Farmer's Lung: A Review

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Pulmonary disability among agricultural workers handling mouldy hay has been known for generations and it seems probable that Ramazzini's description in 1713 of the Diseases of Sifters and Measurers of Grain included the condition now known as farmer's lung. However, it was first described as a clinical entity by Campbell in 1932 and by Fawcitt in 1936. Both these authors collected their material from the North-west of England, an area of high rainfall, but it exists in other areas as well. In 1953 Fuller reported on 32 cases from the Devonshire area and Williams and Mulhall (1956) collected 10 cases in Radnor and Breconshire. In 1961 Staines and Forman reported the results of a survey conducted on behalf of the College of General Practitioners; 444 cases had been reported to them but the criteria of diagnosis were defined broadly, and some of these cases may have been due to other pulmonary disabilities. This work demonstrated the difficulty of diagnosing the condition and also a correlation between the incidence of the disease and rainfall, both geographical and seasonal. Cases have also been reported from Ireland, Iceland, Switzerland, Norway, Sweden, Finland, and New Zealand. In the United States Dickie and Rankin (1958) examined 39 patients with farmer's lung and obtained lung biopsies in eight of them. Rankin, Jaeschke, Callies and Dickie (1962) published a pathological and physiological study on 34 additional subjects.

The relation of the disease to the inhalation of mouldy vegetable matter has never been questioned but the view that it was a true pulmonary mycosis was challenged by Duncan in 1945 and subsequently by Fuller in 1958 and 1962. In 1962 Pepys, Riddell, Citron, and Clayton, in a detailed report dealing with the immunological investigations into the agent in mouldy hay responsible for farmer's lung, demonstrated antigens in mouldy hay which seemed to be responsible for the disease, and in a further report the following year Pepys, Jenkins, Festenstein, Gregory, Lacey, and Skinner (1963)

traced the responsible organism to thermophilic actinomycetes which developed in damp hay as it became overheated due to moulding. Characteristic precipitin reactions were obtained only from the serum of subjects exposed to mouldy hay and from a high percentage of patients with the clinical features of farmer's lung.

Incidence

The true incidence of farmer's lung must be largely conjectural. However, it is certainly not a rare condition in some parts of the country. A consultant dealing with a Welsh rural population of 90,000 saw 195 cases in eight years. There is a marked regional variation, and the survey carried out by Staines and Forman on behalf of the College of General Practitioners disclosed an incidence of 10.5 per 100,000 general population and 193.1 per 100,000 farming population in Wales while the figures for East Anglia were 1.2 and 11.5 respectively. The authors of this report put forward a 'speculative incidence' in the British Isles of about 1,000 cases a year.

Cases occur at any time of the year, with the exception of a few weeks in midsummer, but there is a noticeable preponderance of cases diagnosed in the early weeks of the year when stored hay is being used. A study of meteorological data shows an increase of cases following a summer of heavy rainfall when hay has presumably been stored with too high a moisture content leading to overheating, moulding, and suitable conditions for the growth of the incriminated actinomycetes. The evidence is conflicting on whether the incidence of the disease is changing. It is reasonable to suppose that modern combine harvesters and drying facilities will do much to reduce the risk as far as grain crops are concerned. On the other hand, it has been suggested that the practice of baling hay in the fields may have the opposite effect.

The ratio of males to females among reported cases is about 20:1, but this probably reflects the

preponderance of males over females in the dustier agricultural jobs.

