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THE NOVA SCOTIA MEDICAL BULLETIN

Environmental Medicine

Do you hear the children weeping, oh my Brothers
Ere the sorrow comes with years?
They are leaning their young heads against their mothers,
and **that** *cannot stop their tears.*
The young lambs are bleating in the meadows,
The young birds are chirping in the nest,
The young fawns are playing in the shadows,
The young flowers are blowing toward the west—
But the young, young children, oh my Brothers,
They are weeping bitterly!
They are weeping in the playtime of the others,
In the Country of the free.

"For 'oh'" say the children "we are weary
And we cannot run or leap;
If we cared for any meadow, it were merely
To drop down and sleep.
Our knees tremble in the stooping,
We fall upon our faces, trying to go;
And underneath our heavy eyelids drooping
The reddest flowers would look white as snow.
For, all day, we drag our burden tiring
Through the coal-dark, underground;
Or, all day, we drive the wheels of iron
In the factories, round and round.

It is more than a century since Elizabeth Barrett Browning wrote this evocative description of child labour. In those days working conditions were atrocious; many children and adults developed bronchitis, tuberculosis and died.

Thanks to enlightened and determined pioneers, we now have legislation and organizations to protect the workers' health and prevent exploitation. Nonetheless hazards still exist. New industrial processes have brought a bewildering variety of pollutants which threaten not only individuals but also our whole biological existence. Mercury, whether it's popped into oranges or swallowed by unsuspecting swordfish, remains an ubiquitous hazard and arsenic can contaminate our own drinking water. Our forests can be sprayed by chemicals of highly debatable toxicity and our farm produce contaminated. We are therefore grateful to Dr. Mastromatteo for his comprehensive review of a subject that particularly emphasizes the role the general practitioner can play in this field, and the need for further education.

National and regional organizations are developing to cope with this expanding branch of medicine. Dr. Rodney May recently Director of Occupational Medicine, stimulated many activities in Nova Scotia but has been transferred to Ontario. The Medical Society of Nova Scotia Occupational Health Committee has also played an important role. Dr. Albert Prossin, past chairman, is now a full-time Industrial Health physician to the Cape Breton Development Corporation. He explains that there are at least 30 part-time physicians engaged in Environmental Medicine and occupational nurses are employed in at least seven different locations. Nurses, physicians, technicians and toxicologists fulfil many duties. Preemployment physical examinations are performed. Levels of dust are monitored and threshhold limits of toxic substances are checked. Sites include the Heavy Water Plant, Michelin Tire Factory, coal mines, Gulf Oil Refinery and the Nova Scotia Power Corporation.

From atomic radiation to inhalation of hydrogen sulphide, physicians need to coordinate their efforts with technicians and government to maintain a healthy community and to protect our future quality of life.

It is hoped that the forthcoming workshop on Occupational Medicine in Cape Breton, in which Dr. Mastromatteo is guest speaker, will be well attended.

Dr. John M. Williston is chairman of the Society's Committee on Occupational Medicine and will welcome enquiries or contributions on this important topic. □

B.J.S.G.

An Appreciation

DR. CARLETON LAMONT MACMILLAN

Carleton Lamont MacMillan was born in Goldborough in 1903; educated in Sydney Academy, Acadia and Dalhousie, where he attained his M.D., C.M. in 1928. He practiced in Rose Bay for two or three months, and then opened a practice in Baddeck in 1928. From then until he retired in 1966, he conducted a general practice (in the true sense of that word) in Baddeck and a wide surrounding district, including all of Victoria County and much of Inverness. His exploits in bringing medical care and attention to a wide section of rural Cape Breton (with appalling travel conditions) are richly documented in his own book "Memoirs of a Country Doctor", which has enjoyed wide sale since its publication in 1974.

Monty Senior was much too modest to tell of more than his efforts to reach the sick and disabled, and his procedures in bringing them his skill. What he does not say in his book was the kindness, the steadfastness and the reassurance that he carried with him. While he could not be designated as charismatic, Monty had an aura of concern and confidence which he brought to the bedside and, while often troubled and uncertain himself, he did not burden his patients with his doubts.

In addition to serving the population as a doctor, confessor, friend and companion, he also represented his constituency in the Nova Scotia legislature for eight years. There he was respected by all members and was a welcome guest in any group regardless of political stamp.

In his later years he spent a great deal of time enthusiastically prospecting in areas near Baddeck. His verandah was rarely free of a large quantity of ore samples which he lugged from many miles off the road to his car. He himself attributed his remarkable recovery from cardiovascular difficulties (prior to his retirement) to his efforts in this area. The several years prior to the publication of "Memoirs" were busy with collating, editing and reducing the mass of data down to a readable size book. Unfortunately, many episodes, some humorous and some tragic, had to be omitted. However, enough survived to be a lasting monument to this fine man.

Monty had many honors in his life. The people of Baddeck recognized him, The Canadian Medical Association tendered him a Senior Membership and he was admitted to the Order of Canada in 1972. Truly this Prophet achieved honor even in his own country.

His family will miss him and we extend our sympathy. The section of Cape Breton that he served will miss him. The many friends across Canada and the United States will miss him, and we in the profession have lost an exemplary member.

He was a great soul and we are all diminished by his death.

". . . and therefore never send to know for whom the bell tolls; — it tolls for thee."

— John Donne

□

J.F.N.

Environmental Hazards to Health*

The Private Physician's Role

E. Mastromatteo,**M.D., D.P.H., D.I.H.,
Toronto, Ontario.

John Stewart was an illustrious son of Nova Scotia who served a long and distinguished career in medicine as a researcher, surgeon, military physician, medical administrator and humanist. I am greatly honoured to have been asked to present the John Stewart Memorial Lecture which is a tribute to his memory.

In preparing my text I wondered what John Stewart would have thought of the relevance of environmental hazards to the practice of medicine. I am sure that he would agree that the two are relevant. John Stewart commenced his medical career at the time of the *sanitary revolution* when the role of micro-organisms in human diseases was being first determined. He pioneered in the development of antiseptic techniques. We are today in the midst of the *environmental revolution*. If John Stewart were alive he would have been involved in the elucidation and control of those environmental factors affecting the health of his patients.

Pollution, environment, occupational health and environmental health are currently "in" words. From all sides the public's attention is being called to the health risks resulting from air pollution in our cities, the use of pesticides, the contamination of the food and water we drink, and the environmental agents in the workplace. Increasing technology has increased the number and complexity of environmental agents to which our patients are exposed. Along with these technological changes, social changes of no less importance are taking place with such things as increasing use of drugs and alcohol, permissiveness, women's liberation and changing values among the young people.

Almost daily there are pronouncements about the effects of environmental agents upon the health and well-being of people. Insidious hazards with a long latent period, such as occupational cancer or mutagenic effects, cause special concern. Although life expectancy in infants has shown dramatic increases, there has been little real improvement in that of middle-aged males. The major health problems in developed countries — cardiovascular diseases, cancer, death and injury from accidents and mental illnesses — are not adequately controlled by conventional medical treatment. The rising cost of medical and hospital care is causing closer attention to health care costs. What is needed is more emphasis on the identification and removal of environmental hazards and on the modification of personal life styles, where these are known to have health risks.

Many of the environmental health effects are controversial or political issues. In terms of human health it is difficult for the public to sort out the truths from the half-truths. The practising physician, particularly in smaller communities, is looked upon to give advice and to answer questions on the effects of environmental agents encountered in his community. With this viewpoint in mind, I hope to present a general overview of the environmental hazards to health and to comment on the role of the practising physician.

The role of physicians in environmental health is not new. Hippocrates in 500 B.C. said: "That the physician is an honour to his profession who has led through the whole circle of the sciences; who has due regard of the seasons of the year, and the diseases which they are observed to produce; to the states of the winds peculiar to each country, and the quality of its waters; who marks carefully the localities of towns, and of the surrounding country, whether they are high or low, hot or cold, wet or dry; who moreover, neglects not to mark the diet and regimen of the inhabitants and, in a word, all the cause which may produce disorder in the animal economy."

The study of occupational health hazards is not a new subject. The first complete text book on occupational diseases was written in 1713 by Dr. Bernardo Ramazzini, Professor of Medicine in Italy. Ramazzini wrote: "Various and manifold is the harvest of diseases reaped by certain workers from the crafts and trades that they pursue; all the profit they get is fatal injury to their health. That crop germinates mostly, I think, from two causes. The first and most potent is the harmful character of the materials that they handle, for these emit noxious vapours and very fine particles inimical to human beings and induce particular diseases; the second cause I ascribe to certain violent and irregular motions and unnatural postures of the body, by reason of which the natural structure of the vital machine is so impaired that serious diseases gradually develop therefrom."

Ramazzini's book has remarkable relevance to today's concern about occupational diseases. Ramazzini gave sound practical advice to the physicians of his day which may be paraphrased as follows: "When a physician goes into the house of a working man he should be content with a three-legged stool instead of a gilded chair and in addition to the questions recommended by Hippocrates he should ask one more: 'what is your job?'." In his book Ramazzini was familiar with noise-induced hearing loss. He also described a lawsuit brought by citizens against a chemical company on the basis of health hazard to the surrounding residents. Medical testimony was used in deciding this case.

Occupational cancer is not a new subject. In 1775, Sir Percival Pott described occupational cancer in chimney sweeps resulting from exposure to coal tars in their work. Percival Pott was an English surgeon who is also known for the ankle fracture and the spinal deformity which both bear his name.

*The John Stewart Memorial Lecture presented at the 51st Dalhousie Refresher Course, November 15, 1977.

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AIR POLLUTION

There have been several acute episodes of increased illness and death from urban air pollution. The best known is the London fog of December 1952 with 4,000 deaths. In these acute episodes there was usually a heavy build-up of air contaminants to 5 to 10 times their usual level. In these episodes it is the susceptible who are affected — the young and the elderly with pre-existing respiratory and/or cardiac disease. The measured pollutants were total suspended particulates, total sulfates, and sulfur dioxide.

Other health effects have been related to specific localized sources of air pollution, e.g., Yokohama asthma.

There is little direct evidence that exposure to urban air pollutants have caused chronic health effects. A number of epidemiological studies have shown that mortality from lung cancer and chronic bronchitis are increased in urban residents when compared with rural residents. How much this is due to the effects of exposure to air pollution or to other differences, e.g., cigarette smoking, occupational exposures, etc., is not clear. Some studies with asthmatic children and adults have shown a correlation between air pollution levels and respiratory complaints.

In general, it does seem that urban air pollution is associated with increase in chronic bronchitis and lung cancer. The effects of air pollution, however, are quite small when compared with that from cigarette smoking. More research is needed, however, on the nature of urban air pollutants, other factors in urban residence, and their health effects on city residents.

Some authors have reported an increase in heart disease from carbon monoxide in urban air pollution, in patients with pre-existing coronary insufficiency. More definitive studies are needed on this subject also.

Exposure levels for community air pollutants have been developed by several jurisdictions. In others, air pollution indices have been developed; these should be regarded as a general guideline for the control of air pollutants and not as fine lines between safe and unsafe concentrations.

WATER POLLUTION

Water borne bacterial diseases have largely been eliminated by proper waste treatment and water treatment plants. Water-borne diseases, e.g., typhoid fever, may still occur from inadequate treatment or breakdown in the control system. Viruses are transmitted in water and outbreaks of infectious hepatitis have resulted from drinking of faecal-contaminated water supplies.

Little is known of the effects of long-term consumption of chemical impurities in our water supplies. Many attempts have been made to link the occurrence of kidney stones to phosphates in drinking water but no connection has been demonstrated.

In Japan, there have been a number of water pollution episodes attributed to industrial pollutants. In the Jintsu River basin, residents drank water contaminated with cadmium from mine wastes. They also ate cadmium-contaminated rice which had been irrigated by this same water. After many years, the local residents were found to have renal damage, which resulted in loss of phosphates from the body. Osteomalacia with pathologic fractures were common. The condition was quite painful and the Japanese name for this disease was "itai! itai!", which means "ouch! ouch!" in English.

Also in Japan, the discharge of inorganic mercury effluent into Minamata Bay gave rise to mercury contamination of the local fish and shellfish, which formed a major part of the local diet. Residents who ate this seafood developed a severe neurological disorder with ataxia, paresthesia, visual field defects and paralysis. Women gave birth to deformed offspring and in all, there were about 80 deaths. The residents have developed organo-mercury poisoning. Later, it was found that micro-organisms in the sea sediment were capable of converting inorganic mercury to organic mercury. Organic forms of mercury are very toxic, particularly to the central nervous system.

Contamination of water supplies by mercury in Canada has also occurred. Local residents consuming fish from these waters, have been found with increased blood and hair levels of mercury.

Nitrate contamination of well waters may still be a problem in rural areas where wells have become contaminated with nitrogenous wastes. This should always be considered in rural areas where infants are given well water.

In some localities naturally high levels of fluoride in dug wells have been reported with endemic fluorosis. Occasionally high levels of arsenic or other elements are encountered.

The relationship between soft water and an increased incidence of heart disease has been noted by several authors. Further study on the relationship between hardness of water and heart disease is needed.

There is currently much interest in the degree and significance of environmental exposure to trace elements in water. Drinking water guidelines and standards have been published by several jurisdictions. Practising physicians should refer to these in assessing the significance of water contaminants.

PESTICIDES

The use of synthetic pesticides on a wide scale took place with the development of DDT in the 1940's. The benefits of these chemicals in protecting public health, forest resources and agricultural production, are well known. In Canada about 75 per cent of pesticide consumption is in agriculture.

The chlorinated hydrocarbon insecticides, as typified by DDT, are stable, persistent and tend to collect in the fat of animals and man. There have been reports of harm to wildlife from these chemicals. For these reasons the use of chlorinated hydrocarbon insecticides have been restricted. Chlorinated hydrocarbon insecticides may cause occupational poisoning or accidental poisoning, the latter chiefly in children. Over-exposure may result in convulsions and larger doses of barbiturates may be needed to control and the convulsions.

The organic phosphorus insecticides also cover a wide range of chemicals ranging from those of high toxicity (parathion) to those of low toxicity (malathion). This group of insecticides are similar in their biological action — they inhibit cholinesterase enzymes which are involved in nerve impulse transmission. Acute poisoning may occur from ingestion or skin contact; it is manifested chiefly by tremors and pulmonary edema. Atropine should be given in relatively

large doses. An antidote for organic phosphorus compounds is available.

Carbamates form another group of insecticides and they include Sevin and Baygon. Carbamates also produce cholinesterase inhibition.

HOME ENVIRONMENT

The increasing use of new chemicals and technologies also extends into the home environment, with the use of pesticide strips, ozone generators, microwave ovens, highly alkaline dishwashing compounds, solvent thinners, and polyurethane varnishes. In some areas lead-based paints were used indoors and young children who eat paint chips may develop lead poisoning. Thousands of such cases occur each year in the United States, and isolated cases have been reported in Canada.

The mixing of household bleaches and toilet bowl cleaners may give rise to chlorine gas evolution with adverse health effects.

The home environment is also a common place for accidental ingestion of substances by young children. Over 50 per cent of such accidental poisonings in children are caused by pharmaceuticals and about 10 per cent are caused by pesticide products.

ENVIRONMENTAL CANCER

Cancer occupies a prominent place in Canada as a cause of death. It is estimated that 2 persons in every 5 will develop cancer and 1 in 5 will die from cancer.

It has been stated that up to 90 per cent of cancers are caused by environmental factors. Exposure to environmental agents in the workplace has come to be regarded by some as the major source of environmental cancer. This viewpoint has perhaps been accentuated by the vinyl chloride episode. The finding that workers exposed to vinyl chloride monomer developed angiosarcoma of the liver has served to focus concern on the cancer hazards of the workplace. In actual fact, exposure to agents in the workplace make up only a small proportion of the total numbers of cancers caused by exposure to environmental agents, perhaps 1 to 2 per cent. Environmental agents include such things as X-rays, gamma-rays, sunglight, viruses, natural mould contaminants, diet, cigarette smoking, personal use of drugs and chemicals, personal hygiene and place of residence. *Cigarette smoking alone contributes far more to human respiratory cancer than all occupational causes combined.* This comparison is not intended to minimize the problem of occupational cancer but rather to place it in perspective with other environmental factors related to cancer.

There is considerable controversy concerning dose response relationships to occupational cancer and the extrapolation of experimental data from animals to man. My own view is that extrapolation of such data should be done on the basis of scientific knowledge and understanding of the biological activity, taking into account such factors as species differences, metabolic pathways, dose, route of administration, etc. I believe that a practical dose response relationship exists not only for toxic substances but also for carcinogens, and that acceptable limits of exposure can be developed and used in the control of workers and public exposure. This is the whole basis of toxicology and of the setting of threshold limit values and maximum allowable concentrations.

OCCUPATIONAL ENVIRONMENT

Occupational diseases are diseases arising out of exposure to environmental agents in the workplace. To get some idea of the magnitude of occupational diseases we may examine the experience of Ontario. Ontario has an active labour force of about 3.4 million in a total population of 8.3 million. In 1976, there were over 400,000 claims made by workers for work-related injuries or illnesses; this represents 1 claim for every 8 workers per year. Of these claims about one-third (154,214) were claims for time lost from work. About 24 per cent of all lost time claims are for back injuries. There were 6,236 lost time claims for occupational diseases and 36 fatal occupational disease claims in Ontario for 1976. The types of occupational disease claims in Ontario are shown in Table I.

TABLE I

LOST TIME OCCUPATIONAL DISEASE CLAIMS IN ONTARIO — 1976

	Claims	
	Number	Per Cent
Exposure to noxious gases, vapours and fumes	1,478	23.7
Noise-induced hearing loss	1,454	23.3
Occupational dermatitis	1,245	19.9
Non-ionizing radiation, e.g., "arc eyes"	900	14.4
Peneumoconioses	89	1.4
Silicosis 60		
Asbestosis 29		
Cardiovascular diseases	39	0.6
Occupational cancer	38	0.6
Lead poisoning	16	0.2
All others	977	15.9
TOTAL	6,236	100.0

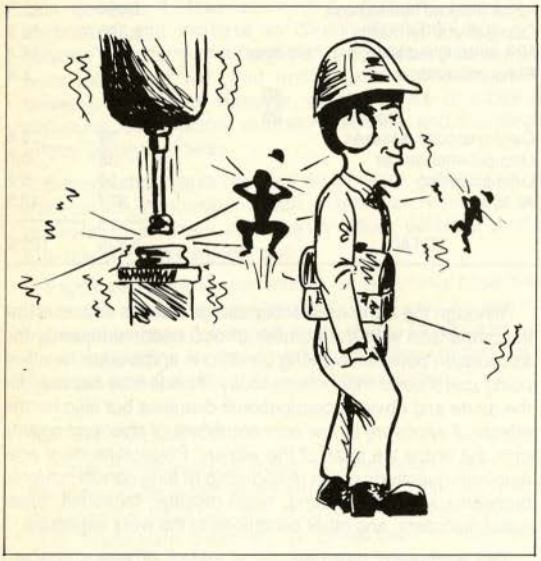
Although the number of occupational disease claims is low in comparison with the number of occupational injuries, the association between working conditions and worker health is being questioned much more today. This is true not only for the acute and obvious occupational diseases but also for the effects of exposure to low concentrations of chemical agents over the entire life span of the worker. Physicians must now deal with questions of the relationship of lung cancer, chronic bronchitis with emphysema, heart disease, leukemia, emotional disorders, and other conditions to the work exposure.

