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Alberta Occupational Medicine Newsletter

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1984

Alberta Occupational Medicine Newsletter: Winter 1983/84

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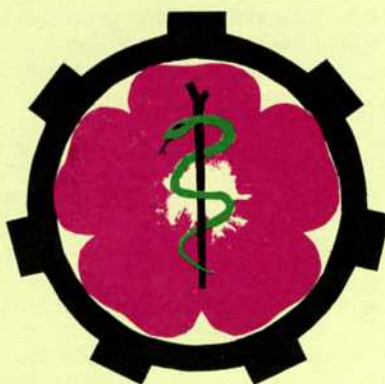
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Vol. 1 No. 4

Winter 1983/84

ALBERTA OCCUPATIONAL MEDICINE NEWSLETTER

EDITOR'S COMMENTS

The widening of occupational opportunities for women has focused some concern about the consequences for their reproductive health, which Dr. Heather Bryant has described and evaluated in our Autumn Newsletter. She balances the series by reviewing "Effects on the Male Worker" in this issue.

We include an overview by Dr. Kirk Barber on "Skin in the Workplace." Dr. Barber, a graduate of the University of Alberta, spent part of his dermatological training at St. John's Hospital in London, England; a well-known centre for diagnosis and research in occupational skin disease. This is of particular interest because occupational skin disease is common and also often preventable or curable by the removal of its man-made cause.

Dr. Tony Waldron, of the London School of Hygiene and Tropical Medicine, was quoted in the Fall Newsletter of the Alberta Heritage Foundation for Medical Research, on a recent visit to our province, as saying that, "Preventive medicine is taught very badly and given low priority in many medical schools." The article implied that this led to a shortage of occupational health researchers. Whether undergraduate medical education is the primary reason for this shortage in Alberta is highly debatable; convenient excuse though it is for the Heritage Foundation. It prompted your editor to present the educational objectives we have used in medical training at the University of Calgary. It is hoped that this will lead to further discussion, including input from the University of Alberta.

The editor of this newsletter will be Dr. W.M. Csokonay for the Spring and subsequent issues. Dr. Csokonay is a Resident in the Department of Community Health Sciences and has considerable experience in family medicine and also in occupational medicine.

John Markham

REPRODUCTIVE HAZARDS OF THE WORKPLACE PART 3 EFFECTS ON THE MALE WORKER

Heather Bryant, B.Sc., M.D., C.C.F.P.

The traditional focus of studies on workplace reproductive hazards has been, until recently at least, the female employee. Because the major issue explored in the past has been fetal risk due to *in utero* exposures, this emphasis seems at first to be only logical and natural. However, due to a series of anecdotal reports which led to formal studies of groups of workers, it has become apparent that the male employee is also at risk for a range of reproductive problems due to worksite toxins. In this article, some of the workplace substances believed to be associated with male reproductive impairment will be discussed. Through this, it will be seen that research in this area is in its infancy; few substances have been studied and the methodological problems are great. At a minimum, however, the information presented here should sensitize physicians to the fact that male reproductive problems can and do result from job-related exposures, and an occupational history should be taken as part of an initial workup of these complaints.

Lead

While lead had long been known to affect the female reproductive system, causing miscarriage and stillbirth, its effects on the male worker have only been studied more recently. In 1975, Lancranjan¹ reported on 150 men, all occupationally exposed to lead at varying levels. This study used semen analysis, as well as a questionnaire concerning sexual functioning to compare the different rates of problems. There was a higher frequency of "disorders of sexual dynamics" and loss

of libido amongst the workers exposed to lead through working in a storage battery plant as opposed to those working in offices and annex workrooms; however, when the plant workers were subdivided into three groups according to physiological criteria of lead absorption, no consistent relationships between these complaints and estimated lead exposure were found. The interpretation of this is somewhat difficult, as men with clerical and non-plant jobs may have had important differences in background or education which may have led them to respond to questioning in this sensitive area differently than from the plant workers. Because the questionnaire was not standardized or used with a control group, we cannot comment on whether the differences found were real or an artifact due to the sample of men studied.

