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File statistics [illegible]

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July 13, 1978

BERLIN EMPLOYEE
STATISTICS ESTIMATE

J. L. Konzen - [illegible]
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We have reviewed the employee records at Berlin relative to the questions raised about potential notification on asbestos exposure for former employees. The data summarized below is an estimate based on a statistical review of approximately 50% of the names on record. These figures exclude retired employees, and covers those people employed prior to 1973. We would expect that at least two-thirds of these people could be notified by their addresses on record or updated by current telephone directories.

1 year or less	1079
1 - 5 years	291
6 - 10 years	92
Over 10 years	46
TOTAL	1508

1) Give exam to retiree (Dick)
2) Greater 1 yr & invite to be exam " " (Jon)
all

/jac

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1. The definition of "abnormal". For example, if radiological abnormalities in category 1 of the ILO U/C classification are regarded as abnormal the proportion of cases found to have asbestosis on this basis will be much greater than if only categories 2 and above are regarded as abnormal (51). Similarly, the level at which a lung function test is said to be "abnormal" will influence the proportion of abnormal cases identified in a study population.

2. The age distribution of the study population. Chest radiographs are more often abnormal, and most lung function tests decrease with age. It is therefore important that any test results reported are adjusted for this factor before the findings are generalized. Many studies report the proportion of workers with abnormal findings without adjusting for age, thus making it difficult to compare study findings (25,37,46).

3. The duration of time since onset of exposure. Although asbestosis may develop within a few years of intense exposure to asbestos - e.g. spraying 100% asbestos insulation (77), the disease usually becomes apparent 10 - 40 years after first exposure (41,60,94,95). The long latent period before asbestosis manifests clinically means that the disease may not develop until some years after exposure has ceased (25). For these reasons, the prevalence of asbestosis will be highest in those workers who are studied 20 or more years following the onset of exposure.

4. The intensity of exposure. Most epidemiological studies have confirmed the existence of a dose-response relationship between exposure to asbestos dust and the development of asbestosis (9,25, 68,69,70,74,75). Furthermore, the risk of premature mortality due to asbestosis seems to be confined to individuals with very high dust exposures (25,36,39,70,78,94). Thus, the nature of the

exposure to asbestos should be reported in any epidemiological study of the prevalence or the mortality attributable to asbestosis.

5. The frequency of exposure. There are few reports which distinguish between constant and intermittent exposure to asbestos in the etiology of asbestosis. Some studies have suggested that asbestosis occurs less frequently in intermittently exposed workers than in continuously exposed workers, but these findings need further investigation (39,135).

6. The fibre characteristics. Exposure to all the major types of asbestos may result in asbestosis and the relative pathogenicity of each type appears to depend on its aerodynamic and chemical properties. The aerodynamic properties of the inhaled asbestos fibres determine their deposition pattern in the lung and the ease with which they are cleared from the lung (25,30,74,84). Although, the various types of asbestos are also known to have differing in vitro effects the clinical implications of these findings remain speculative (62). Finally, it should be noted that fibres with a length greater than 5 microns and a diameter less that 3 microns are thought to be particularly fibrogenic (25,29,30).

7. Coincidental exposure. Workers exposed to asbestos may also be concurrently exposed to other hazardous materials rendering the interpretation of research findings difficult. Consideration should be given to the pathogenic contribution of products such as magnesium carbonate, calcium silicate, diatomaceous earth and fibrous glass which may accelerate the development of pulmonary fibrosis (95,97). There is some evidence, after controlling for exposure to asbestos, that asbestosis develops more rapidly and is more severe in smokers than in non-smokers, and it has been found that asbestosis is more frequently the cause of death among

smoking rather than among non-smoking asbestos workers (42,80,81,82). Thus, it is recommended that asbestos workers be strongly advised and assisted to discontinue the smoking habit.

The need to consider the interaction of all the above factors requires very complex research designs. Comprehensive studies which take all these variables into account are not currently available and existing data must be generalized with caution.

Since there is clear evidence of a dose-response relationship for asbestosis, it is to be expected that the incidence of asbestosis will decline as occupational exposures are reduced. This expectation has been confirmed in a British study of asbestos factory workers. The mortality rate from asbestosis among workers first exposed after the introduction of controls was found to be significantly less than those whose exposure occurred prior to the application of the standards (71,72,73). In another study the prevalence of asbestosis in workers employed for twenty years or more in a U.K. textile factory fell from 81% in 1930 to 2.3% in 1968 (41).

