from virus grown in cell cultures are highly effective. The second is that the risk of infected animals being smuggled into the country under a complete ban is greater than the risk of an animal coming out with the disease after it has undergone the recommended scheme of quarantine. All dogs and cats, the committee recommends, should be retained in quarantine for six months. They should be vaccinated with an approved vaccine on entry into quarantine and again one month later. Finally, they should be subject in the quarantine kennels to a variety of restrictions, which the committee specifies, and the kennels themselves should be run on rules that are also specified. The main object of the vaccination and rigorous segregation in the kennels is to prevent any possibility of the disease being transmitted between animals there by saliva alone. Previous quarantine regulations were based on the idea that it could be transmitted only by bite, and because this is now questioned the committee recommends that isolation of the animals should be more strictly controlled.

The freedom from rabies that the British Isles enjoy,[2] owing to their insular position and sensible regulations, is too precious to lose under any circumstances. But the threat of the disease coming in with a wild animal such as a bat does exist, and the danger of smuggling by irresponsible people against a total prohibition or a very long quarantine period must be faced. The Minister of Agriculture has had to consider whether the measures now proposed are the best compromise, or whether some longer period of quarantine should be substituted, and he has accepted the committee's recommendations. It is reassuring to learn from the report that Continental experts and the World Health Organization have also expressed their agreement with them.

Some Mediaeval Teeth

Nonsuch Palace, built by Henry VIII in 1538 and named to reflect its unsurpassed splendours, covered the site of the old village church and graveyard at Cuddington, which dated at least from 1100. The palace had a short life, passed out of Royal hands, and was demolished in the seventeenth century. Excavations on the site have taken place during the past decade, and the burial area has yielded up a number of children's skeletons. A brief survey of the jaws of 23 children from this excavation has been made by P. M. C. James and W. A. Miller.[1] It is valuable because of the relative dearth of studies of early deciduous dentition.

Twenty-one of the 23 children had teeth which showed considerable attrition, and the authors believe this was due to the coarse, gritty nature of mediaeval diets. That must at least in part be true, for from Roman to mediaeval times hand querns were often made of a friable lava imported from Germany and they shed much grit into the flour. However, another factor worth considering is whether the attrition was due not to grit but to long chewing on tough food. H. Brabant[2] attributes it to a diet which is resistant to chewing rather than abrasive, and A. L. Périer[3] has noted that the teeth of Eskimos show gross attrition, though their diet consists mainly of tough meat and is often virtually free of grit.

An unusually high proportion of the children's teeth were morphologically abnormal, which suggests that they may have been subject to genetic defects as a result of close inbreeding in a small village community. Unfortunately the dating of the skeletons is uncertain. They could range back from 1538 to as early as 1100 and possibly into Late Saxon times. This covers a period of transition in which dental conditions appreciably altered. Of the 222 deciduous teeth recovered 27 (12%) were carious, and of the 193 permanent teeth 11 (6%) were likewise. The latter figure is perhaps rather higher than the average for the period, the former decidedly so. Most authors agree that in mediaeval times the milk dentition was much less prone to caries than the permanent teeth. Until the sixteenth century food did not differ greatly from before the Norman Conquest. At the beginning of this period, for example, rickets is hardly to be identified; by Tudor times it had become commonplace.

Teeth of only four skeletons showed enamel hypoplasia, which seems to be a rather low proportion. Rates of 20% are normally found in Saxon skulls, and the percentage rises during succeeding centuries, until in and after Tudor times up to 80% of teeth found in burial grounds are hypoplastic. Moreover, in contrast to what is most often found today, the location of enamel hypoplasia in many mediaeval and other early populations usually indicates a disturbance of calcification at the age of 3 to 4 years. At Nonsuch two of the skeletons, and perhaps all four, had registered it by 18 months or soon after. Periodontal diseases occurred with a frequency which seems to be about average for the admittedly ill-defined period from which the skeletons date.

In general the jaws show a state which is transitional between that characteristic of earlier pre-Conquest populations and that which appeared in the seventeenth to nineteenth centuries.

[1] James, P. M. C., and Miller, W. A., *British Dental Journal*, 1970, 128, 391.
[2] Brabant, H., *Bulletin de la Société Royale Belge d'Anthropologie et de Préhistoire*, 1968, 79, 105.
[3] Périer, A. L., *Pratique Odonto Stomatologique*, 1949, Fiche No. 141.

Disability and Pneumoconiosis

About 300,000 miners are working for the National Coal Board today. Of these about 10,000 are receiving benefit for respiratory disability attributed to their work by the Pneumoconiosis Medical Panels. Nevertheless after many years of study there is still disagreement about the exact nature of this respiratory problem in coal miners. It is thought to consist of a combination of pneumoconiosis, presumably entirely due to mining, and chronic bronchitis and emphysema, from which other people also suffer.

Assessing the degree of a miner's disability is difficult; allotting the proportion of this disability between pneumoconiosis, chronic bronchitis, and emphysema is more difficult; but deciding what share of the chronic bronchitis or the emphysema or both together is due to mining and what is due to the rest of the miner's environment is an almost impossible undertaking. Yet this is what equitable compensation of the miner requires. At page 481 of this issue of the *B.M.J.* Dr. R. Ryder and colleagues discuss the problem of emphysema by (*a*) comparing the emphysema found post mortem in 247 miners with that in 247 controls from the same area, and (*b*) relating the severity of the emphysema in the miners to their clinical state, radiological appearances, and lung function as recorded by the Pneumoconiosis Medical Panel in life.