Semen analyses were also done, and the proportion of men who demonstrated oligospermia or teratospermia (criteria for these definitions were not stated) was found to be significantly elevated in exposed workers compared with non-exposed.¹ However, historical evidence of infertility or loss of partners' pregnancies was not conclusive. Similarly, a different study of tetraethyl lead workers failed to demonstrate clinical subfertility.² Neither of these studies were designed in such a way that finding a significant difference was likely, however. In view of the spermatic abnormalities found, further studies on the clinical implications of these would be valuable.

Dibromochloropropane (DBCP)

Dibromochloropropane (DBCP) is a chemical pesticide, previously used widely on various types of crops throughout the world. The story of its discovery as a biological reproductive hazard is a particularly interesting one, as the initial suspicion of its effects came about as the result of casual worker conversations on diffic-

ulties many of the men in one DBCP manufacturing plant were having in fathering children.⁵ It was only after this had been known by the workers for some time that scientific study was undertaken. The results indicated increases in rates of oligospermia, lower median sperm counts, and impaired testicular function, with concomitantly elevated FSH levels, amongst exposed workers.^{4,5,6} An additional note was made of not only clinical infertility of individual males, but of a demonstrable statistically significant decrease in fertility of the worker population as a whole, when compared to national fertility standards.^{7,8}

This chemical is currently no longer distributed in Canada, and thus is unlikely to be of clinical significance to patients presenting to Alberta physicians. However, its investigation has provided several methodologic advances in the field of male occupational reproductive hazards, and it is perhaps the most diligently documented reproductive toxin to date. Those interested in further reading in this general area are directed to the referenced papers on DBCP as a worthwhile starting point.

Vinyl Chloride

Vinyl chloride is well-known as a hazardous agent, primarily due to its demonstrated association with hepatic angiosarcoma.⁹ Because of its documented mutagenicity, some researchers have investigated its potential impact on human reproduction by studying cohorts of exposed and unexposed rubber workers.^{9,10} Questionnaires were administered to male workers regarding their wives' reproductive histories; the women themselves were not interviewed, and data on such important factors as the women's age, medical history, and alcohol and drug history were not elicited. The data demonstrated no significant difference between the exposed group and controls in rate of fetal loss in pregnancies occurring before vinyl chloride monomer exposure. However, a significantly elevated rate of loss occurred amongst the same women after their husbands were exposed to vinyl chloride (15.8% loss in "exposed" vs. 8.8% loss in "controls").¹⁰

This study has been criticized primarily because it did not interview the most reliable subjects, i.e., the women themselves, and also for its failure to control for many other variables that could affect spontaneous abortion. However, it is difficult to explain why the bias would result in over-reporting of fetal loss in recent pregnancies only in the "exposed" group. The possible mechanism of such an effect has been attributed to possible male germ cell damage, or exposure of the mother/fetus early in pregnancy to chemicals brought home on the workclothes¹¹ or transmitted post-conceptually in the semen.¹² While the "final answer" on vinyl chloride monomer and spontaneous abortion is perhaps not available, the indica-

tion that male reproductive damage may extend to fetal loss due to post-conceptual exposures is one to be borne in mind.

Waste Anesthetic Gases

The data for the effect of waste anesthetic gases on women was reviewed in the last issue of this newsletter. Similar mailed questionnaire surveys on male dentists and anesthesiologists have also been performed; much of the data were combined and summarized in a single article.¹³ The combined results showed no clinically or statistically significant increase in miscarriage rate for wives of males exposed to anesthetics. There was noted an increase in the rate of congenital abnormalities (excluding skin abnormalities) from 3.7 to 5.0 percent when wives of controls and exposed males were compared. Because these data are from self-reports, it is difficult to validate the "diagnoses" of malformations; there was, interestingly, no increase in any particular type of abnormality, which would make one more convinced of evidence of specific toxic effect. Further monitoring studies, to delineate and validate malformation patterns, if present, and to attempt to identify a particular hazardous agent, have been recommended.¹³

