



**Report to the Workers'
Compensation Board on
Respiratory Complications
among Workers Receiving
Compensation for Non-malignant
Respiratory Disease**

March 1993

Industrial Disease Standards Panel
IDSP Report No. 11
Toronto, Ontario

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Industrial Disease Standards Panel

In 1985 the Ontario legislature established the Industrial Disease Standards Panel (IDSP) to investigate and identify diseases related to work. The Panel is independent of both the Ministry of Labour and the Workers' Compensation Board. At the end of each fiscal year the WCB reimburses the Ministry for the Panel's expenditures.

The Panel's authority flows from section 95 of the *Workers' Compensation Act* and its functions are set out as follows:

- (8) (a) to investigate possible industrial diseases;
- (b) to make findings as to whether a probable connection exists between a disease and an industrial process, trade or occupation in Ontario;
- (c) to create, develop and revise criteria for the evaluation of claims respecting industrial diseases; and
- (d) to advise on eligibility rules regarding compensation for claims.

Decisions of the Panel are made by its members who represent labour, management, scientific, medical and community interests. Once the Panel makes a finding, the WCB is required to publish the Panel's report in the Ontario Gazette and solicit comments from interested parties. After considering the submissions the WCB Board of Directors decide if the Panel's recommendations are to be implemented, amended or rejected.

To assist with its work the Panel has a small staff of researchers, analysts and support people. In addition to its own staff, the Panel relies heavily on the advice of outside experts in science, medicine and law, as well as input from the parties of interest.

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Report to the WCB on Respiratory Complications among Workers NMRD

Panel Membership

<i>Panel Members</i>	<i>Appointment</i>
Ms. Nicolette Carlan (Chair)	May 16, 1991 to May 15, 1994
Dr. Carol Buck	June 1, 1991 to June 16, 1994
Mr. James Brophy	January 23, 1992 to January 22, 1995
Mr. William Elliott	November 7, 1991 to November 6, 1994
Ms. Nicole Godbout	December 16, 1992 to December 15, 1995
Mr. John Macnamara	November 7, 1991 to November 6, 1994
Mr. Homer Seguin	May 28, 1992 to May 27, 1995
Dr. Michael Wills	November 7, 1991 to November 6, 1994

Report to the WCB on Respiratory Complications among Workers NMRD

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March 25, 1993

Mr. Odoardo Di Santo
Chair
Workers' Compensation Board
2 Bloor Street East, 20th Floor
Toronto, Ontario
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Dear Mr. Di Santo:

Enclosed is a copy of the Panel's "Report to the Workers' Compensation Board on Respiratory Complications among Workers Receiving Compensation for Non-malignant Respiratory Disease".

I would be pleased to discuss the Report with you. Please let me know when it would be convenient to do so.

Sincerely,

Nicolette Carlan
Chair

Enclosure

C60776 0009

The Nature of the Issue

It is well known that the recorded cause of death, especially in complex, multifactorial cases, is frequently arbitrary or misdiagnosed. Adjudicatory problems surrounding the recorded cause of death can result in failure to recognize the compensable respiratory disease as a significant contributing factor in the death of the worker with resultant non-payment of survivor's benefits to dependents.

The widespread effects on the body as a result of disease may be more complicated than the effects of a single traumatic injury. Other health problems experienced by workers who have contracted a work-related disease are often exacerbated by the disease. These health problems however, are not a direct consequence of work.

The Industrial Disease Standards Panel has dealt with one such issue in its Report No. 10, *The Report to the Workers' Compensation Board on Cor Pulmonale* (1). In that report, the Panel found a probable connection between a compensable respiratory disease and the development of cor pulmonale, or right heart failure. This condition, though not directly caused by work, nevertheless may develop as a secondary condition.

The Board has in place a policy (Document No. 03-04-02) dealing with secondary conditions resulting from a work-related disability. The policy reads:

"Workers sustaining secondary conditions that are causally linked to the work-related injury will derive benefits to compensate for the further aggravation of the work-related disability or for new injuries" (2).