Despite the development of industrial hygiene control methods and worker education programs, additional cases of asbestosis can be expected over the next few decades. The full effects of significant past exposures will not be apparent until the latent period of the disease has elapsed. The continuing importance of asbestosis, particularly in end-use industries, is highlighted by Selikoff's study of Canadian and U.S. insulators (10), where it was shown that asbestosis accounted for 8% of the deaths of the union members between 1967 and 1973. Most of these deaths occurred among men who had accumulated 20 years or more since first exposure and whose first exposure was in 1953 or earlier (79).

Natural History

Although the life expectancy of persons with asbestosis has improved dramatically since 1930, there is considerable evidence that workers who already have evidence of pulmonary asbestosis are at higher risk than other asbestos workers to develop lung cancer. Studies have reported that between 15 - 50% of workers with asbestosis die of lung cancer (25,36,83) with the proportion of such deaths increasing in recent years (71,78,84). The mean latent period for lung cancer, 25 - 30 years (9,25,36) is greater than that for asbestosis, 10 - 20 years (41) so that it is postulated that improved dust conditions have decreased the severity of asbestosis and increased the proportion of workers who survive asbestosis long enough to experience increased risks of lung cancer (9,85).

Treatment

There is no specific therapy for asbestosis, but some investigators consider that aggressive control of intercurrent respiratory infections, insistence on safe work procedures including the use of protective equipment, and complete avoidance of cigarettes can help to reduce the ill health and disability associated with asbestosis (86, 98,197). Unfortunately, definitive evidence to support these statements is lacking and the probability of developing controlled studies to test the hypothesis seems remote. Consideration might also be given to providing vaccination against influenza and pneumococcal pneumonia for individuals with asbestosis (190).

Similarly, it is not established whether removal of a worker

from exposure to asbestos at the earliest sign of asbestosis prevents progression of the disease. However, since there is evidence of a dose-response relationship for mild and moderate asbestosis and since at least one primate study showed that the disease did not progress following removal from exposure, it is reasonable to assume that discontinuation of exposure might induce a more favourable prognosis (25,74,75,87,88).

The worker with early asbestosis should be made aware of the diagnosis and the possible implications of continued exposure. He should be provided with an opportunity to leave a job where he is exposed to unsafe levels of asbestos. If that is not possible, he should be advised to severely reduce his personal exposure by strict attention to work procedures and conscientious use of personal protective equipment during intermittent exposures. All asbestos workers, and particularly those who have evidence of lung disease, should be advised to discontinue smoking.

Screening for Asbestosis

The presence of basal crepitations was thought to be the earliest sign of asbestosis, and the first industrial hygiene standard was developed on this basis (61). However, more recent occupational health programs have relied heavily on chest radiographs and lung spirometry (39,51, 60), and it is thought that modern laboratory and radiological techniques are more sensitive indicators of early asbestosis than the clinical findings (51,89,90). However, some researchers maintain that the presence of clinical signs, especially basal crepitations, may precede the detection of radiographic or lung function abnormalities in at least some cases of asbestosis (39,91,92). Comparative serial analysis of spirograms and chest radiographs offer the best chance of identifying early asbestosis (25, 54). Inclusion of serial measurements of diffusing capacity might offer some additional benefits (48).

It must be emphasized that all those involved in a screening program should be aware that the tests employed are not diagnostic tests. Any individual with a positive screening test should be referred for more extensive diagnostic testing including a full medical examination. Further, workers should be aware that the tests are non-specific and therefore abnormalities other than asbestosis will be detected. Indeed, as occupational exposures are effectively reduced one might expect that an increasing proportion of the abnormal results will be attributable to non-occupational disease.

Mesothelioma

Malignant mesotheliomas are rare tumours, which arise from the pluripotential mesothelial cells of the pleura, peritoneum, or pericardium. The association of mesothelioma with asbestos exposure was first noted in 1946 (128), but it was highlighted in 1960 when Wagner and his colleagues reported 33 cases who had had occupational, environmental, or domestic exposure to asbestos in the crocidolite asbestos mining area of the Northwest Cape of South Africa (5). It is of interest that a similar excess has not been identified in the Transvaal crocidolite mining area despite intensive investigation (129).

