SUM: The worker appealed a decision denying him a pension for an asthma condition which he developed while employed by a company that produced foam seats for automobiles. He was exposed to toluene diisocyanates in that job. The worker started working with that employer in January 1985 and stopped in March 1985. He was awarded a two-year provisional pension that expired in February 1989. It was not renewed on the basis that the worker no longer suffered from a disability caused by exposure in the course of employment.

There was a conflict in the medical evidence as to whether the worker continued to suffer a disability from occupationally induced asthma. One doctor was of the view that the worker's asthma had resolved after 1985 and that any remaining respiratory impairment was due to the worker's smoking. However, two other doctors were of the opinion that the worker continued to suffer from asthma that was due to workplace exposure.

The Panel found that the worker continued to suffer from a disability caused by asthma. His condition prevented him from accepting employment in environments where he could be exposed to irritants. The worker was unable to participate in vigorous exercise and had to restrict his activities in heat, cold, humidity, and under other circumstances. He was prescribed medication for his condition which he continued to use. This was direct evidence concerning the impairment caused to the worker in his activities of daily living. The preponderance of the medical evidence supported the conclusion that the worker continued to suffer from an asthma condition that was the primary cause of his restrictions. The worker had been exposed to an agent in the workplace that triggered his asthma and he then developed non-specific sensitivity which resulted in asthma that persisted after removal from the occupational exposure.

The Panel did not agree with the medical evidence that there was no measurable disability according to the AMA Guides because the worker's impairment was reversed with medication. The worker's life was circumscribed by his condition, both in the personal and employment spheres. He was prohibited from certain types of employment and recreational activity. He also was forced to continue with medication and he received treatment for his condition. The worker thus had a disability for which he should be assessed by the Board for a pension. The Panel noted that the Board applies the AMA Guides when assessing impairment in asthma conditions. A prior Tribunal decision had found the AMA Guides to be ill-adapted to the measurement of permanent disability in asthma cases. Another Tribunal decision concluded that workers who develop conditions, such as asthma, which restrict their future employment activities are entitled to pensions even if the condition is almost asymptomatic in the absence of exposure.

The appeal was allowed. The worker was entitled to a pension at a rate to be determined by the Board. [16 pages]
TYPE:
DIST:
DECON: 622/90 consd, 740/91 consd
IDATE:
HDATE: 110493
TCO:
KEYPER: L.S. Smith, a lawyer
XREF:
COMMENTS:
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This appeal was heard in London on May 11, 1993, by a Tribunal Panel consisting of:

Z. Onen : Vice-Chair,
R.H. Apsey: Member representative of employers,
F. Jackson: Member representative of workers.

THE APPEAL PROCEEDINGS

The worker appeals a decision of Hearings Officer, N. Holsmer dated August 27, 1992. The Hearings Officer denied the worker entitlement to permanent disability benefits after February 14, 1989, for an asthma condition. The Hearings Officer also determined as the worker no longer had a continuing disability related to workplace exposure. The Hearings Officer also denied continued entitlement to vocational rehabilitation benefits because the worker was deemed to no longer suffer from a compensable condition.

The worker attended the hearing and he was represented by L.S. Smith, a lawyer. The employer did not participate in the appeal.

THE EVIDENCE

The Panel heard testimony under oath from the worker. We also considered the Case Description and an Addendum. In addition, Mr. Smith submitted a research brief consisting of three scientific articles concerning occupational asthma as well as a curriculum vitae for Dr. A. Reinhartz, one of the specialists treating the worker.

THE NATURE OF THE APPEAL

The worker developed asthma in 1985 at the age of 30 while employed with the accident employer as a sorter-packer. The Board accepted entitlement for this disability. The worker was awarded provisional permanent disability benefits at the rate of 10%. These benefits expired on February 14, 1989, and they were not renewed. The Board decided that after this date the worker no longer suffered a disability related to his workplace exposure. The Hearings Officer confirmed this decision on further appeal.