The majority of cases so far reported have arisen within the agricultural industry and ancillary occupations. However, it appears probable that the disease can be caused by the inhalation of dust from a variety of organic substances provided they have been stored for a sufficient time under conditions favourable to the development of the thermophilic actinomycetes or similar organisms. The condition known as bagassosis, first recognized in Louisiana in 1937 and described by Hunter and Perry in 1946 following an outbreak in England, presents a clinical picture very similar to that of farmer's lung. Bagasse is the residue of sugar cane after the sugar has been extracted. The residue is baled and used for making insulating board for the building trade. The outbreak described by Hunter and Perry followed the introduction of a shredding machine to break up the bales. Before the introduction of this machine the bales were opened under water and so gave rise to no dust and no pulmonary disability. A larger outbreak occurred more recently in a newly opened paper mill in Puerto Rico (Buechner, 1961). Pulmonary disability following the inhalation of grain dust has already been mentioned, and similar conditions have been described in workers engaged in the now obsolete process of splitting the dried pods of the paprika plant and in the use of a size derived from tamarind seed in the textile industry (Murray, Dingwall-Fordyce, and Lane, 1957). The serology of these conditions has not as yet been explored and their relation to farmer's lung is uncertain. It has been suggested that the disease known as 'broken wind' in horses is of the same nature as farmer's lung. This condition appears to be more common during the winter months and is confined to stabled horses, but the post-mortem findings suggest hypertrophic emphysema without an interstitial fibrosis. However, 'fog fever' in cattle is probably identical with farmer's lung.

Symptoms and Signs

Acute, subacute, and chronic stages of the disease have been distinguished, but one stage may merge imperceptibly into the next. In about half the cases there is no clearly defined acute stage and the disease develops insidiously following repeated exposure to the dust of mouldy vegetable matter (Pepys and Jenkins, 1965). Acute attacks begin some hours after exposure and are characterized by distressing shortness of breath, fever, malaise, and cough which is usually unproductive. Moist sounds may be heard in the chest, but there may be no radiological signs at this stage. If there is no further exposure the

illness may be short and a rapid and permanent recovery made. It is probable that the majority of patients with these acute symptoms never seek medical help, and, among those who do, the transitory nature of the disability tends to result in the correct diagnosis being overlooked.

If the acute attack is severe, or if exposure to the antigen is continued, the illness may merge into the subacute stage characterized by severe dyspnoea and cough which may then become productive. Slight haemoptysis has been reported. Cyanosis may be present, and fine crepitations are audible over the lung bases with weak breath sounds. The radiological appearances may show a fine mottling, usually more marked in the lower lung fields. Headache, loss of appetite, and evening pyrexia are common, but the symptom that drives the patient to seek medical advice is usually the dyspnoea which may be out of all proportion to the clinical signs. Provided there is no further exposure to the antigen, this subacute phase may last for up to six months and, although there may be some residual pulmonary dysfunction, complete recovery may be expected in the majority of cases. However, each attack requires a longer period of recovery until the chronic third stage is reached. From this stage no recovery is to be expected since it is characterized by a fine interstitial fibrosis frequently accompanied by a honeycomb type of bronchiectasis.

Radiological Appearances

Radiology can provide useful supporting but not conclusive evidence. In the subacute stage many cases of farmer's lung show a mottling—generally fine—most prominent in the middle and lower lung fields. This mottling tends to clear as the reaction subsides but there may be no radiological change at all. A similar picture may be seen in pulmonary sarcoidosis, miliary tuberculosis, and some pneumoconioses. In the more chronic stages the radiographic findings are similar to those seen in interstitial fibrosis of whatever aetiology.

Pathology

The histological appearance of the lung after a recent attack is essentially of sarcoid-like granulomata with epithelioid cells, macrophages, multinucleated giant cells, and occasional eosinophils. There is also a diffuse mononuclear infiltration.

The more chronic cases show a diffuse interstitial fibrosis frequently associated with cystic honeycombing. The fibrosis has no characteristics to distinguish it from any other interstitial fibrosis.

Diagnosis

The farming community is subject to the various pulmonary diseases that occur in the general community. Pulmonary disability in a farmer is not necessarily farmer's lung. In the early stages of the disease it may be possible to diagnose the condition in some cases with tolerable certainty on the history and clinical examination alone, but it must be remembered that the condition is most likely to be met in the early months of the year, a time when acute respiratory infections are most common. A short-lived episode of fever, and cough with dyspnoea out of all proportion to the clinical signs, coming on about six hours after known exposure to the dust of mouldy hay or similar mouldy vegetable matter, possibly with a history of previous similar episodes that can be related to similar exposures, is highly suggestive of farmer's lung. This clear-cut, typical picture is seldom seen in practice as the sufferer frequently does not seek medical advice and recovers within a few days. The subacute and chronic stages of the disease are likely to present much greater difficulty, particularly when the sufferer is a middle-aged person with a pulmonary disability which may be due to farmer's lung, chronic bronchitis, or both conditions simultaneously. Farmer's lung may develop insidiously, and even the history of previous typical attacks following exposure to mouldy hay may be absent. In these cases, which may well prove very common, a clinical examination alone is unlikely to prove sufficient to establish the diagnosis. Fortunately there are aids to diagnosis available.