The association of pleural and peritoneal mesotheliomas with asbestos exposure has now been confirmed in many parts of the world. All types of asbestos, with the exception of anthophyllite, have been implicated (130,131,132,133,134), but there are apparent differences in the risk of mesothelioma depending on the type of asbestos to which the subjects have been exposed. The risk seems to be most evident with crocidolite, less with amosite, and apparently even less with

chrysotile (135,136,137). With both amosite and chrysotile, there appears to be a higher risk in the manufacturing than in mining and milling industries (136,138).

The most disconcerting feature of the relationship between malignant mesothelioma and asbestos exposure is the association of this tumour with apparently low levels of exposure, including domestic or neighborhood exposures (5,139,140,141,142,193). Although some of the domestic and neighborhood exposures described in the South African studies were, in fact, rather heavier than many present-day occupational exposures, the fact that mesotheliomas may develop as a result of non-occupational exposure is a cause for concern. In addition, mesotheliomas may follow relatively brief exposures in the remote past (5,139,140,141,142).

In occupational settings it has been possible to demonstrate the existence of a dose-response relationship - increasing evidence of disease with increasing exposure levels and duration of exposure (143). However, since mesotheliomas can develop in response to very small doses of asbestos, it is difficult, if not impossible, to define a safety threshold which will completely eliminate the risk of this tumour (11,25).

Clinical Features

The commonest presenting complaints of patients with pleural mesothelioma are chest pain and shortness of breath. The pain is often described as dull and aching, but it may be pleuritic and can be very severe. The shortness of breath is progressive and may be due to lung compression, restriction of respiratory movement by the tumour or an associated pleural effusion. Mesothelioma of the peritoneum

may present with diffuse abdominal pain, abdominal swelling or progressive loss of weight. The presenting complaints may be symptoms of obstruction of the bowel or other organs indicating that the disease is far advanced at first presentation.

The most common clinical sign of pleural mesothelioma is a pleural effusion. Distant metastases are rarely observed, but local spread may occur to involve bones, lymph nodes or mediastinal tissues. A peritoneal mesothelioma is usually detected clinically as an ill-defined abdominal mass usually associated with ascites (25,101,144).

Lung function tests are of limited diagnostic value, although evidence of a rapidly progressive restrictive disease suggests the presence of a pleural mesothelioma and should prompt a full clinical examination. Lung function tests are more useful in evaluating the effectiveness of different treatment programs.

A routine chest x-ray may lead to the diagnosis of an asymptomatic pleural mesothelioma, and this condition should be strongly suspected if there is evidence of a pleural effusion and nodular pleural thickening on the chest radiograph (25,84). More advanced tumours usually infiltrate the lung, producing larger nodular lesions which are readily visible on the chest radiograph. In later stages, the hilar and peritracheal nodes may also be obvious radiographically (148).

It is difficult to confirm a diagnosis of mesothelioma histologically during life, because of the wide variety of cell types within the tumour and the need to exclude primary tumours elsewhere (145,146). Histochemical techniques may be of some value in distinguishing between mesothelioma and secondary carcinoma, but these tests have not proved to be useful as was originally believed (147).

The differential diagnosis of mesothelioma should include

metastatic carcinoma (commonly from the lungs, stomach, colon, breasts, pancreas, or ovaries), tuberculous pleurisy, and rheumatoid disease (25, 36). Since some of these conditions can be treated effectively, the diagnosis of mesothelioma should be made by exclusion.

Epidemiology

The overall incidence of mesothelioma in Canada has been calculated as approximately 1 per million population per year or 1/10,000 deaths (16). In order to assess the influence of asbestos exposure, the medical records of all individuals who died as a result of mesothelioma in Canada between 1959 and 1968 were collected. It was found that 23% of the male cases had been occupationally exposed to asbestos (16). However, it should be noted that despite the presence of large asbestos mining and milling operations in Canada, the majority of these men had been employed in industries, manufacturing or using asbestos products (16,85).

