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ALBERTA OCCUPATIONAL MEDICINE NEWSLETTER

EDITORIAL COMMENTS

This issue of the Newsletter appears after a brief hiatus. To those of you who are responsible for the maintenance of library collections (or who are merely well-organized), and who have noticed the off-timing, we apologize. With our resumption, we are pleased to again acknowledge the generous support of Alberta Community and Occupational Health for another year. However, as their departmental budget was trimmed by the ubiquitous budget cuts, so too was our budget trimmed and re-organized. The result is a somewhat smaller — and slightly later — edition of the Newsletter. While we expect to be able to resume a regular schedule for the upcoming year, the size of the Newsletter will continue to be somewhat shorter this year than last.

This issue contains two overview articles of occupational health issues. The very common problem of degenerative spinal disease is concisely summarized in Ron Dufresne and Tinie van Schoor's regular article. In contrast, an uncommon but fascinating environmental health issue — cadmium exposure — is reviewed by Michael Reesal, Ron Dufresne and Ken Corbet. In addition, we are pleased to have another case report, this time from our Edmontonian colleague, Tee Guidotti.

Because this is a Newsletter, we plan to continue our coverage of upcoming conferences, newly available guidelines, and other bits of information of interest to our "target audience." Please feel free to consult us to comment, for more information, or to suggest ideas for further articles.

Heather Bryant, M.D., Ph.D., FRCPC
Editor

NEW GUIDELINES FROM ALBERTA COMMUNITY AND OCCUPATIONAL HEALTH

Seven new Guidelines have appeared since the last issue of the Newsletter. An overview of these follows:

23. Guideline for
Occupational Health Services
This guideline looks at sections of the 1985 International Labour Conference Recommendation concerning Occupational Health Services. As such, it provides an international perspective on the functions, organization, and conditions of operation of such services. It will be useful to health professionals whose responsibilities involve on-site or consultative roles in occupational health settings.

25. Use of Resuscitators in Industry
This ten-page guideline considers many aspects of resuscitator use which should be considered when resuscitation emergencies are possible at any worksite. Covered are indications and contraindications, as well as necessary features of the resuscitator units themselves. A sketch of legal and policy issues is also provided.

26. Use of Inhalators in Industry
This is a three page document, which augments No. 25 above.

27. Medical Monitoring for Workers Exposed to Carbon Monoxide
The occupational and physiological considerations of carbon monoxide exposure, including a summary of high-risk individuals, are summarized in this 10-page guideline.

28. Medical Monitoring for Workers Exposed to Benzene
In the years since benzene's relationship to leukemia was established, its utilization has decreased. However, the number of workers potentially exposed is still substantial. This guideline considers acute and chronic health effects, and the medical monitoring procedures designed to limit the risks.

29. Stress and the Worker
The complex biological and psychosocial perspectives on workplace stress are outlined.

30. Medical Monitoring for Workers Exposed to Tetrachloroethylene (Perchlorethylene)
This overview of health effects and monitoring recommendations will be useful for those providing care to workers potentially exposed to this chemical through work involving dry cleaning or metal degreasing.

31. Health Effects of Fingernail Sculpting with Methyl Methacrylates
Fingernail sculpting can be hazardous to the health! This guideline outlines the effects, and the prevention of ill effects, of this increasingly popular line of work.

* * *

Prepared in the Department of Community Health Sciences, Faculty of Medicine, The University of Calgary through funding by Alberta Community and Occupational Health

COMPENSATION APPEALS FORUM CALL FOR PAPERS, CASE COMMENTS, LETTERS, ETC.

Workers' Compensation Appeals Tribunal

The Workers' Compensation Appeals Tribunal, under the chairmanship of S. Ronald Ellis, Q.C., determines appeals arising under the *Workers' Compensation Act,* and appeals from decisions of the Workers' Compensation Board respecting entitlement to compensation or benefits, and assessments or penalties. It also determines the effect of the Act on workers' rights to take civil actions against their employers.

