STANDING COMMITTEE ON PUBLIC ADMINISTRATION

INQUIRY INTO THE MANAGEMENT OF ASBESTOS CONTAINING MATERIALS IN WESTERN AUSTRALIAN STATE SCHOOLS

TRANSCRIPT OF EVIDENCE TAKEN
AT PERTH
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SESSION ONE

Members

Hon Barry House (Chairman)
Hon Ed Dermer (Deputy Chairman)
Hon Matthew Benson-Lidholm
Hon Vincent Catania
Hon Nigel Hallett

Hearing commenced at 11.15 am

TANDON, DR MAHARAJ K.,
Consultant Thoracic Physician,
Hollywood Private Hospital,
Nedlands 6009, examined:

The CHAIRMAN: On behalf of the committee, I welcome you to the meeting. To begin with, would you please state your full name, contact address and the capacity in which you appear before the committee?

Dr Tandon: My full name is Maharaj Tandon. I am a respiratory physician of 40 years' standing in Australia. I work at Hollywood Private Hospital as a private consultant. I am also a member, for the past 15 years, of the Industrial Diseases Medical Panel, and for the past five years I have been the chairman of that panel. The Industrial Diseases Medical Panel is an independent government-appointed body, under WorkCover, and we look these days almost exclusively at claimants who have asbestos-related diseases from their work exposure. Our job is to assess their disability quite independently of any other body.

The CHAIRMAN: Thank you, Dr Tandon. You will have signed a document titled "Information for Witnesses". Have you read and understood that document?

Dr Tandon: Yes, I have.

The CHAIRMAN: These proceedings are being recorded by Hansard. A transcript of your evidence will be provided to you. To assist the committee and Hansard, please quote the full title of any document you refer to during the course of this hearing for the record. Please be aware of the microphones and try to talk into them. They are for recording purposes and not for amplification. Please ensure that you do not cover them with papers or make noise near them, which will interfere with the audio.

I remind you that your transcript will become a matter for the public record. If for some reason you wish to make a confidential statement during today's proceedings, you should request that the evidence be taken in closed session. If the committee grants your request, any public or media in attendance will be excluded from the hearing. Please note that until such time as the transcript of your public evidence is finalised, it should not be made public. I advise you that premature publication or disclosure of public evidence may constitute a contempt of Parliament and may mean that the material published or disclosed is not subject to parliamentary privilege.

Dr Tandon, you have been provided with a list of questions, which I am happy to ask as chairman of the committee, and we can get the information that way. However, before we do that, would you like to make an opening statement to the committee?

Dr Tandon: With regard to the health risks from asbestos exposure, in my opinion, with minimal exposure to free asbestos fibres at levels of around 0.001 asbestos fibres per millilitre of air, the risk of development of diseases such as asbestosis, lung cancer and mesothelioma is effectively non-existent. Even if children are allowed to play in areas of crumbling asbestos sheeting, such as roofing, the dangers from falling and injury are greater than any risk of disease from asbestos fibre inhalation. Asbestosis requires prolonged heavy exposure of at least a few months, whereas for mesothelioma, a period of short duration - as little as two weeks - can cause mesothelioma, but this exposure has to be intense, not just a casual, isolated fibre inhalation. The question about the risk of exposure in schools and environmental exposure is, in my opinion, minimal, and I have not come across any such case. I would like to state firmly and clearly that inhalation of a single fibre does

not cause asbestos-related diseases. This opinion is based on not only my personal experience, but also extensive searches of the literature. As stated earlier, I have been practising as a respiratory physician for the past 40 years, and I have not come across a single case, nor have I encountered, in the past 15 years as a member of the Industrial Diseases Medical Panel, any claimant who has had occasional environmental exposure, although there have been cases where people have worked in areas of heavy exposure, such as the Midland railway workshops, where there are much higher levels of exposure. In those cases, asbestos-related pathologies have been seen. In terms of this inquiry, as I understand it, looking at the minimal exposure from crumbling asbestos-containing materials in schools where children may be exposed, the level of exposure is not much greater than that observed in the general environment. If the exposure is much higher, as stated, such as at the Midland railway workshops, where there is a hundredfold higher level, then asbestos-related diseases can result.

The CHAIRMAN: Thank you, Dr Tandon. You have covered some of the ground that these questions appear to be seeking information on. Perhaps if I just restate the questions, you may have something to add, and then we can get the other information that you have. First of all, can you explain how asbestos causes disease, and what diseases are caused by particular types of asbestos?

