkers' pneumoconiosis in Australia is due in large measure to geological and engineering factors supplemented by satisfactory hygiene in dust control measures, the effective documentation of the change in prevalence and of related medical data in a form suitable for research, is attributable to the foresight of George & Outhred (Joint Coal Board, 1962).

Although Badham investigated the health of textile workers in 1923 and Robertson surveyed men in the woodworking industries in 1927, comparatively little attention was paid to non-pneumoconiotic disorders of the lungs and bronchi. In recent years Gandevia, first in Melbourne and later in Sydney, and his collaborators at the University of New South Wales, have taken particular interest in bronchial syndromes resulting from a variety of inhalants especially those encountered in chemical (Gandevia, 1963) and textile (Gandevia & Milne, 1965a) industries.

In recent years Australian physicians have become increasingly aware of asbestos as an industrial hazard and cases of asbestosis and mesothelioma of the pleura in asbestos workers have been reported from several states (Joseph, 1960; McNulty, 1962; Elder, 1967; Mortimer & Campbell, 1968).

Pulmonary pathology

Australians have made some notable contributions to the study of lung pathology, but in this review it is only possible to mention a few. R. Webster (1939) when Pathologist to the Children's Hospital, Melbourne, wrote a series of papers on the pathogenesis of tuberculosis and was largely responsible for correcting the previously held belief that the tonsils were the common site of entry of the tubercle bacillus into the body and of the first lesion in alimentary tuberculosis. Lynne Reid, a graduate of Melbourne University, began her long series of contributions to the fundamental understanding of pulmonary pathology with an article in *Thorax* (Reid, 1950) on the nature of bronchiectasis in which she showed that the

saccules arose from the relatively proximal generations and that their subpleural position was due to destruction of the bronchial tree and lung tissue distal to them. This was followed by studies in conjunction with J. Hayward (Hayward & Reid, 1952) on the cartilage of the intrapulmonary bronchi in normal lungs, in bronchiectasis and in massive collapse. She continued her studies at the Institute of Diseases of the Chest at the Brompton Hospital where she has made valuable contributions to the understanding of the pathology of chronic bronchitis and emphysema, culminating in a book on the Pathology of Emphysema (Reid, 1967).

Important papers on the pathogenesis of emphysema were published in 1956 and 1957 by K. H. McLean (1956, 1957a, b, c) in the *Australasian Annals of Medicine*, in which, by detailed meticulous work, he concluded that from the morphological viewpoint nonspecific bronchiolitis is the basic lesion of emphysema and that viral infection in the presence of accessory factors, e.g. prolonged exposure to inhaled chemical irritants (air pollution and smoking) is the usual starting point of this disease.

G. S. Christie (1954) now Professor of Pathology at Melbourne University, was amongst the first to recognize and describe pulmonary changes associated with chronic rheumatoid arthritis. Campbell & MacDonald published in 1965 what is probably the first description of a progressive fibrocystic condition of the upper parts of the lungs in patients with spondylitis ankylopoietica and described its characteristic features.

Respiratory physiology

As elsewhere in the world studies in respiratory physiology began in university research laboratories, but over the last decade they have moved progressively closer to the bedside.

For 25 years two successive occupants of the Chair of Physiology at the University of Sydney were men of distinction in the physiology of respiration. H. W. Davies, a pupil of Haldane and Priestley, who was co-author with Jonathan Meakins in 1925 of *Respiratory Function in Disease* was Professor of Physiology in Sydney from 1930 to 1946. His researches included investigations into alterations in blood gases in exercise and disease and he appears to have been the first in Australia to have correlated clinical observations with physiological laboratory investigations. He was succeeded by F. S. Cotton (1946–55) who before and during World War II carried out a distinguished series of meticulous studies in normal subjects on circulatory and respiratory function; he recorded a number of original observations, the significance of some of which has only recently been appreciated and it is generally agreed that he pioneered the development of the G

suit used by fighter pilots. Professor W. A. Osborne led a team of Melbourne workers in designing an early form of respirator for use against chlorine gas in World War I.

