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GUEST EDITORIAL

Sexually Transmitted Diseases in Nova Scotia

WHAT CAN WE DO BETTER?

Donald B. Langille,* MD, MHSc

Guest Editor

The Community Health Committee of the Medical Society of Nova Scotia is pleased to have been able to bring together individuals from a number of health care disciplines in order to produce this issue of the *Journal* devoted to sexually transmitted diseases. The problem of STDs crosses the boundaries of many disciplines – clinical, laboratory, and community health – involves most often the young, and far too often leads to the loss of one of the most cherished aspects of life – the ability to have children. This reality is clearly brought home by both Arthur Zilbert's article describing treatment of delayed or inadequate treatment of pelvic infection[1] and that by Lynn McIntyre on prevention of infertility.[2]

The message brought to us by Forward *et al* should also be heeded.[3] It is clear that there is a lot of testing for disease due to *C. trachomatis* that is unlikely to be found where it is being sought. When the resources expended are compared with the benefit realized, and both are reviewed in the light of the real possibility of false positive tests, physicians should be seriously considering their testing habits in regard to diagnosis of this infection.

In looking at how best to deal with STDs, we need to ask ourselves more than how we are doing in diagnosing STDs accurately and treating them appropriately. As Heather Young indicates so clearly, we must also ask ourselves other questions, including ones about our level of proactive involvement at both the individual and community levels.[4] Are we ourselves comfortable in talking with patients about this most intimate of subjects? Are we taking the initiative in providing preventive messages to young patients? Are we involved with school boards and other organizations with a part to play in preventive efforts? Are we dealing well with these issues with our own children?

We should also ask ourselves how we are doing in making certain that sexual partners are advised of possible infection, tested and treated. There is evidence, at least in some parts of Nova Scotia, that

* Member, Community Health Committee.

the usual method of asking patients to notify partners is not reliable, and that public health resources are seldom used in attempts at such notification.[5] We should be very emphatic with patients that partner notification take place and, where we are at all uncertain, be quick to utilize public health professionals. To do this it will also be necessary to build good working relationships with public health. This can only be done where both physician and public health practitioner are willing to recognize what each has to offer, and work together to address what is, and will no doubt continue to be, a major health problem for Nova Scotians.

The Community Health Committee hopes that this issue of the *Journal* will serve to remind physicians of the importance of STDs, and that it will help to stimulate thought and concern about how we as a profession can both prevent and treat them. □

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Contact Tracing for Control of Sexually Transmitted Diseases

Donald B. Langille,* MD, MHSc

Halifax, N.S.

In Nova Scotia more than 2000 cases of genital *Chlamydia trachomatis* infection and almost 300 cases of STD due to *Neisseria gonorrhoeae* were reported in 1991.[1] Chlamydial infection, which is currently much more common than gonorrhoea, is the STD with the highest rate of preventable complications.[2]

Reports of infection due to *C. trachomatis* in Nova Scotia have increased dramatically since the early 1980s, when diagnostic testing become more available and more became known about the organism and the consequences of infection with it. Reported cases have been reasonably stable since 1987. There has been probably at least some role played by surveillance bias associated with the increase in numbers of reported cases *C. trachomatis*. On the other hand, reported cases of STD due to *N. gonorrhoeae* are dropping in Nova Scotia, where numbers of cases have dropped from 952 in 1986 to 294 in 1991. There is no clear explanation for this trend, although it conceivably could be due in part to more effective partner notification and treatment.[3]

STDs, particularly those due to *C. trachomatis*, in addition to being common, are felt to contribute substantially to PID, subsequent infertility, and ectopic pregnancy, resulting in much preventable morbidity, mortality and health care resource expenditure.[4-6]

Contract tracing for STD control has for many years been a mainstay of North American public health practice.[7] The objectives of contact tracing may be summarized as:

1) To inform contacts of their risk;

2) To educate contacts about disease transmission and symptomatology; and

3) To ensure that contacts are appropriately managed.

Contacts should, whenever possible, be interviewed face-to-face. It is imperative that the person sought should be correctly identified, that the seriousness and the importance of the situation be stressed, and that confidentiality be maintained.

The questions considered in this review are: a) What is the evidence to support various approaches to contact tracing for STD prevention? and b) How can Nova Scotia physicians best participate in such contact tracing efforts?

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CONTACT TRACING FOR CHLAMYDIAL INFECTION

There is some disagreement regarding appropriate contact follow-up for genital infections with *C. trachomatis*. The Centers for Disease Control recommend the "contract" method of sex partner referral with the patient assuming referral responsibility whenever possible.[8] It is recommended that those with chlamydial infections encourage self referral in all partners from the 30 days prior to onset of symptoms or clinical evaluation. This option is recommended only if the patient is interested, responsible and is able to effectively notify partners.[9] With chlamydial infection (and also with gonorrhoea), high rates of infection in contacts and the fact that many of those who are infected are asymptomatic are seen as major reasons for contact tracing efforts.[10]

The position of Health and Welfare Canada concerning contact tracing for chlamydial infection appears to leave the responsibility with physicians, but allows some role for public health efforts, stating that "Physicians have a responsibility to their patients to ensure that sexual partners are examined and treated. Public Health authorities may undertake the task if requested."[2]

Evidence does exist for the effectiveness of field follow-up of chlamydial infections. Of 678 men with non-gonococcal urethritis, over three times as many partners of infected individuals appeared for evaluation following active field follow-up as compared to two different methods of self-referral. Rates of infection with *C. trachomatis* in those who were evaluated in the three groups did not differ significantly, though significantly higher absolute numbers of cases were detected through the field follow-up method. The chief limitation of the study was an inability to ascertain whether or not those referred by the "informal system" had received treatment at other centers.[11] A cost analysis indicated that field training was the most efficient of the methods used. Data from Quebec indicate that physicians as a group do not do as well as specialized contact tracing services in identifying asymptomatic carriers of *C. trachomatis* who are contacts of those diagnosed with this infection.[12]

CONTACT TRACING FOR GONOCOCCAL INFECTION

Active tracing of female contacts of males with gonorrhoea, in order to provide them with epidemiologic treatment, has been the policy of the Centers for Disease Control since 1950. This policy was modified in the 1970s to include male contacts of culture positive females.[13]

For gonococcal infections, arguments have been made for replacing the relatively expensive and time consuming use of trained investigators with systems using "contact cards" handed out by patients to sexual partners, informing them of possible infection and how to seek help. In one study comparing such a system with field follow-up by trained investigators, similar numbers of male and female contacts reported for treatment in each study group, and no differences in rates of gonorrhoea infection in contacts were found.[14] These findings were comparable to those of Potterat and Rothenberg who replaced a 15 to 20 minute investigative interview followed by a "contract" follow-up arrangement with a self referral card system to bring infected partners of heterosexual males to treatment.[15]

Rates of infection with gonorrhoea in contacts of cases in various centers are cited as ranging from 10 to 25% in men and 53 to 66% in women.[14] There is some evidence to suggest that for asymptomatic men as well as women, active public health intervention is required to identify their infected contacts.[16]

Despite the continuing debate and paucity of research concerning the usefulness of various types of contact tracing, there are situations in which intensive effort should definitely be applied. These would include in the case of gonorrhoea the introduction of PPNG infection into a previously unaffected community, all PPNG infections where control of these infections remains feasible, men and women with repeated STDs, and all STDs in children.[9]

In summary, sexually transmitted diseases are common and account for large proportions of significant illnesses including PID, infertility, and ectopic pregnancy. Large numbers of individuals who are contacts are also infected with *C. trachomatis* and *N. gonorrhoeae*, and many of these are asymptomatic. There is however, debate concerning approaches to contact tracing. Most authorities agree that some form of contact tracing should be carried out and that it should be more intensive under specific circumstances. It is also recognized that the success of contact tracing programs is dependent upon the extent to which contacts come forward for treatment, and that there is often a limited degree of success.[17]

CONTACT TRACING IN NOVA SCOTIA

Sexually transmitted diseases are felt to be both common and often asymptomatic in rural communities in Canada.[18] In Nova Scotia, where many individuals reside in rural areas and small towns, most STD follow-up is carried out by primary care physicians without the use of public health assistance. This usually takes the form of the physician explaining to the patient the need for notification, and leaving the responsibility to that patient. There is some evidence that such informal methods are less than ideally effective, with approximately one-third of contacts not being notified, even though this is not usually due to logistical difficulties.[19] Contact tracing efforts by health unit staff vary considerably across the province, and reports of STDs are rarely made to health units directly from physicians (personal communication, Dr. J. LeBlanc).

IMPROVING CONTACT TRACING IN NOVA SCOTIA

How can contact tracing objectives best be met in Nova Scotia? This clearly depends upon the options, their costs and effectiveness, and the degree of comfort that those trying to make such a system work would have with each.