Concern has arisen however, that workers receiving compensation for a non-malignant respiratory disease [NMRD] sometimes fail to have secondary conditions causing deteriorating lung function recognized and compensated accordingly during life. Furthermore, it is well known that the recorded cause of death, especially in complex, multifactorial cases, is frequently arbitrary or misdiagnosed. Adjudicatory problems surrounding the recorded cause of death can result in failure to recognize the compensable respiratory disease as a significant contributing factor in the death of the worker with resultant non-payment of survivor's benefits to dependents.

In developing this report, the Panel has attempted to address three issues:

- 1) Do workers commonly experience other respiratory problems secondary to compensable non-malignant respiratory diseases [NMRD]¹?

¹ Non-malignant respiratory disease, for the purpose of this report, includes both restrictive and obstructive diseases. Restrictive disease refers to the restriction of the volume of air in the lung. Restrictive disease can involve the alveoli or air sacs and supporting tissue. Examples of compensable restrictive lung diseases would be asbestosis or other pneumoconioses. Obstructive disease indicates an obstruction to the flow of air through the tubes in the lung. Potentially compensable examples of obstructive disease are emphysema, chronic bronchitis and asthma. Some diseases show both patterns. (For a discussion of the normal lung function and how various diseases impinge on that functioning see Appendix A.)

- 2) If the answer to issue one is yes, are these additional respiratory problems likely to result in increased disability and mortality?
- 3) What is the likely effect of a compensable respiratory disease when the worker suffers from other concurrent non-occupational respiratory disease?

The Panel is well aware that these are difficult questions, because although some occupational lung diseases are distinct entities, "most work-related respiratory morbidity and mortality is the result of an increase in bronchitis, emphysema, lung cancer and tuberculosis, in which non-occupational factors also play an important part" (3). Indeed, according to the former Advisory Committee on Occupational Chest Diseases, "concurrent non-occupational respiratory disease is the biggest assessment confounder for the ACOCD" (4).

How the Panel Proceeded

If underlying respiratory disease has damaged this system, then clearance of inhaled infectious agents or of materials produced during the response to infection may be impaired, making it easier to acquire an infection and more difficult to get rid of it.

Background Information

To assist the Panel in developing the rationale for this report, the Panel asked Dr. Linn Holness of the Department of Occupational and Environmental Health, St. Michael's Hospital, to provide, in lay terms, an explanation of normal lung function [Appendix A] and an explanation of the impact of lung disease on lung function (5). The latter text on adverse outcomes, with modifications, follows.

The respiratory tract has a number of ways by which it protects itself against infections. In the airways, these defense mechanisms include coughing and mucociliary clearance. The mucociliary clearance mechanism consists of the appropriate type of mucus to carry inhaled agents out of the lung and the ciliated cells (cells with hair-like projections which beat in sequence) to move the mucus up the respiratory tract to be expelled by coughing or swallowing. If underlying respiratory disease has damaged this system, then clearance of inhaled infectious agents or of materials produced during the response to infection may be impaired, making it easier to acquire an infection and more difficult to get rid of it. Chronic bronchitis and asthma may affect these airway defence mechanisms. If coughing is depressed or ineffective this may decrease the removal of potential infectious agents and also make the clearance of mucus more difficult once the infection has occurred.

In the lower respiratory tract, the air sac (alveolar) and lung tissue (parenchymal) region has several methods of defence. Alveolar macrophages (cells which consume inhaled materials) help to clear the air sacs. Inflammatory and immune responses are also important in defending this area. The impairment of cell mediated immunity is particularly important for tuberculosis. Diseases which affect this region, such as silicosis and asbestosis, may result in impairment of these defence mechanisms and make it more difficult to fight infection.

In chronic lung diseases some of the above mechanisms may be impaired, making it more likely that the affected individual will develop an infection and also making it more difficult to fight the infection. Further, the impairment resulting from the lung infection, added to the impairment from the underlying lung disease may be enough to reduce severely the individual's ability to transfer oxygen and carbon dioxide adequately.