The Workers' Compensation Appeals Tribunal is calling for submissions for a publication called the *Compensation Appeals Forum.* This publication contains analytical comment from the Tribunal's constituencies and other observers concerning the Tribunal's decisions and processes, and general compensation principles related thereto. Submissions will be reviewed by an editorial board established within the Research and Publications Department of the Tribunal, and will not be seen by the decision-making members of the Tribunal until they are published in the *Forum.* We invite you to send papers, case comments, letters, etc., in English or French, to Dr. Roger Rickwood, Chairperson of the *Forum's* Editorial Board. The editorial board reserves the right to reject, edit, or condense all submissions, and does not assume the responsibility for the loss or return of manuscripts. For free sample copies please contact:

Research & Publications Department
Workers' Compensation Appeals Tribunal
505 University Avenue, 7th Floor
Toronto, Ontario M5G 1X4
Telephone: (416) 598-4638

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ADVERSE HEALTH EFFECTS FROM INDUSTRIAL AND ENVIRONMENTAL CADMIUM

Michael R. Reesal, M.M., Ph.D., F.R.C.P.(C).*
Ronald M. Dufresne, M.D., C.C.B.O.M.**
Ken Corbet, M.D., C.C.F.P.***

"All is not gold that glitters — and all is not silver that shines."
(Anonymous)

Alloys of nickel, chrome, and cadmium outshine silver not only in appearance but in toxicity also. Indeed, silver is relatively harmless and apart from the grey cosmetic disfigurement of argyrosis, even excessive medicinal silver causes no recognizable organ or metabolic disturbance. Cadmium does not enjoy this record of safety.

Cadmium poisoning was first described by a Belgian physician in 1858 in three servants who used cadmium carbonate as a polishing compound. From 1939 to 1954, some two hundred people near Fuchu, Japan suffered from a condition termed 'Itai-Itai', literally, 'ouch-ouch' disease. Multiple fractures and severe pain in the legs and lower back affected mainly post-menopausal women; most members of the community showed abnormal levels of glucose, calcium, and amino acids in their urine. Although this collection of signs and symptoms was unusual and persistent, it took investigators 15 years to discover that itai-itai was caused by large amounts of cadmium in the village's water supply; the inhabitants of the community had for years been consuming rice contaminated by the effluent of a lead-zinc mine upstream from their rice paddies.

SOURCES OF EXPOSURE TO CADMIUM

Cadmium is a relatively rare, non-essential trace element, and is obtained by extraction from lead and zinc ores. It is present in the environment largely through the application of phosphate fertilizers and the deposition of airborne contaminants from mining, smelting, and waste incineration operations; such anthropogenic sources outweigh natural sources ten-fold. Within the food chain, cadmium is concentrated in the leaves of plants and in the liver and kidneys of food animals; fish and crustaceans may also show significant levels in their tissues. The primary route of absorption for the general population is therefore gastrointestinal. Regular tobacco smokers, however, can absorb significant amounts through inhalation.

Though cadmium is ubiquitous, it is essentially harmless in the small amounts that are generally present in the environment; it is its use in industry that has intensified exposures to hazardous levels. Cadmium has many industrial applications; the potential for exposure is greatest in its use in a variety of low-melting point and corrosion resistant solders and alloys (used in electroplating and the production of nickel-cadmium batteries), or as an oxide, chloride, or sulfate in the production of pigments and plastic stabilizers.

In Alberta, we might pay particular attention to the few remaining solders that still contain cadmium (Braze 202, Easy-Flow, and TEC contain 5 percent, 20 percent, and 95 percent cadmium respectively); these pose a hazard to both the user and bystanders if adequate ventilation and personal protection are not used. Alloys containing cadmium are usually labelled with warnings and are becoming increasingly expensive.

HEALTH EFFECTS OF CADMIUM

As with any toxic material, the spectrum of health effects will depend on the route of absorption, the dosage and time interval over which exposure occurs, and the metabolic characteristics of the individual. Cadmium is absorbed poorly through the gastrointestinal tract (only 5 percent), but quite well via the respiratory tract (>40 percent).