[11.20 am]

Dr Tandon: I will answer the second part of the question first, because that is relevant. There are two types of asbestos, commonly known as white asbestos and blue asbestos. White asbestos has shorter fibres, is easily cleared from the lung, and has not been shown to cause extensive disease, whereas blue asbestos - crocidolite - is much longer; that is, the fibre size is greater than five microns. This is not easily cleared from the lung, and because it cannot be cleared, it produces irritation. It is ingested by certain cells called macrophages. When these macrophages ingest these fibres through a process known as phagocytosis, they cause further changes to occur. In the case of asbestosis, the fibres turn into fibroblasts, which causes scarring. They also release certain enzymes, such as tumour necrosis factor, which can cause further changes in the DNA and cause malignant changes to occur down the track, after a period of 35 to 40-odd years. These fibres are lying there and are causing chronic irritation and inflammation, especially when these fibres reach the pleural surface. Once they are ingested from the lungs, they travel via the lymph channels to the parietal pleura, where they cause chronic inflammation. Chronic inflammation over a period of 35 or 40 years can, and does, result in the formation of mesothelioma. However, this is not true in all cases. There is some individual propensity for disease to occur, because it is possible for two people to work under identical conditions and for one to develop the more malignant form of disease, which is not as common. What is more common is that simple plaque formation occurs, which is a thickening of pleura and causes only fibrous change in the pleural surface. When these plaques are formed, people are not aware of it. People can have a normal chest X-ray or CAT scan of their lungs for some other unrelated condition, and these are incidental findings. These plaques, if they are very extensive, will cause encasing, so that rather than pliable pleura, the lungs do not move as well, and that can cause some shortness of breath and pain to occur. These are the extreme cases. The important ones are the inhalation of blue asbestos, or crocidolite, fibres, which are longer and are retained in the lung, and which cause fibrosis, or asbestosis; or the more serious and malignant condition, such as mesothelioma. Occasionally, they also cause lung cancer to occur if the same changes that are taking place in the pleura, causing mesothelioma, are happening in the lung. These are some of the things that can happen. However, the most common one is the benign pleural plaques, which are present and are found as an incidental finding.

Hon VINCENT CATANIA: Dr Tandon, I refer to the recent death of Bernie Banton. Would blue asbestos have been the cause of his death?

Dr Tandon: That is exactly what I am saying, because blue asbestos is mined in Australia and South Africa.

The CHAIRMAN: Dr Tandon, you have touched on this, but I will repeat the question. The committee has heard differing views on the health risk of asbestos fibres. One view expressed is that one fibre of asbestos can kill, while another view - for example, a response that the Department of Housing and Works gave to this committee - is that the "scientific and medical communities disagree with this theory. Victims of asbestos diseases usually have had very high exposure levels to asbestos fibres over a long time". Can you comment on this issue any further than what you have already mentioned to us?

Dr Tandon: In my opinion, the view that one single fibre of asbestos can kill is an alarmist view, with no scientific validation. If we believe this particular theory, then we should equally believe that a single passive inhalation of cigarette smoke would cause lung cancer in any single individual, which is almost unheard of, and nobody believes that. I am just trying to draw a corollary between a single fibre and a similar single inhalation of cigarette smoke, either active or passive. It does not happen. However, the reverse view - the second point of view about prolonged extensive exposure, whether it is of short duration for mesothelioma, or for prolonged exposure for the more common asbestosis - is well-established.

Hon ED DERMER: Dr Tandon, I understood from your introductory comments that you were saying that a period of two weeks' exposure might be sufficient to create problems.

Dr Tandon: Intense exposure; it is the intensity that is important.

Hon ED DERMER: Speaking hypothetically, my child may be at school today, and there may be a person doing work in the roof of his classroom, and he may drill three, four, five or six holes through asbestos-containing material. I am talking about blue asbestos, which I understand is more toxic than the white asbestos. An incident such as that would raise the level of asbestos in that classroom for a period of time - maybe an hour or two or a day. Is that something that as a parent I should be worried about?

[11.30 am]

Dr Tandon: No, because the case that I have quoted - the two weeks, which is the only single case reported - involved a person who was working with the production of asbestos inhalation masks. This person was subjected to heavy dust exposure on a continuous basis, not just incidental for an hour or so. The dust comes into the atmosphere and is dispersed. By the time it is dispersed, it becomes so minimal as to be non-existent.