Other Agents

Several scattered reports of male occupational reproductive hazard have been made. Diethylstilbestrol manufacture was related to a high rate of gynecomastia and loss of libido in one study, although no control group was used.¹⁴ Manganese, inorganic mercury¹⁵, and chlordecone (kepone)¹⁶ exposures intense enough to cause severe generalized toxic symptoms also have been reported to be linked to sexual dysfunction and/or oligospermia. It is difficult to extrapolate these anecdotal reports to an assessment of reproductive risks at usual low but chronic occupational exposure levels.

Conclusion

It is apparent from the information above that there is much to be done in this area of research. Many of the study techniques require refinement and validation, and there is a need to establish monitoring techniques to identify potential hazards early, rather than waiting for effects to become so widespread that toxicity is obvious before studies are initiated.

Despite these cautions, however, it is evident that occupational exposures may result in male reproductive impairment. Thus, when complaints regarding infertility or sexual dysfunction are made, it would be worthwhile to include an occupational history as part of the data base.

Because research in this area is progressing rapidly, it is to be expected that most practising physicians will not have at their fingertips the most recent information on the growing list of toxins. There are a number of resources that can be turned

to, however, if further information on particular workplace exposures or toxins is wanted. Mutagenicity, animal, and human reproductive studies on industrial chemicals are now summed up in such review volumes as that of Barlow and Sullivan.¹⁵ In addition, library searches and services available to physicians through the Canadian Centre for Occupational Health and Safety or the Alberta Occupational Health and Safety Library (Librarian: Mr. Keith McLaughlin, telephone 427-3530) may locate information difficult to find in non-occupational libraries. Information so obtained may be used to help individual patients, although more often than not, no direct evidence that the problem is related to workplace exposures is likely to be uncovered. In addition, the use of such resources is a worthwhile practical support to physicians attempting to provide reasonable advice in what has been shown, in these articles, to be a complex and rapidly advancing area of research.

REFERENCES

1. Lancranjan I, Popescu HI, Gavanescu O, Klepsch I, and Serbanescu M. Reproductive ability of workmen occupationally exposed to lead. *Arch. Environ Health* 1975;30:396-401.
2. Robinson TR. The health of long service tetraethyl lead workers. *JOM* 1976;18(1):31-40.
3. Chenier NM. Reproductive hazards at work: men, women, and the fertility gamble. *Can Adv Council on Status of Women* 1982.
4. Whorton D, Krauss RM, Marshall S, and Milby TH. Infertility in male pesticide workers. *Lancet* 1977;2:1259-1261.
5. Whorton D, Milby TH, Krauss RM, and Stubbs MD. Testicular function in DBCP exposed pesticide workers. *JOM* 1979;21:161-166.
6. Glass RI, Lyness RN, Mengle DC, Powell KE, and Kahn E. Sperm count depression in pesticide applicators exposed to DBCP. *Am J Epid* 1979;109:346-356.
7. Levine RJ, Symons MJ, Balogh SA, Milby TH, and Whorton MD. A method for monitoring the fertility of workers. Validation of the method among workers exposed to dibromochloropropane. *JOM* 1981;23:183-188.
8. Levine RJ, Blunden PB, Dal Corso RD, Starr TB, and Ross CE. Superiority of reproductive histories to sperm counts in detecting infertility at a dibromochloropropane manufacturing plant. *JOM* 1983;25:591-597.
9. Infante PF, Wagoner JK, and Waxweiler RJ. Carcinogenic, mutagenic and teratogenic risks associated with vinyl chloride. *Mutat Res* 1976;41:131-142.
10. Infante PF, Wagoner JK, McMichael AJ, Waxweiler RJ, and Falk J. Genetic risks of vinyl chloride. *Lancet* 1976;1:734-735.
11. Strobino BR, Kline J, and Stein Z. Chemical and physical exposures of par-

ents: Effects on human reproduction and offspring. *J Early Hum Dev* 1978; 1(4):371-399.