Coal miners first became eligible for compensation in 1929, when the Workman's Compensation (Silicosis) Acts of 1918 were extended to coal miners who were "drilling and blasting in silica rock." In 1934 this was further extended to "any operation underground in any coal mine." But by 1936 parliamentary concern led to a Medical Research Council investigation into "the form of disabling chronic pulmonary

disease which is believed to exist amongst coal miners but which, not coming within the accepted definition of silicosis, does not qualify for compensation."[1] This M.R.C. report in 1942 referred exclusively to miners in South Wales, because the majority of cases came from the anthracite mines there. The radiological differences between classical silicosis and the coal miner's disease were established. The pathology, based on 42 necropsies, suggested that the disease was a mixed pneumoconiosis and that silica was always present even in the type described radiologically as the "dust-reticulation" type, which had not previously been described. It was also noted that all cases showed some evidence of emphysema and that the dust reticulation, by its tendency to "reduce, disorganize and render ineffective the elastic fibres of the lung," might contribute to the development of the emphysema. There was no mention of chronic bronchitis.

But pathologists in Cardiff were still not satisfied with their understanding of the miner's disease. A. G. Heppleston[2] concluded in 1947 that the primary lesion was the localization of coal dust in the lymphoid tissue at the divisions of the respiratory bronchioles, which, with little fibrosis, was associated with emphysema affecting the adjacent vesicles. He later attributed the dyspnoea to an increase in the dead space of the dilated respiratory bronchioles.[3] Meanwhile in 1949 J. Gough and J. E. Wentworth introduced their whole-lung paper section technique,[4] which led to a clearer definition of the main types of emphysema[5]—focal in miners, centrilobular and panlobular in the general population. Paper sections have since been used in epidemiological approaches to the emphysema problem, but the quantitation of the extent and severity of emphysema in these samples of an infinitely variable organ has proved difficult. Attempts to measure the "internal surface area" of the lung using geological principles may be more successful.[6] A different approach is offered by the finding that the enzyme α_1-antitrypsin is lacking in the blood of patients with familial emphysema.[7] Unfortunately the structural findings, available only after the patient has died, do not correlate well with the functional respiratory measurements in life. Indeed Lynne Reid has doubted whether centriacinar (centrilobular) emphysema, the type which coal miner's focal emphysema most resembles, has any functional importance.[8] She and G. Simon have also shown that only the more severe degrees of emphysema are radiologically detectable.[9 10]

Chronic bronchitis also appears to be more prevalent in coal miners than in the general population,[11] though another M.R.C. report in 1966 said that "intensity of dust exposure does not appear to be a very significant factor in determining the prevalence of bronchitis in coal miners."[12] Chronic bronchitis can at least be defined clinically in terms of sputum production, and hyperplasia of the bronchial mucous glands is its morphological counterpart. Functionally both chronic bronchitis and emphysema are associated with reduction in the one-second forced expiratory volume, and either or both may produce the functional state of "irreversible (by bronchodilators) airways obstruction," to which obstruction of small airways (2-3 mm.) probably makes a substantial contribution.[13] Some progress in disentangling chronic bronchitis and emphysema from each other in life may come from the recognition that pure forms exist—the pure chronic bronchitic having been christened by A. C. Dornhorst the "blue bloater," the patient with pure emphysema the "pink puffer." This distinction draws attention also to the variable effects of irreversible airways obstruction on the cardiovascular system.

Fortunately the miner has not been completely forgotten in all this. Regulations under the National Insurance Acts ensure that, if he is judged by the Pneumoconiosis Medical Panel to have at least 50% disability due to pneumoconiosis, any disability due to chronic bronchitis or emphysema may be taken into account. Of course this is no help to those who have less than 50% disability from pneumoconiosis. Perhaps attention to the patients with the "pure" diseases and those subjected to single aetiological factors, though few in number, may give some further lead to the problem of dividing one man's respiratory disability equitably between the diseases contributing to it.

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Prophylactic Lithium

Two years ago in a leading article[1] lithium was given cautious approval as a prophylactic agent in cases of recurrent affective disorders. P. C. Baastrup and M. Schou[2] had described 88 such patients who had had at least two relapses in the preceding two years and who had been treated with lithium carbonate for periods of one to five years. The relapse rate was significantly lower than in a similar period before treatment began. But B. Blackwell and M. Shepherd[3] pointed out that with such patients, seen at a time of frequently recurring illness, the relapse rate might fall purely by chance. Since then two investigations have been carried out to study this possibility[4 5]; in all 99 patients were selected as having had at least two relapses during a two-year period. They were found to have an equally high relapse rate during the succeeding two years.

In contrast, and in a model of international collaborative study, J. Angst and his colleagues[6] have repeated and enlarged on the study of Baastrup and Schou. They studied 244 patients and confirmed that treatment with lithium reduced the number of episodes and admissions to less than half that in a similar earlier period. They also confirmed previous observations[7 8] that there is a tendency for the episodes of illness to get more frequent but that when lithium was introduced this trend was reversed.

In spite of the rigid criteria for the definition of relapses and the similarity of results in three different centres the claims in favour of lithium could still be criticized for the possibility of bias since the investigations were not double blind. Two such trials have now been reported. In order to overcome the possibility that early side effects might make the "double-blindness" of the trial illusory, Schou[9] selected pairs of patients who had been on lithium for a year or more and then gave half of them placebo on a random basis. He then followed them to their first relapse and terminated the trial as soon as statistical significance had been achieved by the use of sequential analysis. Elaborate arrangements including false serum lithium levels were used to ensure "blindness." Two such trials, one on bipolar manic depressives