A recent review of retrospective studies from the international literature indicated that, among patients with mesothelioma, a high proportion - from 20 to 95% - gave a history of asbestos exposure, albeit often for a short period in the distant past (25). However, it must be remembered that asbestos exposure is common in the general population and, in the above studies, up to 41% of the controls also reported asbestos exposure (25). It should be also noted that a mesothelioma may occasionally develop in individuals with no history of exposure to asbestos (14,15,16,80,84). Although the reliability of each of these retrospective studies depends, to a considerable degree, on the extent to which the researchers controlled for the bias inherent in this type of study, the association of mesothelioma with asbestos exposure is clear.

There is some preliminary evidence to suggest that asbestos workers, whose chest x-rays demonstrate pleural plaques, are at a slightly higher

risk than other workers of developing mesothelioma (149). It is unusual to find asbestosis to any degree in association with pleural mesothelioma; however, in peritoneal mesothelioma, pulmonary fibrosis may be quite evident (25,84).

The importance of mesothelioma among asbestos workers has been clearly demonstrated by a number of prospective studies of occupationally exposed groups. It has been shown that among groups of workers who have had heavy exposures to asbestos approximately 5 - 10% of deaths are due to mesothelioma (18,25,122,136,141,150).

There is evidence of a dose-response reslationship for mesothelioma. It appears that both the degree and length of exposure are significant with the death rate increasing with longer periods of employment and more intense exposure (139,141). It has been reported that workers with long employment and heavy exposures have six times the mesothelioma risk of those with light exposures (141).

More specifically, within the various occupational groups exposed to asbestos, insulation workers and textile workers seem to have the highest risk of mesothelioma (11,133,135,151,153). The incidence of tumours seems to be highest in cities where there are shipbuilding industries (14,25), and lowest in asbestos mining areas (15,152,156).

Animal experiments indicate that all types of asbestos fibre, when injected intrapleurally, are capable of producing malignant mesothelial tumours. Inhalation experiments, on the other hand, tend to produce cancers rather than mesotheliomas (158,159). This finding suggests that respiratory clearance mechanisms are at least partially effective in reducing the proportion of inhaled fibres which reach the lung periphery where they may induce the formation of mesothelial tumours (29,30,74,122).

It is currently considered that the observed differences in the carcinogenic potential of the various asbestos fibre types is primarily due to physical differences between the fibres. Fibre size

is the main factor which determines the degree to which the fibres are deposited and retained in the lung. Chrysotile fibres, being soft, pliable, curly and often quite fine, are more likely to be intercepted high in the bronchial system than amphibole fibres which are rigid, travel readily in the airstream and are thus less likely to be stopped by the airway walls (30,84,122). It is of interest to note that analyses of lung parenchymal tissues obtained from men employed in the Canadian chrysotile mines, who had been diagnosed as asbestotic, have shown the presence of tremolite and other amphibole fibres in excess of chrysotile fibres (85,163,205). The presence of amphibole fibres in commercial chrysotile may thus contribute to the development of subsequent disease in men whose primary exposure is to chrysotile.

Fibres which are less than 2.5 microns in diameter and between 10 and 30 microns in length seem to be particularly pathogenic in terms of inciting mesothelial growth (158,160). The importance of fibre diameter was identified by Wagner et al, who injected rats interpleurally with glass fibre. Two samples of glass fibre were used, one with a median fibre diameter of 0.12 microns, and the other with a median diameter of 1.8 microns. Four mesotheliomas were observed in 32 rats injected with a finer fibre, and none in those given the coarser fibre (160). Fibre diameter is thought to be of particular importance in explaining the different mesothelioma rates in the North Cape and Transvaal areas of South Africa. Mesothelioma is uncommon in the Transvaal, an area where crocidolite has a much larger diameter than that from the Cape Province (30,206). Furthermore, it has been suggested that exposure to fine fibres is more likely in the manufacturing and end use industries than in mining and milling. This may explain the observed differences in the incidence of mesothelioma in these occupational groups.

Other environmental factors may also be responsible for the

development of mesothelioma. In experimental studies, a wide variety of substances including fibre glass, barium sulphate crystals and some polymers have been shown to be associated with this tumour (160). Unlike bronchial carcinoma, smoking does not seem to play a synergistic role in the development of mesothelioma (25,166).

Natural History

The clinical course of mesothelioma is usually one of rapid deterioration and the prognosis is extremely poor. Approximately half of the patients with pleural mesothelioma die within 12 months of diagnosis and very few survive more than two years. The prognosis for peritoneal tumours is also very poor (25,36).