12. Pries CN Reproductive effects of occupational exposures. *Am Fam Phys* 1981;24:161-165.
13. Spence AA, Cohan EN, Brown BW, Knill-Jones RP, Himmelberger DU. Occupational hazards for operating room based physicians: Analysis of the data from the United States and United Kingdom. *JAMA* 1980;238: 955-959.
14. Harrington JM, Rivera RO, and Lowry RK. Occupational exposure to synthetic estrogens — the need to establish safety standard. *Am Ind Hyg Assoc J* 1978;39:139-143.
15. Barlow SM, and Sullivan FM. Reproductive hazards of occupational chemicals. Acad Press London 1982.
16. Cohn WJ, Boylan JJ, Blanke RV, Fariss MW, Howell JR, and Guzelian PA. Treatment of chlordecone (kepone) toxicity with cholestyramine. *NEJM* 1978;298(5):243-248.

SKIN IN THE WORKPLACE

Kirk A. Barber, M.D., F.R.C.P.(C).

Occupational dermatoses represent any abnormality of the skin produced or aggravated by the working environment.

A 1977 survey of occupational skin disease in California documented skin disease (excluding thermal and chemical burns) as the most frequently reported of all occupational illnesses. Skin diseases accounted for 40 percent of 43,888 occupational illness reports by physicians with 2.1 physician reports of skin conditions per 1000 workers. Reports from the United States as a whole revealed similar statistics; 45 percent of all lost workday and non-lost workday cases in the private sector were due to skin diseases and disorders.

The most common occupationally related skin conditions as presented in the California report are:

CONDITION	% AFFECTED
CONTACT DERMATITIS, IRRITANT OR ALLERGIC	92.2
INFECTIONS, PRIMARY OR SECONDARY	5.4
OTHER (acne, folliculitis, pigmentary changes, etc.)	2.4

Contact eczema (dermatitis) accounts for the overwhelming majority of skin conditions. I will therefore devote this presentation to a discussion of this topic.

The term eczema and dermatitis will be used interchangeably.

Eczema is derived from a Greek term meaning to "boil over". It is a clinical diagnosis used to describe a group of morphological changes in the skin. Acute eczemas exhibit erythema, edema, and

vesiculation; chronic eczemas proceed to scaling, lichenification (thickening), and fissuring. Subacute eczemas combine the features of both. The second concept necessary to understand eczemas is that of endogenous versus exogenous eczema. Atopic eczema is an inherited form of eczema and is endogenous in origin, and contact eczema is obviously exogenous in origin. On a morphological basis there two may present identical appearances on the skin.

An irritant contact dermatitis is the result of direct destructive action of any substance, usually a chemical (e.g., acid, alkali, or solvent), on the barrier function of the skin through a non-immunological mechanism. The stratum corneum is a 1.6 mm semi-permeable membrane which retains our body water and provides protection from the environment. The cells of the stratum corneum are "cemented" together by a lipid-based substance and keratin is the major intracellular substance in these cells. They provide a remarkable barrier to chemical and physical insults. Irritants of the skin damage either the keratin or the cement substance producing an eczema.

Irritants of the skin may be graded from strong to weak. Strong irritants are usually corrosive substances (such as strong alkalis and acids) that rapidly injure anyone's skin immediately following contact. Weak irritants may cause irritation in only a small number of individuals; however, given the proper conditions every person exposed will develop a reaction. Soaps, detergents, and organic solvents are examples of weak irritants. Treatment of an irritant contact dermatitis is based on providing excellent protection of the skin from the irritant, removing the inflammation, and rehydrating the stratum corneum. Protective clothing, topical corticosteroids and emollients are the mainstays of therapy.

Many workers note an adaptive phenomenon called "hardening". After daily repeated contact with an irritant, the skin becomes more resistant to the effects of the irritant. This shows on the worker's skin as a localized thickening, scaling and hyperpigmentation. The mechanism is unknown.