The issue before this Panel is whether the worker is entitled to permanent disability benefits for an asthma condition after February 14, 1989.

In preliminary discussions, we established that in the event the Panel allowed continuing entitlement for an asthma condition, the worker could return to the Board and request vocational rehabilitation services. We therefore did not consider the question of the worker's entitlement to
vocational rehabilitation services in 1990. Mr. Smith pointed out that the worker is currently receiving vocational rehabilitation services from the social service agency.

THE PANEL'S REASONS

(i) The worker's employment experience

The worker developed occupational asthma in 1985 at the age of 30. At the time, he was employed by a company which produced foam seats for automobiles. Shortly after starting this employment in 1984 on a part-time basis, the worker started to develop symptoms he attributed to a cold. These symptoms persisted.

In January 1985, he started to work with the employer full-time. His responsibilities included repair and trimming of foam car seats. The worker's symptoms increasingly worsened until March 1985 when he was advised by his doctor that his symptoms were likely due to exposure to isocyanates in the course of employment. The worker was eventually advised by the specialist who treated him that he could not return to his previous occupation with the accident employer as this would reactivate his asthma condition. In May 1986, the Board reviewed the worker's case, and he was awarded provisional permanent disability benefits at the 10% rate. This award was effective for a two-year period. The worker also received supplementary benefits at the rate of 90%. Prior to this, the worker received temporary total benefits.

The evidence is that the worker was unable to return to work with the accident employer due to his asthma condition. He therefore started to receive vocational rehabilitation assistance from the Board in September 1985. Before his employment with the accident employer, the worker's experience was primarily in factories, or with agricultural producers such as tomato canners. The evidence shows that he had grade ten education, and he had operated a punch press, worked on the assembly line in automotive parts plants, and spray painted, again in a factory setting.

The worker's initial goals in vocational rehabilitation in 1985 were to return to work as a spray painter in an industrial or automotive setting, or as a press operator, or to general work in a warehouse setting. In February 1986, the worker was advised by his family doctor that he should avoid work with spray paint, or in similar environments where the air quality was poor. The worker then redirected his job search efforts.

The worker succeeded in finding employment as a glass blower in 1986; however, this was terminated due to lack of work. The worker also found employment mixing plastics in a moulding plant. He experienced some breathing difficulties in this employment. His employment was terminated after six months due to lack of work. Finally, the worker was employed spray painting in a factory in 1990. After three weeks, he found he was experiencing difficulty breathing, and he was provided with additional medication by his family doctor to control his asthma.
In February 1990, the worker terminated vocational rehabilitation services on the basis that the worker no longer suffered from a compensable disability.

(ii) The worker's evidence concerning his asthma

The worker testified that prior to his employment with the accident employer in 1984, he had not experienced any symptoms such as unexplained coughing, breathing difficulties, or wheezing.

The evidence shows that the worker had suffered from childhood asthma which resolved at approximately age six. The worker stated that he cannot recall his childhood asthma experience. The worker's evidence is that he was an active youth, and participated in sports, including soccer.

The worker has smoked approximately one pack of cigarettes per day from age 15 until he stopped approximately eighteen months ago. He also stopped smoking for approximately nine months after the onset of his asthma.

The worker described the onset of his symptoms while working with the accident employer in 1984 and 1985. He initially started work part-time on week-ends only. Approximately two or three weeks after he commenced this employment, the worker noted symptoms which included congestion and a cough. He used home remedies, including cough syrup, to deal with the symptoms. He did not seek medical attention immediately.

In January 1985 he consulted with his family physician, Dr. P.W. Parry, who diagnosed a cold. The worker started full-time employment in January 1985 with the accident employer. He found it increasingly harder to breathe.

According to the worker, his condition worsened after an explosion in the plant which he believes involved the chemical toluene disocynate. There is no other evidence to connect the worker's asthma condition to this event.