In general, individuals with occupational lung disease with significant impairment may still be able to manage if they have no other lung problems. For example, they may have a moderate reduction in their lung function which reduces the amount of oxygen in their blood, but even though it is reduced there is still enough for them to function. However, other additional respiratory problems, either acute problems like pneumonia, or chronic, such as chronic obstructive lung disease, which cause additional impairment in lung function, may reduce their lung function to a critical level. This results in respiratory failure which is characterized by an insufficient level of oxygen in the blood.

Medical Opinions

As part of its investigation, the Panel wrote to three respirologists and posed several questions concerning compensable respiratory illness.

In one of those questions, the respirologists were asked to address the following issue:

Does a compensable respiratory illness affect the worker's ability to deal with other non-work related respiratory illnesses such as pneumonia or bronchitis?

[N.B. The classes of respiratory impairment referred to below are those outlined in the American Medical Association Guides to the Evaluation of Permanent Impairment (6).]

- (1) "Yes, it is well established that many of the occupational lung diseases such as asbestosis, silicosis, asthma, byssinosis are associated with excessively frequent lung infections. It is therefore appropriate that any such acute condition be compensated and if causing additional loss of function, compensation should be applicable as occupationally related. Death under such circumstances should be accepted as work related. Again, I believe that this opinion would reach general agreement for cases in the Classes 3 & 4, but would have to be considered individually for cases in the Class 2" (7).
- (2) "The answer to this is an unequivocal "yes." For example, acute silicosis, as found in sandblasters, predisposes to acute pneumococcal pneumonia. Any chronic lung condition makes an episode of pneumonia or acute bronchitis more dangerous, and may lead to a further decline in lung function" (8).

Any chronic lung condition makes an episode of pneumonia or acute bronchitis more dangerous, and may lead to a further decline in lung function.

- (3) "Were a subject with severe impairment of lung function due to asbestosis, complicated silicosis, complicated coal workers' pneumoconiosis, or hard metal disease to develop pneumonia, it would certainly jeopardize to some extent his chance of recovery. There is little doubt that this is true. In regard to bronchitis, this is not so in that this condition produces little or no effect on lung function. I would stress that even were pneumonia to occur, it would only be those subjects who have occupational disease that is far advanced and associated with fairly severe respiratory impairment that there would be a significant effect either on prognosis or the duration of morbidity" (9).

In summary, there was general agreement that many non-malignant respiratory diseases are associated with excessively frequent lung infections. Opinion differed as to the likely consequences of a bout of acute bronchitis. There was also general agreement that the compensable respiratory condition would have to be fairly severe [AMA Class 3 or 4] before there would be a significant effect on prognosis. One physician felt that workers whose impairment was rated as a Class 2 would have to be evaluated on a case by case basis if death resulted from a complicating lung infection.

The Literature

According to Board statistics, the majority of awarded claims for NMRD are for asbestosis, silicosis and occupational asthma. These diseases will therefore be the focus of our review.

Deaths among Asbestotics and Silicotics:

There is no question that victims of asbestosis frequently suffer from a shortened life-span.

Likewise, compensated silicotics showed reduced life expectancy due largely to deaths from tuberculosis and non-malignant respiratory disease.

There is no question that victims of asbestosis frequently suffer from a shortened life-span. The five-year survival of workers receiving compensation for asbestosis was only 69% of that of age-matched Ontario males. The ten-year survival of these workers was only 53% of that of the age-matched Ontario males (10). Likewise, compensated silicotics showed reduced life expectancy due largely to deaths from tuberculosis and non-malignant respiratory disease (11).

Brown et al. studied "the Homestake cohort" of 3,328 white gold miners who worked full-time underground for at least a year between 1940 and 1965 (12). Exposure to silica as measured by total dust was strongly associated with mortality due to respiratory tuberculosis [SMR = 364] and NMRD [SMR = 279]. The researchers also looked at a subcohort of workers first employed after 1951, when dust levels were thought to have been substantially reduced. Although limited by small numbers [only 109 deaths] the authors report "it appears that the pattern of mortality by latency and length of underground employment for non-malignant respiratory disease is similar to that seen in the entire

cohort". The risk for NMRD was high with an SMR of 286 [based on 4 deaths observed vs. 1.4 expected]. Based on pathology reports, at least two-thirds of the individuals in the large cohort who died from NMRD had a confirmed diagnosis of silicosis. In the post-1951 group, of the four deaths from NMRD, none had silicosis recorded on their death certificates.