Twenty percent of cases of contact dermatitis will be allergic in origin. This is the result of a Type IV delayed hypersensitivity reaction to the allergen producing the features of eczema on the skin. Contact allergens produce sensitization in only a small number of individuals exposed. The most common allergens include nickel, potassium dichromate, epoxy resins, and formaldehyde, each of which is found in many workplaces.

The significance of a diagnosis of allergic contact dermatitis should not be underestimated as the protective and therapeutic measures used to treat eczemas in

general are usually ineffective for such a case. Affected workers experience prolonged periods of disease and of unemployment, and often find it necessary to change jobs or to learn a new trade. The costs to society and the individual are obvious.

The clinical appearance of eczema does not provide many clues to help differentiate between an irritant contact dermatitis and an allergic one. The affected sites are those exposed to the chemical (commonly the hands and face) and the rashes may be identical. The diagnostic tools available to the occupational physician are:

- (1) the proper clinical diagnosis
- (2) cutaneous patch testing
- (3) a survey of the patient's workplace.

Patch testing is diagnostic in allergic contact dermatitis but of little help in irritant contact dermatitis. Cutaneous patch testing requires the allergens to be of a concentration known not to be an irritant and dispersed in a standard vehicle (e.g. petrolatum). These allergens are applied to the back for 48 hours under occlusion provided by small aluminum chambers. If a person is allergic to a substance they will develop the features of an eczema in the area of application.

The technique is simple and the information provided is valuable, however, it requires standardization and expertise in interpretation.

The worker's assessment often requires a direct inspection of the workplace and viewing the worker performing his job. This is helpful in applying relevance to the results of the patch testing and for seeking other possible causes. The practicality of treatment can also be evaluated. There is no quicker way to lose a worker's confidence than to suggest protective measures that are impractical or perhaps even dangerous.

Each occupation can be rated according to its relative risk to the worker. Of equal importance is the fact that there are high risk workers for certain occupations. These high-risk workers are most commonly those workers with either a previous contact allergic sensitivity or those with previous skin disease.

Contact allergy to nickel is the most common allergen in females affecting approximately 10% of all women. This allergy may limit a person's ability to handle nickel-plated hand tools. Table I outlines other high risk occupations for such people.

TABLE I
High Risk Occupations for Nickel

Hairdresser
Nickel electroplating
Jewellers
Tailors
Dental Assistant

Atopic eczema is an endogenous eczema which makes a person a high-risk worker for certain occupations. These occupations are usually those involving frequent prolonged exposure to soaps, detergents, solvents and wet work. Examples of low risk occupations for patients with atopic eczema are outlined in Table II. As you will note, these are mostly "dry" jobs.

TABLE II
Low Risk Occupations With Atopic Eczema

Computer Assistant
Security Guard
Typist/Receptionist
Teacher
Policeman

Psoriasis and chronic hand eczema are also skin diseases that place an individual in a high risk category for certain occupations.

It is well recognized that the extent of occupational skin diseases is now under reported. The Report of the Advisory Committee on Cutaneous Hazards to the Assistant Secretary of Labour in the United States contains the opinion that the true incidence may be as much as 10 to 50 times the numbers of reported cases.

We have now established an Occupational Dermatology Clinic at The University of Calgary. The primary purpose of our clinic will be to provide a province-wide referral center for physicians treating individuals with an occupational skin disease. It is our aim to use the experience and information gained in this clinic to improve our treatment and the knowledge of environmental and occupationally related skin conditions present in our province's work places.

References available upon request.

EDUCATIONAL OBJECTIVES IN OCCUPATIONAL MEDICINE FOR THE MEDICAL UNDERGRADUATE/RESIDENT CONTINUUM

John Markham, F.R.C.P.(C), C.C.B.O.M.