Acute effects of exposure usually occur from the inhalation of fumes or smoke produced by the heating, cutting, or incineration of cadmium-containing materials; acute ingestions, though they have been reported, are rare. In inhalational exposures, there are often no clues that exposure is occurring (except that cadmium imparts a yellow color to smoke or fumes). Several hours elapse before the onset of mucosal irritation (rhinitis, pharyngitis, and tracheitis), nausea, fever and rigors, and myalgias of the back and limbs; these early symptoms thus resemble the relatively benign "metal fume fever" or "welders' shakes" not uncommonly seen with the inhalation of zinc fumes.

Cadmium, however, is particularly toxic to alveolar cells (so much so that it was once considered for use in chemical warfare) and can produce a chemical pneumonitis, marked by progressive dyspnea, tachypnea, and cyanosis, diffuse crackles, and ill-defined infiltrates on the chest radiograph. Treatment is supportive. Fatalities are due to massive pulmonary edema that begins 24-48 hours after exposure; if recovery ensues, then it is usually complete - a reduced forced vital capacity (FVC) and diffusion capacity (DL_{co}) are more the exception than the rule.

Chronic occupational or environmental exposure to cadmium (either gastroin-

testinal or respiratory) may eventually cause renal injury. Once in the bloodstream, the metal (as Cd^{2+}) binds to metallothionein, a medium weight protein thought to serve as a 'scavenger' of excess cations to prevent their interference with cellular enzyme systems.

The cadmium-metallothionein complex is deposited in the liver and the kidney; it is the latter organ that is most sensitive to chronic toxic effects. Because of its small size, the complex passes through the glomerulus, but then is reabsorbed by the cells of the proximal tubule. The half-life of cadmium in the kidney is long (approximately 30 years); renal burden therefore increases from zero at birth to an average (non-occupational) value of 25-50 micrograms/gram of renal cortex in a 50 year old.

Because the threshold value for toxicity is near 250 micrograms/gram of cortex, the reader can appreciate that there is a large margin of protection conferred by the metallothionein system. When this threshold value for renal burden is reached, tubular and/or glomerular dysfunction occurs, signalled by the appearance in the urine of:

- a high proportion of low molecular weight proteins (most notably β2-microglobulins and retinol-binding protein)
- increased uric acid, amino acids, glucose, calcium, and phosphates
- elevated cadmium levels (greater than 10 μg/gm creatinine).

It is the loss of calcium and and phosphates in the urine that produces the upset in calcium metabolism and nephrolithiasis. Progression to chronic renal failure is uncommon and if exposure is stopped in the initial stages of insult, some degree of recovery is usually observed.

These changes in the urine are markers of tissue injury, and so have limited value in monitoring the ongoing exposures of workers. In an occupation or environment that poses a risk for cadmium exposure, serial measurements of urinary and serum cadmium are appropriate; collection and analysis techniques can markedly affect the interpretability of the results, however, and consultation is recommended when considering these tests. Substitution by less hazardous substances, engineering containment of processes and contaminants, safe work practices (such as not eating or drinking in the work area), and personal protection should all be applied before undertaking biological monitoring of exposed persons.

Other signs and symptoms noted in advanced cadmium poisoning can include weight loss, mild anemia, peripheral neuropathy, anosmia, testicular atrophy, yellow discoloration of the enamel, and hepatic injury. Controversy surrounds many of the other purported health effects of cadmium. Animal experiments have demonstrated both carcinogenic and teratogenic potential; inconclusive epidemiologic evidence exists for a relationship of cadmium to tumors of the prostate, lung, and kidney. There is a possible role of cadmium in hypertension and emphysema, but again there is no firm evidence for or against this relationship.

This uncertainty as to whether cadmium insidiously affects human health emphasizes the need for further study of the use of cadmium in industry and of those processes that release cadmium into the environment. Alberta's current 8 hour exposure limit for cadmium dusts or fumes is 0.05 mg/m^3; Ontario is considering a more stringent 0.02 mg/m^3. Sweden has taken the boldest step to date by banning the import or use of cadmium in certain industrial applications.