Hon ED DERMER: Would not even a day's exposure at that intensity be likely to be a threat, or a possible threat?

Dr Tandon: Unlikely. I have never come across one, nor is it reported.

The CHAIRMAN: Doctor, are you in a position to indicate to us how much of the asbestos-containing material in schools is blue asbestos-based and how much is white asbestos-based?

Dr Tandon: There is usually a mixture of the two. It is difficult. I am not an occupational physician or a scientist and I do not have actual analysis of the proportional results. But the main thing is that only 10 or 15 per cent of the total material is bound by cement and results in the production of asbestos sheeting. It is difficult to say what proportion of fibres are blue or white. As I said, I am not an occupational physician; therefore, I am unable to comment on that.

The CHAIRMAN: What do you and the medical and scientific community consider is a safe level of asbestos fibres in the atmosphere? What level carries a negligible risk of harm, and is there a level that carries no risk of harm?

Dr Tandon: The level of risk that carries no level of harm, as accepted by the World Health Organization's report of 1986, is less than the 0.001 fibres per millilitres of air. In schools, what we observe is even less. It is 0.0002, which is tenfold less. These levels of fibre concentrations are safe and they are no more than what is seen in the general environment. However, levels higher

than 0.01, which is nearly more than a thousandfold per millilitre of air, are not safe, as I stated in my opening comments may be observed in places such as the Western Australian Midland government railway workshops.

Hon ED DERMER: On that point, Dr Tandon, are you aware of measurements being taken of the concentration of fibres in the ambient level of air in metropolitan Perth or country districts of Western Australia? Can you give us any data on what the normal ambient air concentrations might be?

Dr Tandon: As I said earlier, the ambient concentrations are less than 0.001 fibres per millilitre of air.

Hon ED DERMER: The danger level is 0.01. Therefore, the ambient level of air is about one-tenth?

Dr Tandon: One-tenth.

Hon ED DERMER: Does the ambient level of air vary from district to district? Obviously in a place like Wittenoom the level would be higher.

Dr Tandon: Of course, and that is a different story altogether. Wittenoom would have much higher levels, and it is not safe.

Hon ED DERMER: If we were to go from suburb to suburb or country town to country town -

Dr Tandon: No, I do not think that there would be, other than the area where the fibre is mined. There were studies in Italy that looked at where there was heavy production mining. As they moved away 10 kilometres or so, the fibre concentration was almost non-existent.

Hon ED DERMER: They are returning back towards normal levels?-

Dr Tandon: Yes, towards the normal levels.

Hon ED DERMER: Okay. Therefore, other than in areas where mining has occurred, would the ambient level of air be fairly uniform in different parts of the world?

Dr Tandon: Absolutely.

Hon ED DERMER: Thank you.

The CHAIRMAN: Doctor, this has been covered principally, but the Department of Education and Training has advised that action is taken if airborne asbestos fibre levels exceed one-tenth of the national occupational exposure standard; that is, action is taken if asbestos levels exceed 0.01 fibres per millilitres of air. Do you have any further comment to make on that?

Dr Tandon: No. I would concur with that view, because we are approaching the danger levels of causing disease in the community. In those conditions, actions such as protective coating are essential to minimise that risk.

The CHAIRMAN: To sum it up, would you consider the Department of Education and Training's response to be responsible and safe?

Dr Tandon: As far as I know, they have observed different buildings, and wherever there has been crumbling fibres, they have taken the action of protective coating, and that is a responsible response.

The CHAIRMAN: As far as you know.

Dr Tandon: Yes, as far as I know. As I said, I am a clinician not an occupational or environmental scientist.

The CHAIRMAN: Just moving on to the area of the asbestos survey of schools, as a preamble, you may be aware that a survey to identify and assess the risk of asbestos-containing materials was conducted in WA state schools between November 2006 and July 2007. The survey involved the

visual identification and assessment of asbestos-containing materials. Identified asbestos-containing material was assessed in terms of its physical condition and probability of disturbance, which determined the risk ranking given to the asbestos-containing material. Air monitoring was not undertaken as part of the survey, but is conducted in certain circumstances. Do you have a view on the methodology followed in assessing that asbestos-containing material in state schools?

Dr Tandon: No, I do not have any view on the methodology, because, as I stated, I am a clinician, not an occupational physician or environmental scientist, so I am unable to comment on that.