Contact tracing may be divided into *formal* tracing and *simplified* tracing.[20] In the former, trained staff interview patients, obtain names and addresses of sexual partners, locate such partners, and offer examination and treatment. The major objective of formal contact tracing is to identify the source of the patient's infection, though during the process others to whom the disease may have been spread will also be identified. The advantages of such as system include comprehensiveness, an ability to reach contacts unlikely to seek care without encouragement, and increased ability for evaluation to take place. The major disadvantage is cost, which inevitably means the diversion of resources from other activities, particularly education, and also specialized facilities such as STD clinics.

Simplified contact tracing refers to the situation where patients inform partners of possible infection in an attempt to ensure their examination and treatment without revealing their identities to physicians. This may also be referred to as partner notification. As indicated, this is the approach generally used in Nova Scotia today. Such a system is inexpensive and acceptable to patients. However, there are major disadvantages, which include difficulty with evaluation, and low success rates with unreliable patients.

What can be done to improve contact tracing and partner notification in our province? It can fairly be assumed that the responsibilities of primary care physicians for ensuring that sexual partners receive appropriate advice and treatment will not be lessened, and that the informal system will be maintained for some time. Physicians can, however, do several things which will help to ensure that this responsibility is carried out as effectively as possible.[21] These include:

1) Knowing the epidemiology of STD in their region of practice to more effectively target those at risk for STDs so that the diagnosis can be made and partners notified. Some physicians will, due to the nature of their practices, see more STDs than others. However, even in "low-risk" populations, "high-risk" individuals may be identified.

2) Educating patients, particularly younger patients, about STDs as part of routine care. Most STDs occur in those aged 15 to 24 years, who may be provided with information aimed at preventing STD's during any office visit, when the physician feels this would be appropriate.

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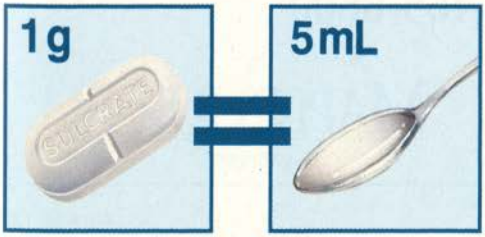

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3) Thoroughly educating patients with STDs about the necessity to notify contacts, and follow-up on their efforts to do so.

4) Building good working relationships with local health units so that where difficulties are anticipated or encountered with partner notification, Department of Health staff can become involved in reaching those who otherwise may go unaware of their infection, and the risk they pose to others. This resource is under-utilized currently.

This last point is an essential one if we are to ensure that partners of those infected are reached. Those who are difficult to reach are also those likely to belong to the "core" groups of carriers of STD organisms felt to be responsible for their continued spread.[22] It is only through the combined efforts of physicians and the public health system that effective prevention of sexually transmitted diseases can take place. □

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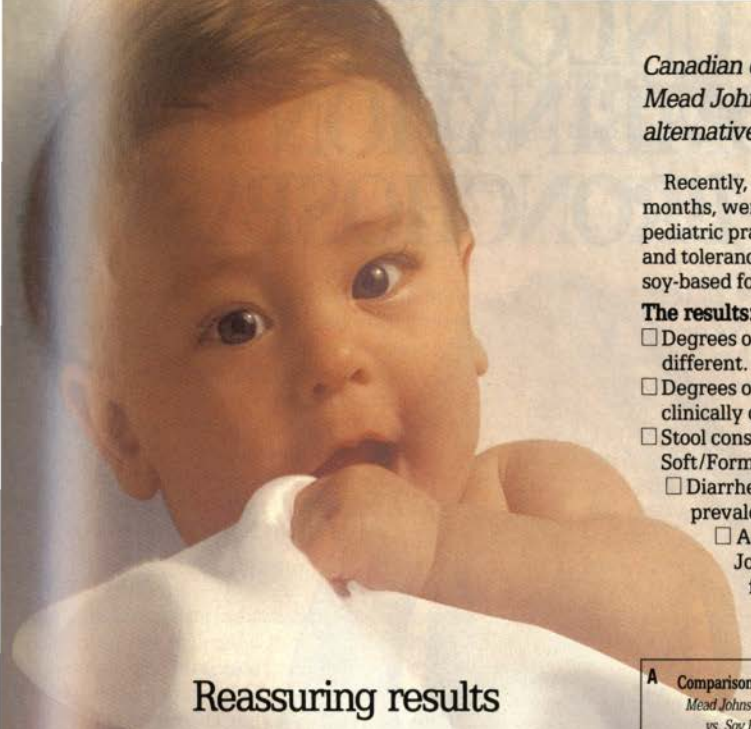

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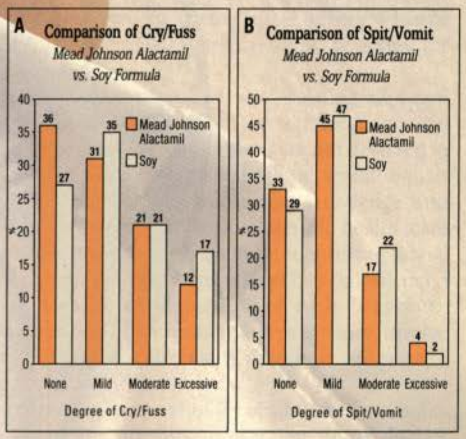

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A PUBLIC HEALTH CHALLENGE

Heather J. Young,* RN, MScN

Halifax, N.S.

Canadian teenagers are sexually active but generally do not use contraceptives reliably during their teenage years.[1,2] These unsafe sexual practices have a high probability of resulting in pregnancy and/or a sexually transmitted disease. Both of these problems are amenable to primary prevention with public health interventions. An overview of the literature which discusses the psychology of sexual risk behaviours, knowledge, attitudes and intervention experiences has provided the foundation for the development of a Sexual Health Program in Nova Scotia.

PREGNANCY

Nova Scotia data indicate that the pregnancy rate and abortion rate decreased from 1980 to 1988 in 15 to 19 year olds, and increased from 1988 to 1989. In Canada, the pregnancy rate for 15 to 19 year olds in 1987 was 38.8/1000. In Nova Scotia, the pregnancy rate for the same age group in 1987 was 40.2, and in 1989 was 46.7.[3] In the *Canada Youth and AIDS Study* (1988) it was found that sexual activity increases with age - 39% of grade 9 students, 56% of grade 11, and 80% of those more than age 20 have had intercourse at least once.

For unintended teenage pregnancy the resulting problems are many and can include parenting challenges, interrupted education and low level of educational attainment, which in turn contribute to the literacy and job problems facing our society. Low birth weight babies and welfare dependence both contribute to the high cost of the health care and social services systems.[4]

SEXUALLY TRANSMITTED DISEASES

Sexually transmitted diseases and their sequelae also have profound affects on our communities. Canadian gonorrhoea rates from 1986 indicate that women 15 to 19 are at second highest risk, and that this rate is increasing.[5] More than 70% of reported cases of infection with *Chlamydia trachomatis* occur in those aged less than 25 years.[6] Sequelae of STDs have serious consequences that can include involuntary infertility, tubal pregnancy and chronic pelvic pain.

The costs related to STDs for teens and society are great. These include the costs of screening and direct care, educational programs, research and cost of consequences that include future new reproductive technologies.

TEEN SEXUAL RISK-TAKING BEHAVIOURS

Nova Scotia teens are generally well informed about contraception and pregnancy, but this knowledge is not being translated into behaviour change.[1] Approximately 31% of sexually active females attending Nova Scotia high schools consistently do not use contraceptives.[7] In comparison with AIDS education, teens are less informed and less concerned about STDs.[8,9] Knowledge of prevention with condoms is generally high, but again, this knowledge does not always lead to preventive behaviours, as use of condoms by teens only ranges from 40% to 50%.[10] The main sources providing education to teens on these topics are schools, health care professionals and family. The informal systems providing such information include peers and media. These sources provide valuable information with regard to who requires targeting for education programs.

While knowledge is evident, the alarming data and negative sequelae of unintended teenage pregnancy and STDs indicate that Nova Scotia teens are not deterred in continuing to practice high risk-taking sexual behaviours. The challenge of transforming knowledge into behaviour change is one with which all health care professionals must be concerned. In identifying sexual risk-taking behaviours in teens, it must be acknowledged that the negative consequences of such behaviour, whether they be STD or unintended pregnancy, involve similar behavioural courses and, therefore, will require similar preventive interventions.[11]

Understanding the psychology of adolescent sexuality is a necessity for planning prevention. If we do not understand teen behaviour, we will continue in ways that obviously have not been effective. Teens in general, perceive themselves as "the 3 I's" - Immune, Infertile and Infallible - in whatever behaviours they undertake. Fisher identifies the problem as one which relates to the need for "information, motivation and behavioural skills" which will then lead to safer adolescent sexual behaviour and sexual health practice.[12]

REASONS FOR SEXUAL RISK-TAKING

Some factors that have been associated with early adolescent sexual activity and lack of contraceptive use (for both pregnancy and STDs) include low self-esteem, low sexual self-concept, drug use, lack of parental support and strong peer and social pressures.[13] As mentioned above, approximately 31% of Nova Scotia young women who are in high school indicated that they did not consistently use contraceptives. Those students who were sexually active but did not use contraceptives stated

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reasons such as "it would never happen to me".[7] The other most common reason for not using contraceptives was that they (teens) "did not expect to have sex".[13] Fisher, attributes these high risk behaviours to structural and psychological barriers, the first barrier being that teens do not accept that they are sexual beings. Teens need to acknowledge their own sexuality before they can plan for prevention. Parents and health care professionals must also accept and acknowledge these facts if they intend to engage in primary prevention strategies. The next barrier is for teens to recognize signs that sexual intimacy is approaching and that decisions must be made. It is only the acceptance of these factors that will then lead them to learning relevant preventative information such as pre-sex discussion/negotiation of prevention and consistency in practising prevention. This in turn requires a shift to alternative behaviours and practices.[11] If the reasons for risk-taking behaviours can be identified, then it is feasible to plan for reducing the risk.