The workplace has become a matter of major concern about health hazards — particularly these long-term subtle hazards. There is also concern about other aspects of the workplace — the stress produced from fixed assembly line production schedules the monotony of assembly work, the provision of adequate health services, including health surveillance, to workers on the job. The workplace represents in microcosm some of the major concerns of society about the meaning of work itself, the quality of life in a rapidly changing environment and the health protection of workers.

The Ontario experience indicates that acute exposure to a large number of gases and vapours forms a significant proportion of all of the occupational disease claims. The noxious gases may cause simple asphyxiation, e.g., nitrogen; chemical asphyxiation, e.g., carbon monoxide or cyanides; respiratory irritation, e.g., ammonia, chlorine, nitrogen dioxide and sulfur dioxide; systemic effects, e.g.,

carbon tetrachloride and benzene; or allergic bronchitis, e.g., isocyanates. In the case of the irritant gases, the point of their action is largely determined by their solubility in tissue fluids. For example, ammonia, which is highly soluble, carries marked irritation of the mucosa of the eye, nose and throat. This affords adequate warning of the presence of hazardous levels in the workplace. Nitrogen dioxide is much less soluble and thus penetrates more deeply into the respiratory system, and it may cause delayed or serious pulmonary edema.

In Ontario, the number of claims for noise-induced hearing loss has shown a marked increase in the last 5 years. Noise-induced hearing loss has been recognized as a compensable occupational disease in Ontario since 1947. In 1950 there was 1 claim, in 1976 there were almost 1500. Increasing noise levels have resulted from technological changes with faster and more powerful equipment. But there is also increasing noise exposure in general society — from amplified rock music to recreational use of snowmobiles. It will become more difficult to sort out the importance of non-occupational noise exposures (the so-called sociocusis) in the hearing loss found in workers. The increased number of noise claims is due in part to changes in the approach to compensation in Ontario.



At one time occupational dermatitis accounted for over two-thirds of all compensable occupational disease claims. In Ontario it now comprises about 20 per cent of the occupational disease claims. A wide variety of chemical and physical agents capable of causing dermatitis are found in the workplace. About 80 per cent of the occupational dermatitis is due to skin contact with primary irritants, e.g., oils, solvents, caustics, cement dust, acids, etc., and about 20 per cent to skin sensitizers, e.g., epoxy hardeners, chromates, etc. Some chemicals are both irritants and sensitizers.

The chief cause of "arc eyes" is the welding arc. The arc emits copious amounts of ultraviolet radiation (UV). UV may cause skin burning and skin tanning but it also may cause an intense conjunctivitis. These eye effects clear readily with no after effects.

Exposure to toxic dusts such as lead, arsenic, fluoride, beryllium, cadmium, and other toxic dusts, are still prevalent in industry. Physicians may be asked about the health effects of health monitoring for these toxic substances. For this, the physician will need ready access to information on the diagnosis, treatment, health surveillance and prevention of poisoning from these agents.

A number of dusts in the workplace, when inhaled into the lungs, are capable of producing tissue reaction in the lung. Crystalline free silica in the form of quartz is the most important of these dusts. Silicosis from long-term exposure to silica is found chiefly in foundry workers, granite cutters, and hardrock miners. Workers with silicosis are very liable to develop tuberculous infection. Other important cuases of pneumoconiosis are asbestos and coal dust. Sometimes mixed exposures may result in "mixed dust pneumoconiosis" with a radiological picture peculiar to each dust. Asbestos has become an important occupational health hazard as asbestos is capable of causing asbestosis, lung cancer and mesothelioma. The lung cancer found in asbestos workers is largely — perhaps completely — associated with those who smoke cigarettes. The prevention of peneumoconiosis depends on good engineering control of the dust. Medical supervision with periodic medical examination, chest examination and X-ray and pulmonary function testing are important.

Environmental cancers have been mentioned earlier. While the number of workers who develop occupational cancer is relatively small, this subject has assumed a major importance. Occupational cancer has been associated with many exposures. For example, lung cancer has been reported in workers exposed to certain nickel and chromium refining operations; the manufacture of chromate pigments; arsenic exposure; radioactive gases and dusts in mining; asbestos exposure; and coal tars in coke oven workers. Sinus cancer has been reported in workers exposed to certain nickel refining operations and in furniture makers exposed to hardwood dusts. Leukemia has been reported in workers exposed to benzene and to ionizing radiation. Cancer of the urinary bladder has been observed in dye workers and in rubberworkers exposed to beta-naphthylamine. Bone cancer has resulted from exposure to radioactive substances. I have also referred earlier to the occupational skin cancer in chimney sweeps exposed to coal tars. Skin cancers have also been reported in workers exposed to shale oils, heavy petroleum oils, ionizing radiation and ultra-violet radiation. Mesothelioma has occurred in asbestos workers and angiosarcoma of the liver in workers exposed to vinyl chloride monomer. This listing serves to illustrate the variety of occupational cancers which have been reported.

Allergic respiratory disease from inhaled allergens also exist in the workplace. I will mention but two examples: allergic bronchitis and bronchial asthma from exposure to wood dust from British Columbia red cedar and from exposure to isocyanate vapours and dusts (e.g., TDI, MDI, PAPI) in remarkably low concentrations. Isocyanates have come into widespread use in the last 10 years in the manufacture of plastic foams, paints and coatings. Some researchers are convinced that isocyanate exposure results in gradual and permanent impairment in pulmonary function.

I have not dealt with the occupational health hazards related to biological agents, e.g., undulant fever in packing house workers and serum hepatitis in laboratory workers. I have not dealt with the wide range of other physical agents encountered at work such as vibration, heat, ultraviolet, infrared, micro-waves, electricity, X-rays, and changes in pressure — all subjects in themselves. Nor have I dealt with dusts and fibres of vegetable origin, some of which have well-recognized hazards, e.g., cotton dust and byssinosis; flour dust and baker's asthma; and mouldy grain and farmer's lung. And, finally, I have not dealt with the stress of the workplace and its possible relationship to diseases such as peptic ulcer, hypertension and emotional disorders.

This cataloguing of some of the major occupational health hazards will serve to underline the importance of Ramazzini's advice in history-taking — "What is your job?".

THE ROLE OF THE PRACTISING PHYSICIAN

What is the role of the practising physician in relation to these environmental health hazards? The physician in family practice has the important role. Th family practitioner is consulted by the worker and his family when they are ill. The family practitioner has the viewpoint of assessing the "whole man in his total environment" of work, home and play. He has the opportunity to provide advice and counsel about lifestyles which may be important in the work environment — drug or alcohol intake with cholorinated hydrocarbons and cigarette smoking with asbestos or pulmonary irritants. The family physician can assess the family situation as well. Some hazards are carried from the workplace and may affect children and spouses in the home, e.g., lead dust, asbestos and isocyanates. Of more concern are the scattered reports of possible mutagenic and teratogenic effects in offspring related to the occupational exposure of their parents. The observation that operating room personnel in hospital produced offspring with a higher incidence than normal of still-birth and congenital defects is one such observation. The observations and enquiries of family practitioners have often provided the first spark in our unraveling of the effects of environmental agents on people.

Physicians should thus enquire into possible environmental causes for diseases which they find in their patients, particularly in those who have unexplained contact dermatitis; pulmonary fibrosis; cancer of the lung, sinus, bladder or skin; leukemia; aplastic anemia; peripheral neuropathy; mesothelioma; angiosarcoma of the liver; non-infectious liver or kidney damage; peripheral neuropathy; Raynaud's phenomenon; and allergic bronchitis or bronchial asthma of recent onset. The family physician should not relate these diseases to the workplace by exclusion of other known causes — rather he should do so on the significant history of exposure to agents known to cause these diseases.

Family phsicians play another important role because they provide most of the occupational health service for workers in small plants — where most workers are employed. Family practice which includes part-time service for small plants in the area can be rewarding and helpful. I would make the plea that these part-time services be more than pre-employment and periodic examinations or emergency medical care. The family physician should be able to take time from his busy schedule to walk through and see the workplaces where many of his patients spend more than one-third of their waking hours. The only hope of providing the prevention, treatment and counselling health services needed for small plants is by the provision of such services on a part-time basis, according to the needs of the particular plant. The approach in Sweden, where the health insurance fund pays 50 per cent of the cost of these services to small plants, is worthy of note.

To fulfill his role in meeting the problems presented by health hazards at the workplace, the family physician will need backup and support. First of all, I believe that the basic medical undergraduate medical education has spent too little time on occupational and environmental diseases. I am advised by my academic colleagues that about 4500 hours of undergraduate education are required to prepare a physician. I would be surprised if formal training in occupational health took more than 2 to 6 hours of undergraduate instruction in most medical faculties. This situation is, I am sure, changing. The teaching of these subjects at undergraduate level should be reviewed in terms of today's priorities. The appointment of professors and/or directors of occupational medicine in university health science centres also helps give visibility and attention to this field.

In dealing with the harmful effects of environmental agents and personal lifestyles on health at the postgraduate level, there is also need to increase the training opportunities in occupational medicine. Optional programs in occupational medicine could well be included in the training of family physicians, internists, community medicine physicians and others. For physicians in practice, short courses on occupational medicine, either on a full-time or part-time basis should be available. The full-time course could range from a single day to those lasting 2 to 3 weeks. Part-time courses could involve 1 to 2 evenings per week over a period of several months. In this way, family physicians could obtain the specialized information needed in this field.

This approach has been used in many European countries where legislation has required that all plants having over a certain number of employees must retain a physician. Such legislation revealed the lack of trained physicians in this field and sparked both undergraduate and postgraduate training of physicians in occupational medicine. I personally would also like to encourage the development of national institutes, perhaps supplemented by regional ones, as needed. These institutes could serve to give the professional and public visibility needed in this field, assist in the necessary training, conduct or arrange for research in this field, provide consultation in occupational medicine, and provide the specialized laboratory support which may be needed. Whatever arrangements are made, the family physician will have to know where to turn to get quick information on the toxicity of substances, where he may send samples for analysis, where he can obtain assistance to make environmental measurements in the plant, or where he can obtain medical consultation or advice in the handling of an occupational health problem in his patient.

I believe that family physicians have other key roles above that of providing the treatment and preventive health service for small plants. Family physicians should have input into the development of occupational and environmental exposure standards — maximum allowable concentrations (MAC's), threshold limit values TLV's), ergonomic standards in the design of work equipment and layout, and physicial requirements of the job.

I have mentioned the need for long-term health studies to detect and measure the influence of environmental agents and stresses at work on such diseases as chronic bronchitis, hypertension, and cancer. These diseases are now more prevalent with our aging population. They have multi-factoral causes among which occupational environmental factors have been assumed by man to play to part. These health studies are needed not only for academic reasons. The data are needed not only to settle questions of workmen's compensation but also to build programs to prevent such diseases. In doing these health studies, I believe that better use could be made of physicians' records, and health insurance records, as well as case registers and vital statistics data. The financing of such studies raises problems. Here again, the example of Sweden is of interest. By payroll assessment of all industries the Swedish Work Environment Board was created to provide funds for research in occupational health.

Family physicians also play their part in community service, by serving as medical advisers on public advisory bodies or community groups concerned about the work environment and the general environment. The general public is concerned primarily about the effect of occupational and environmental agents on their health and well-being. The family physician with his background of education, training and experience in health and human biology, has the background to provide the needed advice and help. As noted above, the practising physician will require quick access to specialist help in environmental health as needed either at university health science centres, government institutes, or both.

In closing, I would like to repeat the advice of Hippocrates, who said that the physician who takes account of environment factors is a credit to his profession in investigating medicine properly. In relation to work hazards, Ramazzini's advice is still direct and useful — remember to ask "what is your job?". So many occupational diseases, once developed, are irreversible, e.g., noise-induced hearing loss, Raynaud's phenomenon of occupational origin, and occupational lung cancer. As physicians, we must stress the recognition of adverse environmental factors and their control in order to prevent environmentally-induced diseases. This applies to personal factors such as cigarette smoking as well as to factors in the occupational and general environment. Practising physicians of today who follow this path would be following in the medical traditions of John Stewart. □

ACKNOWLEDGEMENT

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Bibliography

1. Health Hazards of the Human Environment. World Health Organization, Geneva 1972.
2. Man and Environment — Crisis in the Strategy of Choice. Robert Arvill — A Pelican Original, 1972.

The Development of Occupational Health Services In Cape Breton Mines*

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A modern Occupational Health Service in mining must highlight the prevention of disease, illness and injury.

Appropriate programs are more important than quantitative determinations of personnel and facilities, although these latter increase as the needs and numbers of employees increase. Enlightened senior management of the Cape Breton Development Corporation have assessed the needs and have encouraged the development of appropriate and responsive programs in a modern Health Service for the miners of Cape Breton. This includes programs relating to pre-employment exams, job placement exams, primary attention to on-the-job injuries and illness, both occupational and non-occupational, rehabilitation hygiene and sanitation, periodical physicals, physical-fitness programs, alcoholism and drug-abuse counselling, first-aid training, training for emergency technicians, mass screening, toxicological problems and health education. Advice to operational management and the personnel department regarding compensation problems is also a function.

The developing Health Service has received maximum cooperation from union leaders, who have provided input in planning, along with the Department of Labour. A close liaison with the Accident Prevention Department has been stressed, as well as a continuing health education program for management and union personnel and, of course, on-going and continuous training for health staff personnel.

Credibility is achieved with the passage of time and is measured by program responses. The cost effectiveness of such a modern Occupational Health Service can eventually be measured, not only in dollars and cents, but as well in the optimum mental and physical health of the employees and the organization as a whole. All of this is reflected in better community acceptance and mutual growth.

INTRODUCTION

Over a 21-month period, a perceptive senior management has encouraged the development of a modern, responsive Occupational Health Service, where nothing previously existed. The service has struggled through an early period of distrust and resentment into areas of attention to illness and injury, counselling services, rehabilitation, health education, emergency services, sanitation and hygiene and toxicological problems — to the very important areas of mental hygiene and human resource development — in an attempt to create better motivation, improve job satisfaction and increase team spirit. All of this is part of the answer to further personal-corporate growth and harmonious community relationships.

HISTORICAL PERSPECTIVE AND PHILOSOPHY

It was an unfortunate fact that the Corporation inherited a legacy completely devoid of the smallest fragment of a health service. Nothing existed health-wise, except the memories of older doctors, who had served the old company as "compensation doctors" only. Consideration had not been given to the wide constellation of occupational health needs.

In developing modernization plans for the industry in 1972, the senior management of the Corporation determined that this vacuum should be filled by creating a modern Occupational Health Service for the industry. The director of health services has attempted to reflect their philosophy in developing such services for the Corporation.

INITIAL DEVELOPMENT

Primary planning began in June of 1973. An early attempt was made to explain to all sectors of the industry what was being attempted. Because of the above-mentioned legacy, it was necessary to dispel rumours and untruths, and to explain the ingredients for the development of a modern Occupational Health Service. "Credibility" was felt to be necessary in order to develop a service that would be responsive to the health needs of all employees. Also, it is reasonable to suggest that when employees are functioning at maximum mental and physical wellbeing, that is, healthy, their life spans are prolonged, their families benefit, industrial productivity is enhanced and this is reflected in community development.

The task has been continuously facilitated by enlightened senior management and union leaders, and the utmost cooperation from all sectors of the industry.

THE GUIDING PRINCIPLES AND FUNCTIONS OF DEVCO HEALTH SERVICES

Our Occupational Health Service has been structured on definite industrial health needs and philosophy, which includes:

1. Pre-Employment Examinations — these are carried out in detail, to ensure that personnel entering the industry are fit for the jobs which they are about to undertake, and to determine where they may best be placed, when the need exists.

2. A Primary Treatment Service — for those who are injured or become ill at work; because a comprehensive "medicare" program exists in Nova Scotia, it would serve no

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useful purpose to duplicate the services of the family physicians of our employees for on-going care, beyond the primary stage.

3. Occupational Hygiene — to advise on any matter in the working environment which may introduce a health hazard, such as —

A. Dust problems as these relate to health:
(1) to define attainable and reasonable dust levels;
(2) to advise on the development of dust control techniques to attain these standards;
(3) to measure the amount of potentially dangerous dust in the working atmosphere;
(4) to examine periodically men exposed to dust;
(5) to deal effectively with men whose examinations disclose evidence of developing pneumoconiosis.

B. Noise pollution:
(1) monitor potentially hazardous areas;
(2) assessment of hearing of men in these areas;
(3) advise on protective hearing devices.

C. Toxicological health problems, such as:
(1) carbon monoxide problems;
(2) methane problems;
(3) lead problems;
(4) benzene problems;
(5) etc.

4. Close Relationship with the Accident Prevention Branch — in order to jointly investigate and correct hazards to health in the workplace, related to occupational injuries and accidents.

5. First-Aid Training — to organize, and to assist in providing training for, prompt and effective first-aid procedures.

6. Counselling Services — counselling on problems of alcoholism and other drug-related problems, as well as other emotional-medical problems related to absenteeism and work performance. An industrial health worker will be joined by a social service worker. Also, nurses perform many counselling functions.

7. Compensation — to assist in ensuring that men do not work when they are unfit, and therefore that compensation and sickness benefits operate fairly and effectively for this purpose, without being abused; accordingly, to carry out examinations necessary to advise management and to co-operate with family physicians and union representatives.

8. Rehabilitative Procedures — to supplement public services where available, and otherwise to provide equipment personnel training required for rehabilitation measures following injuries and sickness.

9. Periodic Physical Examinations — to all employees, including those at sedentary occupations, and those at jobs requiring physical and mental strain. As well, providing more frequent examinations of employees working in high-risk areas, or in area of hazard.

10. Physical Fitness — to encourage employees to keep fit, through advice, training programs, diets and explanation of organ functions.

11. Research — to familiarize ourselves with research that has been done in all matters of occupational health related to our industry, and initiate programs, within our capabilities, related to our particular problems.