The CHAIRMAN: Therefore, you cannot comment on air monitoring?

Dr Tandon: No. It was done by appropriate environmental scientists, and I would accept their view. As I said, as a practising clinician, I do not have a view.

The CHAIRMAN: That covers most of the questions. Dr Tandon, do you have a view on whether asbestos-containing materials that have been identified in schools should be labelled and identified?

Dr Tandon: Yes, they should be labelled and identified so that particular attention is made regarding the state of those, and if they are found to be crumbly and possibly releasing any materials, protective coating should be applied, but that is only possible if there is regular monitoring and inspection of those areas.

The CHAIRMAN: Where would you put the threshold?

Dr Tandon: The threshold would be where you find on visual inspection that the surface is loose and crumbly. If something is nice and smooth without any crumbly appearance, it is possibly safe. If it is at all crumbly, it must be protected by adequate protective coating, which may last 10 years or so.

Hon ED DERMER: Is it essentially like a varnish or lacquer?

Dr Tandon: Exactly.

The CHAIRMAN: Do members have any further questions on the survey?

Hon VINCENT CATANIA: Not on the survey. Doctor, you said you have been practising for about 40 years. You may have said this in your opening statement, but during the time that you have been practising, have you come across any schoolchildren or teachers who have contracted any disease through their exposure to asbestos?

[11.40 am]

Dr Tandon: No, I have not yet come across a single case.

The CHAIRMAN: Where do most of your cases originate?

Dr Tandon: Most of the cases I have seen, as I said, were in the past 15 years when sitting on the Industrial Diseases Medical Panel, where the claimants who have had disease from occupational asbestos exposure come up for disability assessment. That is where most of my experience has been, although, as I said, as a practising clinician we see all kinds of diseases. However, I have not come across anyone - an occasional teacher or schoolchild - who has had asbestos-caused problems.

Hon VINCENT CATANIA: Have those cases you have come across in the past 15 years been solely here in Western Australia?

Dr Tandon: Yes.

The CHAIRMAN: Is it possible for you to provide the committee with a breakdown? I know you have to be careful about patient confidentiality, but is a breakdown available in generic terms of the origins of people who have sought treatment at that level?

Dr Tandon: No. As I said, I do not keep any records as to how many patients, where they work, and what has happened. Most of these cases have involved three categories: Wittenoom workers,

James Hardie, and plumbers and other carpenters and where they have been working. The fourth category is the handyman, building an asbestos-sheeting shed and cutting with power tools which release a lot of these things. All of these have required some significant ongoing exposure. These are the classes: number one, Wittenoom; number two, James Hardie; and number three, plumbers and other tradespeople who regularly used power tools - not hand cutters, but power tools - which released the fibres into the atmosphere, which they inhaled, and those occasional, unfortunate people who might have built a little beach shack and have done a lot of cutting of the asbestos fibres during three or four days. That is where the intense exposure has taken place.

The CHAIRMAN: Would any of those patients that you have had dealings with have been contractors working in state schools?

Dr Tandon: They may possibly have been. I do not have any figures in that regard.

Hon VINCENT CATANIA: With regard to power tools and cutting asbestos, if you were to cut the asbestos sheeting of, say, a fence and you were present among a lot of dust from the asbestos, how long would it take to get exposed to the disease - over what period?

Dr Tandon: The question about the fence is an important one, because there you are working in the open with the flow of air, and it is how intensely you are going to inhale that, compared with working in a building in a closed environment, where there is not the free flow of air and there are higher levels of concentration there. This is where it is difficult to work out the proportionality. Again, I come back to the intensity of exposure.

Hon VINCENT CATANIA: I say that because I helped my grandfather on many occasions with an asbestos fence, and I used to hold the nut on the other end while he unscrewed it.

Dr Tandon: Not only you, but I myself in 1977 was helping the installer of a James Hardie fence. I was holding the sheet while he was drilling the holes. I have done that myself.

The CHAIRMAN: Are there any parallels between fibreglass and asbestos?

Dr Tandon: Yes. I have had one patient who had no asbestos exposure. He was a boat builder who presented with fluid on the lung - mesothelioma. That is the only case. It is so vivid when you see it that it sticks in your mind. I have seen one case of boat builder-fibreglass exposure. Fibreglass involves exactly the same mechanism. It does get absorbed and not cleared from the lung.

