Opening Remarks at Meeting of Research Committee at Cambridge 15th December 1958 by Dr. J.F Knox.

I am permitting myself the indulgence of a few preliminary remarks on the occasion of the first meeting of our second year of effort on our problem and our objectives.

The asbestos industry continued on its way without much thought for its occupational health aspects from the turn of the century when Dr. Montague Murray, Physician to Charing Cross Hospital, described the first case of asbestosis till 1924 when Dr. W.E Cooke, pathologist of Wigan, described the second case. Murray considered that he had seen spicules of asbestos in the slides made from the lungs in his case while Cooke found the "curious bodies". It was not at first apparent that the "curious bodies" were spicules of asbestos modified by a covering of protein. Cooke thought they were fungi but McDonald, Gloyne, and Stewart investigated them so completely that their descriptions - especially that of Gloyne - remain among the best in the world today. [The Americans were second in the field here.] Lanza and Lynch followed up the English work, and while confirming it, did not add anything. Lanza, indeed, did edit a textbook called "Silicosis and Asbestosis" in which the section on pathology was written by Gloyne and the engineering and legislative aspects were described by Middleton of the Factory Department in London. However, the experimental section of this book was written by Leroy Gardner and was very good indeed. With his untimely death in 1942 the United States lost a pioneer research worker who has never been replaced by one of equal gifts. E.J King working in Hammersmith Hospital, London, with a grant from Turner Brothers Asbestos

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Company Limited, repeated Gardners work on the experimental animal with a wide measure of agreement but he differed in one specific and important matter. Gardner was unable to give his animals fibrosis of the lungs with inhalation of short fibres and ball milled asbestos but King, using intra-tracheal injection methods with fibre sizes around 2.5 microns was able to give a reticular fibrosis to his animals. The Americans critised these results on the grounds of overdosage but the point has never been resolved. Even King has not claimed to have induced asbestosis by inhalation of short fibre asbestos.

After the report of a survey of the industry in 1930 by Dr. E.R.A Merewether legislation was introduced for dust control but following Cooke's case in 1924 the industry started to introduce dust reduction measures so that when Merewether did his tour, preventive measures were under-way. Just to remind you of what Merewether found in the examination of 363 workers with various exposures; there were 95 or 26.2% with evidence of fibrosis of the lungs while, of the group whose exposure had exceeded 20 years, 80% had such evidence. Further evidence of the effects of the severe exposure of the earlier days was provided by a mortality study published by Doll in 1955. He showed that in a group of 113 men employed for 20 years and upwards the number of deaths which occurred was 39 when the number expected to occur on the rates for Great Britain during the same time would have been 15. This was obviously excessive and the excess was entirely in the group of diseases of the respiratory system and the heart.

The industry persisted with its efforts to reduce the dust

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in workrooms and there is no doubt that some success has followed these measures. For ourselves in Turner Brothers Asbestos Co. we have reviewed the mortality of 418 men and women who have worked in scheduled areas for 10 years and upwards since 1/1/33. All deaths were included up till 30/12/57. The number of deaths which had occurred in this group was 20 and the number expected on the rates for Great Britain as a whole was 21.8. This certainly represents some measure of control. But there have been some suspensions for asbestosis in the group though there were no deaths directly attributable to it. The number of suspensions up to the present is 5 but I would be prepared to include another 3 as likely to be suspended in the next few years.

The position therefore is that there has been a substantial reduction but a problem persists. Even in our best areas there is some fibrous dust but we are nearer threshold limits than we were. The question is - how near? Our aim therefore is not just - how does asbestos in bulk cause fibrosis of the lung? - a purely qualitative approach, but, by how much do we require to reduce our dust so as to fail to cause asbestosis at all? - a quantitative approach.

It has been said by Wyers and others that we are seeing a milder form of the disease today and this may well be so. In the mining areas rock dust is mined with fibre and the resultant cases which occur there seem to be milder according to Cartier at Thetford mines. The possibility that asbestosis may be modified in its course by admixture with other materials such as cement, magnesia, diatomaceous earth or even plastics is

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of great practical interest. In Copenhagen Frost has described abnormalities in the lungs of insulating workers and Ahlborg in Gothenburg has done so also. Magnesium carbonate and hydroxide would probably be the diluting agent here. The possible preventative effect of aluminium or aluminium hydroxide might well be further explored.

To sum up we are attacking the problem of the etiology of asbestosis hoping to understand the process better with a view to modifying it and we have the knowledge that reduced dust exposure has lessened the incidence.

Another recent trend in pneumoconiosis is to study more fully the link up between dust exposure and general body states. This is important to try and understand why one individual has an apparent immunity while others are susceptible.

It was an observation of Caplan of the Cardiff Pneumoconiosis Panel in 1953 that in cases of miners with rheumatoid arthritis there were lung shadows on the X-ray film similar to those caused by pneumoconiosis alone but possessing characteristic differences. Enough work has been done on this by Gough and others to show that in rheumatoid arthritis generally there are occasionally pulmonary changes and in cases of pneumoconiosis the two conditions may be superimposed, the one aggravating the other. One case of rheumatoid lung disease associated with asbestosis has been described by Rickards and Barrett which had a fatal termination.

Gough's conclusion on this matter in a recent paper is that the only relationship between the collagen diseases (rheumatoid arthritis is now considered a collagen disease) and pneumoconiosis established so far is that which occur in rheumatoid

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subjects. He also considers that there is suggested evidence that silicosis may be a modified antigen - antibody reaction. Incidentally Gough quotes P. [illegible]t of Reading with the claim that silica can enter into combination with protein and the belief that inter-action occurs between collagen precursers and silicic acid molecules. These observations and studies may be of considerable importance in the investigation of asbestosis.