SUMMARY

Cadmium is widely distributed in the environment but is rarely accumulated to such a degree as to pose a health hazard (even in smokers). In an industrial setting, acute inhalational exposure can cause a chemical pneumonitis, whereas chronic exposure affects the proximal tubule of the kidney. Cadmium's role in the causation of cancer, birth defects, hypertension, and emphysema is unclear. While today's medical practitioner is unlikely to see illnesses due to cadmium, it is prudent, as always, to inquire about a patient's present and past occupations, and to be aware of those occupations that carry a risk of cadmium exposure.

** Alberta Workers' Compensation Board*

*** Alberta Workers' Compensation Board and Clinical Assistant Professor, Department of Community Health Sciences, Faculty of Medicine, The University of Calgary*

**** Resident in Community Medicine, Department of Community Health Sciences, Faculty of Medicine, The University of Calgary*

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THYROID FOUNDATION OF CANADA

Everyone is welcome to attend the first meeting of what will be the Edmonton Chapter of the Thyroid Foundation of Canada.

DATE: Tuesday, October 20, 1987
TIME: 7:30 p.m.
PLACE: 2nd Floor, 9321 - 48 Street, Edmonton (East of Edmonton Journal building in Eastgate Industrial Park)
SPEAKER: Dr. Jody Ginsberg

Thyroid malfunction affects one out of every 20 adults in Canada. Thyroid Foundation of Canada now has over 1700 members across Canada, with approximately 100 in Edmonton alone. We are a self-help group whose goals are:

- To awaken public interest in, and awareness of, thyroid disease;
- To lend moral support to thyroid patients and their families;
- To assist in fund raising for thyroid disease research.

For more information contact:

June Layman
3555 - 107 Street
Edmonton, Alberta
T6J 1A9
Telephone: (Bus.) (403) 427-4788
(Res.) (403) 436-8859

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UPCOMING CONFERENCES

WELLNESS '86 CONFERENCE

Toronto, Ontario, November 29 to December 1, 1987

Contact:
Ms. Laura Palmer Korn
c/o North York Parks and Recreation
5100 Yonge Street, 3rd Floor
North York, Ontario M2N 5V7

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ANNOUNCEMENT

NOW AVAILABLE: A new Canadian text on preventive Medicine with an Alberta flavour! This text addresses preventive aspects, community health, long term and acute care, providing an overview of historical and current forces in the delivery of prevention care. *Think Prevention!* (J.M. Howell, ed.) can be ordered from the Health Unit Association of Alberta, Suite 412, 9707-110 Street, Edmonton, Alberta T5K 2L9, at a cost of $17.50 (plus $2.50 for postage and handling). Telephone (403) 482-2391.

* * *

THE CASE OF THE PHANTOM DRIVER

Tee L. Guidotti, M.D., M.P.H., C.C.B.O.M.

Occupational medicine presents us with many interesting challenges but seldom are we called upon to distinguish the difference between reality and illusion. In this intriguing case, the issue arose whether an episode on a deserted highway was an unreported toxic incident or was the product of a lesion affecting the patient's ability to perceive reality.

CASE REPORT

The patient was a 40 year old truck driver whose work in Northern Alberta took him through sour gas fields. One day in October 1986 at noon he was hauling gravel from a pit to a gas plant when he encountered a strange sight. A truck was parked at an odd angle on the road in icy conditions. Behind the steering wheel a man was slumped unconscious. The patient found a secure place to park his own truck, called for assistance on a mobile phone and walked toward the person. With vigorous shaking, he awakened the other driver, who then began to speak and then to swear at him. Ultimately, the driver drove off in great anger, weaving as he drove down the road.

Perplexed and not a little angry, the patient returned to his own truck only find that the cabin was filled with the stench of rotten eggs. Since he had not smelled this odor earlier, the thought crossed his mind that someone was playing a trick on him and had put foul smelling material inside his truck. He searched furiously for it but found nothing and the odor went away. He went on to his destination and spoke to workers at the plant about what he had just experienced. It was agreed that the driver who had been found unconscious would be in danger driving in an impaired condition on the icy and deserted road. A search was organized that should have overtaken the truck but it was never found. The patient and those he spoke with concluded that the incident was best explained by both men having entered a cloud of hydrogen sulphide. A nearby gas plant was thoroughly searched but no source of emission was found and no incident of release had been reported earlier that day.