Hon ED DERMER: You may have answered this question, but I want to ask it specifically. In your 40 years of clinical experience, have you ever had a patient presenting who could not identify any history of exposure for, say, the two-week period that you said earlier was probably the minimum period to provide a risk?

Dr Tandon: I have not come across, although it has been reported, people who have said they have never had any exposure. Do not forget that besides asbestos there are other materials that also cause mesothelioma. We really do not know. We have to be sure, based on pathology, that the disease-causing pollutant is asbestos, not something else.

Hon ED DERMER: You referred to the gentleman involved in the boat building industry and that it may have been fibreglass.

Dr Tandon: Exactly.

Hon ED DERMER: I suppose what I am getting at is that if, in your clinical experience, you say that asbestos-related diseases have arisen only with two weeks' exposure, or more, that is understandable - that is the result of your 40 years' experience. However, if during that 40 years' experience there were patients who were presenting to specialists such as yourself and who arrived with no explanation for where they were exposed, that might raise concern in people's minds that perhaps a lesser period in those instances was the cause of the problem.

Dr Tandon: That may well be so, but I am not able to comment. I can speak only from what I have observed.

Hon ED DERMER: In your time, you have not struck anyone with a total mystery cause?

Dr Tandon: Quite right.

Hon MATT BENSON-LIDHOLM: With regard to children who in the past have been exposed to blue asbestos in places like Wittenoom, as they advance in years are you seeing an increase in the number of people who are reporting with asbestosis and then mesothelioma?

Dr Tandon: Absolutely so.

Hon MATT BENSON-LIDHOLM: What is the time frame within which we can expect those sorts of cases to come to notice?

Dr Tandon: It is a long latent period of 35 to 40 years.

Hon MATT BENSON-LIDHOLM: It does not necessarily have to be that, does it?

Dr Tandon: Not that long.

Hon MATT BENSON-LIDHOLM: I refer to a former friend who grew up in Wittenoom. I think he passed away at about 35 years of age. Would that be unusual?

Dr Tandon: If he was there as a child of five or 10 years and he has had a 20-year latent period, that is quite in keeping with the current knowledge that it can be caused by as low as 20 years, although the usually accepted period is 35 to 40 years. However, it can be a shorter period of up to 20 years following someone who was resident as a child and grew up in the Wittenoom area. As you said, he was 35 years of age. He may have been five or 10 years of age at the time, so 20 years has elapsed before the disease has occurred.

The CHAIRMAN: We have mentioned asbestos and fibreglass. What other materials can lead to mesothelioma?

Dr Tandon: There is another substance called Aronite that can also cause it, but it is not used in the Western Australian environment.

Hon VINCENT CATANIA: Is that a glue?

Dr Tandon: It is like a glue.

Hon VINCENT CATANIA: You can buy that as a glue, I think, in shops.

Dr Tandon: I do not know about that.

Hon ED DERMER: I do not think it is Araldite

The CHAIRMAN: Are you aware of any studies or papers relating to asbestos risk and how to manage asbestos risk that you would like to inform the committee about or provide the committee with?

Dr Tandon: I have provided references from two textbooks; that is, the textbooks of pulmonary diseases, occupational -

The CHAIRMAN: Doctor, can you identify those articles for the purposes of Hansard?

[11.50 am]

Dr Tandon: I have the textbook *Pulmonary Diseases* in which chapter 32 is about asbestosis and silicosis, and the standards of exposure are stated at page 661.

The pathogenesis is also stated; how the asbestos-related disease was caused following inhalation; and the release of various enzymes and how the malignant mesothelioma developed. It is stated quite clearly and underlined that asbestos-induced mesothelioma developed only after intense exposure and not following minimal exposure experienced by urban dwellers. I have taken that

from *Occupational Lung Disorders*, third edition, by Raymond Parkes. It explains how asbestos causes asbestosis. In this regard, it is important to note there is a certain variation, which I stated in my opening remarks. Some people will develop simple benign pleural plaques; others will develop mesothelioma. There is the host response; that is, certain immunological changes that might make individuals more predisposed to the disease as compared with their counterparts who do not have that inherent propensity to develop the disease. The host response is also stated on page 469. As I said earlier, mesothelioma, unrelated to asbestos exposure, is also a recognised factor. Not all mesothelioma is asbestos caused. I have stated the case about a gas worker; that is, he reported two weeks only exposure. I have underlined that. That is where I have taken this book from. There is, of course, the "Asbestos Exposure and Disease - Notes for Medical Practitioners", which was produced by the Occupational Safety and Health Service of the Department of Labour of New Zealand, as well as the asbestos-related diseases article by T. Mosman, published in the *New England Journal of Medicine*, which gives an insight to asbestos diseases and the level exposure required.