The way in which occupational medicine is best taught varies according to the format of the curriculum and the available teachers. Some will incorporate it in general systems and clinical teaching, as for example Dr. Alex Herbert of the University of Alberta does with respiratory medicine. Some will be taught before graduation and some during residency training. At the University of Calgary, we have given about 16 hours in the course on "The Family" (chairman: Dr. Michael Tarrant), starting with the occupational health of physicians and going on to the health protection of their patients in relation to their jobs. Because of the necessary flexibility in method, this brief article will focus on educational objectives. Although the

training of physicians has important attitudinal components, these are difficult to define and even harder to evaluate. Therefore, the objectives listed will be limited to those involving knowledge and skill. They are designed for future clinicians.

1. Job-Related Patient Management

The student should be able to:

- a) identify and analyse those relationships in employing organizations which affect the rehabilitation of the employees.
- b) explain the financial implications of job changes associated with ill health.
- c) describe the ethical problems liable to occur for a family physician in communicating with employers.
- d) describe how to relate to occupational health workers in this respect.
- e) explain the harmful effects of "sheltering" disabled employees in contrast to the beneficial effects of providing modified but productive work.
- f) explain how Workers' Compensation operates and the implications for the management of patients and the provision of the necessary professional opinions.
- g) explain the social security and other benefits to which work people are entitled.

2. Protection of the Patient as a Worker

The student should be able to:

- a) discuss the principles of protecting a patient from known hazards by the investigation of his working environment and/or providing him with protective equipment.
- b) explain the limitations of personal protective equipment in workplaces.

3. Occupational Health Conditions

The student should be able to:

- a) describe the signs, symptoms and pathology of the more common and/or important occupational diseases and injuries.
- b) know where further information can be obtained.
- c) explain to his/her patient what action should be taken concerning prevention of the effects of (for example):
 - i) some dusts (silica, asbestos)
 - ii) solvents (benzene, carbon tetrachloride)
 - iii) pesticides (organic phosphorus compounds)
 - iv) heavy metals (lead and mercury)
 - v) other metals (cadmium, zinc)
 - vi) gassing (H₂S, CO)
 - vii) radiations (including noise)
 - viii) biological hazards (Hepatitis B, mouldy hay)
 - ix) psychological and lifestyle hazards.

4. Occupational Epidemiology

The student should be able to:

- a) analyse the determinants, distribution and frequency of health and disease in relation to selected occupations.
- b) discuss how the body's organ systems may be damaged by exposure at work to certain examples of chemical, physical and biological agents.
- c) explain how work may be etiologically important in common illnesses like coronary heart disease, chronic bronchitis, and anxiety and depressive states; and conversely to explain how work may have a therapeutic value.

5. Occupational History Taking

The student should be able to:

- a) take an occupational history.
- b) discuss its importance in the diagnosis and management of occupational and non-occupational illnesses.

6. Occupational Health Services

The student should be able to:

- a) describe the aims of occupational health services in places of employment and analyse their relationship to other health services.
- b) discuss the financial and social constraints under which such services sometimes operate and the conflicts of interest with which they may have to grapple.
- c) know how to attempt to work constructively with such services.

These objectives were developed over 15 years of teaching at the Universities of Saskatchewan and Calgary. They owe much to my old teacher, Professor R.S.F. Schilling, Professor Emeritus of Occupational Health at the London School of Hygiene and Tropical Medicine. Professor Jim Spooner, Medical Educator at the College of Medicine, University of Saskatchewan, and Dr. Elizabeth Kaegi, Medical Director of C.I.L. Paints, also made valuable contributions.

COMING EVENTS — ALBERTA

May 1-3, 1984

Canadian Radiation Protection Association, Annual Conference; Banff, Alberta, Canada.

COMING EVENTS — ELSEWHERE

May 7-9, 1984

International Conference on Occupational Ergonomics; Toronto, Ontario, Canada.

July 12-13, 1984

Second Annual Conference on Occupational Hazards to Health Care Workers, University of Washington, Seattle, Washington, U.S.A.

September 9-14, 1984

XXI International Congress on Occupational Health, Dublin, Ireland.