The next day, while preparing a meal, the patient lost memory for an entire hour during which he apparently ate, cleaned up his dishes, and started to watch a sporting event on television. On suddenly realizing that an entire hour had passed during which he had no recollection of his activities, he made an appointment to see his physician. While sitting in the examining room, he suddenly experienced the overwhelming smell of rotten eggs and thought that he was being observed by his physician through a peephole in the door. Angry, he started to leave the office when he suddenly realized that there was no peephole and that the incident had been a visual hallucination.

Subsequently, he had a number of olfactory, visual, and auditory hallucinations, sometimes with paranoid content, such as a revelation that he should not trust a long standing business associate. He became fixed in his suspicion that he had experienced a toxic exposure of hydrogen sulphide and that long-term sequelae of the exposure were responsible. He had had no history of alcohol abuse or drug abuse (other than marijuana smoked in his early twenties). He had sustained some head trauma, including loss of consciousness once in an accident in which he was hit by a metal rod, and in amateur boxing as a teenager, with no knockouts. He was not under extraordinary stress and between these episodes demonstrated normal thought processes and affect.

Involvement of occupational medicine began when a safety officer from the sour gas plant contacted the company's occupational physician and asked for his opinion about the validity of the man's claim that his problems were due to hydrogen sulphide. The truck driver welcomed the phone call from the company doctor who investigated the history by confirming details with the man's wife. Although the presumptive diagnosis (later confirmed) was not related to a toxic agent, toxic exposures remained an etiologic possibility. Hence, a referral was made to the nearby University of Alberta Occupational Health Program for further evaluation. During the first week of February 1987, just prior to keeping his appointment for evaluation, he experienced a generalized motor seizure and was hospitalized.

An extensive neurologic evaluation performed in Grande Prairie and Edmonton demonstrated a large mass localized in the left temporal lobe without obstruction of ventricles or midline displacement on computerized tomography, without flushing after injection of contrast media. A biopsy of the mass revealed a grade I astrocytoma. The patient is presently doing well and is awaiting radiotherapy.

DISCUSSION

The patient had clearly been having temporal lobe seizures. These seizures had been remarkably complex, mixing olfactory, visual, and auditory elements. Although it is not unusual for the smell of rotten eggs to be among the odors present in the aura, its presence in this patient led to a confusing situation since the possibility also existed of exposure to hydrogen sulphide.

Until the diagnosis of his brain tumor, the patient maintained adamantly that he had been able to appreciate the difference between reality and hallucination throughout the course of his illness and that the incident on the road with the mysterious driver had been as real as any event in daily life. However, the incident in the doctor's office was just as real to him at the time that it occurred. Features of the encounter make it unlikely to have been a sour gas exposure. While it is true that "knock-downs" can result in almost instantaneous unconsciousness and return to consciousness, in this episode the affected driver "came to" only slowly and would have had to have retained control over his truck initially in order to have stopped it without leaving the road. The patient did not smell the odor of rotten eggs until after he had encountered the driver, suggesting that he had not entered a cloud of hydrogen sulphide earlier with subsequent olfactory paralysis.

Did this patient encounter an unconscious person on a lonely, wind-swept, deserted road? Did he act as a Good Samaritan, awakening the man and warning him of the dangers of being stalled on the icy road? Was a sour gas release that could have led to a fatality concealed and never reported? Did the events in question really happen, or did they exist only in the mind of the patient, stimulated by his then-unknown tumor? With proper treatment and care, the patient may do well. He will never be entirely convinced, however, that the events of that day in October were played out on the left side of his brain and not in the real world.

Acknowledgement:
I thank Dr. Don Johnston of The University of Calgary for referring this case to me for evaluation and Dr. Walter J. Vanast, neurologist, for accepting it in referral from me.

* *Occupational Health Program, Department of Health Services Administration and Community Medicine, Faculty of Medicine, The University of Alberta*

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DEGENERATIVE SPINAL DISEASE AND COMPENSATION

J. Tinie van Schoor, M.D., C.C.F.P.*
Ronald M. Dufresne, M.D., C.C.B.O.M.**

INTRODUCTION

Back problems account for over 20 percent of all industrial injuries reported annually to the Workers' Compensation Board. Approximately 30 percent of all permanent disability awards are for backs.