12. Prevention of other Diseases — Related to Occupational Environment or General —

A. Sanitation & Hygiene — Inspection of plant work areas, shower areas, toilets, eating areas, etc.

B. Plant Health Protection Programs —
1. Blood pressure and weight screening checks.
2. Diabetic urine checks.
3. Mass Screening — (a) chest X-rays
   (b) hearing defects
   (c) vision defects
   (d) electrocardiographic changes
   (e) lung function testing
4. Influenza and Tetanus Immunization Programs

STAGES IN THE DEVELOPMENT OF HEALTH SERVICES

1. Nursing Stations

Because of the widespread geographical nature of our Corporation, after initial planning it was felt that this first stage was necessary. Five Nursing Stations have been designed and provided to meet these needs. The Nursing Stations have now been in operation since January, 1974. Each Station is staffed by a nurse around the clock. It is equipped to handle any immediate emergency. At each mine, this is the central health facility for primary treatment of injuries or illness, with subsequent referral to family physicians. The nurses have familiarized themselves with underground travel. All nurses have received a preliminary two-week comprehensive course in occupational health problems, with lecutres from experts in many fields. Ongoing training programs are being provided.

The nurses provide counselling services, review sanitation in the plant, assist in mass screening procedures as mentioned, discuss diets and health matters, and provide the manager with information regarding their initial treatment of injuries or illness. The nurse is also in contact with the family physician of the employee. Confidentiality of medical information is always stressed.

Physiotherapy equipment is being provided at the Stations to assist in our function of early rehabilitation of an injured workman. The family physician or specialist will advise on the type of treatment.

Health education is stressed, utilizing discussion and pamphlets.

First-aid training is performed at each plant. The director of health services visits each Station at regular intervals, as well as the head nurse and the industrial health worker — in order to attend to individual functions regarding personnel and to discuss problems.

Close contact is kept with the manager and the industrial relations officer, as well as the accident prevention officer.

The nurses at our Stations relate to social service agencies in the communities. Personnel at Stations also relate to and are part of the Emergency Measures Organization in each community.

Reports from all quarters seem to indicate that the Nursing Station personnel have been a welcome adjunct to each plant.

2. Diagnostic Center

This was planned, designed and constructed at the Devocal Building in Glace Bay. It serves as a coordinating center for the entire health service. Facilities are provided for the director of health services, the head nurse, the medical record librarian, the industrial health worker, the first-aid training officer, the medical secretary, the chief of emergency underground technicians, the occupational hygienist and the industrial social worker.

In addition to basic primary treatment facilities, for those not employed at the major plant sites, diagnostic equipment has been provided, such as audiometry, electrocardiographic and vision testing equipment. Also, a spirostat lung function determinator has been provided, as well as an automatic lung function machine.

Most pre-employment and periodic examinations are done in this area, as well as pension examinations and executive physical examinations. Various types of necessary occupational health forms have been designed, to be utilized not only for present needs, but for future research needs. Many employees with occpational health problems are interviewed and examined here, as well as at the Nursing Stations.

The staff functions at this location, as well as periodically at Nursing Stations, using a definite schedule for utilization of their time and services.

Most of the diagnostic equipment is portable, so that it may be utilized at Nursing Stations for mass screening procedures, such as vision testing, audio-metric testing, pulmonary function testing, etc.

Haemoglobins, white blood cell determinations and urinalyses are performed at the central area as well as at Nursing Stations. Blood for more complicated determinations may be collected and sent to the general hospital in the area when required.

Health Service personnel meet on a monthly basis to discuss matters pertinent to the functioning and development of the service. A monthly report is required from all personnel. The director of health services submits a monthly report to the vice-president of the Coal Division, outlining activities as well as recommendations for further discussion.

The central facility mentioned above has been operative since July of 1974.

3. Development of Underground Emergency and First-Aid Health Services

This further stage in the development of appropriate services to meet the needs of men who are injured or become ill at the underground workings has been achieved. A multidisciplinary committee was formed to advise the director of health services on this aspect of occupational health. The committee was composed of —

(1) the director of health services as chairman;
(2) the director of mines planning;
(3) the U.M.W. safety coordinator;
(4) the Corporation accident prevention director;
(5) medical specialist — traumatology — Victoria General Hospital, Halifax;
(6) president of Supervisor's Association;
(7) president of Shotfirers Union;
(8) mine rescue superintendent;
(9) first-aid officer;
(10) Department of Labour representative.

At each mine, the committee spent the mornings travelling underground, and reviewing the facilities and equipment. The committee was accompanied by the Manager and Union Safety Committee at each mine.

Afternoons were spent discussing present layout and proposed changes. As a result of the deliberations of this committee, which was representative of all sectors of the operation, proposals (which embrace types of mobile stations, as well as deployment of underground personnel, who would have first-aid responsibilities) were considered. Underground emergency technicians have been hired and trained, prior to assuming underground duties.

ONGOING PROGRAMS AND FUTURE DIRECTION OF HEALTH SERVICES

There are several areas which are now receiving further consideration, in order to continue the progress and orderly development of our Health Service.

1. Underground emergency and first-aid services, as discussed above, have been initiated.

2. Occupational health programs, which relate to a wide variety of occupational hazards. An occupational hygienist is being trained to assist in the development of a Hygiene Section.

A. Pneumoconiosis or Coal Miners' Lung Disease — "Total Dust Program":

(1) In October of 1974, a "Coal Miners' Lung Disease" Conference was held in Cape Breton, with leading experts in this field from Canada, Britain and the U.S.A. utilizing a multidisciplinary approach — medical and engineering — to this problem.

As a result of knowledge gained at this Conference, we are proceeding in the further development of up-to-date monitoring and measuring techniques for our respirable dust hazard.

Unfortunately, in previous days, there was not much attention paid to this. However, the Corporation is actively pursuing the institution of this program. Technicians have been hired and trained.

(2) Dust control measures — much has been done by the Corporation to bring up-to-date techniques in this area into practical application.

(3) Periodic assessment of personnel working in high-exposure areas is being carried out.

(4) The coordination of the above three measures, in order to continuously remove the hazard of dust in an optimum fashion, is proceeding.

B. Noise Pollution Problems — and their hygienic problems — must be approached in an organized monitoring fashion, as earlier mentioned. This has now been initiated.

3. Further development of health through physical fitness programs.

The federal Department of Health is showing much interest in this aspect of occupational health, and has called for a conference in the near future to discuss this problem. We now have programs operative in three geographic areas.

4. Continued coordination of various departments in the Corporation to further reduce the number of injuries at the workplace.

The Accident Prevention Department has instituted programs of training, reporting of accidents and injuries, and continuing assessment of the types and number of injuries and manner of occurrence. However, many physical and psychological factors relate to the cause of injuries, all the way from poor housekeeping to accident-proneness. Factors varying from problems at home, to alcoholism, to all sorts of medical or psychological disorders may underlie injuries, particularly recurring ones. We are developing counselling services so that cases requiring medical or psychological counselling and treatment are referred to the Health Services.

Incentive programs have proved to be very effective in many major industries. They should be carefully planned and administered in small groups. They add to *esprit de corps*, and make employees more conscious of the prevention of injuries or accidents.

In short, the Corporation's philosophy is that co-ordination of accident prevention, industrial relations and health services, along with liaison with managers and supervisory personnel, can do much to lessen the incidence of both minor and crippling injuries in our industry.

5. Mental hygiene and human resources development.

It is easily appreciated that an organization is only as functional as its degree of mental and physical health. The Health Services Branch realizes that mental health, within the work organization, and the utilization of human resource potential becomes very important. These factors are reflected in work performance, work satisfaction, motivation, handling of stress, individual and corporate growth, teamwork, promotional factors and recognition for a job well done, etc. A combined interdepartmental, multidisciplinary approach to this all-important area is very appropriate in the modern work organization, and its changing patterns and roles. The Health Services Branch and its personnel have a vital role to play in this particular approach.

URGENT CHALLENGE

I would like to throw out to you what I consider to be an urgent challenge. We can all take a great deal of pride in the successes of the mining industry and in the development of our natural resources. We must now, and with a sense of urgency, look at all we are doing to develop the *human resources* in the mining industry. What are we doing to help fulfill the basic human needs of all our employees for growth and development? What are we doing to harness the tremendous human potential that exists within our industries and turn this into a very productive and satisfying force for human and corporate growth? What are we doing to increase the motivational factors within our industries? What are we doing to increase the job satisfaction within our industries, and what factors have we introduced or are in existence to develop to the maximum the mental health within our work organizations?

Some will say that nothing can be done in this direction. It is my feeling that a tremendous amount can now be done along these lines if we will address ourselves to the particular area that needs development. I also feel that we must do this with a sense of urgency if we are to see the further growth and development of the mining industries in our country, and if we are to avoid a depletion of our work force because of the fact that increasing opportunities for personal growth exist in other areas.

In this part of the twentieth century, it behooves us to look closely at this problem for these reasons. Another factor is that we must take an intedisciplinary approach in order to bring together expertise. The Cape Breton Development Corporation, in October of 1974, was confronted with an acute problem on top of a chronic existing problem of coal miners' lung disease, a problem indigenous to and inherent in any coal mining operation. We called together a Conference utilizing the multidisciplinary approach and bringing together engineers, doctors, technicians and scientists. Initially, we were told that this approach would not prove useful in the solution of our dust problem. We have now found this to be a totally inaccurate statement. Based on the results of our Conference, we are now able to put togehter an extremely useful "total dust program" for our industry. Hopefully, this will cut down tremendously and perhaps even eliminate coal miners' lung disease. This remains to be seen. However, the multidisciplinary approach to this problem has proved to be very effective and all segments have harmoniously co-operated in the solution of the problem.

We must now apply this same approach to the very important development of human resource potential and growth in our industries.

CONCLUSION

An outline of the development of modern Occupational Health Services in the Cape Breton Development Corporation has been given. The aim is to make the service as responsive as possible to the employees. Because of an inherited climate of mistrust, credibility is achieved with the passage of time and is measured by responses to programs. The cost effectiveness of such a modern Occupational Health Service can eventually be measured, not only in dollars and cents, but as well in the optimum mental and physical health of the employees and the organization as a whole. All of this is reflected in better community acceptance and mutual growth. □

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Shedding New Light on an Old Problem

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During the last twelve years in Nova Scotia the incidence of neonatal death† has decreased from 11 to 4 per 1000 live-births and of perinatal death from 24 to 10, decreases of 64 and 58%. As this epidemic comes increasingly under control physicians involved in the delivery of perinatal care have the challenge to turn their skills from the now preventable diseases such as respiratory distress syndrome, asphyxia neonatorum, and fetal malnutrition to some of the less common causes of death, such as fetal and neonatal infection.

We are fortunate indeed that fifteen years ago one of our most able scientists took up the TORCH against intrauterine infection. Dr. Juan Embil, Dr. Shabtai Affias and their many co-workers have struggled many years to produce for us a solid resource of investigational techniques to throw light on this increasingly prominent problem of intrauterine infection.

Those of us in the field of clinical perinatology are extremely grateful for this definitive reference article, which will be available to all maternity health workers in Nova Scotia. As well as being a review article it is a summation of a series of articles in the world literature which the senior author is too modest to mention. It is only one of the benefits which have been produced by the staff of the Infectious Diseases Research Unit at The Izaak Walton Killam Children's Hospital. □

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†Death during the first 7 days of life in infants weighing more than 1000 gm at birth.

Congenital Infections and the TORCH Syndrome

PART I

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PERINATAL INFECTIONS

Toxoplasmosis
Other — syphilis
listeriosis
group B beta haemolytic streptococcus
Rubella
Cytomegalovirus
Herpes Simplex Virus

Infections during pregnancy are receiving increasing attention for several reasons: new methods and techniques allow detection of the infectious agents; the potential devastating effects of these infections on the foetus and newborn are now realized; and there is suspicion that many infectious agents that do not seem to cause disease in parents are sexually transmissible and harmful to the foetus. Organisms such as Group B beta haemolytic streptococcus, mycoplasma, chlamydia and cytomegaloviruses are examples. Further, there are new approaches (including vaccination, chemotherapy, therapeutic abortion and caesarian section) to preventing the birth of diseased infants.

When infection is diagnosed in the pregnant patient, information is needed on the risk of transmission to the foetus or newborn and the consequences in terms of possible congenital abnormalities, clinical manifestations and long term complications that may be unapparent at birth, so that a balanced decision can be made on managing the pregnancy.

Since many of these infections produce similar foetal and neonatal effects, clinical differentiation of the causative agents is difficult. They have, therefore, been collectively called the "TORCH complex" of perinatal infections and include Toxoplasmosis; Other, eg. syphilis, listeriosis, group B beta haemolytic streptococcus; Rubella; Cytomegalovirus infection; and Herpes simplex infection.

The wide range of infecting organisms includes bacterial, chlamydial, fungal, viral and parasitic agents (Table I). They may be acquired during the embryonic, foetal, natal and neonatal periods. The mode of transmission varies accordingly; it may be intrauterine (transmitted transplacentally or via an ascending infection), or it may occur during parturition by ascending infection or during the passage of the infant through the birth canal, or in the neonatal period from the mother, nursery personnel and other persons.

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TOXOPLASMOSIS

The Organism

Toxoplasma gondii is a unicellular parasite (a coccidian). The definitive host for this organism is the cat, while swine, sheep, chickens and possibly cows serve as intermediate host and man is only an accidental host. Man is infected by ingesting either meat infested with cysts or oöcysts excreted by cat. The organism exists in human tissue in 2 forms: trophozoites and tissue cysts.

Transmission

The infection is acquired by the ingestion of insufficiently cooked meat or by accidental ingestion of oöcysts (from soil or contaminated food). Toxoplasmosis may be acquired by WBC or whole blood transfusion. Congenital infection is acquired during maternal primary infection; the organism first infects the placenta and subsequently the foetus. Congenitally infected infants are a potential hazard, since organisms have been recovered from their urine, faeces, tears and saliva.

Epidemiology

The organism is ubiquitous and human infection is common. The prevalence of toxoplasma antibodies varies in different populations of pregnant women and may reflect social and culinary preference. In Halifax the prevalence is 13.7%, essentially the same as that in Berlin (13.2%). This is in contrast to 32.8% in New York City and 87.5% in Paris. The maternal risk of acquiring toxoplasmosis during pregnancy varies from 0.2% (in New York City) to 0.7%

TABLE I

INFECTIOUS AGENTS OF THE FETUS AND NEWBORN

Bacteria	**Viruses**
Group B beta Haemolytic streptococcus	Cytomegalovirus
Listeria monocytogenes	Herpes simplex
Escherichia coli	Varicella-zoster
Other enteric and gram negative bacteria	Variola, Vaccinia
Treponema pallidum	
Neisseria gonorrhea	Hepatitis B
Mycobacterium tuberculosis	Rubella
	Enteroviruses
Mycoplasma	Rubeola
Ureaplasma urealyticum (T. mycoplasma)	Mumps
Mycoplasma pneumoniae	Influenza A
Chlamydia	**Fungi**
Chylamydia trachomatis	*Candida albicans*
	Protozoa
	Toxoplasma gondii

pregnancies in Paris. However, only 36% of gestational toxoplasmosis results in foetal infection.

The gestational age when infection occurs is an important consideration. Incidence of foetal infection during the first trimester is low (about 17%), but 80% of infected foetuses manifest congenital abnormalities. During the second trimester foetal infection is 25% with half of those infected being abnormal. Acquisition of congenital infection during the third trimester is 65% but only 8% of those infected will be symptomatic. The effect of maternal chronic infection is not clear. Some studies claim increased incidence of repeated abortions in chronically infected women.

Pathogenesis

The ingested parasite penetrates the cells lining the gastrointestinal tract and local multiplication occurs. The trophozoite may persist in macrophages and be carried in the blood stream and seed many organs including the muscles, heart and lungs. In congenital toxoplasmosis the CNS is invaded as well as the eye tissues, resulting in chorioretinitis. Congenital chorioretinitis may be a major component of hypersensitivity that can manifest itself many years after the original invasion.

Pathology

In fatal cases the CNS is never spared. Examination reveals necrosis of brain parenchyma due to vascular involvement. Periaqueductal and periventricular vasculitis and necrosis are prominent, with subsequent calcification. Stenosis of the aqueduct may result with hydrocephalus as a consequence.

Clinical Manifestations

Where the disease is overt in the neonatal period, two major clinical forms can be distinguished: neurological disease where chorioretinitis, abnormal CSF, intracranial calcification, hydrocephalus and convulsions are very frequent and the "generalized disease" where hepatosplenomegaly with jaundice, fever, lymphadenopathy, anemia, eosinophilia, abnormal bleeding and rash predominate. Some manifestations may not be evident at birth and may actually be progressive; these include deteriorating vision, deafness and mental retardation.

Diagnosis

Isolation of the organism is difficult but is achieved by mice inoculation.

Serology

Several tests are available, for example, the Sabin-Feldman dye test which is the most reliable, but is difficult technically; indirect hemagglutination which is not reliable for congenital toxoplasmosis and the indirect fluorescent antibodies (IFA) test which compares favourably with the "dye test". The fluorescence technique has been adapted for detection of specific toxoplasma-IgM. Demonstration of this IFA-IgM in cord blood if there is no maternal blood contamination is a good presumptive diagnosis of congenital toxoplasmosis. However, up to 75% of newborns with congenital toxoplasmosis may be negative for IFA-IgM. Thus, maternal blood must be examined. The presence of high titres in the dye test and positive IFA-IgM make the diagnosis of congenital toxoplasmosis probable. Extremely high dye test titres with negative IFA-IgM make the diagnosis possible. Otherwise, serological diagnosis is difficult. Serological diagnosis of maternal acute infections is indicated by a high titre at dye test, and positive IFA-IgM indicates recent primary infection.

Treatment

On an investigational basis, two regimens have been tried in pregnant women: 1) a combination of pyrimethamine and sulfadiazine and 2) spiramycin. However, caution is necessary since the administration of pyrimethamine to experimental animals resulted in teratogenic effects; this treatment is therefore contraindicated during the first trimester of pregnancy. Treatment of congenital toxoplasmosis with pyrimethamine and sulfadiazine alternating with spiramycin is also under investigation.

Prognosis

Five year follow-up in clinically apparent congenital disease by Eichenwald[1] revealed a 12% mortality rate. Of the survivors, 85% were mentally retarded and most of these had severe CNS symptoms as well. Severely impaired vision occurred in 50% and severe hearing loss in 13%. In subclinical cases evaluation is difficult.

Prevention

For the seronegative pregnant women strict hygenic habits are needed. Consumption of thoroughly cooked meats and eggs; avoidance of handling cats, faeces, sand or soil, and thorough washing of fruits and vegetables are recommended.

RUBELLA

Interest in intrauterine diseases was rekindled in 1941 when rubella, usually a mild exanthematous disease, was linked to the prenatal acquisition of infection causing severe foetal malformation.

The Organism

Rubella virus is a togavirus. It consists of an RNA core, and a lipid-containing envelope, measures 50-100 nm, and is pleomorphic.