REDUCING THE RISK

A solution to teen risk-taking postulated by Fisher involves changing the above components, information, motivation and behavioural skills.[12] It is therefore of benefit to review which educational approaches have been used over the years in an effort to prevent pregnancy and STDs. In the mid 80s efforts to reduce the numbers of young people at risk for pregnancy and STDs increased when concern for HIV disease began to grow. The first set of sex(uality) education curricula concentrated on increasing teens' knowledge about sexuality and emphasising the risk and the consequences of pregnancy. With this knowledge it was thought that teens would choose to avoid unprotected intercourse.[14]

The next curriculum moved beyond information on "the plumbing" to more information on values clarification, decision-making and communication skills. The intent of this approach was that if teens' values became clearer and their decision-making skills were improved they would be more likely to avoid risk-taking behaviours. If communication skills were improved it was thought that teens could communicate their decisions more effectively to their partners.[14] However, teaching of the specific skills that would really enable teens to avoid unprotected intercourse and practice safe sex was not provided. When evaluated, these curricula did find an increase in knowledge, but generally not behaviour change. Although knowledge has been identified as a foundation for behaviour change, knowledge alone has not been strongly related to reducing risk-taking behaviours.[1,15]

The current curriculum of sexual health has merged the lessons learned from past approaches and the need to incorporate new information. It is suggested that teens not only need information but that they need to acquire and practice skills with which to apply the information. As well, the motivating factors required by teens to put all this into practice need to be identified. These changes may be quite challenging for sexuality educators when they are planning prevention strategies.

PUBLIC HEALTH INTERVENTIONS

The Nova Scotia Department of Health is attempting to address the public health challenge of unintended teenage pregnancy and infection with STDs through the Sexuality Education Program. The focus of this program is not only to contribute toward reducing the incidence of unintended teenage pregnancy and STDs, but also to contribute toward the positive effects of increased self-esteem, and family and peer relationships. The Sexuality Education Program is carried out by Sexuality Education Resource Nurses (SERNS) in each of the six Public Health Units across the province. These Public Health Nurses (16 in total) have increased their knowledge and skills in this specialized area of practice involving sexual behaviour, sexual orientation, information and skill needs as well as issues such as assault and sexual acting out. Such professionals also need to identify "user friendly" persons to whom teens can be referred. These persons include general practitioners and counsellors.[12] Presently, SERNs provide sexuality education to parent and teen groups in a variety of settings. The foundation of the education programs provided is based on the Fisher model of changed information, motivation and behaviour skills.

Education can be very effective but it is acknowledged that it is only one part of a comprehensive sexuality education program which also should include counselling and clinical services. At the present time the Department of Health promotes adolescent sexual health through educational strategies alone. To ensure other components of a comprehensive program are available, the establishment of partnerships in communities has been essential. These partnerships include Planned Parenthood of Nova Scotia and its affiliates, other community groups, teachers and medical practitioners, and have been very beneficial in promoting adolescent sexual health. These relationships will continue and others will be established to insure teens are being provided the necessary services to meet their own needs and those of society.

SUMMARY

Unintended teenage pregnancy and STDs have similar behavioural causes which lend themselves to public health interventions. Understanding the psychology of sexual risk-taking behaviours in teens and some evaluation outcomes of past educational curricula can result in proactive sexuality education programming. Professionals working with teens have a responsibility to ensure that their knowledge, attitudes and above all, "askability" are appropriate to this age group. To answer in the affirmative the question of being an "askable professional" we need to ensure that: 1) books and posters addressing the issue of sexuality are on desks and on walls of our offices to make teens feel comfortable; 2) questions are asked

such as "do you have any concerns about sexually transmitted diseases or pregnancy" to establish that the professional is an appropriate person to address sexual concerns.

Preventing unintended teenage pregnancy and STDs constitutes a challenge to health professionals. With each new approach and program intended to address these issues evaluations are required to assess "what works". The negative impact and costs of such programs to teens and society, if not evaluated, may be tremendous. □

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CHLAMYDIAL DIAGNOSTIC TESTING

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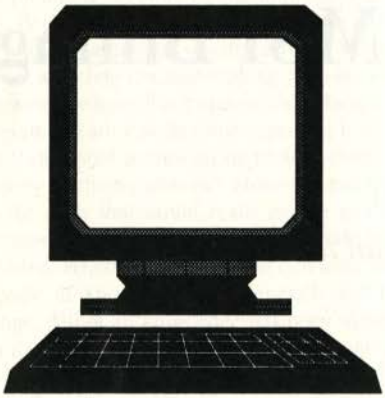

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Human Immunodeficiency Virus Infection in Nova Scotia

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It is over ten years since the first cases of the acquired immunodeficiency syndrome (AIDS) were reported in the medical literature.[1] What began as a syndrome noted in 15 individuals in the United States has evolved into a worldwide pandemic.

The past decade has seen the discovery of the causative agent, the human immunodeficiency virus (HIV); a basic understanding of the pathogenesis and natural history of infection; the availability of accurate diagnostic tests; development of a number of antiretroviral agents; and renewed interest in preventive and therapeutic strategies for the previously recognized infections which are the major cause of morbidity and mortality in HIV infected individuals.

The pandemic, however, has highlighted our continued difficulty with influencing personal behaviours that impact on health. It has exposed the discrepancies in access to therapies between the developed and developing worlds and the lack of adequate resources to cope with catastrophic illness even in developed countries. In many areas the illness has been met with unwarranted fear and discrimination requiring ongoing action.

This article will give an overview of the epidemiology, natural history and treatment of HIV infection. The infectious diseases clinic at the Victoria General Hospital in Halifax has provided care to all the adult patients diagnosed in Nova Scotia with AIDS. Information regarding AIDS in Nova Scotia is drawn from these records.

EPIDEMIOLOGY

More than 418,000 cases of AIDS have been reported world-wide, although it is estimated that the true number is closer to 1,500,000 (Source: World Health Organization). In Canada there have been 5647 reported cases with 3432 deaths (Source: Laboratory Centre for Disease Control). Ninety-six adults and one child have been diagnosed with AIDS in Nova Scotia and there have been 68 deaths (Fig.1).

The number of AIDS cases represents only a small proportion of HIV infections. The Department of Health reports that 296 Nova Scotians tested HIV positive between 1985 and 1992. (Personal communication: Dr. J. LeBlanc). This number underestimates the true prevalence of infection since some individuals choose to be tested out of province or not at all. The infectious diseases clinic has cared for 337 HIV infected patients since 1983 with 196 currently followed on a regular basis. Anonymous, unlinked seroprevalence surveys in sentinel populations promise better estimates of the prevalence of HIV infection in specific geographic areas or groups of individuals.

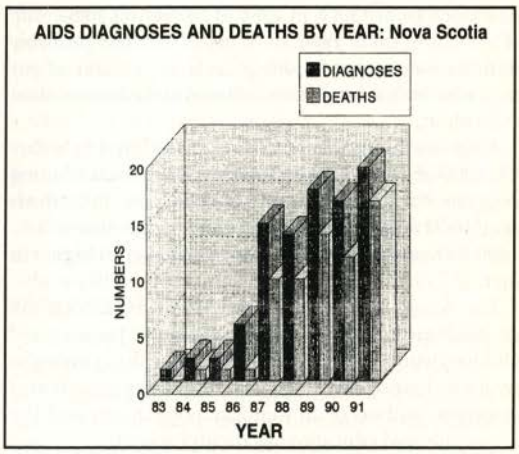

Figure 1

In Nova Scotia, as in Canada and the United States, most cases of HIV infection have occurred through sexual contact between men (Table I). In the United States, intravenous drug use now accounts for 23% of AIDS cases. It is a much smaller percentage for cases in Nova Scotia. Since introduction of universal blood screening in 1985, virtually all new adult HIV infections in North America have been the result of sexual intercourse or needle sharing. Heterosexual transmission accounts for 8% of AIDS cases in Canada and 6% in the United States.