Physicians in occupational medicine are often requested to express an opinion on the relationship between a worker's back condition and the workplace. Most often the question relates to a specific incident at work, but the general effects of work activities may also require evaluation. In addition, the physician may be asked to express a view on apportionment of causation between occupational and non-occupational factors.

With the exception of major trauma (e.g. diving accidents), few protrusions or extrusions occur in normal, previously healthy discs. Therefore, "simple slipped discs" are often the consequence of degenerative changes which have been precipitated by a minor lifting strain or even an awkward posture.

It is as essential, therefore, to understand the natural history of degenerative spinal disease for the medical opinion to be helpful in the compensation process as it is in the establishment of a cost effective preventive program.

DEFINITION

Degenerative disease of the spine is referred to under a variety of descriptive terms. These include: spondylosis, arthritis of the spine, degenerative disc disease, facet joint syndromes, osteophytic lipping or spurring, segmental instability and secondary spinal stenosis.

Localized changes are more prominent in areas of maximum mobility (C5-6) or at the point of maximum weight bearing (L4 to S1). Cracks in the annulus of the disc have been noted as early as 15 years of age while many octogenarians have remarkably normal looking spinal X rays.

SIGNS AND SYMPTOMS

These depend on the stage of the disease and range from localized backache (which may be constant, intermittent, acute or chronic), nerve root compression and spinal stenosis, to spontaneous fusion with limitation of movement but little or no pain.

ANATOMY AND PATHOLOGY

Each intervertebral articulation consists of a three joint complex: one disc and two posterior apophyseal joints. Pathologic changes usually start in one of these three joints and then progress to involve the other, eventually spreading to the level above, below, or both.

The earliest changes in the disc are chemical and are age related. Increased intradiscal pressure may accelerate these chemical changes. Eventual weakening of the annulus occurs with circumferential bulging and loss of disc height. This in itself does not constitute disc protrusion or extrusion and is frequently seen on CT scan of asymptomatic individuals.

The apophyseal joints are true synovial joints. With wear and tear, these joints show typical signs of degenerative osteoarthritis. Any loss of disc height causes subluxation of the apophyseal joints and thus accelerates the development of osteoarthritis.

A second consequence of the loss of disc height is the development of relative laxity of the spinal ligaments. This causes instability between vertebral units and the formation of traction osteophytes at the vertebral body margins which may be the first radiological evidence of disease.

STAGES OF DEGENERATIVE DISEASE OF THE SPINE

1. **Stage of Dysfunction**
   The majority of low back strains fall into this category and pain is probably musculo-ligamentous in origin. The lesion is usually too subtle to be detected by objective tests (X ray, CT scan, etc.) and surgical treatment is generally not indicated.
2. **Stage of Instability**
   Physical signs and early X-ray changes begin to appear. These may include nerve root irritation, disc space narrowing, disc protrusion, traction osteophytes and evidence of osteoarthritic changes in the apophyseal joints. Bracing or muscle strengthening exercises may be useful. If instability is severe or nerve root compression does not resolve, *spinal fusion* or *discectomy* may be indicated.
3. **Stage of Stabilization**
   Spinal movement becomes restricted because of the formation of osteophytes, narrow disc spaces, and subluxation of apophyseal joints. Many patients experience spontaneous improvement of their symptoms. This may be as the result of "spontaneous spinal fusion" when adjacent osteophytes meet, bridging the disc space and thus reducing instability and pain. Entrapment of spinal nerves may still occur and decompressive surgery for spinal stenosis may be indicated.

CAUSES OF DEGENERATIVE DISEASE OF THE SPINE

It has been suggested that wear and tear, as well as functional strains, produce cracks in the annulus of the disc. Any force which suddenly or continually increases the pressure within the disc, may eventually cause the posterior fibres of the annulus to give away. The susceptibility to such forces increases in the presence of degenerative changes.