Transmission of Congenital Rubella

Transmission is transplacental, usually early in the first trimester, although transmission during the second trimester has been reported.[2]

Postnatal Transmission

Transmission is person to person. Congenitally infected infants present a significant risk, especially to pregnant attendants.

Epidemiology

Man is the only host. Rubella is more prevalent in the spring with peak attack rates from March to May. Small epidemics occur every 6-9 years, and major epidemics every 10-30 years. The attack rate is difficult to assess because of the mildness of the disease and the high ratio of unapparent infections, but in closed communities 90-100% of susceptibles will become infected. It is most prevalent in the 5-14 years age group. About 10-15% of women of child-bearing age are susceptible to infection. Of 3658 pregnant women surveyed at the Grace Maternity Hospital, Halifax, in 1976, 442 (12.1%) were susceptible.

Report to the Minister of Labour

Committee on Medical and Adjudicative Procedures in Pneumonoconiosis Claims before the Workmen's Compensation Board.

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INTRODUCTION

In early 1977 the Minister of Labour for the Province of Nova Scotia wrote to Dr. John F. L. Woodbury instructing him, "please consider this letter as your authority to review and assess medical and adjudicative procedures relative to Silicosis and Pneumonoconiosis cases that go before the Workmen's Compensation Board and make recommendations to me regarding any changes that you consider necessary."

A committee was formed to carry out this charge comprising Dr. Woodbury as Chairman together with Monsignor Malcolm A. MacLellan and Dr. F. Murray Fraser. Mr. Daniel M. Campbell acted as legal counsel and secretary to the committee.

There had been controversy over compensation of coal miners in Nova Scotia. Occasional instances of apparent inequity had been identified and publicized. In addition, in certain areas of the United States, it had been legislated that coal miners who had spent a given number of years underground who had become totally disabled should automatically be assumed to be suffering from industrial injury to their lungs and compensated on that basis.

The definition of Pneumonoconiosis contained in the Workmen's Compensation Act had been amended from time to time, most recently in 1975, to read:

"74. In this Act, pneumonoconiosis means permanent alteration of lung structure due to inhalation of dust and the tissue reactions of the lung to its presence."

Also in 1976, s. 9A had been inserted, reading:

"9A. Any coal miner who has worked at the face of a mine, or in similar conditions for 25 years or more and who suffers from a demonstrable loss of lung function about which there is doubt as to whether or not it is Pneumonoconiosis or Silicosis and which cannot be attributed to any other cause will be compensated according to his disability."

PROCEDURE

The committee proceeded by reviewing relevant medical literature and interviewing selected medical specialists concerned with the problem. The committee met with the Workmen's Compensation Board, its Executive Director, and subsequently with its Chief Medical Officer. A meeting with the Workmen's Compensation Appeal Board was held. The committee heard presentations in Sydney by representatives of "the Committee for Automatic Assumption of Black Lung", a coalition of interested groups, DEVCO, representatives of United Mine Workers of America District 26, and local unions in the district, the Glace Bay Medical Society, and concerned coal workers. The committee examined Workmen's Compensation Board files of all claims which had been dealt with by the Workmen's Compensation Appeal Board, and a number of other selected files. The committee also received written submissions from a number of other people.

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THE WORKMEN'S COMPENSATION ACT

As authors of various reports on Workmen's Compensation Acts throughout Canada have noted, any examination of the program must be conducted in light of its history and the evils it was designed to rectify. While we have no mandate to consider and recommend on the policies of the program, we consider such background information equally vital to proper consideration of the procedural aspects of the program.

Before the enactment of the various Acts, an injured workman's sole remedy was by a law suit against his employer. This inevitably involved a considerable delay during which the employee was without pay and was required to pay his own medical expenses. He was placed in an adverse position to his employer, which might result in loss of his employment. He could recover only if he could establish negligence on the part of his employer or someone for whom the employer was responsible, the sole duty resting upon the employer being to take reasonable care for the safety of his employees. Even when such negligence was established, the employer could escape entirely or in part by proving negligence on the part of the employee An employee who was so fortunate and persistent as to win at trial frequently found himself faced with an appeal by an employer much more financially able to pursue such an appeal than he. Under financial pressure the employee was frequently faced with the necessity for surrender or compromise. A further hazard was that the successful employee might face difficulty in collecting if his employer was financially unsound. In addition, the employee so fortunate as to recover was faced with the not inconsiderable legal expense of such proceedings.

To rectify this obviously unjust situation the Workmen's Compensation Acts were enacted, including principles of what we have come to call "no fault insurance." The essential features which are included in our Act are:

1. Entitlement to compensation regardless of fault.
2. Collective liability of all employers through the "accident fund" (in the Nova Scotia coal mining industry the dominant employer, DEVCO, is individually liable respecting claims by its employees by federal-provincial arrangement under the Government Employees Compensation Act).
3. Accident fund maintained by employer contribution without any direct or indirect employee contribution.
4. Payment of compensation not affected by default or insolvency of employer.
5. Right to compensation is in lieu of right of action against employer or third person (in third party cases the Workmen's Compensation Board has a right of subrogation).
6. Level of compensation is determined in accordance with legislative standards and is subject to limits. There is no entitlement to compensation for pain and suffering but only for lost income earning capacity.

The Workmen's Compensation Board is a ministerial agency designated by the Act as a body corporate having responsibility for administration of the program as provided for in the Act. It does not receive funds from the government, but rather all funds are supplied from the accident fund.

Social conditions have changed dramatically since the Workmen's Compensation Acts were conceived sixty years ago, especially in the last twenty years. Today, with the gradual encroachment of external influences caused by legislation covering health care, income maintenance, job placement and training services, and as a result of greater interest of the general population in the nature and administration of social programs, the Workmen's Compensation Board has become a high profile organization.

Public expectations are that the Workmen's Compensation program is part of a general scheme of social benefits. This leads to demands on this program which go outside its original insurance-based principles. This divergence between public expectations of the program and its actual operating policies and principles have led to some dissatisfaction with the program.

THE PROBLEM

The history of mining, and of coal mining in particular, has been marred by accidents and diseases of a serious and severe nature. Coal miners, by reason of dust exposure, have been highly susceptible to pulmonary ailments and specifically the disease known as Coal Miner's Pneumonoconiosis. The wide spread prevalence of lung disorders among the coal miners is a matter of grave concern for the mining population and the coal miners union. Increasing awareness of the problem has resulted in demands for more generous compensation for industrial injury. Alterations to the Workmen's Compensation Board Act including the setting up of the Workmen's Compensation Appeal Board have greatly increased the expectations of coal workers concerning Pneumonoconiosis compensation.

Consequently, a rapid increase in the number of claimants has occurred in recent years. This resulted in delays of a few months in obtaining appointments for examination by lung specialists. In the 1976 report of the Workmen's Compensation Board of Nova Scotia it is stated (page 17):

"With the increase in the number of cases being reported, a problem had developed for a few years of not having examination and clinical findings undertaken as promptly as might be desired. This was due to the lack of chest specialists in Canada particularly in eastern Canada. In the summers of 1972 to 1976 inclusive the W.C.B. arranged for a highly qualified specialist to come from England for the purpose of examining and evaluating a large number of open cases.

This specialist was a highly qualified medical man who had spent many years dealing with silicosis and pneumonoconiosis in the U.K. in the coal fields of England and Wales.

By the Fall of 1973 the backlog of examinations and clinical findings for cases was up-to-date and new cases reported are now dealt with as received.

Another factor in handling the large increase of pneumonoconiosis cases is the fact that there are only three pulmonary function machines availabe in Nova Scotia that can evaluate the type of lung disease that inhalation of coal and silica dust produce. These machines are located in Halifax hospitals and are in heavy demand. With these chest conditions, it is essential that pulmonary function tests be undertaken."

In Nova Scotia since 1970 about 3800 claims were made and considered for Workmen's Compensation. So far about 1400 claimants have been or are being paid compensation benefits due to the compensable conditions of Pneumonoconiosis or Silicosis. Around 1150 of these are still receiving benefits.

THE DISEASE

The Pneumonoconioses are complex diseases presenting difficulties in diagnosis and assessment. They are more difficult to understand for persons who have no scientific training in the field. Particular problems are:

a. The very slow progression of the disease, especially in the early stages.

b. The difficulties of early positive diagnosis.

c. Individual susceptibility to reaction to inhaled dust resulting in wide variation in time of exposure required to produce evidence of permanent alteration of lung substance.

d. The wide variation and intensity of exposure in different mines but also in different areas of the same mine.

e. Individual susceptibility to progression to "complicated Pneumonoconiosis", a much more disabling condition.

f. The frequency with which Pneumonoconiosis is associated with other pulmonary disease.

g. Differing medical opinions as to the characterization of the disease.

The trachea, bronchi and bronchioles are tubes that allow transport of air to the essential or functional cells of the lungs. The functional cells are arranged in sac-like structures closely applied to capillary blood vessels. Through the functional cells, oxygen diffuses into the blood and carbon dioxide diffuses from the blood into the air. A little fibrous tissue is present to provide form to the lung. Some cells move about in the walls of the air sacs and protect the functional units from noxious agents, including dust particles. These cells may give adequate protection but their protective function can be overwhelmed and in that case what is normally protective may become injurious. When this happens material collects in the functional units of the lung. Such material may be opaque to an X-ray beam and seen as nodules on the film of the chest X-ray. Such nodulation only occurs in the functional units of the lung which are beyond the terminal bronchioles.

There is agreement among the best medical authorities that roentgenographic (X-ray) assessment, although not infallible, is the only satisfactory method for assessing the presence and degree of permanent alteration of lung structure during life. Rejection of a claim on the basis of complete lack of roentgenographic evidence of Pneumonoconiosis, in the absence of compelling evidence in the report of the claimant's physician, is therefore appropriate.

Pulmonary function tests provide a fairly objective measure of the degree of disability but do not identify the cause. The committee has considered carefully the opinions of those medical authors who claim that pulmonary function studies can diagnose lung disease of coal miners. We consider that they are dealing with the sum of all the lung conditions affecting the groups of subjects studied by them and that their observations are not confined to Pneumonoconiosis.

We believe that the definition of Coal Workers' Pneumonoconiosis, used by our medical experts and set forth in the Act describes a condition peculiar to the coal working industry, therefore justifying compensation.

Schedule "A" of the Nova Scotia Workmen's Compensation Act of June, 1976 lists the industrial diseases which are compensable under the Act, including "Coal Miners' Pneumonoconiosis."

Coal Miners' Pneumonoconiosis can be defined, for medical purposes, in many ways, see Appendix 1. The most inclusive definition is one which attempts to include almost all respiratory conditions found in coal miners in the term "lung disease of coal miners." Our committee requested comments on the legal definition of Coal Miners' Pneumonoconiosis by several chest physicians. One answer was particularly interesting. It came from Dr. J. C. McVittie, employed by the British National Health Services in relation to its compensation program, a member of the London Panel on Pneumonoconiosis. This medical authority wrote:

"You may be interested to know that in the United Kingdom in 1968 the question, whether in the light of experience and current knowledge any adjustment should be made to the definition of Pneumonoconiosis in the Industrial Injuries Act (fibrosis of the lungs), was referred by the Industrial Advisory Council to their Industrial Diseases Sub-Committee. After 29 meetings between January 1969, and March 1972, and hearing oral evidence and considering written evidence from many sources, including all the experts with knowledge of the clinical and radiological aspects and the pathology of Pneumonoconiosis, the committee recommended: 'The expression Pneumonoconiosis means permanent alteration of lung structure due to the inhalation of mineral dust and the tissue

reaction of the lung to its presence, but does not include Bronchitis and Emphysema . . .' however, no action was taken on the suggested definition by the Conservative Government at that time nor by their successor the Labour Government."

It is the view of our committee that since the bronchi form part of the lung and since permanent alteration of the bronchi could be produced by Bronchitis, the present definition under the Act could be interpreted to make industrial Bronchitis produced or aggravated by coal mine dust a compensable disease. Although there is considerable evidence that coal mine dust is capable of producing Bronchitis and measurable impairment of pulmonary function, it is not possible to prove that these changes were not due in any individual to other causes. Identical changes in broncho-pulmonary functions can be caused by smoking cigarettes, by asthma and other respiratory diseases. The incidence of asthma in the general population is high. It can lead to serious respiratory impairment and disability. Similarly Chronic Bronchitis affects a large proportion of our population. It may occur without obvious cause, it may result from widespread atmospheric pollution, probably the greater number of cases are induced by tobacco smoking. This disease can lead to serious respiratory impairment and disability.

General emphysema (known medically as Panacinar Emphysema) occurs sometimes without known cause and is a seriously disabling disease.

We recommend **that the definition of Pneumonoconiosis in the Act be amended to add the words "but does not include Bronchitis and Emphysema."**

We do, however, agree with the present policy of the Workmen's Compensation Board which is, we have been told, that the disability awarded to a workman granted compensation for Pneumonoconiosis is enhanced when Bronchitis or Emphysema is associated with the disease. This conforms to British practice as described by Dr. McVittie:

"Pneumonoconiotic miners who also suffer from these diseases have the assessment of their disability weighted by the aggravating effects of Bronchitis and Emphysema on the effects of the Pneumonoconiosis."

It also conforms to advice rendered to a former Minister of labour of Nova Scotia that:

"It is not possible to determine accurately what proportion of the Bronchitis present in an individual workman who has been exposed to industrial dust is due to the dust, and what part is due to atmospheric pollution, tobacco smoking, infection and other factors somewhat ill-defined and ill-understood of a socio-economic nature. This knowledge should be taken into consideration when determining compensation. The decision must be arbitrary, but to ignore it would be unjust and lead to grave dissatisfaction on the part of the workman concerned."

We therefore recommend **that when Bronchitis or Emphysema is associated with identifiable Pneumonoconiosis the workman should be compensated for the amount of disability arising from both respiratory conditions.** This is in accordance with the present practices of the Board and its consultants.

A great deal remains to be learned about Pneumonoconiosis. The prolonged inhalation of coal mine dust, comprising carbon, some silica and undoubtedly many other potentially irritating substances eventually overwhelms the normal mechanisms of the bronchi and bronchioles for cleansing themselves. Deposition of dust in the lungs can then be detected by X-rays. Similar dust deposition in the lungs will be found in persons dwelling in atmospherically polluted areas and does not cause appreciable disability. In both these instances the lungs appear black. In the case of the coal worker, coal dust is coughed up in the sputum.

The term "disease" implies the recognition of a disorder that produces symptoms or disability. It is when the X-ray commences to show nodular shadows that the expert can state that the condition of dust inhalation has progressed to permanent alteration of lung structure.

Identifiable factors of individual susceptibility correlate well with progression of Pneumonoconiosis to its complicated form, in which large areas of lung are damaged and ultimately replaced by massive accumulations of fibrous tissue surrounded by general emphysema. Some factors so identified include antibody to altered gamma globulin ("rheumatoid factor"), cellular immunity to mycobacterium tuberculosis, and allergic constitution. There are other factors not yet identified. Progressive massive fibrosis is now referred to as complicated Pneumonoconiosis, a term which recognizes its relationship to simple Pneumonoconiosis, which in turn is clearly an industrial disease in that it is "peculiar to or characteristic of a particular industrial process, trade or occupation."

Factors of individual susceptibility also underlie chronic bronchitis and emphysema. Undoubtedly, industrial atmospheric pollutants contribute to the causation of chronic bronchitis and emphysema in coal workers. Other pollutants, most convincingly tobacco smoke, also participate in causation of chronic bronchitis and emphysema. In the absence of evidence of the presence of Pneumonoconiosis, however, no way exists of demonstrating that a miner's bronchitis or emphysema is primarily a result of his working conditions. The principle of the Act is to compensate workmen suffering from disabling diseases caused directly by their working conditions rather than diseases personal to the workmen. This is illustrated by the definition of "industrial disease" in s. 1 (1) (i) of the Act.

Not to compensate coal miners for bronchitis may seem to be unjust to the workman. However, since it is impossible to prove that the principal cause of a miner's bronchitis is material to which he is exposed in the mine, to compensate for bronchitis in the absence of Pneumonoconiosis would be at variance with the intent of the Act and unjust to all disabled bronchitic individuals not employed in coal mines.

CLAIMS PROCEDURE

The vast majority of claims to the Workmen's Compensation Board involve temporary disability from physical injury and are handled by the claims staff of the Board. Because of the difficulties inherent in Pneumonoconiosis claims, and because the condition is invariably permanent, a different procedure has been evolved from these claims:

1. The claim is initiated by the claimant, usually on the advice of his family physician. Forms are completed by the claimant, his physician, and his employer outlining the claimant's work history and, on the doctor's form, the doctor's findings of the claimant's physical condition.

2. Chest X-rays taken in the claimant's home area are submitted together with the three above-mentioned forms to the Workmen's Compensation Board. These are reviewed by the Chief Medical Officer of the Workmen's Compensation Board and are referred by him to two specialists. If either of these specialists feels there is sufficient indication of Pneumonoconiosis, the claimant is brought to Halifax for further examination. If both agree that there is no indication of Pneumonoconiosis the claim is denied.

3. In the cases of claimants brought to Halifax, further X-rays are taken and reported on by a radiologist in Halifax. The claimant undergoes pulmonary function tests under the auspices of either Dr. Charles Gordon or Dr. John Dill. He is physically examined by Dr. Dill or Dr. Gordon, who notes his findings and prepares his report. The X-rays, pulmonary function tests results, and physical examination findings are referred to the other consultant who prepares his report on the basis of these. Both reports are returned to the Chief Medical Officer of the Board.

4. The Chief Medical Officer of the Board reports to the Board, and the claim is either allowed or denied at that time. It is to be noted that all Pneumonoconiosis claims are considered by the Board. This is not the case in other claims which are typically handled by the staff of the claims department.

5. Whether a claim is allowed or denied, the claimant is referred for re-examination at a future time, usually 3 to 5 years off.

6. It was formerly the practice of the Board to make the specialists' reports available to the claimant's family physician. This is no longer done and the Board now sends only a statement of whether Pneumonoconiosis was or was not diagnosed. The Board gives no

reasons for its disallowance of the claim other than this notification that the disease was not diagnosed.

7. It was formerly the practice of the Board, in cases where two specialists disagreed, to refer the information to a third specialist whose opinion would determine the result. Pursuant to decisions of the Workmen's Compensation Appeal Board, apparently based on the theory that if the physicians disagree there is "doubt" and s. 20 applies, the Board has adopted a practice of allowing the claim if either of the specialists diagnoses Pneumonoconiosis.

8. The decision of the Board as to both the existence of an industrial disease and the degree of disability is based on the reports of the experts.

9. The claimant (the term is to include relatives of a deceased workman) who is dissatisfied with the decision of the Workmen's Compensation Board has a right of appeal to the Workmen's Compensation Appeal Board. This tribunal operates quite separately from the Workmen's Compensation Board. Every claimant can appeal as of right and is entitled as of right to a hearing.