TABLE I

RISK FACTORS FOR AIDS IN NOVA SCOTIA

	Male	Female
Sex with gay/bisexual men	78	0
I.V. needle sharing	2	1
Both of above	1	0
Blood or blood products	4	7
Heterosexual intercourse	0	1
Partner from endemic area	1	0
Unknown	2	0
TOTAL	88	9

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Women account for 77% of heterosexually acquired AIDS in the United States. Of these, most are black or Hispanic.[2] HIV seroprevalence surveys in the United States show rates in women giving birth to vary from 0/1000 (Montana and New Hampshire) to 5.8/1000 births (New York State) with a nationwide estimate of 6000 births to HIV infected women in 1989.[3] The Ontario seroprevalence study has reported a rate of 2.8/10,000 live births.[4] Such information is not yet available for Nova Scotia. In 25-35% of pregnancies involving infected women, HIV is transmitted to the child during pregnancy or delivery.

Prospective studies of seronegative gay men in the United States have shown that annual infection rates decreased from 13.8% in 1983 to a relatively stable rate of 1.1-2.2% from 1986 to 1990.[5] This rate probably underestimates that in the general population of gay men who are less intensively followed and educated than this cohort.

A serosurvey between October 1, 1987 and February 28, 1990 of United States Job Corp (a federal training program for disadvantaged, out-of-school individuals aged 16-21 years) applicants had a seroprevalence of 3.6/1000.[16] Overall seroprevalence was only slightly higher in men (3.7/1000) than women (3.2/1000) in this study.

The future of HIV infection in North America will probably see increasing numbers of infected women and children with the majority of infections still occurring in men who have sex with men. An increasing proportion of infections will occur in minority populations and the financially and educationally disadvantaged.

PATHOGENESIS AND DISEASE PROGRESSION

The human immunodeficiency virus is an enveloped RNA virus belonging to the family of viruses known as Retroviridae. It is composed of a single strand of RNA, structural and regulatory proteins, and the genes that encode these proteins. HIV antigens to which antibodies, used in diagnostic serological testing, are formed include the viral core antigen (p 24), envelope proteins (gp 120/160 and gp 41), and less consistently the DNA polymerase (p 55).

A number of different cells in the body are targeted by this virus, including macrophages, monocytes, microglial cells, and cells of the gastrointestinal tract.[7] The major target is the CD_4 lymphocyte.[7] AIDS is characterized by profound CD_4 lymphocyte depletion. The exact mechanism whereby cell death occurs has not been fully determined.

The mean latency period between infection and development of AIDS is 10 years.[8] Little is known about the factors that determine the duration of the asymptomatic period. As might be expected, CD_4 lymphocyte counts are predictors of HIV progression and hence prognosis.[9] Several studies have suggested that the percent of CD_4 lymphocytes is a more reliable indicator of disease status than is the absolute number of lymphocytes.[10-11] Detection of serum p 24 antigen is associated with a more rapid decline in CD_4 counts and, when combined with CD_4 lymphocyte results, identifies individuals at greater risk for disease progression.[12] By itself, p 24 antigen testing, because of its low sensitivity has limited usefulness in the clinical situation.[13]

Other tests that have been used to assess disease activity include serum beta-2 microglobulin and neopterin levels. Both these proteins are byproducts of increased cell activation and, indirectly, an indicator of increased viral activity. Although the presence of these markers appears to identify individuals at greater risk for progression of disease,[14] their usefulness in the clinical situation is not yet clear.

HIV TESTING

Tests for detection of antibodies to HIV have been available since 1985. The screening test is an enzyme immunoassay (EIA or ELISA). Several commercial test kits are available.

A repeatedly positive screening test is confirmed by Western blot or a similar confirmatory test. The Western blot allows for detection of antibodies to the individual viral antigens. A positive test requires the presence of two bands indicative of antibodies to p 24, gp 41, and/or gp 120/160.[16] Some individuals will have an "indeterminate" Western blot. This occurs when some of the bands are present but do not meet the two band requirement for a positive Western blot. A negative Western blot shows none of the accepted protein bands.

An indeterminate Western blot may occur in early HIV-1 infection when antibodies are being formed (the window period) or with infection with HIV-2.[17] More often, indeterminate Western blots are seen in healthy blood donors with no risk factors for HIV infection. Persistent indeterminate Western blot tests over several months or years do not reflect HIV infection.[18] The current recommendation for follow-up of indeterminate tests in asymptomatic individuals with no risk factors for HIV is repeat testing at six months. Individuals at risk for HIV or with a p 24 or envelope (gp 120/160 or gp 41) band require closer follow-up as they may represent individuals in the window period.

CLINICAL MANIFESTATIONS OF HIV INFECTION

Several different classification schemes for HIV infection have been developed utilizing a variety of clinical and laboratory findings. The Centers for Disease Control (CDC) classification, a clinical staging scheme is one of the most widely used.[19] To some extent this classification follows the natural progression of illness from asymptomatic to the development of the major opportunistic infections and malignancies associated with end stage disease.

CLASS I HIV

It is estimated that 50-70% of individuals will have a clinical illness associated with primary HIV infection although it may not be recognized as such at the time.[20-21]

Clinical presentation may be with fever, malaise, lymphadenopathy, mucocutaneous ulcerations, maculopapular rash, and/or headache.[22] An aseptic meningoencephalitis may also occur. Laboratory findings with primary HIV are nonspecific and include thrombocytopenia and the presence of atypical lymphocytes.

There is some data to suggest that individuals with primary HIV symptoms lasting longer than 14 days are at higher risk for more rapid progression to AIDS than those with shorter duration of illness.[20] Whether these individuals would benefit from immediate initiation of antiretroviral therapy is not known. Patients with prolonged symptomatic acute HIV infection probably warrant closer follow-up for evidence of disease progression and need for therapeutic intervention.

CLASS II HIV

The great majority of HIV infected individuals are asymptomatic with no abnormalities on physical examination. Nonetheless, laboratory testing often reveals abnormalities in immunological parameters with diminished total and CD_4 lymphocyte counts and percentages.[23]

CLASS III HIV

Persistent generalized lymphadenopathy (PGL) is defined as lymph node enlargement persisting for more than three months and for which no other etiology exists. The presence of PGL is not predictive of more rapid HIV progression.

CLASS IV HIV

This stage of infection is characterized by a variety of symptoms, infections, and malignancies attributed to infection with the virus itself or to the immune deficiency resulting from infection. AIDS is the final stage of HIV infection and defined by the presence of one of several opportunistic infections or malignancies.

The incubation period from time of infection to development of AIDS is variable. In the absence of antiretroviral therapy 45% of infected individuals develop AIDS within 8 years of infection.[8] Median survival after diagnosis of AIDS is variable and related to the AIDS-defining diagnosis. Survival for AIDS diagnoses made in 1987 was 18 months following first episode *Pneumocystis carinii* pneumonia (PCP) or diagnosis of Kaposi's sarcoma, and 10 months after development of other diseases such as lymphoma and cryptococcosis.[24]In general, survival has improved since 1984. This has been attributed to use of zidovudine and preventive therapies for PCP. More favourable prognostic indicators are male sex, younger age, white race, and Kaposi's sarcoma or PCP as the AIDS defining diagnosis.[25]

In Nova Scotia, the average survival for patients diagnosed with AIDS has ranged from 14 months in 1988 to 19 months in 1985 and 1989, with no trend toward improved survival having been seen over the years.

PNEUMOCYSTIS CARINII PNEUMONIA

PCP has been and continues to be the most common index AIDS defining diagnosis.[26] In Nova Scotia, PCP has been the index diagnosis in 55% of patients. Similar to other cohorts, 73% of our deceased and 48% of our living patients have had at least one episode of PCP.

The clinical diagnosis of PCP should be confirmed by detection of the organism on special stains of induced sputum or specimens obtained at bronchoscopy. The standard therapeutic regimes remain trimethoprim-sulfamethoxazole (oral or intravenous) and pentamidine (intravenous). Alternative regimes are clindamycin/primaquine and dapsone/trimethoprim. Trimetrexate and difluromethylornithine have been used primarily as salvage therapy.[27] A new agent, 566C80 produced by Burroughs Wellcome, looks promising in early clinical trials.[28] Adjunctive treatment with corticosteroids is recommended for patients with $P_a 0_2$ under 70 mm Hg on room air.[29]

Preventive therapy should be initiated in all HIV infected individuals who have had PCP or with CD_4 counts under 200 cells/mm^3. Sulfatrimethoprim is the drug of choice with aerosolized pentamidine and oral dapsone alternatives for patients intolerant of sulfatrimethoprim.[30]

CYTOMEGALOVIRUS

Cytomegalovirus (CMV) infection in the HIV infected patient is associated with retinitis, colitis, esophagitis, pneumonitis, and wasting syndrome.[31] CMV retinitis remains the most important of these infections. Similar to other studies, our patient cohort has had CMV retinitis as the index diagnosis in 1% of patients. CMV retinitis ultimately occurs in 25-30% of AIDS patients (only 10% in our group).