The worker's continuing breathing difficulties resulted in a diagnosis of asthma, and he stopped working with the accident employer in March 1985.

The worker stated that after he stopped working with the accident employer, his condition improved somewhat. A raw feeling in his throat disappeared; however, he was left with breathing problems and a persistent cough.

According to the worker, his condition has essentially remained the same since 1985, with some deterioration in his respiration on exercise. The worker described his condition in testimony. He stated that his condition largely consists of a heavy feeling in his chest as well as an inability to catch his breath on exercise or when exposed to heat, humidity, cold, dust or other such irritants. The worker testified that he walks long distances regularly because he does not have a car. He finds he experiences increasing chest pain while trying to catch his breath while walking. His activities have been reduced. He is unable to accompany his sons to watch hockey games because the cold air activates his condition. During hot and humid days, he must remain in an air-conditioned room to avoid increased problems. His search for employment has been narrowed to the retail or clerical sector,
because he has been advised any work in a factory is likely to aggravate his condition.

The worker testified that his current medication consists of two different types of inhalers. He uses each inhaler four times per day.

(iii) The medical evidence

There is no question that the worker developed occupational asthma during his employment in 1984 and 1985. The Board has accepted this, and the three medical specialists who provided reports concerning the worker all agreed that the employment triggered an asthma condition. There is, however, conflict in the medical evidence as to whether the worker's continued complaints are attributable to asthma and, if so, whether they are disabling.

The evidence shows that as a child, the worker suffered from bronchitis and asthma. All the evidence suggests that the worker's childhood asthma condition did not continue after the age of six. This is the worker's testimony and it is supported by a report from his family physician, Dr. Parry, in June 1985. In this report, Dr. Parry stated that he had treated the worker since October 1978, and that the worker had never complained of difficulty with asthma, tightness in the chest, wheezing or upper respiratory infections except in two documented cases of chest cold.

The worker's first treating specialist was Dr. D. Singh. He first saw the worker in April 1985. He reported to Dr. Parry on his consultation:

**ASSESSMENT AND RECOMMENDATIONS**

1. From history alone one makes the impression that he probably suffers from asthmatic bronchitis on exposure to car foam seats in the factory where he works. It appears to be job related. He has been of work for two weeks and symptoms have settled. On examination there are no objective signs in the chest at present. ...

Relying on the history alone one would recommend that in case he develops recurrent respiratory symptoms on exposure to environment in the factory where he works it will be best for him to change the job. I did not think that he needs any active treatment at present.

Dr. Singh was satisfied, therefore, that the worker's condition was asthmatic bronchitis, likely caused by exposure in the course of employment. Dr. Singh also recommended the worker change jobs.

The worker was next referred by the Board to an internal medicine specialist, Dr. J.H. Toogood. Dr. Toogood's first report to the Board is dated July 30, 1985. In this report, he gave an extensive description of the worker's experience with exposure with the accident employer and the symptoms at that time. Dr. Toogood also noted that the worker smoked cigarettes for 14 years at the rate of a pack a day. After conducting tests including spirometry, Dr. Toogood went on to state:
The changes are very small, but are consistent with obstructive impairment, which was worse at the end of the work shift; improved after a weekend off work; and improved further after 4 months off work. He is not yet quite up to his predicted normal FEV1 ...

This man's history and pulmonary function findings strongly favour a diagnosis of occupational asthma due to exposure to some unidentified factor at his workplace - possibly TDI. Although he describes one instance in the workplace of a "chemical spill" that reputedly released TDI, this did not account for the onset of his symptoms, nor did it exacerbate their clinical course. This happened in February '85.

His persisting mild ventilatory impairment is likely related to his chronic smoking rather than to the presenting complaint of occupational asthma.

Therefore, Dr. Toogood was also satisfied in 1985 that the worker suffered from occupational asthma, together with a coexisting mild ventilatory problem which he thought resulted from the worker's smoking habit.