Finkelstein et al., studied the mortality of 1190 Ontario miners with compensable silicosis and found an SMR of 180 for all causes (11). For NMRD, the overall SMR was 737, and the subgroup with the longest standing disability, and hence the most complete mortality profile, showed an enormous SMR of 1,140.

Schuler and Ruttner studied selected causes of death among 2,399 certified or compensated silicotics (13). Forty percent of the deaths were from NMRD including bronchitis and emphysema while the corresponding figure for the general population was reported as at most 10-15%.

In Italy, Zambon et al. also investigated 1,234 compensated silicotics with 697 deaths (14). There was a large excess of deaths due to infectious disease (8th/ICD: 001-136), SMR of 1,960 including tuberculosis and silico-tuberculosis. There was also an excess due to respiratory diseases including silicosis (ICD codes 460-519), with an SMR of 741. The authors state that tuberculosis, silico-tuberculosis and silicosis account for almost all of the excess mortality in the silicotics.

Cookson, Musk, et al. studied the mortality experience of 354 claimants for compensation for asbestosis who had worked in the Wittenoom crocidolite mine and mill in Western Australia (15). All-cause mortality in the cohort had an SMR of 265, and the authors state that "pneumoconiosis, malignant pleural mesothelioma, respiratory cancer, bronchitis and emphysema, and tuberculosis all contributed to excess mortality." They speculate that the increase due to bronchitis and emphysema could be "due to unrecognized pneumoconiosis and misclassification of the cause of death, or to a high proportion of cigarette smokers in the population". However symptoms of bronchitis have been reported among non-smoking asbestotics (16) and air-flow limitation may be caused by peribronchial fibrosis resulting from asbestos itself (17-20). Number of radiographic opacities, age at claiming compensation, work in the mill, and degree of disability awarded by the Pneumoconiosis Board were predictors of survival but estimated total exposure to asbestos was not.

The mortality of 1072 workers from the register of compensated silicotics in Quebec who first received compensation between 1938 and 1985 was investigated by Infante-Rivard, Armstrong et al. (21). Only 27.7% of those compensated were miners, with 44.9% from foundries, 13.2% granite workers, 4.8% pottery, and the rest miscellaneous or unknown. The all-cause SMR was 216 with a 3-fold risk of death from lung cancer, SMR = 347. Other elevated SMRs included infectious diseases at 2974, and non-malignant respiratory disease at 975. Causes of

death in the cohort were malignancies (22.6%), pneumoconiosis (18.6%), tuberculosis (13.9%), myocardial infarction (10.6%) and chronic lung disease (7.1%). Other causes accounted for 23.5% of deaths.

In one of the few studies that provides a detailed breakdown of deaths from NMRD, Ng, Chan and Lee assessed mortality among 1419 subjects in the Hong Kong Silicosis Register from 1980-1986 (22). The following are the relevant results obtained:

<i>Cause of Death (ICD9)</i>	<i>SMR</i>
All cause	302
Infectious Diseases	252
Pulmonary Tuberculosis	383
Respiratory Diseases	1269
Pneumonia	295
Chronic bronchitis	
Emphysema and Asthma	745
Chronic airway obstr.	770
Pneumoconiosis	60994
Others	236

Additional Analyses on Ontario Workers Receiving Compensation for Silicosis or Asbestosis

At the request of Panel Staff, Dr. Murray Finkelstein of the Ministry of Labour Health Studies Branch reviewed his data on compensated asbestotics and silicotics to ascertain if the excess mortality in these two cohorts were attributed to the compensated respiratory disease alone (23). For the asbestotics, the respiratory causes of death were all coded to pneumoconiosis, except for bronchopneumonia and tuberculosis. The SMRS for all non-silicosis respiratory diseases were as follows:

<i>SMRs for Non-Malignant Respiratory Disease</i>				
<i>Cohort</i>	<i>Obs</i>	<i>[Total]</i>	<i>Exp</i>	<i>SMR</i>
1940-49	21	[118]	10.9	193
1950-59	13	[78]	11.3	115
1960-69	20	[54]	9.3	215
1970-78	5	[14]	2.8	179
<i>Total</i>	59		34.3	172

Thus out of a total of 264 deaths attributed to non-malignant respiratory disease in this cohort, 59 or 22% were attributed to respiratory disease other than silicosis.

Occupational Asthma

The inherent difficulty in assessment of impairment/disability in patients with occupational asthma is widely recognized. The American Medical Association guidelines (Guides) for evaluation of impairment are widely used by Compensation Boards but have come under criticism as inappropriate for patients with asthma who have variable air-flow obstruction (24). Morbidity and mortality from asthma have been increasing and there has been speculation that one of the contributing factors to the deaths has been a failure on the part of physicians to recognize the true severity of the condition in an individual patient (25). Older adults with asthma show increased mortality in the winter months which may be due to the frequent association with chronic obstructive lung disease (26). Respiratory infections contribute to mortality among asthmatics (27).

From 1979 through 1988, the annual asthma mortality rate in Prince Edward Island exceeded the national rate for all years except 1980. This prompted Sweet et al. (28) to undertake a study, one of the aims of which was to validate the diagnosis for all asthma deaths in P.E.I. between 1984 and 1988. The researchers obtained information from autopsy reports, death certificates and hospital records. A total of 34 deaths were reported to have been caused by asthma. Although the number of cases was small, the study is relevant in that it showed that the recorded deaths of patients who die at age 50 or less are more likely to be valid than the records of older patients, who typically have other co-morbid conditions such as chronic obstructive lung disease and cardiovascular disease. The authors conclude that the recorded cause of death on the certificate which was used and is the standard form for all of Canada was frequently invalid due to physician error and misclassification of the causes of death resulting from the correct use of international coding rules which give precedence to certain causes of death regardless of the way they are recorded on the death certificate. Once again, the impossibility of relying on death certificate data alone to determine the true cause of death is highlighted.

Many patients diagnosed with chronic obstructive pulmonary disease have a combination of chronic bronchitis, asthma, and emphysema (29). Complicating upper respiratory infections are frequent (30). Although smoking is the most prominent etiologic agent, other contributing factors identified include air pollution, respiratory infections and occupational exposures (31). Furthermore, it is estimated that only one-half of all patients dying of chronic airways obstruction are correctly designated on death certificates (31).

Panel Conclusions

It is clear that workers suffering compromised lung function due to occupational respiratory disease are more likely to suffer from more frequent and more severe respiratory infections such as pneumonia than the general population.

For compensation purposes, the issue remains, did the compensable non-malignant respiratory disease lead to complications during life resulting in more severe lung impairment, and did the NMRD make a significant contribution to the worker's death?

The Panel is in agreement with the statement that the boundary between occupationally caused, work-aggravated and non-work-related lung diseases is "indistinct, reflecting the multifactorial nature of the diseases" (27). Nevertheless, in our view, it is clear that workers suffering compromised lung function due to occupational respiratory disease are more likely to suffer from more frequent and more severe respiratory infections such as pneumonia than the general population. This is supported by the medical opinions we solicited and by the medical literature.

In addition, the consistently elevated SMRs in the literature for all manner of respiratory complications among workers with compensable respiratory disease strongly supports the position that such workers are much more likely to die from such complications than the general population. Even when studies among workers compensated for a respiratory disease indicate a deficit of deaths from a particular type of NMRD such as emphysema or bronchitis, one cannot conclude that this is in fact the case. It is possible that such deaths are actually subsumed among other groups, such as deaths attributed to silicosis, for example, reflecting the frequently multifactorial nature of the deaths.