Skin Tests Skin tests with extracts of mouldy hay are of little diagnostic value. No immediate hypersensitivity reaction occurs except in patients who have a superimposed allergic asthma. Most hay extracts give rise to a non-specific irritant effect and are therefore unsuitable for skin testing. However, skin tests with other extracts may have a place in the differential diagnosis of conditions such as asthma and pulmonary aspergillosis which might be confused with farmer's lung.

Lung Function Tests The assessment of lung function is of major importance in the diagnosis of the disease. Cases of farmer's lung exhibit the changes in lung function that are associated with interstitial pulmonary disease. These changes also occur in pulmonary sarcoidosis, berylliosis and other granulomatous conditions of the lungs, the Hamman-Rich syndrome, cystic fibrosis, alveolar proteinosis, and alveolar calcification, and they may also be found in asbestosis and bagassosis. In these

conditions as in farmer's lung the main feature is the replacement of normal lung by granulation tissue and subsequently by fibrosis, thus reducing the distensibility of the lung and the area of the alveolar capillary membrane available for the exchange of gas. This reduction in functional area of the membrane is associated with a reduction in the diffusing capacity of the lungs, especially the diffusing capacity of the alveolar capillary membrane, and the condition is sometimes referred to as 'alveolar capillary block'. Pulmonary function studies demonstrating a reduction in the diffusing capacity of the alveolar capillary membrane in interstitial pulmonary disease such as farmer's lung are distinguishable from those obtained in cases of obstructive lung disease. Obstructive lung disease is characterized by a persistent rise in airways resistance due to a narrowing of the lung airways, their premature collapse on expiration, and also maldistribution of ventilation. There is, of course, no reason why a reduction in the diffusing capacity of the alveolar capillary membrane should not exist together with obstructive lung disease, and a farmer with chronic bronchitis demonstrating obstructive lung disease may also have a superimposed interstitial pulmonary fibrosis due to farmer's lung with a consequent reduction in the diffusing capacity of the alveolar capillary membrane. Indeed it is this combination that is likely to cause the most difficulty in diagnosis.

Inhalation Tests Williams (1963) described inhalation tests for the diagnosis of farmer's lung. Aerosols of mouldy hay extracts provoked reactions in 12 out of 15 patients allegedly suffering from farmer's lung, while no reaction occurred in 20 controls. Reactions were characterized by a delay of several hours when typical acute attacks of farmer's lung occurred with a fall in pulmonary diffusing capacity but no evidence of airways obstruction reversible by isoprenaline. Similarly designed tests on 38 allergic asthmatics using appropriate antigens resulted in the immediate onset of bronchospasm. The inhalation of extracts of clean hay or extracts of the fungi of mouldy hay caused no reactions, and it was concluded that the responsible antigen was neither in the hay itself nor the contaminating fungi and bacteria but was a product of the interaction of the two. Subsequent to this investigation it was shown by Pepys *et al.* (1963), as has previously been mentioned, that the source of the responsible antigen was certain thermophilic actinomycetes, and inhalation tests with the most important of these, *Thermopolyspora polyspora*, have produced farmer's lung reactions in affected subjects.

Corticosteroid therapy suppressed the reaction to the inhalation of the antigen in susceptible subjects and this observation may be of value in the management of the disease.

Deliberate challenge with antigens to which the subject may be susceptible is not without danger and should be attempted with caution in the elderly or respiratory cripple.

Serological Tests The demonstration of precipitins against extracts of mouldy hay in the sera of patients suffering from farmer's lung by Pepys *et al.* (1962) has not only provided valuable supporting evidence for the diagnosis of the disease but has also made it possible to identify the source of the antigens responsible. However, the authors point out that the investigations are at an early stage, and refinements of technique are still being developed.