The diagnosis of CMV retinitis is primarily a clinical one and both patient and physician must be alert to the potential for this to develop. Screening ophthalmological examinations may be of benefit for patients with $CD_4 < 200$.

Intravenous ganciclovir has been the standard therapy for CMV retinitis. Foscarnet appears equally effective for retinitis and one study has shown improved survival as compared to ganciclovir therapy.[32] Long-term maintenance therapy is required to delay relapse of retinitis.

It is not clear that CMV colitis and esophagitis benefit from treatment. The exact role of CMV in pulmonary disease in AIDS patients is also not clear. It is rarely the sole pathogen associated with pulmonary disease and at this time well substantiated recommendations for treatment cannot be made.

TOXOPLASMOSIS

CNS toxoplasmosis is the most common cause of space occupying cerebral lesions in AIDS patients.[33] It is usually a result of reactivation of latent infection. The cumulative risk of an AIDS patient with latent *Toxoplasma* infection developing CNS toxoplasmosis is 28%.[34]

Sulfadiazine or clindamycin and pyrimethamine are the two drug regimens most often used to treat toxoplasmosis. The new agent BW 566C80 looks promising.[35] As with other opportunistic infections in AIDS patients, long-term maintenance therapy is required. The role of preventive therapy has not yet been defined.

MYCOBACTERIA

Mycobacterial infections, both typical and atypical, have been seen with increasing frequency over the last decade and directly related to the AIDS epidemic.

Tuberculosis (TB) may occur at any stage of HIV infection and does not necessarily occur only with profound immunosuppression.[36] Extrapulmonary TB is more common in HIV than in non-HIV infected patients.[37] Of recent concern has been the increase in multiresistant TB in the United States with documented nosocomial transmission.[38] Physicians must be aware of the potential for TB reactivation in their HIV infected patients. Preventive therapy with isoniazid should be initiated in all HIV infected individuals with a positive ($\geq$ 5mm induration) PPD.[39]

Infection with *Mycobacterium-avium* complex (MAC) organisms in HIV-infected patients was first reported in 1982. It is now recognized as a major cause of morbidity and contributor to mortality in those AIDS patients with severe immunosuppression as indicated by $CD_4 < 100/mm^3$.[40] Fourteen percent of our deceased AIDS patients have had MAC infection.

The usual clinical presentation of MAC is persistent fever and weight loss, with or without night sweats, chronic diarrhea, or abdominal pain. Patients are frequently anemic. Diagnosis is confirmed by the isolation of these organisms from blood and/or tissue.

Susceptibility testing demonstrates a greater degree of resistance of MAC to standard antimycobacterial drugs than exists for *Mycobacterium tuberculosis.* Although in vitro results do not necessarily reflect patient treatment outcome, therapy for MAC is generally not as successful as for TB. Multidrug regimes consisting of various combinations of ciprofloxacin, ethambutol, rifabutin, rifampin, clofazimine, and amikacin have been used.[40] Clarithromycin, one of the new macrolide antibiotics, looks promising in early clinical trials.[41] Rifabutin has recently been demonstrated as effective in preventing MAC in AIDS patients with $CD_4 < 200/mm^3$.

MALIGNANCIES

It is postulated that loss of immunosurveillance associated with the immunosuppression of HIV contributes to the development of malignancies, primarily Kaposi's sarcoma (KS) and non-Hodgkin's lymphomas.

Kaposi's sarcoma may involve any body surface and is characterized by continued, often rapid, progression. Visceral involvement is common. The skin lesions are pigmented (red to blue), palpable and asymptomatic. They frequently start in the head and neck area, including the oral cavity. Although the prognosis in AIDS is better for patients presenting with KS than with most of the opportunistic infections, survival in the KS group has actually decreased since 1983.[25] Visceral disease, particularly pulmonary KS, is associated with a poorer prognosis.

The diagnosis of cutaneous KS can be made clinically by an experienced physician. Biopsy should be done if there is any doubt about the diagnosis.

Radiation therapy plays an important role in the management of cutaneous and intraoral KS. Interferon has been of benefit in the subset of patients with no systemic symptoms and better preserved immune system.[42] Combination chemotherapy with more conventional cytotoxic agents has been reserved for patients with extensive disease. However, there has been some success using single agents in AIDS patients with KS.

High-grade non-Hodgkins lymphomas have been seen with increasing frequency in AIDS patients.[43] There have been 8 confirmed lymphomas in our cohort, all diagnosed since 1988. The response of these malignancies to treatment is poorer than for non-HIV infected individuals, usually because of the coexistent immunosuppression from HIV. The use of lower-dose chemotherapy with central nervous system prophylaxis may be a better approach to management of these patients.[44]

TREATMENT FOR HIV INFECTION

Most promising of the therapies to date have been the reverse transcriptase inhibitors. Zidovudine is recommended for use in HIV infected individuals with $CD_4 < 500/mm^3$. At the doses (300-600 mg daily) currently used, it is generally well tolerated with the major toxicity being bone marrow suppression. Prolonged use of zidovudine has been associated with resistant viral isolates, possibly indicating a decline in efficacy.[45]

Two other reverse transcriptase inhibitors that have undergone considerable study are dideoxycytidine (ddC) and dideoxyinosine (ddI). Both produce little bone marrow suppression. The major toxicity is peripheral neuropathy for both, oral aphthous ulcers (ddC) and pancreatitis (ddI and, less so, ddC). Recent reports suggest that both ddC and ddI are effective second agents in patients who have already been on zidovudine (NIAID update April 13, 1992).[46] Decreased survival has been seen when ddC rather than zidovudine is used as the initial antiretroviral therapy. There are ongoing trials looking at the safety and efficacy of combinations of reverse transcriptase inhibitors used either sequentially or in combination.

A number of other agents acting at the level of reverse transcriptase are under investigation as are agents that target other steps in HIV replication. None has yet proven effective in slowing progression of disease. It is anticipated that combination therapies acting at different viral target sites or used in conjunction with immune modulators (such as interferon) will ultimately prove the most effective.[47]

There is continued research into the development of HIV vaccines for both primary prevention and treatment to delay progression once infection has occurred.[48,49]

In Nova Scotia, single agent treatment with zidovudine or ddI is provided for HIV infected individuals with $CD_4 < 500/mm^3$. These drugs are available through the Infectious Diseases clinic at the Victoria General Hospital. Dideoxycytosine is available on a compassionate use basis for individuals intolerant of or resistant to zidovudine and/or ddI.

There is currently one trial evaluating antiretroviral therapy available to Maritime patients: gp 160 vaccine in HIV infected individuals with $CD_4 > 500/mm^3$. Clinical HIV trials are conducted through the Canadian HIV Trials Network, Atlantic region based in Halifax. □

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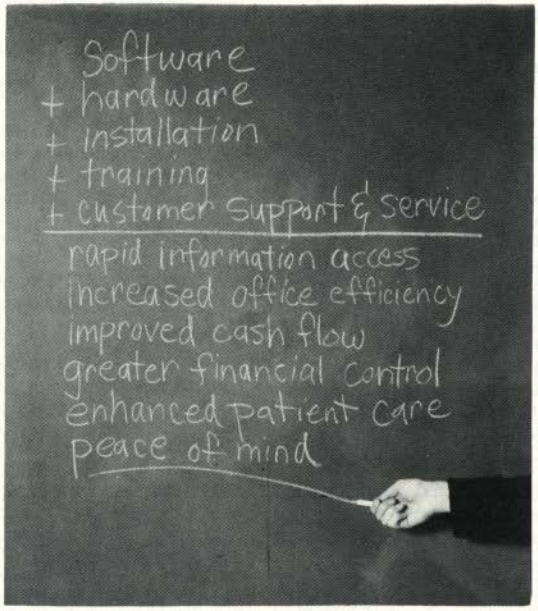
The whole is greater than the sum of its parts.



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Chlamydia trachomatis Infections in Nova Scotia

A LABORATORY PERSPECTIVE

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The number of specimens submitted to clinical laboratories in Nova Scotia for identification of *C. trachomatis* increased from 6464 to 50952 between 1985 and 1990. During the same period the proportion of positive tests declined from 7.95% to 3.99% and between 1989 and 1990 the number of positive tested decreased from 2820 to 2039 (27.7%). In women more than 30 years of age the positivity rate has declined to 0.25%. These changes in the epidemiology of genital infections due to *C. trachomatis* should prompt physicians to reexamine testing habits so that testing is both cost-effective and takes into consideration limitations of presently available testing methods.