The average latent period for the disease is said to be 30 - 40 years from the onset of exposure (5,79,150). In view of the ubiquity of asbestos in our industrial society and the long latent period of mesothelioma, increasing numbers of this disorder can be expected for at least the next three to four decades.

Treatment

No effective treatment for mesothelioma is currently available. Radiotherapy and chemotherapy have not proved useful to date, although it remains to be seen whether multiple chemotherapeutic agents alone

or in combination with radiotherapy will affect the course of this disease. Treatments based on the hypothesis that mesothelioma is, in part, an immunologically determined disorder seem promising, but have yet to be evaluated (25,35).

Screening

Since there is no effective method of achieving the early diagnosis of mesothelioma and no effective treatment for this condition, screening for mesothelioma is neither possible nor practical. The prevention of mesothelioma depends on the avoidance of exposure to asbestos.

Employers and employees must be made aware of the hazards of low doses of asbestos and a program should be developed to ensure that domestic exposure to asbestos is also minimized.

Lung Cancer

An association between asbestos exposure and lung cancer was first identified as a result of the clinical observation that a high proportion of asbestotic workers died of lung cancer (6,36,71). It has been, however, much more difficult to document the association between cancer of the lung and asbestos exposure, since cancer of the lung is not exclusively attributable to asbestos exposure. It is well known

that a significant proportion of individuals in the general population with no occupational asbestos exposure die from cancer of the lung.

General Epidemiology of Lung Cancer

That lung cancer is a significant cause of death in our modern Western society is clear from a review of Albertan statistics. In Alberta, lung cancer is responsible for approximately one death per day. Furthermore, in 1976, 24.1% of all male cancer deaths and 7% of all female cancer deaths were attributable to cancer of the trachea, bronchus and lung.

Over the last few decades lung cancer has become increasingly common and, because it is now a major cause of death, a great deal of research effort has been directed towards identifying both causal and contributory factors.

Asbestos is only one of a large number of factors which can be linked aetiologically to the development of lung cancer. The multifactorial nature of lung cancer makes it much more difficult to determine the magnitude of the contribution of asbestos exposure to this disease than is the case with a basically unifactorial disease such as asbestosis.

Some of the predisposing factors which have been shown to influence the occurrence of lung cancer are:

1. Increasing age. This effect is clearly demonstrated in Table I.

2. The presence of scar tissue in the lung (102,103,196,197).

3. Congenital cystic fibrosis of the lung (104,198).

TABLE I

AGE/SEX SPECIFIC INCIDENCE RATES

FOR

CANCER OF THE LUNG/100,000 POPULATION

ALBERTA 1976

AGE GROUP	INCIDENCE/100,000 POPULATION	
	MALES	FEMALES
35 - 44 yrs.	8.7	7.2
45 - 54 yrs.	45.4	19.1
55 - 64 yrs.	123.1	39.9
65 - 74 yrs.	314.3	56.3
75 - 84 yrs.	348.1	70.1
85 +	301.9	15.2

4. A wide variety of organic and inorganic industrial substances including uranium, nickel, chromates, as well as asbestos (105,106,207).

5. Atmospheric pollution with such carcinogenic compounds as benzpyrene. Industrial contamination of urban air is thought to contribute to the increased frequency of lung cancer in urban areas (101,107,108,109,207,208).

6. Cigarette smoking. This is by far the most extensively studied aetiological factor for lung cancer. Epidemiological evidence is convincing and has established the causal relationship between the inhalation of tobacco smoke and bronchogenic cancer. Numerous retrospective and prospective studies have been done and have shown that the relative risk for cancer of the lung for all smokers (regardless of amount smoked) is ten times that of non-smokers (108,110,115). For heavy smokers the rate of lung cancer may be up to sixty times greater than the equivalent mortality rates for non-smokers (109,116,117).

Prospective studies carried out in England and in the United States have shown a drastic reduction in death rates from cancer of the lung among those who have stopped smoking. For example, it has been reported that the risk remains high for three years after cessation of smoking but becomes significantly lower after ten years (116,118,120,125,209,210).