The method now being used is the immuno-electrophoretic test in which the antigens of the test extracts are diffused throughout the agar-gel by electrophoresis and are then allowed to react with the test sera. This test has shown a characteristic precipitation pattern with the sera of most of the typical cases of farmer's lung. The main source of the antigens responsible has been shown to be *Thermopolyspora polyspora* and to a lesser degree *Microspora vulgaris*. Certain other actinomycetes and streptomycetes may occasionally be involved, but they have not as yet been identified with certainty. A characteristic precipitin pattern is obtained with about 90% of sera from cases believed to be of farmer's lung. The remaining 10% may be explained either as examples of an incorrect initial diagnosis or more probably as due to the responsible antigen being one of the actinomycetes or streptomycetes awaiting identification. It is already evident that the final figure of positive reactions will be above 90% when the tests are performed with an adequate series of antigens.

Sera from patients who have been exposed to mouldy hay and have either no pulmonary disability or who are suffering from bronchitis, asthma or other pulmonary disorders give precipitin reactions to extracts of mouldy hay in about 20% of cases. These cases are the ones most likely to present difficulty when one is deciding how much weight can be placed on the serological results in determining a diagnosis of farmer's lung; the final decision must rest on the information that accrues from the other aids in diagnosis. However, it must be remembered that sufferers from some other pulmonary disorders may also be suffering from superimposed farmer's lung. The presence of positive precipitin reactions in a few farmers without obvious

pulmonary disability will require further investigation.

It will be apparent that, although a negative precipitin reaction does not support a diagnosis of farmer's lung, it does not absolutely exclude it, and a positive reaction, though strongly suggestive, is not by itself conclusive evidence.

Differential Diagnosis

Chronic Bronchitis and Emphysema The typical physiological, radiological, and pathological features of farmer's lung are absent in these conditions. The usual auscultatory findings are rhonchi which are not typical of farmer's lung. Precipitation tests with sera will, in the majority of cases, show no reaction to the prepared farmer's lung antigens, and inhalation tests will not show a reaction delayed by a few hours and culminating in an acute farmer's lung episode with a fall in pulmonary diffusing capacity. Furthermore, the history of the illness will not suggest any special relation to exposure to mouldy hay.

It has previously been pointed out that there is no reason why the disease of farmer's lung should not be superimposed on a case of chronic bronchitis, and it is these patients who are likely to cause most difficulty.

Asthma Asthma is a common condition in the general population and, although a farmer may well develop this as a result of occupational exposure to an allergen to which he is hypersensitive, there should be no difficulty in distinguishing uncomplicated asthma from farmer's lung since the two disorders differ strikingly.

An attack of asthma develops within minutes of exposure to the offending substance. An attack of farmer's lung develops some hours after handling mouldy hay. Exposure to mouldy hay followed by an immediate hypersensitivity reaction suggests asthma since this type of allergic response is uncommon in cases of farmer's lung. Asthma results in tightness of the chest, wheezing, prolonged expiration, and rhonchi quite distinct from the dyspnoea of farmer's lung which is out of all proportion to the auscultatory findings. There are no typical radiographic findings in asthma, and pulmonary function studies show an airways obstruction, reversible with antispasmodics and not a reduction in the diffusing capacity of the lungs unaffected by antispasmodics. Skin sensitivity tests using extracts of the responsible allergen show an immediate weal reaction in cases of asthma. The allergen may be contained in mouldy hay, and in these instances a hypersensitivity to fungal extracts

may be found. No such immediate weal reaction occurs in cases of farmer's lung. Inhalation tests using extracts of the relevant allergen give immediate reactions reversible by antispasmodics in asthmatics whereas in similar tests on patients with farmer's lung a delayed reaction unaffected by antispasmodics occurs.

Precipitin reactions in asthmatics are negative since the antibodies responsible for the immediate hypersensitivity reaction are non-precipitating. Patients suffering from farmer's lung have circulating precipitins and the test is therefore positive.