The Committee has been given a copy of the Executive Director's memo entitled "Policy Guidelines for the Pneumonoconiosis/Silicosis Claims" effective as of and from June 1, 1977. These guidelines specify that each claim finally has to be considered on its own merits or on any characteristics or elements peculiar to a particular claim or claimant.

The committee's interview with Dr. Thomas E. Dobson, Chief Medical Officer of the Workmen's Compensation Board, confirmed that he does not personally recommend rejection of any claim by the Board until the X-rays have been seen by two chest specialists, usually Dr. C. A. Gordon and Dr. John Dill, who have reported unanimously that the claim should be rejected.

Doctors Gordon and Dill agree that they rely upon the chest X-ray examination as the indicator of permanent alteration of lung structure and they rely upon the pulmonary function tests as the objective measurements of the degree of disability.

The Chief Medical Officer studies the two reports and if they are in agreement that compensation should be paid at a certain level, he reports this to the Board, which decides the amount of compensation. The workman and the family physician receive notification that a diagnosis of Pneumonoconiosis has been made and the workman is told the level of his pension.

Should both consultants agree that Pneumonoconiosis is not present, the recommendation to the Board would be that the workman should not receive compensation.

In the event that the chest consultants consider that Pneumonoconiosis is present, but is not causing significant disability, the committee was told that at least one of the consultants would recommend compensation at the 15% level. If the two consultants agree that compensation should be paid, but recommend different levels, the Board would accept the recommendation at the higher of the levels recommended.

The consultants sometimes recommend review after a period of years. Although the committee heard evidence of dissatisfaction with the long periods recommended to elapse before further chest X-rays were arranged by the Board, there is strong evidence that coal workers' Pneumonoconiosis is a very slowly progressive condition especially in early phases. Claimants and their advocates should not assume that the recommendation of further chest X-rays after a period is an indication that the presence of disease has been established. "Many cases reported result in findings of little or no evidence of Pneumonoconiosis. However, claims once made are subject to review as long as the claimant remains in employment." (Report of the Workmen's Compensation Board of Nova Scotia 1976). The committee finds current procedures fair to claimants.

In the event of a conflict of opinion between the two consulting respirologists, it has been the practice of the Board to obtain a third opinion, usually in recent years from Dr. McVittie. The purpose of the third opinion, was originally to establish a majority decision and this practice had been recommended by a committee on Pneumonoconiosis, reporting to the Minister of Labour in 1969. The policy of obtaining a third opinion has been changed pursuant to decisions of the Workmen's Compensation Board primarily on the basis that the workman should receive the benefit of doubt existing concerning his diagnosis or disability.

In fact the workman is being given the benefit of the doubt at every level, even at levels where it is questionable whether it is appropriate. If a radiologist sees nodulation in the chest X-ray and is not quite sure whether to grade it # 1 or # 2 he will err on the side of calling it the more advanced condition. Each chest specialist reports that he does the same thing. The medical specialists recommend compensation of grade # 1 coal workers' Pneumonoconiosis cases, despite prevailing medical opinion that grade # 1 is not disabling. The Workmen's Compensation Board gives the workman the benefit of the doubt if there is a conflict between the opinions of the specialists both as to diagnosis and as to the amount of compensation.

APPEALS PROCEDURE

The Workmen's Compensation Appeal Board:

1. Examines the file of the Workmen's Compensation Board including the reports of the experts upon which the decision appealed from was based.

2. Conducts hearings at which the claimant may present his case with the assistance of a Workmen's Counsellor.

3. Refers the claimant for further medical examination if it is deemed necessary.

The proceeding is not an adversary one in that there is no one opposing the appeal, and there is no cross-examination of the appellant. While the Board has power to subpeona witnesses, it has never done so. It is most unusual for physicians to give their evidence in person. The Workmen's Compensation Appeal Board is a lay-tribunal, including no medical professionals on the Board or on staff. The Board is obligated to hear every appeal unless a time limit has been exceeded. It has authority to extend the time period for filing of an appeal and would tend to do so. With the launching of an appeal all relevant data are obtained from the Workmen's Compensation Board, reviewed and studied by Board members, before and during the hearing.

Most appeals are against rejection by the Workmen's Compensation Board or seeking an increase in permanent partial disability. Appellants are advised to be represented by counsel which is provided without charge.

The Act provides that notice of hearing must be served on "all interested parties", but in practice notices are served only on the Workmen's Compensation Board, the appellant, and his representative. The Supreme Court has held that an employer is not entitled to be a party to an appeal. Cross examination of witnesses is allowed, but there is no person present to do so. Witnesses are, however, questioned by the Board. Evidence is given under oath but the Board exercises wide latitude in accepting hearsay evidence.

Hearings are held and are open to the public. Only the appellant, however, is entitled to make representations to the Board.

Decisions in favour of the Appellant are based primarily on finding evidence of medical doubt in specialists' reports. A statement that one of the consultants suspects that Pneumonoconiosis may be present and recommends review in two years leads to referral to a further consultant for an opinion.

The Appeal Board feels that the presence of physicians on their Board would influence their interpretation of the medical evidence and this would not be desirable.

BENEFIT OF DOUBT

There is much misunderstanding of the meaning and effect of s. 20 of the Act. That section provides that the Board(s) shall be entitled to draw from all circumstances of the case, the evidence and medical opinion, all reasonable inferences in favour of the applicant. We interpret that this is to mean that where there is medical doubt as to whether disability was caused by industrial conditions or incidents or by some other cause, then this doubt may be resolved in favour of the applicant. We believe it does not mean that where there is a mere

possibility or suspicion the question will be decided in favour of the applicant.

The Boards have responsibility to consider the reliability of medical opinions and the qualifications of all witnesses to interpret medical evidence.

The weighing of medical evidence and the assessment of its credibility becomes the specialty of Workmen's Compensation Board staff physicians. Some consultants have been able to cultivate a scientific attitude of neutrality which allows them to give a sound and balanced opinion to third parties. Staff physicians assist the Workmen's Compensation Baord in weighing all the evidence available.

The Workmen's Compensation Appeal Board, however, feels no need to have similar medical evaluation of the scientific language of medicine employed in physicians' reports. Consultants' reports are studded with medical terminology making inferences obscure to persons without medical training.

Receiving and considering evidence is a specialized and difficult function. It is particularly difficult when the evidence received is expert evidence on a complex medical subject. Where the expert evidence is on the very point that the Board must decide, the Board must be particularly careful in assessing the evidence. This is impossible to do where there are merely written reports. In difficult cases, the expert witness should also be called before the Board.

These principles apply even more stringently to the Workmen's Compensation Appeal Board, since it deals exclusively with controversial cases.

In accordance with s. 20 of the Act the members of the Appeal Board feel very strongly that the workman must be given the benefit of any doubt. Sometimes the consultants' reports may be misinterpreted as expressing a doubt. If there is any clear evidence of Pneumonoconiosis, the medical specialist will state that it is present. With lesser findings, he may suspect that Pneumonoconiosis is present, but not recommend compensation. Occasionally the Appeal Board has accepted that statement of suspicion as constituting a doubt to be resolved in favour of the Appellant. It is our belief that an explanation by the consultant would place the statement in perspective. We recommend **that in Pneumonoconiosis cases, the medical consultants appear before the Appeal Board to explain their reports.**

BOARD PROCEDURE — GENERAL

Since Pneumonoconiosis was first recognized as a compensable disease the legislation has been amended several times. Board procedures to evaluate claims have changed over the years in the light of the Board's experience and in response to alterations of the Act. At present they are as described above.

After a review of the medical literature and discussions with many competent specialists, the committee is convinced that the present medical procedures of the Workmen's Compensation Board in evaluation of Pneumonoconiosis claims are sound and fair to workmen.

However, while the procedures are not unjust, they are not altogther satisfactory. Justice must not only be done, it must be seen to be done. We believe that the present medical and adjudicative processes of the Board do justice in the administration of the Act. Justice has not, however, been seen to be done in the eyes of many members of the public. The Board is perceived by many as a remote and impersonal institution, and in an adversary position to workmen.

We **recommend that the Board be given a human face in its relationships with claimants.** This might be done by assigning each claim to a named claims officer who would have personal contact with the claimant to explain the procedures, requirements, and decisions of the Board. In addition to gathering information about the claim, the officer would receive questions from the claimant and attempt to deal with them. These officers should be located in locations convenient to claimants, and particularly in the industrial Cape Breton area. We feel that such personal contacts would do much to assist claimants and to eliminate the feeling of frustration which frequently develops in dealings with the Workmen's Compensation Board.

Another reason that justice is not seen to be done by the Board is that its decisions are based entirely upon evidence gathered by it, albeit from independent consultants of high standing. There is no opportunity for the claimant to make representations on his own claim except in completing Form 6S, the application form. His only opportunity to press his claim is by appeal to the Workmen's Compensation Appeal Board.

We feel it is desirable to resolve claims at the Board level to the satisfaction of the claimant, if possible. We feel that this will be accomplished if claimants have an opportunity to present their claim at the Board level. We recommend **that claimants be advised of the recommendation of the Board's consultants, either against compensation or in favour of compensation at a specified level. If this recommendation is not satisfactory to the claimant, he should be given an opportunity to submit further evidence to the Board.** The Board should receive and consider all such relevant evidence. We believe that dissatisfaction might frequently be resolved in meetings between the claimant and the Board.

BOARD RELATIONSHIPS WITH THE PUBLIC

While in the past the image of the Board in the view of the miners was a negative one, we noted a feeling among miners' representatives and union leaders that things have improved recently and are likely to improve further. The Board is to be commended on this progress and the process is to be encouraged.

The purpose of the Board is to serve the needs of disabled workmen by prompt and fair administration of the compensation program provided for by the Act. The Board must be eminently fair, completely impartial, immune to pressure groups, and the essence of justice in all respects. At the same time, the Board has to exude a spirit of understanding, sympathy and helpfulness, and be so disposed as to give every claimant a full and complete hearing. Its relationship with the miners and with all concerned, including the public, is a matter of paramount importance. Fundamental in this respect are openness and good communication with the public and all interested parties. Knowledge and complete explanation of what the Board is, its structure, function, and purpose, as well as what it is not, should constitute the core of a public information program by the Board. Thus misunderstandings would be corrected and public confidence reinforced.

Despite the fact that the Board has to make difficult and sometimes unpleasant decisions, it needs to overcome the unfortunate image of always being in the role of the adversary. As noted above, we feel this might, to some extent, be accomplished by having claims assigned to identified officers to deal personally with each claimant's case, and thus to provide personal recognition and concern for the individual.

The quality of the service by the Workmen's Compensation Board and Workmen's Compensation Appeal Board to the people of Nova Scotia is dependent on the membership of those Boards. The Minister must continue the practice of appointing members of high integrity and ability to the Boards.

RELATIONSHIPS WITH THE MEDICAL PROFESSION

The family physician has primary responsibility for the medical care of disabled workmen and a high level of co-operation must exist between the Board and the physician. To improve the quality of medical care we feel that it is essential that all medical consultants' reports and test results be made available to those having primary responsibility for the claimant's care. At present it is the practice of the Board to provide only a brief report as to whether or not a diagnosis of Pneumonoconiosis was made. We recommend THAT ALL CONSULTANTS' REPORTS AND TESTS RESULTS OBTAINED BY THE BOARD RELATING TO PNEUMONOCONIOSIS CLAIMS BE PROVIDED TO THE CLAIMANT'S DESIGNATED PERSONAL PHYSICIANS.

Family physicians of claimants can contribute to proper settlement of Pneumonoconiosis claims. The family physician's report filed in support of a Pneumonoconiosis claim should be based on a thorough physical examination and be in sufficient detail to be of value in evaluation of the claim. To ensure that this is done

conscientiously, the family physician should be adequately compensated.

There is a feeling among family physicians that their reports to the board are ignored. Physicians, on the other hand, must appreciate the role and function of the Board, and that compensation is awarded strictly in accordance with the Act and not based on social need. Occasionally, unreasonable expectations are given to miners by the ill-founded assurances by family physicians that a miner's lungs are "full of rock dust" and that he therefore has a compensable disease.

We recommend THAT A PROGRAM BE UNDERTAKEN IN CONJUNCTION WITH DALHOUSIE MEDICAL SCHOOL AND/OR THE DIVISION OF CONTINUING MEDICAL EDUCATION TO IMPROVE THE UNDERSTANDING OF PNEUMONOCONIOSIS AMONG PHYSICIANS IN NOVA SCOTIA.

We recommend that THE BOARD EMBARK ON A PROGRAM OF CONTINUING COMMUNICATION WITH THE MEDICAL SOCIETY OF NOVA SCOTIA CONCERNING WAYS AND MEANS OF IMPROVING COMMUNICATIONS BETWEEN THE BOARD AND PHYSICIANS AND OF FOSTERING MUTUAL RESPECT.

PROVISION OF REPORTS

If a miner whose application for Pneumonoconiosis benefits has been denied is dissatisfied with the decision of the Board he must at present launch an appeal to the Workmen's Compensation Appeal Board simply to gain access to the material upon which the Board based its decision. The result may be acceptance or denial of the appeal, but once the appeal is commenced it is almost invariably carried through. If the claimant had access to the medical reports, it is probable that some of the appeals would be unnecessary.

The committee believes that it is a matter of basic right that each claimant should be entitled to know on what material the decision involving his rights is based. We recommend **that claimants be entitled upon request to receive copies of reports of consultants to the Workmen's Compensation Board, and that the consultants be so advised.**

MEDICAL INFORMATION AND RESEARCH

Coal workers' Pneumonoconiosis has been a sizable health problem in Nova Scotia. Although some study and discussion among lung specialists has taken place locally, (e.g. in October 1966 a conference on Silicosis and Pneumonoconiosis was convenied in Sydney, Nova Scotia to consider the problems associated with these industrial diseases in the coal mines of Nova Scotia) the committee perceives need, not only for closer communication among persons concerned with diagnosis and treatment of miners suspected of having coal worker's Pneumonoconiosis, but also for systematic studies of correlations between autopsy findings in deceased miners, clinical, roentgenologic and laboratory test results. Indeed the need for improved communications, among physicians, systematic collection of data, regular performance of autopsies, and for correlative studies have been emphasized by most of the individuals and groups who have made representations to the Committee.

Recently Dr. David Janigan, Professor, Department of Pathology, Dalhousie University, has returned from a period of study of Pneumonoconiosis in Great Britain. He has advised us that he intends to set up a division of Pulmonary Pathology. The work of this division could be greatly enhanced by a uniform policy of performance of autopsies on coal miners, with suitable preparation of lungs for careful assessment.

We recommend **that the Workmen's Compensation Board promote the establishment of periodic conferences among physicians involved in the diagnosis and treatment of lung disease and the continual systematic collection of clinical and autopsy data from coal miners.**

We recommend **that consideration be given to possible methods for a miner and his relatives to record a decision before the death of the miner concerning the performance of post-mortem examination of his body.** Legislation may be required to recognize the validity of such autopsy permission. While it is beyond the terms of reference of this committee we would like to observe that such legislation would presumably not be restricted to coal miners but rather would affect the public generally. If legislation can be designed that does not interfere with the rights and privileges of citizens it would be of considerable benefit to the public by virtue of its effect in increasing the understanding of disease process.

DIAGNOSES AND TREATMENT

Workmen see the necessity of travelling to Halifax for examination as an imposition, notwithstanding that expenses are paid. While it is not strictly within our term of reference, we repeat recommendations of other committees and commissions **that pulmonary care facilities in Cape Breton be improved.**

This might be done by:

In the past two decades several well trained internal medicine specialists have settled in Sydney. It happens that their specialization has been in the fields of rheumatology, cardiology, gastrointerology and general internal medicine. The specialty of diagnosis and treatment of lung disorders (respirology, pneumonology) probably will commence to receive attention in Cape Breton when appropriate diagnostic and treatment facilities are made available in the local hospitals. This implies the placement of complex instrumentation in a designated hospital to be attended by highly qualified technicians. Such technicians, if not in contact with the challenges associated with a university atmosphere would be prone to become somewhat disinterested. It would therefore be desirable that a Cape Breton unit be closely associated with the university medicine environment in Halifax. This could be arranged. It might happen that one of the well qualified internists in Cape Breton could be encouraged to study towards special competence in respirology. Alternatively, a lung specialist would be encouraged to establish himself in Cape Breton. Such a physician should practice independently of the Board and of the employers, but his opinion ought to be available to both on the same basis that it would be available to a private referring physician.

The complexity of the pneumonoconioses demands that the medical evaluation of patients be very complete and include X-ray examinations and pulmonary function tests. Pulmonary function test instrumentation has become very complicated. The machines measure air flow to the functional units, the amount of air that does not ordinarily move out of the lungs on expiration, and various other things, the most important of which, from the standpoint of Pneumonoconiosis, is the ability of the lungs to allow diffusion of oxygen into the blood stream. Of the various meansurements made by pulmonary function testing machines, this is the most difficult and the operation of such machines must be extremely well understood by the technician operating them.

There is a need for the chest physician to have some understanding of the processes going on in the machines. In addition, it is generally necessary to have the technicians involved in pulmonary function testing overseen by someone who has an excellent understanding of lung physiology.

The situation in the university teaching hospitals of Halifax exemplifies this. Dr. C. A. Gordon, the most senior of our respirologists, learned pulmonary physiology in New York and was responsible for training of the first pulmonary function technicians in this area.

There are now pulmonary function laboratories at Camp Hill Hospital (under the guidance of Dr. C. A. Gordon), at the Halifax Infirmary, also under his guidance, and at the Victoria General Hospital, where the professor of bio-physics, Dr. Josenhans, has recently been appointed to oversee the operating of the pulmonary function laboratory.

In the near future, Dr. R. Lemoine, a brilliant physician, who has been studying pulmonary medicine in California, will be returning to Halifax and will be concerned with the operations of the clinical testing pulmonary function instruments at the Victoria General Hospital and also with research into lung disease.

Dr. Lemoine's concern with pulmonary physiology will beautifully complement Dr. Janigan's work in the pathology of lung disease (the one studying the living patient and the other studying tissues removed from patients, living or dead). In addition Dr. John Dill is studying the defence systems which are involved in protection of persons from injurious substances and the disorders that occur when the protective systems fail or go awry.

This sort of concentration of researches and clinical facilities tends to occur at the medical schools of universities. It would be difficult to maintain the standards of excellence achievable in this atmosphere in a pulmonary function laboratory in Cape Breton.

The tremendous opportunity available in Nova Scotia for study of coal miners' Pneumonoconiosis should be exploited.