The strength of the relationship between smoking and lung cancer renders it essential that in epidemiological studies of the effect of other carcinogenic materials, such as asbestos, the effect of smoking is controlled for as far as possible. Unfortunately, relatively few studies of asbestos workers do, in fact, attempt to

control for this factor with few exceptions (9,10,11,139,211).

Clinical Features

The clinical features of both occupational and non-occupational lung cancers are essentially similar. The location, histologic type and intrinsic rate of growth of the tumour determine whether symptoms and signs develop early or late in the course of the disease. The early symptoms rarely prompt the individual to seek medical attention because they are usually non-specific (an irritative cough with increasing sputum production) and not particularly alarming. The non-specificity of the symptoms and signs may lead to additional medical delays until the persistent nature of the complaints leads to further investigation and a definitive diagnosis (212). If the sputum is blood-tinged or if there is a frank haemoptysis, an individual usually obtains prompt medical attention, however, haemoptysis is the initial symptom in only 5 - 6% of these patients (118,119). A presenting symptom of chest pain is found in up to one-third of patients. This symptom is particularly frequent among those patients who are subsequently shown to have a centrally located carcinoma, since chest pain usually indicates that a segment of the lung has become completely occluded or atelectatic (118,119).

Clinical signs are rarely helpful and may be absent until the disease is well advanced, at which time the signs are those of a space occupying lesion or atelectasis. The x-ray findings in patients with lung cancer may be caused by the tumour itself, changes in the distal lung tissue secondary to tumour-induced airways obstruction or both. The findings are extremely variable depending on the location, the cell type and the length of time the tumour has been present. It is

known that chest x-ray abnormalities may antedate the first signs or symptoms of the disease by seven or more months. These early features are, unfortunately, subtle in nature, and often are appreciated only in retrospect (114,213,214,215,216).

The cytologic examination of the sputum is a valuable adjunct in the diagnosis of bronchial carcinoma, but it is generally a poor technique for the diagnosis of peripheral tumours unless special manoeuvres are employed (100,119,217).

The relative importance of the various histological types of lung cancer is disputed. Squamous cell tumours are said to be the most common (40 - 70%) followed by adenocarcinomas (10 - 25%), bronchioloalveolar tumours (5 - 15%), and small cell or oat cell tumours (5 - 10%) (125,164). The cell type is thought to be more closely related to the anatomical site of the tumour than the nature of the aetiological agent (123,164,218,219). There is some preliminary evidence that adenocarcinomas are more frequent than expected in asbestos workers with lung cancer (123), but the mechanisms responsible for this trend are not clear and observer variation is known to be a contributing factor (124).

In the general population centrally located lung tumours are said to outnumber peripheral tumours by 3:1 (125). In asbestos workers there seems to be an increase in the proportion of peripheral tumours and furthermore, asbestos-related tumours seem to have a predilection for the lower lobes (84,124,198).

Treatment

The best hope of recovery from bronchial carcinoma is surgical removal of early lesions. Overall, five year survival is approximately 8% *, but the survival is considerably better for patients who have asymptomatic, surgically-resectable, peripheral tumours. The five year survival for such a group of patients has been reported to be as high as 38% (127). A recent literature review of reported surgical results found that 31 - 42% of all resected cases survive five years after diagnosis (101).

Unfortunately, at the time of diagnosis, a substantial proportion of patients (50 - 75%) are found to have inoperable tumours on the basis of widespread metastases, cell type (e.g. oat cell tumours) or poor general health. These patients may, however, be offered palliative treatment with radiotherapy or chemotherapy (125,164,212,220,221).

The influence of lymph node spread and cell type on prognosis is marked. In one major study patients with well-localized squamous cell carcinomas or adenocarcinomas had five year survival rates in the range of 50 - 60%, whereas, five year survival dropped to approximately 10 - 15% in the presence of hilar lymph node involvement (222). Oat cell or undifferentiated small cell carcinoma has the worst survival pattern; only 5% survive 18 months and less than 1% survive more than 3 ½ years from diagnosis (164,221).

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* Alberta-specific data obtained from the Provincial Cancer Hospitals Board indicate that the survival rates for lung cancer have remained fairly constant over the years 1956 - 1971. The five year survival rate for lung cancer in Alberta is 8.6%, the ten yhar survival is 6.6% and the fifteen year survival at 1.6%. This survival data is comparable to rates elsewhere in the world (101).