The worker was once again referred to Dr. Toogood by the Board in August 1988. After this examination, Dr. Toogood reported the worker complained of subjective sensations of dyspnea and that he often had a transient sensation of weight on his chest associated with anxious hyperventilation. He also noted that pulmonary function tests on this examination showed essentially normal values which had not materially changed from those recorded three years previously. Dr. Toogood concluded:

This patient's continuing respiratory complaints are unrelated to either his previous occupational asthma, or to Chronic Smoker's Bronchitis. The exertional dyspnea I would attribute to poor cardiorespiratory fitness plus the gradual weight gain during the past few years. The drop in the PEFR values I would attribute to operator error in his use of the Peak Flow Meter. The non-specific chest discomfort at rest is a mild anxiety symptom likely attributable to situational (family) stress.

We note that Dr. Toogood indicated in this report that there was essentially no change from his review of the worker in 1985. Nevertheless, he concluded that the worker's continuing complaints were no longer due to asthma.

In 1989, the worker was referred to Dr. Singh once again by Dr. Parry. Dr. Singh conducted an examination in September 1989 and he reported the results to Dr. Parry. Dr. Singh noted the worker's complaints included the following:
He continues to complain of wheezing in chest with congestion and cough, worse in the morning, but he also complains of wheezing in chest on climbing one flight of stairs. No nocturnal wheezing. No history of sinus congestion or postnasal drip.

Dr. Singh went on to note that for the past one-and-one-half years, the worker had been experiencing heaviness in his left anterior chest as well as skipping heart beats.

Dr. Singh went on to report the results of pulmonary function tests conducted on this examination:

Pulmonary function tests had shown mild reduction in mid expiratory flows, suggestive of mild small airways obstructive ventilatory defect with significant reversibility after ventolen inhaler.

Dr. Singh then provided Dr. Parry with his assessment of the case:

1. He is suffering from chronic wheezy bronchitis or asthmatic bronchitis. In April 1985 when he had developed these symptoms, he was working at [the accident employer] and it was thought to be occupational asthma. He had stopped working there and was on Compensation Board benefits. He was extensively investigated by Dr. Toogood in London at that time and was reviewed by him again at a later date. The allergy skin test done in 1985 had shown a nonatopic test pattern. Serum IgE level was also normal. Review of his pulmonary function tests in London by Dr. Toogood showed that the values in August 1988 when he was seen by him last time were within normal limits. The pulmonary function tests done in 1985 in Dr. Toogood's clinic were also close to normal. The fact that he continues to have symptoms while he is not working in the same factory makes one wonder about extrinsic asthma unrelated to his occupation. Though his allergy skin tests were negative in 1985, one cannot completely rule out possibility of allergic asthma, though small airways disease due to chronic smoking and exercise asthma may co-exist. He gave history of asthma when he was child, but he grew out of this by age five. He probably had tendency towards bronchial asthma and was most likely precipitated by exposure to chemicals or fumes at [the accident employer] and now he continues to have symptoms even though he is not exposed to the same environment.

Dr. Singh recommended the worker continue with Ventolin inhaler as well as Beclovent. He also concluded that the worker's complaints of chest pain with shortness of breath may be due to anxiety resulting in hyperventilation.
The worker started to see a new family physician, Dr. A. McDonald. Dr. McDonald referred the worker to a new specialist Dr. A. Rheinhartz. Dr. Rheinhartz' curriculum vitae indicates that he has specialised in the practice of neurotoxicology and the effects of solvent exposure, behavioural toxicology, and occupational lung disease.

The worker first saw Dr. Rheinhartz on January 25, 1991. Dr. Rheinhartz conducted a full examination and test of the worker for his respiratory complaints. He conducted a histamine challenge test which showed a moderate histamine induced bronchial spasm. Dr. Rheinhartz concluded:

On the basis of these tests, [the worker] still has symptoms of bronchial hyper reactivity. He still does have asthma and if in fact the Workman's Compensation Board is saying that his asthma is now because he is not exposed to T.D.I.'s is not accurate.