Pulmonary Aspergillosis The pathogenic member of the species is usually *Aspergillus fumigatus*, although exceptionally other members may be responsible for the disease. The fungus proliferates on dead organic matter and it is not infrequently a saprophytic invader complicating existing pulmonary disease such as carcinoma, tuberculosis, lung abscess, unresolved pneumonia, and pneumoconiosis. The differential diagnosis in these instances will be that of the underlying condition. There is, however, an allergic variety of pulmonary aspergillosis in which inhalation of the fungal spores gives rise to episodes of asthma and fever accompanied by areas of pulmonary consolidation and collapse, but the radiographic picture is entirely different from that of farmer's lung. Sufferers may in fact be chronic asthmatics who, on exposure to the fungal spores, suffer episodic aggravation of their asthma. The attacks usually resolve within a few days but exceptionally they may go on for months.

In these chronic cases an interstitial pulmonary fibrosis may result. The sputum contains tenacious plugs incorporating filaments of the fungus and eosinophil cells. There is also an eosinophilia in the peripheral blood. Pulmonary function tests show the airways obstruction observed in asthmatics and not a diminution of diffusing capacity except in those rare cases where the disease has progressed to interstitial fibrosis. Skin tests with appropriate extracts of *A. fumigatus* are positive giving an immediate weal reaction followed by a nodule, biopsy of which shows the cellular reaction to be eosinophilic. The trial inhalation of extracts of *A. fumigatus* provokes immediate asthmatic reactions, reversible by antispasmodics. Sera from these patients do not show precipitin reactions to the appropriate antigens of farmer's lung. However, sera from patients with farmer's lung may react with extracts of *A. fumigatus* but these reactions can be distinguished from those due to the farmer's lung antigen.

The recovery of *A. fumigatus* from the sputum is

of limited value since, disregarding the saprophytic type of infection, normal people inhale and expectorate these widely dispersed spores without suffering any disability.

Chronic Diffuse Interstitial Fibrosis of the Lungs This comparatively rare disease of unknown aetiology was first described by Hamman and Rich (1944). The condition became known as the Hamman-Rich syndrome but the disease, as described by these authors, covered only a small variant of the much wider condition now accepted as a single clinical entity. A compromise is frequently used whereby the disease is known as chronic diffuse interstitial fibrosis of the Hamman-Rich type. Fibrosing alveolitis has recently been suggested.

The outstanding clinical features are progressive dyspnoea, unproductive cough, râles at the bases of the lungs, clubbing of the fingers, and loss of weight. The early radiographic changes are not unlike those seen in farmer's lung with a fine mottling starting in the lower zones and spreading upwards as the disease progresses. The mottling becomes coarser and more confluent and may show cystic spaces usually noticeable first at the bases.

The pathology is essentially an oedematous thickening of the alveolar walls followed by an exudate in the alveolar spaces which appears to retract and become organized with the oedematous thickening as a fibrosis of the alveolar walls.

A clear-cut case of this disease will not provide the history of exposure to mouldy hay followed by the episodes typical of farmer's lung. However, difficulty may be encountered in differentiating those cases of farmer's lung with an insidious onset. Pulmonary function tests will show a reduced distensibility of the lung with a reduction in the diffusing capacity of the alveolar capillary membrane in both diseases. Serum precipitin reactions are of great value, and a positive result provides presumptive evidence that the interstitial fibrosis is in fact a late sequela of farmer's lung. If any doubt remains, inhalation tests using extracts of mouldy hay should clinch the diagnosis.

Silo-filler's Disease This puzzling disease is as yet uncommon in this country but it is worth bearing in mind since it is an acute respiratory disease of the agricultural community and is therefore to be distinguished from farmer's lung. The disease was described by Lowry and Schuman in 1956 and is believed to be due to the inhalation of nitrogen dioxide given off during the early stages of fermentation of silage. It is alleged that the use of artificial nitrogenous fertilizers and drought conditions at the time the silo is filled increase the danger.