Finally, it would seem only common sense that an individual's lung function reflects the cumulative impact of the insults it has received. As our consultant has stated: "individuals with occupational lung disease with significant impairment may still be able to manage if they have no other lung problems. However, if they have additional impairment, [including non-work related impairment], their lung function may be reduced to a critical level". The Panel is convinced that fair public policy demands compensating those individuals whose ability to survive their cumulative lung damage is seriously compromised by the existence of a compensable respiratory impairment.

Panel Recommendations

The Panel has already considered the occurrence of Cor Pulmonale among those with recognized claims for non-malignant respiratory disease. In some ways, cases of Cor Pulmonale can be viewed as a subset of what would appear to be the larger problem of correct determination of those complications of compensable NMRD that lead to increased disability or death among workers. The Panel is aware that certain respiratory diseases can and do arise quite independently of any workplace exposure.

The Panel has already discussed many of these issues in its deliberations surrounding Cor Pulmonale. The logic requiring a thorough investigation into the cause of death to identify Cor Pulmonale among workers in receipt of benefits for non-malignant respiratory disease is equally applicable to the larger group.

The Panel is aware of the Board Policy (Document No. 03-04-02) which deals with secondary conditions resulting from a work-related disability. The policy reads:

"Workers sustaining secondary conditions that are causally linked to the work-related injury will derive benefits to compensate for the further aggravation of the work-related disability or for new injuries".

However, the Panel is of the opinion that this policy and its implications for additional compensation are not well-known to either workers or their physicians.

As a result of these deliberations the Panel was able to make findings and recommendations aimed at assisting the Board in the adjudication of these difficult claims.

The Panel finds that workers who suffer from compensable non-malignant respiratory diseases for which WCB benefits for impairment have been paid, are likely to suffer from additional respiratory problems. Specifically for those workers the Panel finds:

1. A probable connection between the occurrence of non-malignant respiratory disease and the occurrence of respiratory infections.
2. A probable connection between the increased incidence of infection and an increased impairment during life. Furthermore, among these workers there is a greater probability of death resulting from respiratory infections.
3. An increase in mortality from concurrent non-compensable non-malignant respiratory disease as a result of the cumulative impact on lung function.

Report to the WCB on Respiratory Complications among Workers NMRO

In accordance with these findings the Panel recommends:

1. The Board should notify workers in receipt of a WCB award for an impairment due to non-malignant respiratory disease and their physicians of the existence of Policy Document No. 03-04-02. This will ensure that eligible workers are aware that additional entitlement may be payable for secondary respiratory conditions.
- 2a. The Board should investigate the cause of death for all workers who during their lifetimes were in receipt of benefits for a non-malignant respiratory disease.
- 2b. Notwithstanding the recorded cause of death, if the compensable non-malignant respiratory disease is judged to have significantly contributed to the death of the worker, the appropriate dependency benefits should be paid.

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Appendix A— Lung Function

The tissues of the body require oxygen to function. The carbon dioxide they produce must also be removed. The transfer of oxygen and carbon dioxide between the tissues and the external environment is accomplished by the coordinated action of the lungs and cardiovascular system under the control of the nervous system.

The main function of the lungs is the transfer of oxygen and carbon dioxide between the blood and the air. Air which contains oxygen is inhaled into the lungs and transferred to the blood and red blood cells in the small blood vessels in the lung. Similarly, the carbon dioxide produced in the body is transported through the blood vessels to the lung where it is transferred to the air which we exhale.

The air moves into and out of the lungs through a branching structure of tubes or airways. As the airways branch the individual airways become progressively smaller in diameter. The large central airway is called the trachea which divides into bronchi and the small airways are bronchioles. The air moves through the airways by the bellows-like action of the chest. The airways end in many tiny air sacs called alveoli which have thin walls and a rich supply of small blood vessels called capillaries. Because of the large number of alveoli there is a large surface area for the transfer of the oxygen and carbon dioxide.

There are three components involved in the normal functioning of the lungs.