After this, the worker was once again referred by the Board to Dr. Toogood. Dr. Toogood examined the worker and reported on his examination on August 15, 1991. He noted that the worker continued to complain of wheezing and an unproductive cough as well as sensations of chest discomfort. He went on to state that:

Spirometry shows mild obstructive pulmonary impairment which is partially reversible to inhaled bronchodilator ... mild airways hyper responsiveness ... slight hyper inflation, a reduction in diffusing capacity and pa02, and a slight and persisting base access.

Dr. Toogood then went on to consider the results of pulmonary function tests during the past six years. He noted that this comparison showed accumulated damage to the peripheral small airways due to chronic bronchitis and possibly early emphysema. He also noted that the blood gases indicated chronic hypoventilation which could be due to drug usage or specific types of tobacco or other smoke inspiration. He went on to state:

The claimant shows evidence of slowly progressive pulmonary impairment due to advancing chronic airways inflammation, over the 6 year period since I first saw him. It is unlikely this is a consequence of his short period of occupational exposure to TDI in the past. Nor is it likely to be an adverse pulmonary reaction to a drug (clonazepam). More likely it is due to 20 years of exposure to inhaled tobacco smoke - his own and his wife's. The auscultatory crackles are not likely a consequence of repeated micro or macroaspiration of stomach contents since there are no historical features to suggest these have occurred (although he does have symptoms of gastric reflux, and may have a convulsant disorder). They are a very unusual finding in Smoker's Bronchitis, in my experience. They are more likely to be heard in association with chronic marijuana usage, where they reflect damage due to the very deep inhalation followed by a prolonged breath-hold
commonly used to augment peripheralization of the inhaled smoke and systemic absorption of the psycho-active agent. If this same inspiratory technique is used with tobacco, it would be expected to produce the same kind of damage to the peripheral airways—probably mainly in the region of the respiratory bronchioles.

Therefore, Dr. Toogood was of the view in 1991 that any respiratory impairment suffered by the worker was most likely due to his use of tobacco. There is no evidence to indicate that the worker used marijuana or other similar drugs.

We have two reports before us both prepared in 1992 by Dr. Reinhartz. In his report of March 31, 1992, Dr. Reinhartz analyzed Dr. Toogood's reports. He noted that Dr. Toogood reached a different conclusion in his second report of 1991, although the pulmonary function test results were essentially the same. Dr. Toogood was of the view that the worker suffered from occupational asthma in 1985; however, by 1988, he had changed his view and concluded that the worker's impairment was due to smoking. He went on to state:

**DISCUSSION**

1. It is unclear why Dr. Toogood changed the diagnosis first from occupational asthma, to physical deconditioning, to smoker's bronchitis, considering the symptoms and the pulmonary function studies were not materially changed. This point was made by Mrs. N. Holsmer in Memo #92, paragraph 4. In fact, the tests for nonspecific bronchial hyperreactivity were positive in 1988 and in 1991, indicating that his bronchial hyperresponsiveness was increased. This by itself is not diagnostic of asthma, but when used in conjunction with a history of paroxysmal cough, wheezing and shortness of breath, especially brought on by specific triggers like cold frosty air, then this makes the clinical diagnosis of asthma likely. [emphasis in the original]

Dr. Reinhartz provided a further report on November 17, 1992. This report was based on a recent examination of the worker. The worker reported to Dr. Reinhartz that his condition had remained unchanged. Dr. Reinhartz noted that a histamine challenge test which was conducted on this examination showed a reversible reduction in the worker's capacity:

At the time of the histamine challenge, his FEV1 dropped to 1.6 liters at a PC 20 of 2 mg/ml. This reproduced his morning symptoms identically and he has felt short of breath and wheezy with this. He was given some saline and ventolin afterwards, which brought his FEV1 up to 2.4, indicating reversibility.
Dr. Reinhartz went on to state:

As I have mentioned in the past, patients with documented occupational asthma do not, necessarily, recover completely and symptoms may persist, even after a long time out of the exposure. There are no appropriate guidelines for the assessment of disability in patients with asthma and the ones which are used mostly are from the American Medical Association. These guidelines do not take into consideration that patients with asthma have bronchial hyperresponsiveness, which makes it difficult for them to work in areas where there are non-specific lung irritants or cold air. Their asthma can be aggravated by non-specific irritants, even though it was originally caused by a specific sensitising agent. Because of [the worker's] persisting bronchial hyperreactivity, he would not be able to work in an environment high in irritants and in conditions where he is exposed to cold.

The evidence also includes reports by Board doctors on the worker's condition and its cause. The worker was examined by Dr. D.W. Dyer on April 17, 1986. According to Dr. Dyer's report of the examination, the worker stated that his condition had completely resolved and he felt fine. We note that the evidence also shows that, during this period, the worker was attempting to return to employment with the accident employer. He had asked to be placed in an environment where he would not be exposed to TDI, and he had asked about monitoring of his condition on return to work. Dr. Toogood was asked about the worker's desire to return to work as well as a monitoring program. He stated in a letter to the worker dated August 30, 1985:

... I can only recommend that you not return to your previous job in the plant. If a low exposure job could be found for you, now or in the future, you would most likely be able to tolerate that without difficulty.

The worker's evidence is that he continued to experience his asthma symptoms after the onset in 1984. His symptoms did not resolve. On the basis of all of this evidence, we can only conclude that the most likely explanation for the worker's report of resolved symptoms to Dr. Dyer in 1985 is that the worker hoped this would assist in his return to employment with the accident employer. The worker was keen to return to this employment, and the evidence indicates that he made attempts to do so. In any event, he was advised by Dr. Toogood not to do so.

On May 30, 1990, Dr. C.C. Gray, the Board's chest disease consultant, provided an opinion on the worker's condition. He noted that the worker had stated he was free of symptoms in 1985, and that the symptoms resulting from TDI exposure had now resolved. Therefore, in his view, the worker was not entitled to further compensation for any subsequent symptoms. He noted that Dr. Toogood was of the view that any subsequent symptoms were due to the effects of smoking.
Dr. C.R. Woolf of the Board, also a chest disease consultant, provided a further opinion concerning ongoing complaints on November 1, 1991. He stated that he had reviewed Dr. Toogood's report, and he agreed with his analysis that the worker's main problem was progressive airway obstruction resulting from his smoking habit. He noted that the worker exhibited some reversible restriction, but he was persuaded that this was not due to an occupational cause.

In this appeal, we were provided with scientific and other background information concerning asthma, its diagnosis, etiology and assessment. This included a discussion paper prepared for the Tribunal by Drs. R.L. MacMillan and S. Tarlo. The following is an excerpt from this paper:

In suspected occupational asthma, if the worker is removed from the sensitizing agent, the disease will usually remit. It has also been noted that certain asthmatic workers will improve during the weekend when they are out of the workplace, only to have their symptoms return when they begin working again on Monday. This draws attention then to the connection between the exposure and the asthma, but in any individual patient, it can be very difficult to tell whether the agent is responsible or whether the development of the asthma is independent of the exposure. Not everyone with asthma who is removed from the workplace will recover completely. In some workers, the symptoms of asthma persist for the rest of their lives. Once having developed non specific factors, such as exercise, a common cold, irritating dusts and fumes including tobacco smoke, and ingestion of Aspirin. Thus, occupational asthma resulting from sensitization in the workplace may leave the bronchial tree permanently in an irritable state.

A review of the scientific papers before us also shows us that it is generally accepted in the scientific and medical community that substances referred to as isocyanates which are commonly used in certain manufacturing processes result in asthma for some workers. Toluene diisocyanates (TDI) is a form of isocyanate which is commonly found in certain types of manufacturing. Approximately 5% to 10% of workers exposed to TDI develop asthma. The symptoms can develop within weeks or months after the commencement of exposure. Symptoms can develop after exposure to extremely low concentrations of TDI. As we noted earlier, the worker was exposed to TDI while in the employ of the accident employer.