The typical history is one of being in a silo, or in the vicinity of one, from two hours to six days after it has been filled. Immediate cough, dyspnoea with a sensation of choking, and severe weakness are noted, and these symptoms do not entirely clear up for the following two to three weeks. At the end of this period there is an acute illness accompanied by fever, rapidly progressive dyspnoea, cyanosis, and cough. Clinical findings consist of moist inspiratory râles heard over both lungs. There is a marked neutrophil leucocytosis, and the radiographic picture shows scattered, small, discrete opacities throughout both lung fields, very similar to those seen in miliary tuberculosis. The lungs in fatal cases contain very numerous uniformly distributed nodules of miliary size which on histological examination are seen to be bronchioles filled with a cellular fibrinous exudate with ingrowth of fibroblasts from the bronchiole walls. The pathological picture is similar to that of the condition known as bronchiolitis fibrosa obliterans.

It is reasonable to suppose that, if this condition is due to the inhalation of nitrogen dioxide, the above description will vary according to the degree of exposure, and the inhalation of more concentrated gas may well result in the more familiar picture of 'nitrous fume' poisoning. Delaney, Schmidt, and Stroebel (1956) reported two patients with silo-filler's disease, one of whom died in acute pulmonary oedema 36 hours after exposure to the fumes.

Pulmonary Sarcoidosis The epithelioid-cell pulmonary granuloma, characteristic of sarcoidosis, is found as a response to a variety of agents, and it may well be that the disease is a non-specific host-tissue reaction rather than a single clinical entity. The list of agents capable of producing the sarcoid-like tubercles is a long one, including acid-fast bacilli, protozoa, fungi, beryllium, and zirconium. Recently an acid-fast lipid material obtained from pine pollens has been shown to produce sarcoid-like lesions in experimental animals.

The essential pulmonary lesion of acute and sub-acute farmer's lung is indistinguishable pathologically from that of sarcoidosis. The radiological picture is also similar, but hilar node involvement is not found in farmer's lung. Both diseases may show hyperglobulinaemia and reduced sensitivity to tuberculin. Pulmonary function tests may show a reduced distensibility and a diffusion defect in both conditions. The presence of evidence of sarcoidosis elsewhere in the body would of course exclude farmer's lung and the Kveim test may also be of assistance. The diagnosis is likely to depend largely on the history and the results of the immuno-electrophoretic precipitin test which, in experienced

hands, can distinguish the two conditions in a high percentage of cases. A typical farmer's lung reaction to test inhalation of extracts of mouldy hay would be conclusive.

Other Pulmonary Diseases The fibrotic stage of farmer's lung may be confused with other pulmonary diseases giving rise to fibrosis. Further conditions to be borne in mind are miliary tuberculosis, unresolved pneumonia, the collagen diseases, histoplasmosis, pneumoconiosis, and byssinosis. The occupational pneumoconioses present as an alternative diagnosis more frequently than might be expected since it is not uncommon for farming and agricultural work to be a part-time or retired occupation in mining and industrial districts.

Conclusions

The disease of farmer's lung is probably as old as the settled agrarian civilizations of temperate climates and has probably been responsible for serious ill-health through the ages. That it may be a serious disease is beyond question. Staines and Forman (1961) recorded the length of illness in 111 cases; in 44 of these there was more than three months' incapacity, and five patients were still incapacitated after a year. The assessment of the final disability after recovery from the illness showed that, out of 121 cases, 35 were judged to have a moderate or severe disability in following their regular occupations and 10 in doing other jobs. Only 51 were judged to have been left with no disability at all. If the 'speculative incidence' in the British Isles of about 1,000 cases a year put forward by these authors is correct, it seems that about 580 persons a year are left with some assessable disability and about 290 of these experience a moderate or severe disability in following their regular occupations. Such estimates of the total yearly incidence in the British Isles cannot, of course, be used as an indication of the number of successful claims that may be expected from the disease being prescribed under the Industrial Injuries Act. Broadly speaking, persons eligible to claim industrial injuries benefit are confined to those who are employees, and the evidence so far available suggests that a significant proportion of sufferers from farmer's lung are to be found among self-employed persons and their families.