Factors believed to predispose to degeneration of the spine include:

1. Degenerative changes of the "three joint complex" due to *aging.*
2. Poor *posture.*
3. Repeated trauma of normal *daily living* and *recreational activities,* etc.
4. Acute or chronic repetitive trauma, including *occupational* factors such as repetitive or heavy lifting, carrying and bending; especially when done to the point of fatigue.
5. Epidemiologic studies reveal that the condition is more prevalent in *men* than in women.
6. Some epidemiologic studies suggest that the condition is more prevalent in *labourers* than in sedentary workers.
7. Back pain is more common in individuals with a *previous history* of back pain or surgery.
8. Some individuals develop degenerative changes at an earlier age and to a much greater extent. A *genetic* or inherited predisposition has been postulated.

SUMMARY

A simple explanation of the causes of degenerative spinal disease and its progression is not available. Many factors are involved and lead to the eventual need for treatment (surgical, medical, and rehabilitative).

Some of these causes are occupational and some are not. In Workers' Compensation claims for back disability, an administrative adjudicative decision must be made and it is very often based on the best available medical opinion.

Physicians are often asked to state whether an accident or incident under the claim has, as a matter of medical fact, caused or contributed causally to the pathology. As the causes of degenerative spinal disease are almost always multiple, the real problem which has to be faced is one of apportionment of responsibility.

However, in order to have a better understanding of how the various worker's compensation systems in North America presently view the problem of medical *causation,* it may be appropriate to quote Professor Arthur Larson, on the topic of Medical Causation:

> "It must be shown by evidence, opinion or otherwise, that the exertion attendant upon the duties of employment, no matter how slight or how strenuous, and no matter with what other factors (such as preexisting disease or predisposition to attack) it may be combined, was sufficient to contribute toward the precipitation of the attack. Where

evidence as to the work engaged in shows it to be sufficiently strenuous, or of such a nature that, combined with the other facts of the case, it raises a natural inference through human experience that it did so contribute, this is sufficient...

"In other cases, the opinion of experts that the exertion shown by the evidence to exist would be sufficient is also sufficient to authorize a finding on the part of the fact-finding tribunal that it did. But, in one way or another, the fact must appear."

Occupational health physicians should be aware of these facts in designing a cost effective preventive program or in dealing with workers' compensation.

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Specific

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* *Coordinator of Medical Services, Alberta Workers' Compensation Board,*

** *Director of Medical Services, Alberta Workers' Compensation Board and Clinical Assistant Professor, Department of Community Health Sciences, Faculty of Medicine, The University of Calgary*

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UPCOMING CONFERENCES

INTERNATIONAL INJURY PREVENTION CONGRESS

Melbourne, Australia, February 29 to March 4, 1988

Contact:
The Secretariat
I.I.P.C.
P.O. Box 29, Parkville
Victoria 3052, Australia

BACKS FOR THE FUTURE, 1988

Calgary, Alberta, March 16 to 18, 1988

Contact:
Janice Moore
Director of Education
Alberta Hospital Association
10025 - 108 Street
Edmonton, Alberta T5J 1K9
Telephone: (403) 423-1776

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UPCOMING CONFERENCES

RECENT ADVANCES IN OCCUPATIONAL CANCER

Department of Medicine, University of California, San Francisco Unit of American Cancer Society, Northern California Occupational Health Center, December 3 to 5, 1987.

Contact:
Labour Occupational Health Program
Institute of Industrial Relations
University of California
Berkeley, California 94720

Lela Morris (415) 642-5507
Lynn Duncan (415) 476-8474

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UPCOMING CONFERENCES

MANAGEMENT OF SAFETY AND HEALTH PROGRAMS

Northwest Center of Occupational Health and Safety, Department of Environmental Health, SC-34, December 7 to 8, 1987.

Contact:
Janice B. Schwert,
Program Manager
Northwest Centre for Occupational Health and Safety
Department of Environmental Health, SC-34
University of Washington
Seattle, Washington 98195
Telephone: (206) 543-1069

We invite your input!

As always, we encourage your comments on current topics, ideas for future content, and short articles from your own knowledge and experience. In addition, short announcements of interest to our readers will be inserted as space allows. Please contact the Editor (address on page 1) with any comments or contributions. In addition, we remind you that when articles state that "further references are available", these can also be obtained through the Editor.