1. Ventilation—the movement of the air in and out of the lungs
2. Diffusion—the transfer of gases between the alveolus and the capillary
3. Perfusion—the flow of blood through the lungs

Ventilation depends on three factors;

1. the size or volume of the lungs;
2. the expansibility or elasticity of the lungs, and
3. the ability to move the air through the airways which is related to the resistance to flow in the airways.

The factors may be assessed by various lung function tests. A number of different volume measurements can be made. Some of the common lung volumes assessed include:

Static Lung Volumes

1. Total lung capacity (TLC)—the total amount of air in the lungs at the end of a full inspiration

2. Vital Capacity (VC)—the amount of air that can be expelled after a full inspiration
3. Residual Volume (RV)—the amount of air left in the lungs at the end of a full expiration
4. Tidal Volume (V_T)—the amount of air expired in a normal breath
5. Functional Residual Capacity (FRC)—the amount air left in the lungs at the end of a normal expiration

Dynamic Lung Volumes

(volumes measured with a forced, rapid expiration)

1. Forced Vital Capacity (FVC)—the amount of air that can be forcibly and rapidly exhaled after a full inspiration
2. Forced Expiratory Volume in 1 Second (FEV_1)—the amount of air expelled in the first second of expiration

The expansibility or elasticity of the lungs can be measured as the compliance of the lung. This test is more difficult to do (the individual has to swallow a balloon) and is not done on routine lung function testing.

The ability to move air through the airways can be assessed in several ways. A simple measure is the ratio of FEV_1 to FVC which compares the amount exhaled in the first second with the total amount of air exhaled during a forced, rapid expiration. The flow of air can also be measured at various lung volumes. Common flow rates measured include:

1. Forced Expiratory Flow between 25% and 75% of the FVC (FEF_{25-75})
2. Forced Expiratory Flow at 50% of the FVC (FEF_{50})
3. Forced Expiratory Flow at 75% of the FVC (FEF_{75})

The FEF_{75} represents flow in the smaller airways. Airway resistance can also be measured. Most of the resistance to airflow occurs in the larger airways. Therefore, airway resistance will not necessarily detect abnormalities in small airways unless the abnormality is large.

Diffusion or the transfer of gas between the air in the alveolus and the red blood cells in the capillaries depends on four factors:

1. the difference in the pressure of oxygen between the alveolus and the capillary

2. the size of the barriers that the gases have to pass through which include the fluids and cells lining the alveolar space, the alveolar membrane, the capillary membrane, the fluid or plasma in the capillary, the membrane of the red blood cell and the fluid inside the red blood cell,
3. the surface area available for the transfer to take place,
4. characteristics of the blood (e.g., the number of blood cells available).

The diffusing capacity for carbon monoxide ($D_L CO$) provides a measure of the overall diffusion of gas from the inhaled air to the red blood cell.

Perfusion depends upon the flow of blood through the blood vessels in the lung. Diseases which affect the blood vessels in the lungs such as blood clots (pulmonary emboli) may impair perfusion.

Another simple test which is performed which provides an overall assessment of the function of the lung is the measurement of the amount of oxygen and carbon dioxide in arterial blood expressed as the partial pressure oxygen and carbon dioxide (PaO_2 and $PaCO_2$).

There are many different types of lung disease which may adversely affect lung function. Two main types of abnormalities which are often described are obstructive and restrictive defects.

An obstructive defect results in the decrease in the FEV_1/FVC ratio and flow rates and an increase in resistance. These abnormalities would be found in chronic bronchitis with associated airway obstruction, asthma and emphysema.

A restrictive defect results in a decrease in lung volumes. The FEV_1/FVC ratio is maintained even though the actual volumes are decreased. Typical restrictive lung diseases are interstitial disease such as idiopathic pulmonary fibrosis, asbestosis, and hypersensitivity pneumonitis.

Defects in diffusing capacity may be found in some obstructive disease (emphysema) and restrictive disease (idiopathic pulmonary fibrosis, asbestosis, and hypersensitivity pneumonitis).