Chlamydia trachomatis, infection of the genitourinary tract is an important cause of morbidity both in Nova Scotia and in the rest of Canada.[1] It not only produces symptoms associated with acute infection but unrecognized or inadequately treated infections may lead to the development of pelvic inflammatory disease, ectopic pregnancies and vertical transmission to infants born to infected mothers.[2-5] In addition to significant morbidity there is a considerable social and economic cost to the individuals and to the health care system in general.[6]

Diagnostic testing for *C. trachomatis* using cell culture technique began at the Victoria General Hospital (VGH) in 1983. Since that time the most frequently used diagnostic tests have shifted from the isolation of *C. trachomatis* using cell culture, to direct fluorescent antibody staining using the MicroTrak® system, to the detection of *C. trachomatis* antigen by enzyme immuno assays (EIAs) and more recently to EIAs of increased sensitivity and specificity. In addition, there has been a marked increase in the number of tests performed by hospital laboratories in the province. Accompanying these changes has been significant change in the epidemiology of *C. trachomatis* genital infection. These changes in testing and prevalence prompted us to examine the use of tests for *C. trachomatis* genital infection by Nova Scotia physicians.

MATERIALS AND METHODS

Records of *C. trachomatis* testing performed at the Victoria General Hospital were reviewed from 1985. Information gathered included a number of tests performed, the method used and the positivity rate. We also examined in more detail tests performed using the Kallestad Pathfinder® EIA performed at the VGH laboratory between August 1, 1989 and March 31, 1990. Results were analyzed by gender, age, specimen source and the location of the clinic where the specimen was collected.

Senior technical staff of other laboratories in Nova Scotia where *C. trachomatis* testing was performed were asked to complete a data collection form outlining the history of *C. trachomatis* testing at their site, the number of tests performed, the methods used, and the positivity rate.

Data are presented as absolute numbers or proportions. Data were analyzed using both test results and individuals as the unit of analysis. With respect to test characteristics and use, the test result was used. When describing characteristics of individuals, e.g., those who tested positive versus those who tested negative, analysis by individuals was undertaken. This is to ensure statistical independence of the outcome measure and to guard against a slight bias being introduced by counting several times the same individual who had several tests performed.

RESULTS

The number of tests for *C. trachomatis* increased from 6,338 in 1985 to 50,952 in 1990 (Table I). In 1990, 48,181 tests performed at six laboratories were performed using an EIA, 2,771 tests in two laboratories were examined by direct fluorescent antibody testing and 158 tissue cultures for *C. trachomatis* were performed at the Victoria General Hospital. Despite a 94% increase in the number of tests performed between 1987 and 1990, the number of positive tests declined from 2,517 to 2,039. This represents a decrease in the overall positivity rate from 9.6% to 4.0%. Although this decline was greatest at the VGH laboratory the positivity rate at other laboratories also declined from 7.2% in 1987 to 5.0% in 1990.

Data on *C. trachomatis* EIA tests on male urethral and female cervical tests performed using the Kallestad Pathfinder EIA at the VGH in 1989-90 are shown in Figures 1 and 2. Of note, there were only 9 positives tests of the 3,962 tests performed on cervical specimens from women over the age of 30 and only one test was positive of the 804 performed in women over the age of 40 years. The overall positivity rate in specimens collected from the cervix was 397 of 15,777 (2.5%) whereas 46 of 775 (5.9%) of male urethral specimens were positive. Figures 3 and 4 show the cumulative percept of positive and negative tests performed (repeat tests excluded) in males and females. Tests were most frequently positive in females between

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TABLE I

SUMMARY OF CHLAMYDIA TESTING IN NOVA SCOTIA: 1985 TO 1990

	1985 Tested (Positive)	1986 Tested (Positive)	1987 Tested (Positive)	1988 Tested (Positive)	1989 Tested (Positive)	1990 Tested (Positive)
Victoria General Hospital	6338 (504)	12390 (1183)	24517 (2393)	26610 (2022)	27383 (1683)	26898 (838)
Camp Hill Medical Centre				3440 (190)	12108 (740)	16411 (724)
Cape Breton Regional		680 (72)	1556 (110)	2097 (140)	2102 (116)	1838 (128)
Colchester					426 (32)	1990 (101)
Highland View Regional				478 (59)	735 (87)	954 (99)
Yarmouth			173 (14)	518 (34)	752 (52)	933 (71)
Aberdeen				534 (43)	1045 (69)	1061 (46)
Soldiers Memorial Hospital				117 (9)	751 (41)	867 (32)
TOTAL	6338 (504)	13070 (1255)	26246 (2517)	33794 (2497)	45302 (2820)	50952 (2039)
Overall % positive	7.9%	9.6%	9.6%	7.3%	6.2%	4.0%
VGH only	7.9%	9.5%	9.8%	7.6%	6.2%	3.1%
VGH excluded		10.6%	7.2%	6.6%	6.4%	5.0%

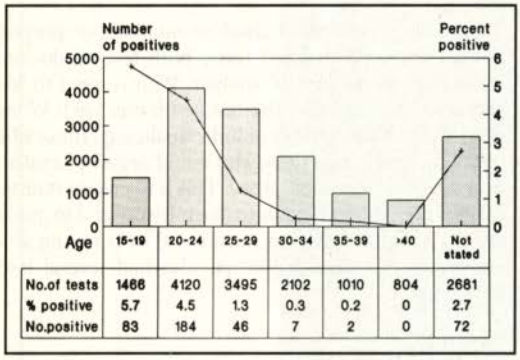

Figure 1

Results of testing of female cervical specimens at the Victoria General Hospital, 1989-1990 using an enzyme immunoassay.
Shaded bars indicate number of tests performed. The solid line represents the percent of tests which were positive.

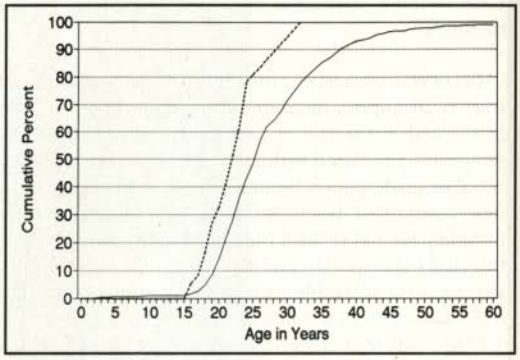

Figure 3

Cumulative percent of males whose urethral specimens tested positive or negative by age.
Solid line represents the cumulative proportion of negative tests. The dotted line represents the cumulative proportion of positive tests.

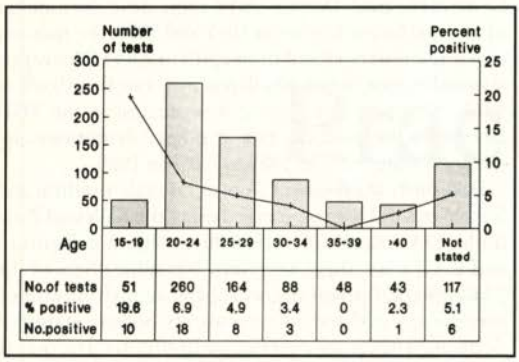

Figure 2

Results of testing of male urethral specimens at the Victoria General Hospital, 1989-1990 using an enzyme immunoassay.
Shaded bars indicate number of tests performed. The solid line represents the percent of tests which were positive.

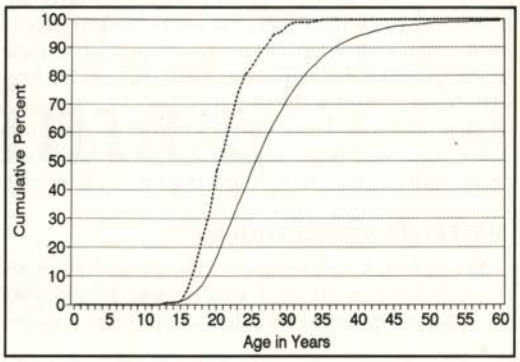

Figure 4

Cumulative percent of males whose urethral specimens tested positive or negative by age.
Solid line represents the cumulative proportion of negative tests. The dotted line represents the cumulative proportion of positive tests.

10 and 14 years, however numbers in this age bracket were small since physicians were encouraged to request *C. trachomatis* culture rather than antigen tests in this age group. Among females the next highest positivity rate was between the ages of 15 and 19. Between the ages of 15 and 19, 83 (5.7%) of 1,446 tests were positive.

The highest positivity rate among men was in the 15-19 year age range from whom 10 (19.6%) of 51 were positive. There was only one positive test in a male over the age of 34 years of the 89 tests that were performed.

From cervical specimens positivity rates varied somewhat (Table II): 9 (1.9%) of 482 specimens submitted from the Grace Maternity Hospital; 82 (2.6%) of 3,190 specimens from women seen at Dalhousie Student Health, a university health clinic available to all students at the university; 186 (3.3%) of 6,879 specimens sent from private physicians' offices in the Halifax metropolitan area; 30 (4.2%) of 709 from an urban core clinic.

TABLE II

RESULTS OF *CHLAMYDIA TRACHOMATIS* TESTING OF FEMALE CERVICAL SPECIMENS FROM SELECTED LOCATIONS PERFORMED AT THE VICTORIA GENERAL HOSPITAL BETWEEN AUGUST 1989 AND APRIL 1990 USING AN ENZYME IMMUNOASSAY*

Location	Number Tested	Number Positive (%)
1 Halifax County **	6,879	186 (2.7)
2 Dalhousie Student Health	3,190	82 (2.6)
3 North End Clinic	709	30 (4.2)
4 Lunenburg County	499	10 (2.0)
5 Grace Maternity Hospital	482	9 (1.9)
6 VGH Emergency	462	10 (2.2)
7 Dartmouth	358	20 (5.6)
8 Colchester County	264	7 (2.7)
9 Other Locations	2,888	43 (1.5)
10 All locations	15,731	397 (2.5)

* Kallestad Pathfinder(R) EIA with confirmatory testing.
** Private physician offices.