(iv) Conclusions

In 1985, the Board accepted that the worker had sustained a compensable disability which was identified as occupational asthma. He received permanent disability benefits on a provisional basis until February 1989, when these benefits were discontinued on the basis that he no longer suffered from a disability caused by exposure in the course of employment.

There is conflict in the medical evidence as to whether the worker continues to suffer a disability from occupationally induced asthma.

As we already noted, Dr. Toogood was of the view that, after 1985, the worker's asthma had resolved, and that any remaining respiratory impairment was due to his smoking, or other factors. Indeed, Dr. Toogood appears to be of the view that the worker in fact had virtually no appreciable impairment.
The worker was also assessed by Dr. Singh and Dr. Reinhartz. Both of these doctors indicated that the worker presented with a clinical history indicative of asthma. Both doctors essentially reported these complaints throughout their periods of treatment. In 1989, Dr. Singh reported that the worker probably continued to suffer from asthma which was probably due to exposure in the course of employment with the accident employer in 1985. He went on to state that it was possible the worker's asthma coexisted with a small airways disease due to smoking. In this respect, Dr. Singh, like Dr. Toogood, suspected the worker's smoking habit had led to some permanent damage to his lungs which were exhibited on testing. Dr. Singh's 1989 report indicated, however, that he accepted the worker's complaints as asthma symptoms.

Dr. Reinhartz started to treat the worker in January 1991. He conducted a histamine challenge test at that time. This test indicated that the worker had bronchial spasm induced by histamines. He concluded on the basis of the tests as well as his clinical examination and history, that the worker continued to suffer from asthma. Dr. Reinhartz continued to hold to this view in two subsequent reports in 1992. In reporting his conclusions, he noted that workers whose asthma initially started with a workplace exposure can go on to develop non-specific asthma symptoms after they have been removed from the employment and the initial sensitising agent.

The worker's evidence is that he continues to suffer from symptoms which limit his activities. The worker's testimony concerning his symptoms in the course of the hearing was echoed in the reports of Dr. Singh and Dr. Reinhartz. Dr. Toogood reported most of these same symptoms; however, his reports appear to attribute the symptoms in large part to anxiety, or smoking, rather than asthma.

The question before the Panel in this appeal is whether the worker continues to suffer a disability relating to his asthma condition which resulted from exposure in the course of employment. We have concluded, on the basis of our examination of the evidence, that the worker continues to suffer from a disability caused by asthma.

First, the evidence shows that his asthma condition prevents him from seeking or accepting employment in environments where he could be exposed to irritants. Secondly, he is unable to participate in vigorous exercise, and he must restrict his activities in heat, cold, humidity, and under other circumstances. He is prescribed medication for his condition, which he continues to use. All of these facts are direct evidence concerning the restriction or impairment caused to the worker in his activities of daily living. Finally, we are satisfied that a preponderance of the medical evidence supports the conclusion that the worker continues to suffer from an asthma condition which is, at least in part, controlled by his medication, and that this condition is the primary cause of the restrictions suffered by the worker. The evidence persuades us that the worker's asthma condition was initially triggered by his occupational exposure, and that the symptoms have persisted since that exposure. Both Drs. Singh and Rheinhartz were satisfied that the worker's condition was attributable to the worker's occupational exposure. Dr. Singh stated that the worker may also be exhibiting symptoms resulting from changes due to smoking; however, this does not negate his opinion concerning the worker's asthma.
In 1988 and 1991, Dr. Toogood was of the view that any respiratory impairment in the worker was due to his smoking. We note, however, that Dr. Toogood’s view in 1985 was that the worker suffered from asthma which was occupationally induced. Dr. Toogood’s reports do not indicate a clear distinction between the findings in 1985, and those in 1988 or 1991, which would explain the change in his opinion by 1988. He himself stated that the tests, as well as the complaints, remained essentially similar, with some new complaints in 1988 which both he and Dr. Singh attributed to anxiety.