The disease normally presents a distinct clinical picture and, with the aid of the history and ancillary investigations, it can be diagnosed in the majority of cases with reasonable certainty. Difficulty may be encountered in those patients who have a chronic non-specific lung disease of some duration and on

which some degree of pulmonary disability due to farmer's lung becomes superimposed. However, even in these patients it should be possible to recognize the presence of serum precipitins against suitable extracts of mouldy hay, and the decision as to how much of the disability should be attributed to farmer's lung will be assisted by further investigating the distensibility and diffusing capacity of the lungs.

APPENDIX

Prescription of Farmer's Lung under the National Insurance (Industrial Injuries) Act, 1946

In June 1963 the Minister of Pensions and National Insurance referred the following question to the Industrial Injuries Advisory Council:—

'Whether, having regard to Section 55 (2) of the National Insurance (Industrial Injuries) Act, 1946 the respiratory complaint known as "farmer's lung", due to exposure to the dust of mouldy hay, mouldy straw or similarly contaminated agricultural produce, should be prescribed under the Act and, if so, for what occupations.'

The matter was referred by the Council to the Industrial Diseases Sub-Committee for detailed examination. In due course the Sub-Committee submitted a Report, which was adopted, to the Council and that Report was subsequently published by the Minister (Farmer's Lung, 1964).

The Report in brief found that the farmer's lung syndrome appears to be caused by exposure to the dust of mouldy vegetable matter; that a considerable number of cases occur, almost exclusively within the agricultural industry; that the condition often leads to prolonged incapacity and serious disability, so that a considerable sum in industrial injuries benefit might be involved in an individual case, and that the accident and pneumoconiosis provisions of the Scheme do not appear in practice to be adequate to deal with the cases that occur.

Moreover, the judicious combination of the techniques available enabled the disease to be diagnosed with reasonable certainty, and once diagnosed it can be accepted as being due to the nature of the occupation. The relevant Section of the Act was therefore satisfied, and the Report recommends that farmer's lung should be prescribed.

The Report dealt at length with the definition of farmer's lung for the purposes of prescription. It considered that the disease, if prescribed, should include pulmonary disability due to the inhalation of the dust of other mouldy vegetable produce, the disease bagassosis being provided as an example. It

was further considered that a precise definition in terms of the effects of the disease might be advantageous, not only because of the number of synonyms by which farmer's lung is known but also because in this way the important distinction between farmer's lung and bronchitis may be kept in mind. The definition of the disease suggested in the Report is therefore:—

'pulmonary disease due to the inhalation of the dust of mouldy hay or of other mouldy vegetable produce and characterized by symptoms and signs attributable to a reaction in the peripheral part of the broncho-pulmonary system and giving rise to a defect in gas exchange.'

The Report, in considering the terms in which the occupation cover should be drafted, pointed out that the majority of cases arise within the agricultural industry. However, evidence suggests that occasional cases may occur in other employments. Some of these involve occupations ancillary to agriculture, such as threshing engineers and persons engaged in the transport of hay, but others do not; for example, if bagassosis is to be covered, persons manufacturing hardboard from bagasse would have to be provided for. The Report considered that it would be possible to cover all such cases without extending the field of cover too widely by the use of the following formula for the second column of the Prescribed Diseases Schedule:—

'Any occupation involving exposure to the dust of mouldy hay or other mouldy vegetable produce.'

This formula was, for technical reasons, subsequently amended by the Council. The final occupational cover for the second column of the schedule reads:—

'Any occupation involving exposure to the dust of mouldy hay or other mouldy vegetable produce by reason of employment (a) in agriculture, horticulture or forestry; (b) in loading, unloading, or handling in storage such hay or other vegetable produce; or (c) in handling bagasse.'

The Report further recommended that, owing to the complexities likely to arise over the diagnosis and assessment of the disease, the existing organization of the Pneumoconiosis Medical Panels should be used in settling these questions, though in other respects the disease should be treated as an ordinary prescribed disease.

Finally the report drew attention to the need for providing some way to protect workers against this serious disease.

The recommendations of the Report were accepted, and the disease was prescribed and added

to the list of prescribed diseases by regulations (S.I., 1965) which came into operation on June 21, 1965.

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