At present Chlamydia cultures are only performed at the VGH. In 1990, 158 were performed primarily in children in whom sexual abuse is being considered. None of those performed on children were positive.

DISCUSSION

Until recently the incidence of *C. trachomatis* genital infection appeared to be increasing in many North American cities.[7] This trend appears to have reversed in the province of Nova Scotia. Despite a marked increase in the number of tests performed and the wider availability of testing facilities to Nova Scotia physicians, the total number of positive tests has declined slightly and the proportion of tests positive has declined markedly both in urban and rural settings. This decline reflects a decrease in the overall prevalence of *C. trachomatis* genital infections. There may be a number of causes contributing to the decline. It may relate to safer sexual practices, to other changes in the sexual habits of Nova Scotians and the use of effective treatment strategies.

It seems likely that the significant decline in the proportion of positive tests is related to the more frequent use of antigen detection tests for screening asymptomatic women. We were unable to determine what proportion of tests performed were done in accordance with recently published guidelines for testing.[8] These guidelines suggest testing asymptomatic women with evidence of mucopurulent or purulent cervical discharge, high risk women under the age of 25, STD clinic patients not given anti-chlamydia treatment and other groups such as family planning clinic patients if the prevalence exceeds 7%.

It appears that Nova Scotia physicians continue to screen many women who do not fall within these guidelines. This may be due to a failure to recognize that the positivity rate has fallen to the extent that it has. In our population this is especially true in women over the age of 30 or men over the age of 35 in whom the positivity rates were only 0.25% and 1.12%, respectively. By providing appropriate feedback to physicians ordering tests for *C. trachomatis* testing rates may decline.

During the last several years it is likely that the specificity of non-culture methods for *C. trachomatis* has increased somewhat.[9-11] Using presently available EIA tests initial positive and borderline test results are usually confirmed with a second test.[12] The confirmatory test involves the use of a "blocking antibody" which is reacted with the clinical specimen before a second EIA is performed. If *C. trachomatis* antigens are present in the specimen, the antigenic sites are blocked by the "blocking antibody" and the second EIA test is negative. Using the other confirmatory test, a specimen positive by the initial EIA is centrifuged and the pellet examined microscopically by direct immunofluorescence in a manner similar to that used for direct "MicroTrak®" type of antigen detection. In some cases the initial EIA and subsequent direct fluorescent examination use antibodies to different *C. trachomatis* antigens. This may further serve to increase specificity.

Older EIAs not using confirmatory tests have specificities usually varying between 93% and 98%.[9] Laboratories using the MicroTrak® direct fluorescent antibody test to test clinical specimens have a specificity rate between 94% and 99% and results can only be confirmed by collection of a second clinical specimen. Actual specificities may be slightly higher for both EIA and MicroTrak® testing since occasional specimens from infected individuals are antigen positive and culture negative. As the prevalence of *C. trachomatis* declines in the population, specificities lower than 98% would no doubt result in very low positive predictive values, ie a low

probability that a test result reported to a physician is truly positive and not a false positive.[13] If for example 6% of tests are reported positive and the test has a specificity of 96% there is only a 33% chance that a positive test result is correct. It is fortunate that the specificity of newer EIAs is higher because in the setting in which most chlamydia testing is performed, false positive results cause unnecessary stress and difficulties between the patient, physician, family and sexual partners.

There is evidence from our own experience that the performance of EIA tests for the detection of *C. trachomatis* has improved. In women over the age of 40 who had cervical specimens tested with the Kallestad® chlamydia EIA, we observed the remarkable finding of 0 positives among 804 tests. This suggests that the false positivity rate is in a range of 1 in 500 to 1000 in this age group, in other words, a specificity of 99.8% or more. It is possible that this high specificity will not be seen in young age groups where cross reactivity with antigens from other bacteria may decrease specificity.

In a recent comparison of the Syva and Kallestad Pathfinder® EIA carried out on 1,008 specimens in our laboratory, the specificity of each test was 100% when borderline positive EIAs were confirmed with a direct fluorescent antibody test (unpublished observations). Positive predictive values do however remain a concern, especially in the most recent year when the positivity rate fell to 4% for the province and to 3.12% of the 26898 tests performed at the VGH. In females the over 30 age group in whom the positivity rate has fallen to 0.23% there appears to be a need for a very critical review of test ordering. This background prevalence calls question both the cost-effectiveness and clinical utility of routine testing and confirms the recommendations of the expert panel assembled by the Laboratory Centre for Disease Control.

In males, positivity rates are higher than females. This probably reflects the infrequently use of *C. trachomatis* antigen testing in screening the asymptomatic male. Asymptomatic women frequently undergo regular pelvic examinations and endocervical specimens are often collected as part of the regular genital examination whereas routine testing in men is infrequently performed. Men do not tend to have regular genital examinations if asymptomatic, specimen collection is painful and men are not subject to the more serious complications of asymptomatic infection in women.

In summary, recent years have seen a marked increase in the number of tests performed and a decrease in the number of positive test results. It is not clear to what degree this reflects a decrease in prevalence in the population. It is possible that some of the decline relates to increased specificity of tests and that fewer false positive results are being reported. As the positivity rates decline further, physicians should reexamine ordering practices, keeping the cost and limitation of current testing methods in mind. Given the low prevalence in many regions, laboratories in areas of low prevalence should use methods that allow for confirmation of positives in order that the high predictive value of positive tests might be ensured. □

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Chlamydial Diagnostic Testing

A COMPARISON OF THE TWO ENZYME IMMUNOASSAY TESTS CURRENTLY USED IN NOVA SCOTIA

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The study was undertaken to compare the sensitivity and specificity of two commercial enzyme immunoassays (EIA) for chlamydia testing currently used in hospital laboratories in N.S. A total of 1,008 genital specimens from 4 clinics in the Halifax metropolitan area were tested in parallel with both the Pathfinder® and the MicroTrak® EIA systems. All EIA positives were confirmed by direct immunofluorescence tests with reagents supplied by the manufacturers. Chlamydia culture and/or clinical data were used to verify discrepant results. Both chlamydia antigen detection EIA systems are highly specific. The MicroTrak® EIA is perhaps the most sensitive of the two since it detected 11 more true positives than the Pathfinder® EIA. The importance of confirming EIA-screening test results is emphasized in this study.

Rapid and accurate diagnosis of chlamydial genito-urinary infections is essential for both therapeutic and epidemiological purposes. For a number of years, the direct isolation of *Chlamydia trachomatis* in cultured mammalian cells has been looked upon as the standard procedure for diagnosing infections. It is now generally recognised that although highly specific, the cell culture method lacks sensitivity, speed, versatility and is not cost-effective. In the past decade, non-culture methods such as the direct fluorescent antibody (DFA) and the enzyme immunoassay (EIA) now serve as a suitable alternative to cell culture. These non-culture methods are amenable to high volume testing, permit same day result reporting and are better suited to clinical laboratories that lack the facilities or expertise in cell culture technology. As the manufacturers improve the performance of their products, especially with the development of confirmative tests, specificity has increased significantly. However, at present the cell culure method remains the method of choice in all sexual assault or sexual abuse cases for medico-legal reasons.[3]

In recent years, the prevalence of *C. trachomatis* genital infections has declined markedly in Nova Scotia and emphasis has shifted from the diagnosis of symptomatic infections to the recognition of patients who are asymptomatic.[4] Changes in the testing habits of physicians have placed increasing demands on the tests employed in the diagnostic laboratories. In this testing environment, methods need be more sensitive and, as the prevalence declines, tests have to be highly specific in order to assure that the predictive value of a positive test remains high.

Many hospital laboratories in Nova Scotia are presently testing for *C. trachomatis* using commercially available non-culture systems. Some laboratories handling relatively small volumes use the Syva MicroTrak® DFA test. The VGH and other laboratories dealing with larger volumes have found the EIA test more practical and cost-effective.

In this communication, we considered the suitability of the two EIAs currently used in Nova Scotia, namely the Pathfinder® EIA and the MicroTrak® EIA to screening our low prevalence population for *C. trachomatis.* The consideration was based on evaluating the two EIAs in parallel for their sensitivity, specificity and the predictive value of a positive and negative test results.

METHOD

We collected 1,008 specimens from four clinics in the Halifax metropolitan area. Based on previous laboratory data, two of the participating laboratories have a relatively low prevalence rate of about 3%; and two have a moderate prevalence rate of about 10%. The order of specimen collection for each of the test systems was rotated weekly in order to obtain maximum randomness. In some instances, ie. when the physician considered the patient to be at particularly high risk, a specimen for culture was also collected. All specimens were tested in parallel using the MicroTrak® and Pathfinder® EIA systems.