Dr. M. R. MacDonald, Executive Director of the Victoria General Hospital, was once a Medical Officer of Health employed by the Nova Scotia Department of Health in Cape Breton. Dr. MacDonald has written a letter noting that progress in development of respiratory investigative units has been remarkably slow in the past twenty-five years in spite of the increase in knowledge and better diagnostic aids. Dr. MacDonald is seeking support for a lung investigative unit at the Victoria General Hospital. This would be the setting in which Dr. Lemoine would operate his research laboratory and in which pulmonary function studies would be done. Dr. MacDonald suggests that this should become a provincial centre with satellite units in areas such as Sydney or Glace Bay.

This appears the sensible way to upgrade the level of respiratory medicine in Nova Scotia and to create facilities in Cape Breton which can be of high standard. Dr. MacDonald points out that in addition to the current operating budgets of the Hospital, considerable financial support for such a unit is required. "A start in development has been made through the generosity of the Nova Scotia Lung Association but addtional financial help will be required . . . the potentiality for development of first class quality investigative facilities and curative measures for these unfortunate people with respiratory illnesses is here, all it needs is the financial support of philanthropic agencies and agencies such as the Workmen's Compensation or industries whose personnel are genuinely interested in preventing and combatting occupational health hazards and treating diseases accruing therefrom."

PREVENTION

While it is not within our mandate to report on aspects of the Workmen's Compensation program other than those related to claims procedure we would repeat the recommendations made by previous committees and made forcefully to us by representatives of both employers and workmen **that programs be instituted to prevent Pneumonoconiosis and other lung ailments of miners.**

These could include:

1. Implemention of procedures to reduce dust levels.
2. Establishment of standards for dust levels.
3. Stringent pre-employment physical examination of miners.
4. A program of regular physician examination of miners.
5. Use of comfortable and convenient masks.

Because Devco is a federal employer, there are certain constitutional and jurisdictional problems relating to implementation and enforcement of such programs. These can presumably be solved by federal-provincial consultation and co-operation. Progress already achieved in this respect (under Devco auspices in co-operation with the UMW) is most creditable and to be highly encouraged.

SUMMARY

A committee comprising Dr. John Woodbury as Chairman together with Monsignor Malcolm MacLellan and Dr. Murray Fraser counselled by Mr. Daniel M. Campbell has reviewed and assessed medical and adjudicative procedures relative to Silicosis and Pneumonoconiosis cases that go before the Workmen's Compensation Board.

The committee studied the diseases, medical and legal definitions, their diagnosis and the assessment of disability in those afflicted. It was found that the Workmen's Compensation Board procedures relating to diagnosis and assessment were appropriate and in keeping with current medical understanding of the Pneumonoconioses.

The adjudicative procedures of the Workmen's Compensation Board and the Workmen's Compensation Appeal Board were examined. The committee found that these were entirely fair to the claimants and appellants.

Changes in the adjudicative procedures are recommended, chiefly for better communication between the major participants in the process, claimants, Boards, and physicians.

RECOMMENDATIONS

1. That the definition of Pneumonoconiosis in the Act be amended to add the words "but does not include Bronchitis and Emphysema."
2. That when Bronchitis or Emphysema is associated with identifiable Pneumonoconiosis the workman should be compensated for the amount of disability arising from both respiratory conditions.
3. That in Pneumonoconiosis cases, the medical consultants appear before the Appeal Board to explain their reports.
4. That the Board be given a human face in its relationships with claimants.
5. That claimants be advised of the recommendations of the Board's consultants, either against compensation or in favour of compensation at a specified level. If this recommendation is not satisfactory to the claimant, he should be given an opportunity to submit further evidence to the Board.
6. That all consultants' reports and test results obtained by the Board, relating to Pneumonoconiosis claims, be provided to the claimant's designated personal physician.
7. That a program be undertaken in conjunction with Dalhousie Medical School and/or the Division of Continuing Medical Education to improve the understanding of Pneumonoconiosis among physicians in Nova Scotia.
8. That the Board embark on a program of continuing communication with the Medical Society of Nova Scotia concerning ways and means of improving communication between the Workmen's Compensation Board and physicians and of fostering mutual respect.
9. That claimants be entitled upon request to receive copies of reports of consultants to the Workmen's Compensation Board, and that consultants be so advised.
10. That the Workmen's Compensation Board promote the establishment of periodic conferences among physicians involved in the diagnosis and treatment of lung disease and the continual systematic collection of clinical and autopsy data from coal miners.
11. That consideration be given to possible methods for a miner and his relatives to record a decision before the death of the miner concerning the performance of post-mortem examination of his body. (Legislation may be required to recognize the validity of such autopsy permission.)
12. That pulmonary care facilities in Cape Breton be improved.
13. That programs be instituted to prevent Pneumonoconiosis and other lung ailments of miners.

APPENDIX I.

DEFINITIONS

Butterworth's Medical Dictionary by MacNalty, London, 1965.

"Pneumonoconiosis: Disease of the lungs, bronchi and pleura, caused by inhalation of small metallic or mineral particles. The chief symptoms, chronic induration or fibrosis, are usually preceded by bronchial inflammation or cough, after which nodules like those occurring in miliary tuberculosis appear."

"mineral: Essentially any inorganic sustance found in the earth and obtained by mining: extended to include purely organic sustances like coal and petroleum."

Dorland's Medical Dictionary, 25th ed. 1974 W. B. Saunders Co.

"Pneumonoconiosis: A condition characterized by permanent deposition of substantial amounts of particulate matter in the lungs, usually of occupational or environmental origin, and by the tissue reaction to its presence."

Stedmans Medical Dictionary by Williams & Wilkins, Baltimore, 1976.

"Pneumonoconiosis of coal workers: a form caused by deposition of large amounts of coal dust in the lungs, and typically characterized by centrilobular emphysema; called also coal miner's or miner's lung, black phthsis and miner's phthisis c.f. anthracosis.

"Anthracosis: A usually asymptomatic form of pneumonoconiosis caused by deposition of coal dust in the lungs; it is present in most urban dwellers. When the coal dust accumulates in large amounts it may result in pneumonoconiosis of coal workers."

Black's Medical Dictionary, London, A & C Black, 1976.

"Pneumonoconiosis is the general name applied to a chronic form of inflammation of the lungs which is liable to affect workmen who constantly inhale irritating particles at work. It has been defined by the Industrial Injuries Advisory Council as: 'Permanent alteration of lung structure due to the inhalation of mineral dust and the tissue reactions of the lung to its presence but does not include bronchitis and emphysema . . .' "

"Pneumonoconiosis: Inflammation commonly leading to fibrosis of the lungs, due to the irritation caused by the inhalation of dust incident to various occupations, such as coal mining, knife grinding, stone cutting, etc.; the most prominent symptoms are: pain in the chest, cough, little or no expectoration, dyspnea, reduced thoracic excursion, sometimes cyanosis, and fatigue after slight exertion."

Occupational Lung Disease, Morgan and Seaton, W. B. Saunders, Phila. 1975. p. 161.

"Coal workers pneumonoconiosis is best defined as the accumulation of coal dust in the lungs and the tissue's reactions to its presence. Necessary for establishing a diagnosis of CWP are a history of exposure to coal dust — usually at least 10 years underground and certain relatively characteristic abnormalities on the chest radiograph. CWP is separated into simple and complicated forms according to the appearances of the chest film. The pathological basis for the distinction between the two forms of CWP was laid by the pioneer investigations carried out by the Cardiff school."

"It is often suggested that coal dust, or at least the carbon in coal dust, cannot account for the opacities present in the lung. Some persons feel that the radiographic opacities are mainly a consequence of the collagen and fibrous protein content of the lungs, and indeed there may be an element of truth in this claim. Nevertheless, the one outstanding fact that emerges from these studies is the excellent relationship between the coal dust content of the lungs and its radiographic category. The chest film, therefore, despite drawbacks in its interpretation remains the only feasible method of determining and quantifying dust exposure in life."

Gould Medical Dictionary, Blakistons, McGraw Hill, 1972.

"Any disease of the lung caused by inhalation of dust, especially mineral dusts that produce chronic induration and fibrosis."

APPENDIX 2.

List of Knowledgeable Persons Interviewed

Dr. John Cooper
Professor of Pathology, Dalhousie University.

Dr. John Dill
Assistant Professor of Medicine, Dalhousie University, Consultant in Respirology to WCB.

Dr. Charles Gordon
Assistant Professor of Medicine, Dalhousie University, Consultant in Respirology to WCB.

Dr. David Janigan
Professor and Head of Department of Pathology, Dalhousie University.

Dr. Paul Landrigan
Professor of Medicine Dalhousie University, Program Director for Respirology in Department of Medicine.

Dr. Stewart Manchester
Professor of Diagnostic Radiology, Dalhousie University.

Dr. Albro McKeen
Professor of Diagnostic Radiology, Dalhousie University.

Dr. John Guyett Scadding
Emeritus Professor of Medicine, University of London (England), Past President Thoracic Society, Visiting Professor Dalhousie University 1977.

Workmen's Compensation Appeal Board:

Mr. Hugh MacLeod, Chairman
Mr. John J. O'Brien, Executive Officer
Mr. Daniel Almon, Member

Workmen's Compensation Board:

Mr. John Lynk, Chairman
Mr. S. E. Watt, Executive Director
Mr. Willard MacKenzie, Member
Dr. Thomas Dobson, Chief Medical Officer

Dr. J. C. McVittie
Member Pneumonoconiosis Panel of Great Britain, consultant in Respirology to Workmen's Compensation Board.

APPENDIX 3.

List of Important Documents Found Helpful

Black's Medical Dictionary,
London, A & C Black, 1976.

Butterworths Medical Dictionary,
MacNatty, London, 1965.

Dorlands Medical Dictionary,
25th Ed., 1974 W.B. Saunders Co.

Gould Medical Dictionary,
Blakistons, McGraw, Hill, 1972.

Stedmans Medical Dictionary,
Williams & Wilkins, Baltimore, 1976.

Occupational Lung Disease,
Morgan & Seaton, W. B. Saunders, Phila., 1975.

Report of the Royal Commission on the Health & Safety of Workers in Mines, Ministry of the Attorney General, Province of Ontario, 1976.

Notes by Dr. C. R. May of Preventive Medicine Dept. Dalhousie University on Occupational Lung Disease, 1976.

Report of the Task Force on the Administration of Workmen's Compensation in Ontario, 1973.

Report of the Royal Commission in the Matter of the Workmen's Compensation Act, 1967, McGillivray, J.

Report of International Conference on:

Coal Workers Pneumonconiosis,
Annals of the New York Academy of Sciences, Vol. 200, 29 December, 1972.

Coal Miners Pneumoconiosis,
Bouhuys & Gee in Harrison's Principles of Internal Medicine, 8th Ed., 1977.

Communications to this Committee from Dr. T. McVittie.

Briefs Were Presented By: **APPENDIX 4.**

1. "The Committee for Automatic Assumption of Black Lung", J. Akerman — Chairman.
2. Devco Officials:
   Mr. Bruce McDade, Vice President and Secretary
   Dr. A. Prossin, Medical Chief
   Mr. Sanderson, Vice President of Finance
   Mr. Ray Graham, Mgr. Employee Benefits
3. District 26, UMW representatives — Wm. Marsh — Chairman Workmen's Compensation Appeal Board — Hugh MacLeod — Chairman
4. Dr. J. B. Thompkins — Representing the Glace Bay Medical Society

In addition, correspondence was received from:
Dr. D. Nathanson Dr. K. Cadegan □

Congenital Rubella

The risk of the foetus being infected and suffering serious damage after maternal primary infection varies with gestational age. When infection occurs during the first 8 weeks of pregnancy 50-54% of foetuses are infected, and 85% of those will be found to have defects. If the infection occurs from 9-12 weeks gestation, 34% of foetuses are infected and about half of those will be found to have defects, either at birth, or after long term follow-up. The rate of foetal infection declines to 10% when rubella occurs at 13-24 weeks gestation, and the percentage of infected infants with defects declines from 16% to 0% at 24 weeks gestation.

If maternal infection occurs very early after conception spontaneous abortion is possible.

Pathogenesis

In postnatal infection the portal of entry is the upper respiratory tract. Local viral replication occurs, but is below the limit of detection. From 9-11 days after exposure viremia occurs; viruses are excreted, and can be detected from the nasopharynx, urine, cervix and faeces. At least one week elapses before the onset of clinical illness. Antibodies are usually detectable when the infection becomes overt, and this corresponds with the cessation of the viremia. The antibody response peaks from one week to a month after the onset of fever, depending on the type of antibody measured. The duration of this response varies from a few years to life depending on the antibody being considered. Although rare, reinfection is possible, but it is thought that in such instances no viremia occurs.

Congenital Infection

This occurs during the maternal viremic stage of the disease, before maternal illness is clinically apparent. The placenta is first infected and then, in most instances, the virus invades the foetus. The virus can be recovered from the foetal pharynx, urine, conjunctivae, faeces, CSF, bone marrow and circulating leucocytes. The striking difference in foetal involvement at early gestational age is not fully understood. Maturation of the placenta, maturation of foetal defense mechanisms and viral preference for very immature cells to establish growth are factors to be considered.

Pathology

Examination of infected tissues reveals that vascular changes predominate. Cytolysis occurs less often, and inflammatory changes are minimal. *In vitro* cytology shows increased chromosomal breaks, reduced cellular multiplication and progressive mitotic arrest.

Clinical Manifestations of Congenital Rubella

It is a chronic infection with a large range of possible pathologic injuries, not all of which are apparent at birth. The major findings include: hepatosplenomegaly, hepatitis, congenital heart disease (pulmonary arterial hypoplasia, patent ductus arteriosus, etc.), retinopathy, cataracts, microphthalmia, glaucoma, purpura with thrombocytopenia, dermal erythropoiesis, adenopathy, interstitial pneumonia, myocardial necrosis, encephalitis, microcephaly, loss of hearing, gastrointestinal anomalies, bone lesions, immunologic abnormalities and other rare manifestations. Infants who survive the neonatal period show evidence of hearing loss, psychomotor deficits, dental abnormalities and possibly increased incidence of diabetes mellitus.

Diagnosis

Clinical diagnosis of congenital rubella can be made when the infant has a typical presentation with corroborating maternal history. Laboratory confirmation including viral isolation and serology is extremely useful. Increased total IgM ($\geq$ 19.9%) in cord blood is a high risk monitor, but must be confirmed with rubella-specific IgM determination. The most difficult problem is to determine probable foetal infection, when a woman is exposed to rubella during the first trimester of pregnancy. Serology plays a major role and the following serological determinations are available:

1) **Hemagglutination inhibition antibodies (HI)** are detectable when the disease becomes overt, peak from 1-3 weeks after the rash, and persist for many years, possibly for life.
2) **Neutralizing antibodies** behave in much the same way as the HI antibodies.
3) **Fluorescent antibodies** behave like HI antibodies, but do not persist as long.
4) **Complement fixing antibodies** usually appear later, 10-14 days after onset of the rash, peak after 1-2 months, and usually disappear after a few years.

Other serological tests have been described but are not readily available.

Any pregnant woman exposed to rubella should have HI titres determined. If the exposure has occured within 7 days of tests in the home (family contact) or within 10 days outside the home and HI antibodies are detected, immunity is present and the foetus is not at risk. If no antibodies are detectable, the patient is susceptible and a second specimen should be secured 3 to 4 weeks later; if HI antibodies are detected then infection has occurred. If blood for serology is taken at a later date, HI as well as rubella specific IgM should be determined. The presence of rubella specific IgM indicates recent infection.

Therapy

There is no specific treatment available.

Prognosis

Of severely affected infants 10-35% die, and most of those who survive have severe CNS problems, ocular impairment and progressive hearing loss.

Prevention by Active Immunization

An effective, live, attenuated vaccine is available. Evaluation of a 7 year rubella immunization programme in the USA has been published recently.[3] Seventy million doses of vaccine were distributed; following immunization the incidence of rubella decreased sharply and the number of reported cases of congenital rubella declined progressively. Side effects, though infrequent, included arthralgia, occasionally fever, adenopathy and muscular pains; these have been reduced with the introduction of improved vaccine. The high level of immunity achieved has not declined in the 5-7 year follow-up. A study by Modlin *et al*[4], of women inadvertently immunized during early pregnancy, revealed

that the risk of the occurrence of a congenital malformation is much less following vaccination than natural infection. In spite of these findings, rubella vaccination during pregnancy is not indicated. The vaccine virus is *not* communicable to other persons. If reinfection occurs after vaccination, it is asymptomatic and *not* accompanied by viremia.

It is desirable to establish a programme that offers rubella vaccine in public health clinics to all children aged 1-10 years and to all seronegative female hospital staff. Women should be screened premaritally and those found susceptible offered vaccination, provided they are not pregnant.

CYTOMEGALOVIRUS (CMV)

Cytomegaloviruses are unquestionably the most common cause of viral congenital infection in man. Most congenitally infected infants are not detected since they are asymptomatic at birth; however long term follow-up has shown that abnormalities may become evident later in infancy and in childhood.[5]

The Organism

CMV belongs to the herpesvirus family. It measures 110 nm, has a DNA core and an envelope. Although *in vivo* it infects principally epithelial cells, *in vitro* it can be grown only in fibroblastic cell culture.

Transmission

Transmission may occur during pregnancy by the transplacental route. Initial studies demonstrated the importance of primary infection and viremia, but subsequent studies have shown that intrauterine infection may occur with persistent maternal infection.[6 7 8] Intrapartum infection is possible during the passage of the foetus through the birth canal in women with cervical CMV but this is usually *not* associated with disease, and the infant who may acquire viruria one month later is asymptomatic otherwise. CMV may be present in breast milk and faeces.

In post natal life CMV is transmitted only by intimate contact in adults, frequently by sexual contact and by fresh blood transfusion. Although, theoretically, babies with congenital CMV may be infectious, there is little evidence that CMV infection is readily disseminated to other patients or personnel in a hospital setting; nevertheless, precautions in the nursery are advisable.

Epidemiology

The prevalence in the general population varies with age and socioeconomic and geographical circumstances. Serological studies have shown the presence of antibodies to CMV in 37% of adults in Rochester, N.Y.; 34% in Nova Scotia, and greater than 75% in Easter Island, Barbados, Tanzania and Birmingham, Alabama. Several studies show that about 60% of women entering child bearing age have antibodies to CMV; in Halifax the proportion is 37%

Incidence of primary infection in a healthy population, as evidenced by seroconversion, is between 0.9 and 4.1%. Similar studies in pregnant women have shown an incidence of 0.3-4.1%. However 3-6% of pregnant women excrete the virus in urine, and 4-28% of pregnant women have CMV virus in cervical secretions. The prevalence of positive cervical secretions is lowest early in pregnancy (1.3%) and rises to 13.4-28% at the third trimester. Intrauterine infection occurs in 0.5-3.4% of all livebirths. A survey of 542 newborns at the Grace Maternity Hospital, Halifax, showed a 0.55% incidence of infection.