The same findings were accepted by both Dr. Sing and Dr. Rheinhartz as indicative of asthma. Their reports indicated that the worker suffered from a reversible airways condition; asthma. There is also other evidence before us to show that a worker whose asthma is triggered by an agent in the workplace, may go on to develop non-specific sensitivity, resulting in continued asthma even after removal from the occupational exposure. We are satisfied that this happened in the case of this worker.

According to Dr. Woolf, the worker has no measurable disability according to the AMA Guides because his impairment is reversed with medication. We do not agree. The worker's testimony indicates that his life was circumscribed by his condition, both in the personal and employment spheres. He is prohibited from certain types of employment and recreational activity, and he is forced to continue with medication and receives treatment for his condition. As such, the worker has a disability for which he should be assessed by the Board for permanent benefits.

We have therefore found that the worker is entitled to continued permanent disability benefits for his asthma condition, and we remit this case to the Board for an assessment of the extent of the disability. In doing so, we note that the Board applies the AMA Guides in assessing impairment in asthma conditions. We note further that the AMA Guides have been found in the past to be ill adapted to the measurement of permanent disability in asthma cases.

In this respect, we point out that a previous decision of the Tribunal considered the question of permanent disability assessments for asthma according to the AMA Guides. In Decision No. 740/91, a panel reviewed the AMA Guides and concluded that they are inadequate for the assessment of impairment caused by asthma. The decision stated in part:

As we noted earlier, we heard expert medical testimony from Dr. S. Tarlo, who had also examined the worker. Dr. Tarlo described asthma as a condition for which there is no one definition which is universally accepted. She defined the condition as one which is manifested by variable airway narrowing, fluctuating spontaneously or due to intervention. This intervention can be either environmental or medical. The narrowing of the airways is associated with inflammation. Diagnosis is usually based on typical symptoms of expiratory wheezing, shortness of breath, chest tightness, and cough which are reported by the patient to the doctor. There are also objective
tests generally referred to as 'pulmonary function tests' to demonstrate airway narrowing.

Dr. Tarlo and others have commented that the emphasis placed by the AMA guides on objective documentation of a reduction in lung capacity is inappropriate for evaluating impairment in asthma. In a condition such as asthma, there may not be any permanent reduction in lung capacity as a result of the condition. By its very nature, asthma is a variable condition.

We note further that other decisions of the Tribunal have concluded that workers who develop conditions, such as asthma, which restrict their future employment activities, are entitled to permanent disability benefits even if the condition is almost asymptomatic in the absence of exposure. In Decision No. 622/90, the panel considered whether a worker who developed mild asthma after exposure to dust in the course of employment was entitled to permanent disability benefits:

... Decisions of the Tribunal have held that the term "disability" as used in section 45 includes situations where a worker is unable to return to a particular job (see, for example, Decision No. 59/90 (1990), 15 W.C.A.T.R. 132). In this case, it would not appear that the worker's disability away from the workplace is significant. However, her condition is such that it would be severely aggravated if she returned to working in a dusty environment. In this way, she would suffer a "disability" which could trigger a permanent partial disability award.

The panel went on to find that the worker was entitled to a permanent disability award.

In this case, we direct the Board's attention to these considerations in its assessment of this worker's level of permanent disability, and any questions arising out of this order may be returned to the Panel for clarification.

THE DECISION

1. The appeal is allowed.

2. The worker is entitled to permanent disability benefits for his occupational asthma. The Board is to determine the rate of the worker's benefits. Any questions arising out of this order may be returned to the Panel for clarification.

DATED at Toronto, this 7th day of June, 1993.

SIGNED:  Z. Onen, R.H. Apsey, F. Jackson.