IN THE WORKERS' COMPENSATION COMMISSION OF NEW SOUTH WALES.

1939.
May 22, 23, 24,
June 19, 30.

Coram: LAMOND, J., MEMBER.

Medical Assessor: Dr. R. M. Mackay.

JONES *v.* JAMES HARDIE AND CO. LTD.

Dust disease—Death results from disease caused by silica dust—Onus of proof—Section 6 (1), Workers' Compensation Act, 1926-1938.

A fibro-mixer died from fibrosis of the lungs complicated by superadded infection, the fibrosis being caused by the inhalation of dust. His widow claimed compensation, alleging that the fibrosis was caused or aggravated by asbestos dust to which his employment with the respondent had exposed him. The respondent denied liability on the ground that the disease was caused by silica dust.

The Commission, *per Lamond, J.*, found that the applicant had not discharged the onus of proving her allegation, but that the respondent had discharged the onus of proving that the dust disease was caused by silica dust.

Parsonage for the applicant.

Kelynack for the respondent.

JUDGMENT.

LAMOND, J.: The applicant claims compensation as the widow and total dependant of Samuel Jones, whose death on the 14th February, 1939, was alleged to have resulted from fibrosis of the lungs caused and/or aggravated by abestos dust with which it was alleged his work over a period of years with the respondent company as a fibro-mixer brought him into contact.

The respondent company alleges that the death of the deceased was not the result of "injury" arising out of and in the course of his employment, that notice of the injury was not given as required by the Act and that the respondent company was thereby prejudiced.

The deceased was born in 1884 and worked in anthracite mines in Wales, Great Britain, from 1907 to 1913. He then came to Australia, where between the years 1913 and 1919 he worked in various capacities, e.g., as a general labourer, concrete mixer, brick pit worker, and coal miner; in 1919 he entered the employment of the respondent company for whom he worked until 1937 when his health commenced to fail, his main symptoms being shortness of breath, failing appetite, and loss of weight.

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Evidence was given by one, Thomas Jilks, who stated that for the ten years prior to deceased ceasing work he did the same class of work as deceased, that such work involved raking out asbestos from certain asbestos rooms or bins and that dust was frequent, that it was blowing all the time and that it would get in one's nose and throat.

W. B. Hunt, Works Manager, stated that the asbestos was milled and the dust taken out of it before it was received by respondent company, that when received it was similar in appearance to kapok and not below one-sixteenth of an inch in size, that asbestos dust could be seen in the asbestos bins when the asbestos was being raked out, but that in the mixing room cement dust but not asbestos dust could be seen. Deceased's working place was well ventilated and no complaints had been made previously by men working in the mixing section.

Dr. A. O. V. Tymms, who investigated the dust conditions of respondent company's factory in July and November of 1938, stated there was a possibility of an asbestos hazard. No evidence was, however, tendered to show what concentration of asbestos dust there was from time to time in the asbestos or mixing rooms, and very little help can be obtained from consideration of the fact, as it is alleged, that no complaints were made by deceased or other workmen.

Dr. A. M. Stanton, of Granville, who was deceased's doctor over a number of years, deposed to having treated deceased between 1931 and 1936 for minor sicknesses. In 1936 deceased was away from work for two months with influenza and thereafter deceased appeared to go downhill. On 30th November, 1937, he attended deceased for what he described as silicosis of the lungs. From March, 1938, to February, 1939, deceased had a very bad cough and was extremely weak and emaciated. Occasionally he had a temperature, but practically no sputum.

An X-ray examination of deceased's chest was made by Dr. D. J. Maitland in September, 1938. This skiagram has been interpreted by Dr. Maitland, Dr. J. G. Edwards and Dr. W. T. Nelson, as evidencing lung changes due to silicosis. Both Drs. Edwards and Nelson were of the opinion that the X-ray evidenced some super-added infection and all were agreed that it was impossible to say whether asbestos had or had not played a part.

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Dr. Nelson stated that the X-ray showed a typical infective granular type of fibrosis due to inhalation of silica-bearing dusts, whereas asbestos fibrosis gave an appearance of ground glass haziness.

Dr. Shearman made eight tests of deceased's sputum without finding any asbestos bodies or tubercle bacilli.

Clinical evidence was furnished by Dr. W. E. Fisher for the applicant and Drs. W. T. Nelson and G. C. Willcocks for the respondent.

Dr. Fisher stated that he admitted the possibility of pneumoconiosis as the result of mining in Wales, but that if the dust exposure in Australia were correct it would be equally possible for the terminal clinical picture to be accounted for by asbestos dust. He further considered that if there had been some progress of a pre-existing pneumoconiosis due to mining in Wales, exposure to dust here must have had an aggravating effect.

Drs. Nelson and Willcocks on the other hand expressed the view that deceased's death could be accounted for entirely by reference to silicosis with super-added infection, and that there were not sufficient facts to found a medical opinion that asbestos dust had aggravated deceased's condition.

Summary.

In attempting to assess the extent to which deceased was affected by asbestos dust, the following facts are outstanding—

(1) While, generally speaking, exposure to asbestos dust is an industrial hazard, there is nothing known as to what degree of exposure is necessary to cause asbestosis.

In this connection, Dr. Willcocks took some slides of the dust where applicant worked which showed large asbestos fibres, whereas anatomically only the very smallest fibres can be injurious. In silicosis it has been stated that only particles below 10 micro-millimetres are capable of producing harmful results by inhalation:

(2) Examination of deceased's sputum revealed no asbestos bodies:

(3) Examination of deceased's lungs by X-ray revealed no appearance consistent with asbestosis.

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In these circumstances the question for the Commission is whether, despite the foregoing facts, it should find that the applicant has weighed down the scales in favour of the view that the death of deceased resulted either by way of causation or acceleration from the inhalation of asbestos dust.

The Commission's view is that such a conclusion would be entirely conjectural and that a completely satisfying explanation for the death of deceased is found in the progress of a condition of silicosis complicated by a super-added infection, the presence of both of which is established by the evidence.

The award is, therefore, made for the respondent in the following terms:

(*a*) the evidence does not establish that the death of the worker, Samuel Jones, which occurred on the 14th day of February, 1939, resulted either by way of causation or acceleration from the inhalation of asbestos dust;

(*b*) the evidence does establish that the death of the said worker resulted from the progress of a condition of silicosis complicated by a super-added infection;

(*c*) the evidence does establish that the dust disease which resulted in the death of the said worker was caused by silica dust.

Solicitors for the applicant: *Abram Landa & Co.*

Solicitors for the respondent: *J. W. Maund & Kelynack.*