Charles George Lambie, the first fulltime Professor of Medicine in Australia, occupied the Bosch Chair of Medicine in the University of Sydney from 1930 to 1956 and developed within his school an approach to clinical medicine firmly based on physiological mechanisms. That, within its historical context, has been one of the dominant influences in Australian medicine. Thoracic disease lent itself so well to this approach that Lambie's influence on two generations of physicians in this regard cannot be overestimated. Her certainly influenced by precept two others in this department. Ralph Blacket between 1953 and 1959 pioneered a small group working particularly in the field of cardio-respiratory physiology and after a term as Director of the Hallstrom Institute of Cardiology, was appointed to the first Chair of Medicine in the University of New South Wales. John Read after 1958, led a group which has been one of Australia's most prolific contributors to thoracic medicine; their studies have covered a wide range from purely laboratory studies in pulmonary blood flow distribution and respiratory control, to original studies in asthma, chest deformity and obstructive and interstitial lung disease. They introduced the concept of 'responders and non-responders' to chronic hypoxia to explain why some patients with chronic obstructive airways disease develop cor pulmonale while others do not (Read & Lea, 1967) and made important observations on alterations in blood gases (Tai & Read, 1967) and lung volumes (Woolcock & Read, 1966) in acute asthma. A notable contributor to this group was K. T. Fowler (Fowler & Read, 1963) who developed the MS4 Respiratory Mass Spectrometer by the use of which cardiogenic oscillations were observed in the records of expired gas tensions and used as an index of pulmonary blood flow distribution.

In regard to various factors influencing the pulmonary circulation and the effects of embolism, the work of D. Halmagyi and H. J. H. Colebatch (Halmagyi & Colebatch, 1961; Halmagyi *et al.*, 1963) is widely recognized.

The application of physiological techniques to epidemiological studies has been exemplified by the work of the Joint Coal Board of New South Wales in the early 1950s in regard to coalworkers' pneumoconiosis, by Gandevia (Gandevia & Milne, 1965; Gandevia & Ritchie, 1966) in his studies of various industrial groups in both Melbourne and Sydney, and by Woolcock & Blackburn (1967) in their work on chronic lung disease in Papua, New Guinea, and also by current work being supported by the Asthma Foundation in several states.

Physiological assessment began tentatively to enter the clinical environment of thoracic wards in the middle 1950s. The first example of profound CO₂ narcosis in Sydney was recognized, documented and treated, though unsuccessfully, in a Drinker respirator in 1954. But by 1960 only one teaching hospital in Sydney and one in Melbourne (largely due to their associations with the University laboratory of Read and Gandevia respectively) utilized ventilatory tests with any frequency and blood gas measurements at all in the assessment and managements of patients with respiratory disorder. The transformation in the last decade has been remarkable with the introduction of routine laboratory assessments of blood gases, spirometry and lung volumes into the hospital wards and clinics.

Respiratory diseases amongst the aborigines

There is no doubt that diseases of the chest together with a variety of other European ailments and influences, contributed to the decline of the Australian aborigine in the nineteenth and early twentieth centuries and to the extinction of the unique Tasmania race (Cleland, 1928; Basedow, 1932). Tuberculosis was amongst these diseases but it is likely that this term as well as the less specific 'consumption' was loosely applied to some acute or chronic broncho-pulmonary diseases of several kinds; owing to the remoteness and isolation of many tribes and settlements diagnostic precision offered by bacteriological and radiological investigations was usually not available even in comparatively recent times. No case of tuberculosis was identified in 'wild' aborigines leading their nomadic existence in small communities and the disease was undoubtedly introduced by European settlers and in the north by Chinese immigrants who lived in close association with the natives, often under appalling social and hygienic conditions. C. E. Cook (1966) in 1925 observed that tuberculosis was focal in its distribution, occurring in unhygienic and unsupervised camps as well as in organized settlements where an open case might be encountered amongst the European staff. The disease often ran a rapid course to fatal termination in a matter of months and the autopsy pattern was consistent with progression of a primary infection. The question of high racial sensitivity immediately arises but this is doubtful, firstly because of associated problems such as poor living conditions, malnutrition, alcoholism and other debilitating diseases, and secondly because experience of adult-type tuberculosis occurring in aborigines living under better circumstances reveals no remarkable features in the natural history of the disease in this race.

By contrast the distribution of non-tuberculous chest disease was not focal but affected all ages and

groups. There is unanimity amongst all earlier observers that the aborigines were peculiarly susceptible to acute respiratory illnesses dating from the first known epidemic of influenza amongst them in 1839. It is of interest that the current epidemic of influenza in New Guinea is causing a high mortality amongst the natives of the Highlands of that country. Influenza amongst the Australian aborigines is likewise characterized by a high mortality and the common occurrence of complications especially pneumonia which was and probably still is, the commonest cause of death in detribalized or settlement natives.