The CHAIRMAN: Are all those documents public information?

Dr Tandon: Quite right.

The CHAIRMAN: Thank you. Do you wish to table them for the committee's consideration? Thank you. Doctor, would you like to make any closing remarks to the committee?

Dr Tandon: Yes, I do. All I can say is that I cannot overemphasise the importance of maintenance of asbestos-containing materials. Protective coating is absolutely mandatory and should be carried out wherever there is any possible risk of exposure to the environment, particularly schoolchildren, because they have a long, latent period to go through in their life.

Hon ED DERMER: That is where there is evidence of deterioration; is that right, doctor?

Dr Tandon: That is correct.

Hon ED DERMER: Thank you.

The CHAIRMAN: Just a week or so ago there was an incident in a Western Australian primary school where the asbestos-containing material apparently had not been identified and a contractor drilled into that asbestos-containing material. How can that be managed without the contractors knowing about it if they are putting a light-fitting or something into some material?

Dr Tandon: Again, that is a short incidental exposure, not persistent. If for two or three weeks he was constantly working in that area for eight hours a day doing drilling and cutting with power tools, then that would have an effect, but just not one incidental, single exposure that he was not aware of. There is the difference. This is where I come back to the intensity of the exposure for a minimum of a certain period of time.

Hon ED DERMER: It is both intensity and duration?

Dr Tandon: Exactly.

Hon ED DERMER: I think what you are saying is that a duration of two weeks -

Dr Tandon: A duration of two weeks has been reported, but the exposure was intense.

Hon ED DERMER: For that two weeks?

Dr Tandon: That is right.

Hon MATT BENSON-LIDHOLM: Can there be a cumulative issue here? I note that very early on in your discussion you talked about the ability of the body to expel fibres. Is there a possibility that some work may be being done at a school, and the child then goes home and perhaps - I do not know - a tree branch is brushing against an unprotected eave of an old house and starts causing fibres to be released there? Can there be a cumulative sort of thing where there is a build up in the

system of a child or a person over time if the fibres are not expelled? Can there be an issue stemming from that?

Dr Tandon: That is quite correct. As I have said, it is if it is prolonged and sustained. It is the duration. There are three factors: one is the intensity, the second is the duration, and the third is how intense it is over that period. It is not just a simple, occasional inhalation. If we are talking about an occasional inhalation of a few fibres, then if that had been seen or recorded we would be seeing a lot more such cases coming to light.

Hon MATT BENSON-LIDHOLM: Can you give us any indication of the capacity of the body to expel fibres? Has any research been done on that?

Dr Tandon: I am not aware of it.

Hon ED DERMER: In a sense this is repetitive, but I just want to make absolutely sure. In your 40 years' experience, in terms of patients who have come to you with asbestos-caused diseases, the shortest duration of intensive exposure was two weeks.

Dr Tandon: Quite right.

The CHAIRMAN: I think you have indicated this, but do you have any observations for the committee on the identification, maintenance and treatment program adopted by the Department of Education and Training and the Department of Housing and Works on public buildings, principally state schools?

Dr Tandon: I am not quite clear on the announcement, if you are asking whether or not they are undertaking protective maintenance on a regular basis.

The CHAIRMAN: Yes. Are you in a position to give us a view on that?

Dr Tandon: Basically, as I said, I cannot overemphasise the importance of proper maintenance by protective coating, which is essential to prevent any possible - at present it is negligible in our current state of knowledge. That is where I am saying that wherever there is any danger perceived, then protective coating and active measures should be undertaken.

Hon ED DERMER: Am I right in understanding that if you had a policy of regularly inspecting asbestos-containing materials, and if there was a sign of deterioration in the material that might release fibres and a protective coating was then applied, the universal application of that policy would provide for a safe learning and working environment?

Dr Tandon: That is exactly what I am saying.

Hon ED DERMER: Thank you.

The CHAIRMAN: Is there anything you would like to finish on, doctor?

Dr Tandon: No, thank you.

The CHAIRMAN: Thank you very much. You have been very helpful to the committee and we appreciate your time.

Hearing concluded at 11.59 pm