CNS damage has been reported in 5-15% of congenitally infected neonates. The intrapartum and natal infection rate is estimated to be as high as 8% but this mode of infection is not considered harmful to the neonate. A recent study[8] showed that the frequency of intrauterine transmission (3.4%) with recurrent maternal cytomegalovirus infection is essentially the same as that in the general population (2.4% in the same study population); thus the value of seropositivity as protection against congenital CMV seems dubious. An infected infant may have viruria for many months.

Pathogenesis

Maternal viremia is more likely with primary than recurrent infections (though recent studies are in disagreement);[6 7 8] with the latter, virus titre may be low. Transplancental passage is possible and the earlier in pregnancy the more likely it is to affect the foetus seriously and cause CNS damage. The virus replicates in the subependymal tissue, mostly with periventricular damage and calcification. There is no evidence that CMV infection causes an excessive number of abortions.

Pathology

In the case of acute fulminant infection death may occur in the neonatal period. Kidney, liver, lungs and CNS are most frequently affected. In the kidney no gross pathology is evident, but desquamation of infected epithelial cells of the proximal tubules may be seen. The liver shows hematopoiesis and general features of neonatal hepatitis. The lungs, as in adult infection, have the features of interstitial pneumonitis with infiltration of lymphocytes and plasmacytes of the alveolar septa. In the brain ependymal and subependymal involvement predominate.

Clinical Manifestations

Only 5% of infected newborns have the "classical" congenital CMV manifestations which include hepatosplenomegaly with hepatitis and jaundice, thrombocytopenia with petechiae and purpura, microcephaly, periventricular calcifications and progressive mental retardation, chorioretinitis, strabismus, optic atrophy and deafness. Pneumonitis is rare in the noenatal period but may become apparent at age 3-5 months. Late clinical manifestations may be subtle and include neurological and psychomotor abnormalities.

Diagnosis

Isolation of CMV in fresh urine is the most reliable indication of CMV infection. Complement fixing antibodies reflect mostly antibodies of IgG type, and may be transplacentally transmitted. Persistence of CF titre 4 to 6 months after birth indicates possible congenital infection. CMV CF titres tend to fluctuate spontaneously.

Indirect fluorescent antibodies test may be used to detect antibodies of the IgG, IgM or IgA classes. The CMV-IgM test gives 95% accuracy in symptomatic newborns, but may be negative in asymptomatic newborns with viruria. Titres may persist for weeks or months and occasionally seem to fluctuate.

The detection of positive inclusion cells in the urine is rewarding in fewer than 50% of cases and confusion with inclusion cells due to herpes simplex and other viruses is possible.

Prognosis

In severe cytomegalovirus inclusion disease (CID) with CNS symptoms, death is likely within one year. Less severe involvement is associated with high (up to 75%) incidence of neurological symptoms. The prognosis for a completely normal course in the asymptomatic newborn is uncertain; more long term follow-up is needed.

Therapy

None is available.

Prevention

Avoid fresh blood transfusion during pregnancy. Therapeutic abortion may be considered in the first trimester of pregnancy in the presence of maternal mononucleosis-like illness, with negative heterophile antibodies, CMV viruria and seroconversion. No vaccine is available, and the usefulness of developing one is controversial. Although the need has not been proven, newborns suspected of having congenital CMV should be isolated, and should not be handled by seronegative or pregnant females.

HERPES SIMPLEX VIRUS (HSV)

The Organism

HSV belongs to the herpesvirus family. It consists of a core containing double stranded DNA, capsid, tegument, and envelope. Two types are described: HSV 1 and HSV 2; they differ in biological and antigenic properties.

Transmission

This is most common during the intrapartum period, either by ascending infection from the cervix or, more commonly, during the passage of the neonate through the birth canal. Transplacental infection is very uncommon. Apparently Herpes simplex virus is not excreted by breast milk.

Epidemiology

70-80% of neonatal herpetic infections are due to HSV type 2. At delivery, 0.05-0.5% of pregnant women have HSV in the cervix or other sites. Forty percent of their infants will become infected, and over 85% of those infected will be symptomatic. There is no conclusive evidence that HSV is associated with abortions.

Pathogenesis

Most neonatal infections occur when the infant passes through the birth canal. The portals of entry are the respiratory tract, eye and skin, and haematogenous spread may follow with the seeding of many organs including the liver, adrenals, gastrointestinal tract and CNS. Newborns are much more susceptible to the disseminated form of HSV possibly because of immunologic defects. The roles of maternal and foetal antibodies and cellular immunity are not understood and the protective mechanism of antibody mediated cellular immunity has only recently been investigated.

Pathology

Visceral organs are seeded with numerous small areas of haemorrhagic necrosis. There is acute disruption of the normal architecture, eosinophilic intranuclear inclusions and disappearance of the nucleolus. Multinucleated giant cells may be present, and moderate inflammatory reaction consisting of mononuclear cells is present. Cell destruction and fibrosis are found in chronic lesions.

Clinical Manifestations

Although considerable overlapping exists, it is useful to distinguish 3 clinical groups: 1) disseminated, 2) localized, 3) asymptomatic. In disseminated form, jaundice, hepatomegaly, purpura, a tendency to bleed and convulsions are accompanied by constitutional signs such as anorexia, vomiting, lethargy and respiratory distress. Vesicular lesions are seen less often in the disseminated group than in the others. In one third of cases CNS manifestations are present, the most important being meningoencephalitis with increased protein and WBC (lymphocytes) in the CSF examination. Intracranial calcification occurs occasionally. Skin and mucosal lesions are often present. In some instances only ocular involvement exists in the form of keratoconjunctivitis, chorioretinitis and occasionally cataracts. Skin involvement includes vesicles or bullae with early denudation, as well as mucosal lesions.

There are isolated reports of possible transplancental infections early in pregnancy that are associated with microcephaly, intracranial calcifications, microphthalmia and skin lesions.

Diagnosis

If there are no skin lesions diagnosis is difficult. Demonstration of multinucleated giant cells with intranuclear inclusions from vesicular scrapings, allows a presumptive diagnosis. Virus isolation and typing is the preferred laboratory procedure.

Serology

CF (not group specific) is available in most laboratories; a neutralization test and an indirect hemagglutination (IHA) test are available in some. Tests utilizing the antibody mediated cellular cytolysis are experimental only. Specific herpes IgM can be detected by the indirect fluorescence technique. However, this immunoglobulin is usually absent in cord blood and during the first two weeks after birth. Positive CSF serology is a good indication of CNS involvement. Assays for cellular immunity to herpes are not yet available.

Therapy

Several antiviral agents such as iododeoxy uridine (IDU), cytoaine arabinoside (ARA-C) and adenine arabinoside (ARA-A) are being evaluated; they should be considered as investigational for the time being. The value of immune-gammaglobulins is not known. The treatment of congenital herpes simplex remains supportive.

Prevention

Termination of pregnancy is not recommended. The pregnant woman should avoid sexual contact with infected men, especially close to delivery. Some authorities recommend caesarian section before or within 4 hours of membrane rupture in women with genital herpes infection. □

References to follow Part II.

PLEASE NOTE

The New Improved Medical Society Insurance Program on the inside back cover.

Phenylketonuria in Nova Scotia Since 1957

J. T. R. Clarke,*M.D., Ph.D., F.R.C.P.(C),
D. M. d'Entremont,**R.N., B.N. and M. S. DeWolfe,***Ph.D.,
Halifax, N.S.

INTRODUCTION

Phenylketonuria (PKU) is an hereditary disease caused by a defect in the metabolism of the essential amino acid, phenylalanine[1]. Unless appropriate treatment is instituted early in life, affected individuals develop characteristically severe mental retardation.

The condition was first described in 1934 by Folling, the Norwegian physician/biochemist. Over the next 20 years, the basic biochemical defect (hyperphenylalaninemia caused by phenylalanine hydroxylase deficiency) and its genetics were worked out, and the first attempts were made to treat the disease by restricting dietary intake of phenylalanine. By the mid 1950s, it was realized that although adherence to a special low-phenylalanine diet could prevent mental retardation in children with PKU, treatment had to be instituted early in infancy to be effective; once established, brain damage due to the metabolic defect is irreversible.

The need for a method to detect PKU in infancy, before any clinical evidence of brain damage is apparent, stimulated the search for an inexpensive but reliable biochemical test suitable for mass screening of infants. The ferric chloride test for the presence of phenylketones in urine was widely used for this purpose. In 1961, with funds from the Department of National Health and Welfare, and sponsored by the Nova Scotia Medical Society, Dr. W. A. Cochrane, then Head of the Department of Pediatrics at Dalhousie University and the Halifax Children's Hospital, initiated a pilot screening program for early detection of various inherited metabolic diseases in children born at the Grace Maternity Hospital and the Halifax Infirmary. The ferric chloride test was one of many biochemical screening tests done on most infants born at these hospitals.

The ferric chloride test is subject to error, for in some cases, it does not become positive until infants affected with PKU are several weeks old and already showing brain damage. The search for a more reliable screening test continued and in 1963, Guthrie described his now famous microbial inhibition assay for direct measurement of phenylalanine in small samples of dried blood[2]. Within the year, the Guthrie test was added to the screening program underway in the two Halifax hospitals and was introduced at 5 additional hospitals throughout the province — again as a research project supported by the Department of National Health and Welfare. At the same time, diagnostic services for the definitive diagnosis of PKU in infants identified with hyperphenylalaninemia were introduced at the Halifax Children's Hospital.

The simple, inexpensive Guthrie test revolutionized mass screening for PKU by making possible reliable detection of the disease within a few days of birth. Subsequently it has become one of the most widely used screening tests in the world. The principle employed in the test has also been adapted in some centres for the detection of other inherited metabolic diseases, such as galactosemia.

From 1963 to 1965, the feasibility of mass screening for PKU in Nova Scotia, using the Guthrie test, was established and, in the spring of 1966, the program graduated from a research project to an established public health routine. The procedure was made available to all infants born in Nova Scotia as a routine screening service through the facilities of regional hospital laboratories, and it was paid for by the Nova Scotia Hospital Insurance Commission. By 1969, it was estimated that over 85% of infants born in the province were being tested within a week of birth.

In 1971, screening for inherited disorders of phenylalanine metabolism was centralized in Halifax in the Department of Biochemistry of the newly opened Izaak Walton Killam Hospital for Children, under the direction of Dr. A. G. Stewart. At the same time, a new, sensitive spectrofluorimetric method for measuring phenylalanine[3] was introduced to replace the Guthrie test. However, the importance of this advance was not widely appreciated, since the blood sampling technique and reporting system were identical to those used in the Guthrie screening procedure. By 1974, an estimated 95% of children born in Nova Scotia were being tested for PKU by the new screening test, which is still used in the provincial screening program.

Over the past several months, we attempted to identify and locate all children with documented hereditary disorders of phenylalanine metabolism born in Nova Scotia since 1957, in order to assess what effect interest in PKU and the scientific and technological advances that made early detection possible, have had on morbidity due to the disease in this province. This paper describes some of our findings.

SURVEY

Searches were made of records kept by Drs. W. A. Cochrane and M. S. DeWolfe, by the Izaak Walton Killam Hospital for Children and by the Atlantic Research Centre for Mental Retardation. A number of medical practitioners assisted us in locating and assessing older patients. Particular attention was directed towards determining the nature of the metabolic defect in each case, the age at diagnosis, the type and duration of treatment and the present

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status of each patient. When possible, parents were interviewed at length; most (17/23) of the patients were examined in detail. Many of the patients underwent detailed psychological assessment by Mr. William Currie of the Izaak Walton Killam Hospital for Children, and some had EEGs recorded. Biochemical studies included measurement of plasma phenylalanine and tyrosine levels and tests for the presence of phenylketones in urine.

RESULTS

A total of 23 children from 20 families from throughout the province (Figure 1) were studied, including one whose family moved here from Ontario when she was an infant. They ranged in age from 2-20 years; 13 were male and 11 female.

The patients were retrospectively divided into 3 groups according to their phenylalanine tolerance (as reflected by serum phenylalanine level on a free diet or the amount of phenylalanine intake required to maintain the level at 5-15 mg/dl), and clinical course[4]. Although more extensive classifications have been reported[5], they generally require more information on the patients' phenylalanine metabolism than was available about our subjects.

The results are summarized in Table I. Of 23 children studied, 15 were considered to have *classical PKU,* although we recognize that this group is almost certainly heterogeneous. At diagnosis, which was as late as 5 years of age in one case, all had plasma phenylalanine levels greater than 28 mg/dl and excreted phenylketones in their urine. All children in this group who were not started on dietary treatment within the first few months of birth, showed signs of irreversible brain damage.

Three other children had *atypical PKU.* Two of these had plasma phenylalanine levels of 20-25 mg/dl. In the third, the level was 41 mg/dl; her case is interesting, for throughout the course of her disease her phenylalanine tolerance steadily improved. Moreover, although she excreted phenylketones in early infancy, she no longer showed the abnormal metabolites in her urine when placed on an unrestricted diet at age 6 years. The other two were classified as atypical because although they excreted phenylketones, their plasma phenylalanine levels at diagnosis were less than 25 mg/dl. In addition, in one case, despite the fact that treatment was delayed until the patient was 17 months old, he is not severely retarded.

The third group of children studied were considered to have *benign hyperphenylalaninemia.* All had plasma phenylalanine levels of 8-13 mg/dl at diagnosis. None excreted phenylketones in their urine, and none were treated with dietary phenylalanine restriction. All these children were of normal intelligence and showed no clinical evidence of their metabolic disorder.

Fifteen of the 23 children reviewed were first detected through PKU screening programs, including the child from Ontario. These included all 5 subjects with benign hyperphenylalaninemia, 8 to 15 with classical PKU and 2 of the 3 with atypical PKU. Two of the 8 children with classical PKU, identified through screening developed severe mental retardation despite early early detection. In one case, the child had a positive Guthrie screening test in the newborn period, but definitive diagnosis and the initiation of treatment were unfortunately delayed several months.

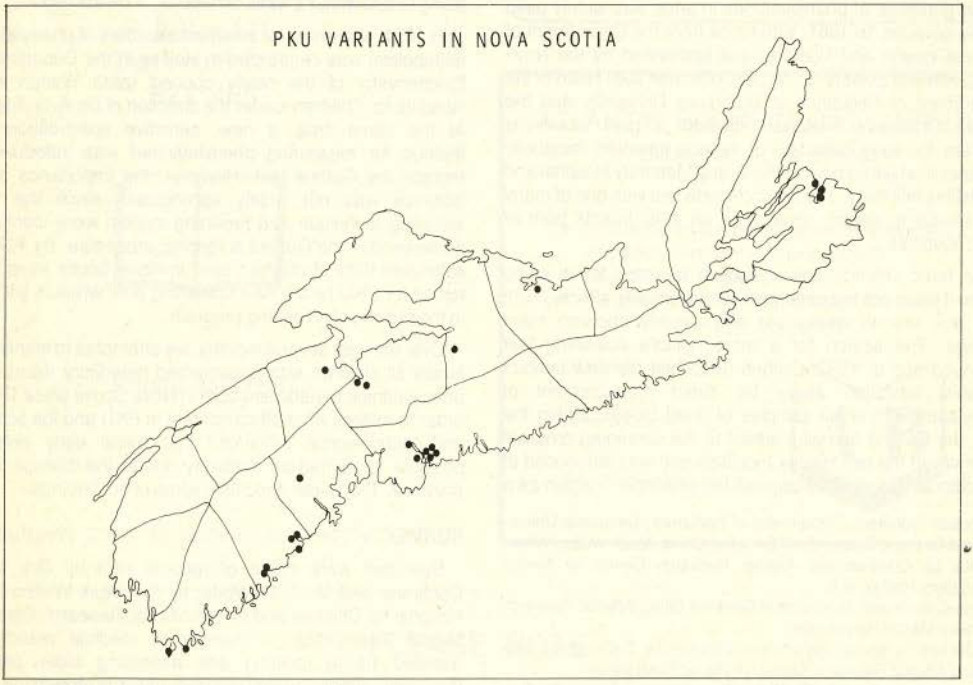

FIGURE 1

Birth places of the children with inherited disorders of phenylalanine metabolism born in Nova Scotia since 1957.

In the other case, the child was identified and treatment was begun within 3 weeks of birth but why she failed to respond is not clear. Her initial plasma phenylalanine level was more than 60 mg/dl and subsequently, on treatment, it rarely rose above 4 mg/dl, even when her dietary phenylalanine intake was increased to 110-120 mg/kg/day after a year on treatment. Her intellectual deterioration was too severe to be explained entirely on the basis of over-vigorous phenylalanine restriction. The relentless neurological deterioration, in spite of apparently adequate therapy and the observation that her phenylalanine tolerance was higher than that seen in children with classical PKU, support the suggestion that she may be suffering from dihydropteridine reductase deficiency[6]. This variant does not respond to dietary phenylalanine restriction; severe brain damage is inevitable. Unfortunately, the child is institutionalized in another province and is not available for examination.

Eight patients were initially identified because of clinical evidence of brain damage. Of these, 5 were born before the Guthrie test screening program was established in 1963; another was born in 1965 before screening became available throughout the province; and 2 were born after 1966 but were initially missed in spite of the province-wide screening program. None of the 8 was intellectually normal. Three, including 1 of the 3 patients with atypical PKU, were mildly to moderately retarded; 5 were severely retarded.

The most prominent clinical findings in these patients were failure to achieve major developmental milestones (e.g. sitting unsupported), and progressive inactivity and apathy. In one case, the child was thought to be deaf and blind when first studied at 10 months of age. In 2 instances, when treatment was instituted at 10 and 17 months, the children showed dramatic improvement initially, as the plasma phenylalanine was lowered to 5-15 mg/dl; however, they were left with some degree of intellectual impairment. Some signs often associated with untreated PKU were absent or inconspicuous in our patients. None had infantile eczema. All had very fair complexions. Although the odour associated with the excretion of phenylketones in the urine is quite pungent, most parents did not comment on it unless asked about it specifically. Even then, some denied ever noticing the unusual smell.

Of the 7 severely retarded children with classical PKU, 4 were examined personally by the authors and detailed records were available from a fifth. All had very fair complexions, white or blonde hair and blue eyes; all were microcephalic. All exhibited a tendency to aggressive, hyperactive behaviour. None could speak intelligibly; all were incontinent of bowel and bladder and required total personal care. Characteristically, severe mental retardation was the only neurological sign. Associated physical findings were generally insignificant. In particular, all were well nourished and of normal stature. Only two had convulsive disorders.

At the other extreme, the children with classical PKU treated appropriatedly from early infancy were indistinguishable from normal children at the same age, though all had fair complexions and blue eyes.