Gandevia (1967) investigated aboriginal groups which had shortly before given up a nomadic existence in small family groups in Central Australia. In 1964 after a prolonged and severe drought in central Australia, a group of Pintubi aborigines migrated to settlements northwest of Alice Springs. The settlements are still remote and isolated by European standards, being mainly inhabited by aborigines with only a few white men. The living conditions are little changed except that food is regularly issued, but there is an important change from a nomadic life in small family groups to a static existence in a large community. The striking physical consequence of this altered environment is the development of chronic respiratory disease which appears to affect over 90% of all ages to some degree in this particular group and carries an appreciable mortality; the evidence suggests that they did not suffer significantly from this disorder prior to migration.

The evolution of the specialty of thoracic medicine

The emergence of chest diseases as a specialty in Australia is a post-war development. Before this there were a number of doctors engaged solely in the diagnosis and management of tuberculosis but these were all Government employees working either in Departments of Health or in Sanatoria. There were however, a few whose private practice was largely based on the management of tuberculosis at a time when collapse therapy in the form of artificial pneumothorax or pneumoperitoneum was a regular form of treatment. These practitioners had mostly undergone a period of training at one or more of the Chest Hospitals or Sanatoria abroad of which the Brompton Hospital was the most popular. One such physician, Dr Cotter Harvey, who had gained a reputation for the treatment of tuberculosis and had been very active in promoting the public health control of this disease through Government instrumentalities and voluntary bodies, decided after he returned from the war to devote himself entirely to chest medicine. He was appointed as Thoracic Physician to the Royal North Shore and the Royal

Prince Alfred Hospitals, at the former being assisted by Dr Bruce White and at the latter by Dr H. Maynard Rennie who in 1944 had established a clinic for the treatment of patients with bronchiectasis. These newly formed units constituted the commencement of Thoracic Medicine as a separate specialty in New South Wales. In Victoria a similar influence was exerted by Sir Clive Fitts who was also one of the first to draw attention to the occurrence of emphysema without significant bronchitis.

A considerable fillip was given to thoracic medicine in Australia by the establishment in 1947 of the Wunderley Travelling Scholarships in Thoracic Diseases. These were endowed by Dr (later Sir) and Mrs H. W. Wunderley for postgraduate study abroad and supported the scholar for a period of at least a year. These scholarships have been awarded to sixteen young physicians most of whom have subsequently become prominent in the field of thoracic medicine and three of whom now hold Professorships.

Before the mid 1930s thoracic surgery in Australia was almost confined to the draining of empyemas and the removal of hydatid cysts from the lungs. About 1934 C. J. Officer Brown of Melbourne, Frederick Clark of Perth and M. P. Susman of Sydney, began to do lobectomies and occasionally pneumonectomies for bronchiectasis and chronic lung abscess. Thoracoplasties and resections for pulmonary tuberculosis followed and a great impetus was given to this form of surgery by the introduction of effective tuberculous chemotherapy. The stimulus supplied by the need to treat chest injuries during World War II led to great strides in the advancement of thoracic surgery in this country as elsewhere, due in no small measure to great improvement in anaesthesia, especially in the hands of Daly, Hotten, Orton and Marshall. In this country, as elsewhere, resectional surgery for bronchiectasis and tuberculosis has become infrequent, most thoracotomies now being done for proven bronchial carcinoma or as a diagnostic procedure.

Associations and societies

As chest medicine emerged as a specialty so did appropriate societies develop. In August 1948, at a meeting of the BMA in Perth the Australian Tuberculosis Association was formed at the instigation of Dr (later Sir) D'arcy Cowan. This body elected Sir Henry Simpson Newland as its first President, an outstanding man who died at the time of the writing of this article, at the age of 96. The Association, which later changed its name to the National Association for the Prevention of Tuberculosis in Australia, was primarily a lay body though it had a large medical membership. In 1955 it organized an Asian Pacific Tuberculosis Conference in Sydney

to which delegates came from twenty-three countries including all those of the Columbo Plan, from WHO and from England. In 1960 in conjunction with WHO officials, it organized a WHO Tuberculosis Seminar also in Sydney to which delegates came from Western Pacific region, south-east Asia region and the east Mediterranean region, and participated in an extensive scientific programme of a fortnight's duration.

In 1952 a medical wing of NAPTA was formed which was given the name of the Australian Laennec Society and from which developed the present Thoracic Society of Australia of which branches exist in each of the six Australian states. Its annual meeting is held in conjunction with the annual meeting of the Royal Australasian College of Physicians.

This necessarily brief survey of the development of thoracic medicine in Australia must inevitably fail to do justice to some of those who have toiled in this field and made valuable contributions to the knowledge and understanding of pulmonary diseases. Thanks to these and to those who are mentioned in this text, the specialty of Thoracic Medicine is now well established in Australia and one can reasonably hope that its practitioners will continue to make worthwhile contributions to this sphere of knowledge.

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