DISCUSSION

From 1957-1977 inclusive, a total of 22 children with identified hereditary disorders of phenylalanine metabolism were born in Nova Scotia. Prior to 1964 (when large scale screening became well established), only one patient with benign hyperphenylalaninemia was identified. With the advent of screening, these children, who would almost certainly have gone undetected before, began to be discovered in significant numbers. Thus, from 1964 to 1973 inclusive, 14 children with disorders of phenylalanine metabolism were detected in 147,000 live-born infants (incidence of 1:10,500 births); 8 had classical PKU (incidence of 1:18,800 births). These incidences are almost identical to those reported for the province of Ontario during a comparable period[7].

Hence, the data indicate that the incidence of classical PKU in Nova Scotia is about the same as that in other areas of Canada. This was surprising on two accounts. First, the proportion of the population of the province of Celtic extraction is higher than in other provinces, and regions with high proportions of people of Celtic descent tend also to have high incidences of PKU[8]. Secondly, the comparative isolation and demographic stability of many Nova Scotian communities might be expected to increase the degree of inadvertent inbreeding and thus increase the incidence of rare autosomal recessive conditions, like PKU. In fact, none

TABLE I

Patients with hereditary disorders of phenylalanine metabolism born in Nova Scotia in the decades before and after wide-spread PKU screening.

Disorder	Total number	Detected through screening	Mentally normal	Mildly retarded	Severely retarded (Number institutionalized in parenthesis)
Classical PKU					
1957-1966	7	2	2 (29%)	1	4 (3)
1967-1976	8*	6*	4 (50%)	1	3*(2*)
Atypical PKU					
1957-1966	2	1	1 (50%)	1	0
1967-1976	1	1	1 (100%)	0	0
Benign hyperphenylalaninemia					
1957-1966	1	1	1 (100%)	0	0
1967-1976	4	4	4 (100%)	0	0
	23	15	13 (57%)	3	7 (5*)

*Includes a patient who may have hereditary dihydropteridine reductase deficiency which is refractory to treatment.

of the patients studied was obviously of Celtic descent, and in none of the affected families was their direct evidence of consanguinity.

Mass screening for PKU in the new-born period has had a major impact on the burden of the disease on both the community and affected families. Early detection through screening contributed directly to successful management of 8 children with classical or atypical PKU, who would very likely have been moderately to severely retarded had their condition not been detected then. In 2 cases, the occurrence of PKU in older siblings generated particular interest in the screening test. Some of these children are now attending school and progressing normally. In the normal course of events, they may be expected to become productive members of the community.

Of the 23 children studied, 7 are so severely handicapped they are unlikely to reproduce. However, one might ask what effect procreation of the remaining 16 would have on the incidence of PKU and associated problems. First, it is important to point out that on the basis of the frequency of hereditary disorders of phenylalanine metabolism in the province, about 1 in 50 Nova Scotians is a carrier of one of these conditions. Thus, the total gene pool is so large that even the most fecund affected individual is unlikely to affect it significantly. In other words, we are in little danger of disturbing the genetic equilibrium of the population for some hundreds of years.

The risks to the direct progeny of individuals afflicted with these conditions are more grave. Because those affected pass a mutant gene to all their offspring, all will at least be carriers. The chances of an affected patient marrying a carrier of PKU or variant of the disease are 1:50, and the probability of such a couple having a child with the condition is 50%. Thus the risk of a patient with PKU producing another affected individual is theoretically 1:100 — small, but still more than a hundred times greater than the risk of PKU occurring in the progeny of other unrelated Nova Scotians. Carriers of PKU can be identified by appropriate testing, and the risk of producing affected offspring can therefore be eliminated by genetic counselling. We strongly recommend that relatives and prospective spouses of individuals with PKU or variants undergo carrier testing before having children.

The risks to the offspring of phenylketonuric women represent a special case that begs further study. The incidence of severe congenital malformations, including microcephaly, among the progeny of women affected with classical PKU is extremely high[9], presumably due to the exposure of the developing fetus to toxic levels of phenylalanine during pregnancy. Whether these can be prevented by placing women with PKU back on dietary phenylalanine restriction during pregnancy is not yet clear. There are innumerable practical as well as theoretical difficulties yet to be overcome before an acceptable approach to this problem is developed. Therefore, for the time being, we generally counsel girls with classical PKU to avoid pregnancy until we know more about maternal PKU and how to manage it safely.

SUMMARY

A total of 23 children with inherited disorders of phenylalanine metabolism born or resident in Nova Scotia since 1957 were studied: 15 had classical PKU, 3 atypical PKU and 5 benign hyperphenylalaninemia. Of these, 7 were severely mentally retarded, 3 were mildly retarded and 13 (including 5 with benign hyperphenylalaninemia) were intellectually normal. The introduction of mass screening of newborn infants for hyperphenylalaninemia in 1966 contributed directly to the successful management of 8 children who would very likely have been moderately to severely mentally retarded had they not been discovered early. The incidence of PKU and variants in Nova Scotia appears comparable to that in other regions of mixed population in Canada. One child was studied who may be afflicted with the recently described hereditary disorder of phenylalanine metabolism characterized by dihydropteridine reductase deficiency. □

ACKNOWLEDGEMENTS

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References

1. **Scriver CR and Rosenberg LE:** Amino Acid Metabolism and Its Disorders, Major Problems in Clinical Pediatrics, Vol. 10, Saunders, Philadelphia, pp. 290-337, 1973.
2. **Guthrie R and Susi A:** A simple phenylalanine method for detecting phenylketonuria in large populations of newborn infants. *Pediatrics* **32**:338, 1963.
3. **McCaman MW and Robins E:** Fluorimetric method for the determination of phenylalanine in serum. *J. Lab. Clin. Med.* 59:885, 1962.
4. **Clarke JTR and DeWolfe MS:** Phenylketonuria: A preventable tragedy. *N.S. Med. Bull.* **54**:87, 1975.
5. **Blaskovics ME, Schaeffler GE and Hack S:** Phenylalaninaemia: Differential diagnosis. *Arch. Dis. Child.* 49:835, 1974.
6. **Smith I, Clayton BE and Wolff OH:** New variant of phenylketonuria with progressive neurological illness unresponsive to phenylalanine restriction. *Lancet* **1**:1108, 1975.
7. **Advisory Committee on Inborn Errors of Metabolism to the Ontario Ministry of Health:** Phenylketonuria variants in Ontario. *Can. Med. Assoc. J.* **115**:509, 1976.
8. **Carter CO:** Factors determining the birth frequency of recessive conditions — a high frequency of classical phenylketonuria in Ireland. *Ir. Med. J.* **69**:386, 1976.
9. **Howell RR and Stevenson RE:** The offspring of phenylketonuric women. *Soc. Biol.* **18** (Suppl.):519, 1971.

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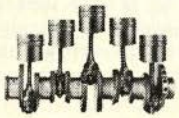

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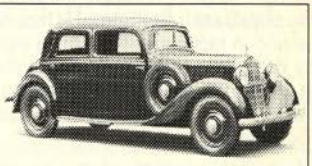

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Correspondence

To the Editor:

Tom Gorman left a legacy. His many accomplishments in the surgical and medical administrative fields are well-documented and have been heralded in the many editorials and eulogies heaped on him. But, to know the man as a close associate and friend over more than 20 years was, indeed, an honor and a privilege. His approach and attempts to solve the many problems of his fellow man with personal involvement and sacrifice was a frequent occurrence. When an issue came forth worthy of involvement — he became involved and researched his subject relentlessly.

Tom Gorman left monetary matters to others — his concern was the maintenance of the dignity of his "brothers and sisters" in the rapidly changing and, as he often phrased it, deteriorating world.

I have lost a good friend, a valuable and close associate — Eastern Nova Scotia has lost a champion of the rights of man. We will all miss his "strength down the middle".

Yours Sincerely;

A Emerson Dunphy, M.D.,
36 Hillcrest St.,
Antigonish, N.S. B2G 1Z2

To the Editor:

There are many wasy of examining the effect of inflation on medical incomes. A recent article in the *CMA Journal*, "Trends in the income of Canadian physicians by province" (1), exemplified this by studying the effects of inflation and fee change on the real disposable incomes of physicians as a group in each province, between 1971 and 1975. In many provinces physicians had suffered a net loss in real disposable income between these years. Though Nova Scotia physicians' real disposable incomes had retained their value and in fact showed a minute increase. However this study only extended to 1975 and unfortunately, as will be seen below, the fee increases experienced in Nova Scotia since 1975 have compared unfavourably with increases in the Consumer Price Index.

I have been looking at the "across the board" Fee Schedule increases in Nova Scotia since April 1973, and comparing these to the Consumer Price Index increases. The Consumer Price Index (CPI) does not necessarily reflect specific increases in costs of medical practice expenses, since it does not tap a number of items relevant of such practice. Nevertheless it is an available and widely used index of inflation.

It is also true that the "across the board" fee schedule increases do not represent the entire increases in fees enjoyed by the medical profession in Nova Scotia. Since, in recent years the Medical Society have sought through its representatives on the Tariff Development Committee to have a small proportion of each increase allocated to raising certain specific fee items, partially with a view to reducing discrepancies between different specialities within the profession. However as will be seen from the following figures, a substantial majority each year has been allocated to "across the board" fee schedule increases. The total percentage increase is given first, and the percentage allocated to "across the board" increases second (in brackets). Consumer Price Index (CPI) increases (for the previous 12 months) are given in a second row.

July 1st, 1974, 10.4% (8%); April 1st, 1975, 12% (9.26%); April 1st, 1976, 8.1% (7.63%) April 1st, 1977, 4% (3.15%).

CPI: March 1974 + 10.36; March 1975 + 11.26%; March 1976 + 9.00%; March 1977 + 7.38%.

I think that most would support the decision to allocate a small proportion of each increase to certain special items, since otherwise the Medical Society could be seen as entirely and indefinitely supporting the status quo. This in turn would possibly result in a loss of confidence in the Medical Society as a negotiating body by those who felt that they were disadvantaged by the status quo. There would then be inevitable pressures by each group to independently negotiate its own fees which, in the long run, would probably be much more disadvantageous than the present situation. Further as regards this smaller amount each year allocated to certain specific fees, I would feel that few would seriously suggest that the Medical Society utilized this sum merely to distribute largesse in an arbitrary way. The specific fees which were specially increased must therefore have contained some anomaly or were unduly low in value at the beginning. Just as a particular fee may sometimes be seen as overvalued in the light of technological changes, as occurred in the case of fibre-optic endoscopies.

If we examine a ten dollar fee as of April 1973, the following "across the board" changes have actually occurred: April 1973 = $10.00; 1974 (+8%) = $10.80; 1975 (+9.26%) = $11.80; 1976 (+7.63%) = $12.70; 1977 (+3.15%) =$13.10.

Whereas to allow for increases in the Consumer Price Index (CPI) (given for the previous 12 months, in brackets below), the following increases would be required: March 1973 = $10.00; March 1974 (CPI change of + 10.36%) = $11.04; March 1975 (CPI +11.26%) = $12.28; March 1976 (CPI + 9.00%) =) = $13.39; March 1977 (CPI) + 7.38%) = $14.37.

In 1977, as far as the "across the board" fee increases are concerned, to allow fully for increases in the Consumer Price Index this ten dollar fee would need to be $14.37, whereas in fact the fee actually stands at $13.10. The implication is that if a physician wished to retain the same financial position, in terms of "across the board" increases, as he would if the increases received had been of the same magnitude as changes in the Consumer Price Index, on the basis of a fee valued at $10.00 in April 1973 he would now need to directly bill his patients $3.24, i.e. $1.97 (representing 15% of $13.10) + $1.27 (being the difference between $13.10 and $14.37). This sum of $3.24 would represent approximately 25% of $13.10, the present fee schedule value. In other words physicians would have to directly bill patients 25% of the present fee schedule values rather than 15%. This, of course, assumes that the physician is in the custom of directly billing his patients the difference between the MSI payment and the Medical Society Fee Schedule, which may not necessarily be the case.

The purpose of this letter is not to suggest that physicians should directly bill their patients at this rate. In fact this would probably be illegal under the AIB regulations. Rather it is to illustrate the deteriorated state of the fee schedule in relation to inflation.

One of the main reasons for this is the cumulative effect of the failure in any year to achieve an increase equal to that of inflation. To illustrate this, suppose in year 1, a particular item has a value of 100. Suppose by the beginning of the next year (year 2) inflation had occurred at a rate of a% but that the fee item was only increased by a lower b%. The fee of 100 would now become the lower sum of (100 + b), rather than (100 + a). Suppose that in the third year the inflation rate was x%, but that the fee item was only increased by a lower value of y%. Then not only would the value be increased by only the lower amount of y%, but this y% would be calculated on the lower value of (100 + b), rather than (100 + a). It is therefore clear that if the above process continued over a number of years, it could have quite a marked cumulative effect.

Net disposable income is of course not completely related to fee schedule values. Since it is possible for a physician to some extent, to try to compensate for the effects of inflation by working longer hours, trying to see more patients in a given time, or both. However, there is obviously a very strong relationship between the two.

Regarding Nova Scotia physicians as a group, it is apparent that in 1974 and in 1975 the *total* increase in fees either did equal or slightly exceeded the Consumer Price Index changes. However, in the last two years they have been somewhat less than the Consumer Price Index increases. As far as the "across the board" fee increases are concerned, they have consistently been lower than the Consumer Price Index since 1973.

I have made no attempt to look back further than 1973, which of course only covers a period of four years.

Yours truly,

Michael J. C. Thomson, F.R.C.P.(C),
Woodlawn Medical Clinic,
110 Woodlawn Rd.,
Dartmouth, N.S. B2W 2S8

Reference

1. **Constantinou S:** Trends in the income of Canadian physicians by province, *CMA Journal*, **117**:1344-5.

To the Editor:

In the recent issue of *The Nova Scotia Medical Bulletin* (Vol. 57, No. 1, Feb., 1978), you published my article on "The Management of the Adult Patient with a Drug Overdose". In that article, I suggested that gastric lavage with a solution of desferrioxamine should be used in the treatment of a patient poisoned with iron. Dr. John Anderson kindly drew to my attention a statement of the Canadian Paediatric Society condemming oral desferrioxamine. Subsequently, I have reviewed the literature on this topic and find the matter to be highly controversial.

The Canadian Pediatric Society currently recommends that intramuscular desferrioxamine be used in moderate iron poisoning and intravenous deferrioxamine be used in severe poisoning as outlined in the Health and Welfare publication "Emergency Treatment".

Because of the lack of good scientific data on this subject, I hope to use an animal model to clarify this question. In order to make your readers aware, however, that the pediatric community is concerned about the use of oral desferrioxamine, I would appreiate early publication of this letter.

Yours truly,

J. Gray, M.D., F.R.C.P.(C),
Assistant Professor,
Department of Pharmacology,
Faculty of Medicine,
Dalhousie University,
Halifax, N.S. □

Personal Interest Notes

The *Bulletin* congratulates **Dr. Lea C. Steeves** for the honor bestowed on him by the Royal College of Physicians and Surgeons at their most recent meeting.

For his contributions in Continuing Medical Education, his devotion to patient care, his services to the Canadian Medical Association and Royal College and Medical Council of Canada, he was chosen for the Duncan Graham Award for 1978.

Dr. Steeves has been President of The Medical Society of Nova Scotia 1969, President of the Canadian Association for Continuing Medical Education and President of the Medical Council of Canada. He was a founding director of the Canadian Heart Foundation and received its Distinguished Service Award in 1973.

Dr. Robert Dickson was appointed as an Officer in the Order of Canada. This order was created in 1967 to recognize outstanding achievement and service in important fields of endeavour.

Dr. Ronald Stewart, formerly practising in Northern Cape Breton as a partner to the Honourable Dr. Maynard MacAskill, recently visited Nova Scotia between appointments. Formerly Director of the Paramedic Rescue Programme for Los Angeles County, he is on his way to take up directorship of Emergency Department at University Hospital, Pittsburg, Pennsylvania. Ron is acting in training premedical personnel, training medical students and M.D.'s to work with these, and acting as a consultant to agencies working to set up ideal emergency care facilities.

The *Bulletin* hopes that some Canadian center, even better, a Nova Scotia Center could attract Ron back.

OBITUARY

Dr. C. L. MacMillan, Sr., (75) of Baddeck N.S. died February 10, 1978 at the Victoria General Hospital. He graduated from Sydney Academy and after one year at Acadia University attended Dalhousie Medical School graduating in 1928. He retired from active practice in 1966. In 1972 he was awarded the Order of Canada in recognition of his contribution to the community. In 1977 Dr. MacMillan was awarded the senior membership in The Canadian Medical Association. The *Bulletin* extends sincere sympathy to his family. □

Notice

Dr. Hans Selye
author of "Stress Without Distress" will present a one day conference on *"Coping With Stress"*

When: May 23, 1978

Where: Chateaux Halifax, Halifax, N.S.

Fee: $95 (including luncheon)

For further information or registration, please contact

Don Mills
Advanced Management Centre
Dalhousie University
Telephone (902) 424-2526

NEW MEMBERS

The Physicians listed below have joined The Medical Society of Nova Scotia between January 1, 1978 and March 31, 1978. A most cordial welcome is extended by the Society.

Dr. J. H. W. Adams	Dartmouth, N.S.
Dr. J. H. Armstrong	Truro, N.S.
Dr. M. J. Brennan	Dominion, N.S.
Dr. Louise Cloutier-Robinson	Dartmouth, N.S.
Dr. J. A. Collins	Halifax, N.S.
Dr. B. F. Fraser	Glace Bay, N.S.
Dr. D. J. Graham	Tantallon, Hfx. Co., N.S.
Dr. T. G. Hand	Dartmouth, N.S.
Dr. H. H. Haysom*	Houston, Texas
Dr. E. B. Holmes	Halifax, N.S.
Dr. B. R. Keith*	Saint John, N.B.
Dr. A. A. Kelland	Kentville, N.S.
Dr. J. W. D. Knox	Halifax, N.S.
Dr. T. J. Marrie	Halifax, N.S.
Dr. J. H. J. Milliken	Wolfville, N.S.
Dr. P. F. Murphy	Sydney, N.S.
Dr. T. S. MacDonald	New Glasgow, N.S.
Dr. R. A. MacLachlan	Halifax, N.S.
Dr. R. G. McLaren	Halifax, N.S.
Dr. J. V. O'Brien	Halifax, N.S.
Dr. J. C. W. Parrott	Bedford, N.S.
Dr. G. K. Payne	Windsor, N.S.
Dr. R. A. Seary	Dartmouth, N.S.
Dr. William Wrixton	Halifax, N.S.

*Recent Dalhousie Graduates.

Lifestyle is keeping in shape or resolving to get into shape by regular physical activity.

ANNUAL MEETING

Canadian Lung Association

Canadian Thoracic Society

Canadian Nurses' Respiratory Society

June 12-14, 1978

Holiday Inn/Winnipeg Convention Centre

For further information: Mr. Hubert Drouin, Executive Secretary
Canadian Lung Association
75 Albert Street, Suite 908
OTTAWA K1P 5E7 Canada

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