Testing was performed in accordance with the manufacturers' instructions. All positive EIA tests were confirmed by direct fluorescence examination of the centrifuged sediment of the residual specimen using the respective DFA reagent supplied by the manufacturers. For the purposes of this study, the presence of 5 elementary bodies in a direct fluorescence preparation was considered positive.

Tests were considered true positive if both the EIA and DFA were positive. If the specimen was EIA positive and DFA negative, the result was considered a false positive. A negative result by both EIA systems was regarded as a true negative. When available, culture results were used to verify disparate results.

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Chlamydia cultures were performed on McCoy cell monolayer using the shell vial method. After 72 hours incubation at 37°C, the McCoy cells were examined by DFA using fluorescein-labelled mouse monoclonal antibodies specific for *C. trachomatis* (Syva MicroTrak®).

Attempts were made to collect clinical data on any discrepant results if cultures were not available.

RESULTS

A summary of test results can be seen in Table I. Of the 1,008 specimens, 954 were negative by both EIA systems. Thirty-seven were positive by both. Eleven specimens, positive by the MicroTrak® EIA were negative using the Pathfinder®. In 10 cases, the Pathfinder® EIA was negative, in one the EIA test was positive; however, it was not confirmed by DFA direct fluorescent microscopy. Six tests were positive by the MicroTrak® EIA but were not confirmed by direct fluorescent microscopy and were negative by the Pathfinder® EIA. These would have been reported to the clinician as negative tests.

TABLE I

SUMMARY OF TEST RESULTS

MicroTrak® Pathfinder®	EIA+ DFA+	EIA+ DFA-	EIA-
EIA + DFA+	37	0	0
EIA+ DFA-	1	0	0
EIA-	10	6	954

The overall agreement between the two systems was 98.9%, the sensitivity of the Pathfinder® was 71.1%, the specificity of DFA confirmed positives was 100% for each. The predictive value of a negative MicroTrak® result was 100%, the negative predictive value of a negative Pathfinder® result was 98.9%.

Table II shows our analyses of the discordant Pathfinder® and MicroTrak® EIA results. Cultures were performed in 5 of the 11 patients with discordant results and were positive in all 5. Three other patients with discordant results had mucopurulent cervicitis but no cultures were collected. Two patients had two sets of specimens submitted. In the first case, the discrepant result was a repeat specimen from a symptomatic patient. In the other case, the discrepant result was from a patient who subsequently had a culture submitted which was positive.

Correlation of results with the order of specimen collection is shown in Table III. More specimens tested by the Pathfinder® system were collected second or third (514 vs 465 for the MicroTrak®). The Pathfinder® was falsely negative in 4 specimens collected first, falsely negative in 3 specimens collected second, and falsely negative in 4 of the 18 collected third. None of the specimens collected third and tested by the MicroTrak® were from patients with *C. trachomatis* infection.

TABLE II

ANALYSIS OF DISCORDANT PATHFINDER® AND MICROTRAK® EIA WITH CULTURE RESULTS

Type of Specimen	Pathfinder® Order of Specimen	Pathfinder® EIA	MicroTrak® Order of Specimen	MicroTrak® EIA	Culture Order of Specimen	Culture Result
Cervix	1	–	2	+	3	+
Urethra	3	–	1	+	2	+
Urethra	1	–	2	+	No culture*	
Cervix	3	–	1	+	2	+
Cervix**	1	–	2	+	No culture*	
Cervix**	2	–	1	+	No culture	
Cervix†	2	–	1	+	No culture	
Cervix†	3	–	2	+	1	+
Urethra	3	–	2	+	1	+
Cervix	1	–	2	+	No culture*	
Cervix	2	+††	1	+	No culture	+

* Showing clinical signs and symptoms.
** Same patient with specimens collected 7 days apart.
† Same patient with specimens collected 3 days apart.
†† Not confirmed by DFA.

TABLE III

EFFECT OF THE ORDER OF SPECIMEN COLLECTION ON RESULTS OF ANTIGEN DETECTION TESTING USING TWO DIFFERENT TEST SYSTEMS

Test System		Order of Collection 1	2	3	Total
MicroTrak®	Number negative	495	456	9	96
	Number positive	21	27		48
	Total specimens	516	483	9	1008
Pathfinder®	Number negative	457(4)*	496(3)*	18(4)*	971(11)*
	Number positive	21	12	4	37
	Total specimens	478	508	22	1008
Culture	Number negative		1	2	3
	Number positive	2	3	2	7
	Total specimens	2	4	4	10

* ( ) Discordant results.

DISCUSSION

The evolution of diagnostic tests for the diagnosis of *C. trachomatis* has resulted in tests of very high sensitivity and specificity. The addition of a confirmatory step using either a blocking antibody or direct immunofluorescence examination of the original clinical specimen has significantly improved the specificity of these systems. In Nova Scotia in 1990, the positivity rate of all testing performed in the province was 4.0%. Tests performed at the Victoria General Hospital had noted that the rate has fallen to 3.12% from 9.8% in 1987. As the prevalence declines, physicians need be much more concerned when tests of lower specificity are employed. For example, if the specificity of the method is 99%, (ie. if 1% of test results are false positive) and if the true prevalence in the

population is 3% then the probability that a positive result is indicative of infection is only 75%. For many asymptomatic patients the consequence of a false positive result can have serious affects into personal relations with other family members or sexual partners and for these patients a positive test probability of 75% would not be acceptable.

Our results suggest that the Syva MicroTrak® EIA system is both highly sensitive and specific in that no patient had the presence of *C. trachomatis* identified by other means which was not identified with the MicroTrak® EIA, ie. the sensitivity was 100%. Similarly, the specificity of the test when direct immunofluorescence was used to confirm the initial result was 100%. Had a confirmatory test not been performed 6 false positive MicroTrak®EIAs would have been reported.

The Pathfinder® EIA was less sensitive. Some of the decrease in sensitivity appeared to relate to the fact that the Pathfinder system was more rigorously challenged in that a greater proportion of positive specimens tested by the Pathfinder EIA were collected second or third. It is probable that decreasing amounts of *C. trachomatis* antigen is collected when multiple samplings are performed. When our results were reported to the manufacturer, they identified a quality control problem associated with the manufacture of the particular batch that we tested. Because of their plans to reformat their EIA system, a repeat evaluation of appropriately quality controlled plates was not performed.

Using the Pathfinder®, only one EIA was positive when the DFA failed to confirm that result. The Pathfinder® EIA itself is more specific than the MicroTrak® EIA and positive EIAs in the latter should always be confirmed using a confirmatory test.

As a result of our evaluation, we have concluded that both chlamydia detection EIA systems presently employed in the province are highly specific and that the MicroTrak® EIA is perhaps the most sensitive of the two since the Pathfinder® detected only 21 of 25 true positives when the specimen was the first collected and only 12 of 15 true positives when the specimen was the second collected. Without further studies of the type we performed, it is not possible to determine whether correction of the quality control procedure has increased the Pathfinder® EIA sensitivity to that of the MicroTrak® EIA.

In summary, physicians should be reassured that the specificity of these tests is very high when specimens are collected from adults and from either the urethra or cervix. Physicians should be reminded that in children, or in circumstances where medical legal issues are preeminent, that a culture for *C. trachomatis* is still the preferred testing method. Physicians should also be aware that specificity, although high for cervical and urethral specimens, is likely lower for vaginal or anorectal specimens and testing of such sites by EIA is not recommended. □

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Management of Tubo-Ovarian Abscess

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Tubo-ovarian abscess occurs most often in younger patients of low parity, and is often difficult to diagnose. Treatment should initially be conservative with intravenous antibiotics, however, early laparoscopy may be of benefit in confirming the diagnosis as well as initiating treatment. Alternatives to total abdominal hysterectomy and bilateral salpingo-oophorectomy should be considered when laparotomy is required.

Tubo-ovarian abscess is recognized as a severe form of Pelvic Inflammatory Disease (PID) that results from inadequate or delayed treatment of pelvic infection. Most cases occur in women of low parity in the third and fourth decades. The location of the abscess may be within the tube and ovary, between the leaves of the broad ligament or a collection of purulent material within the pelvic cavity, generally in the cul-de-sac, in which case the upper wall is usually formed by adherent intestinal coils. Tubo-ovarian masses are reported in 3 to 16% of hospitalized patients with acute PID.[1] Isolated ovarian abscesses are rare, and are more commonly seen as a post hysterectomy infection.[2]

PATHOGENESIS

The pathogenesis of tubo-ovarian abscesses involves the promotion of tissue necrosis by invading organisms, resulting in mucosal deciliation and the production of pus. The pus may occlude the fallopian tube or exude from the fimbriated end, infecting the ovary. Contiguous tissues including omentum, bowel and bladder may serve to contain the pus and in the process become adherent to the tubo-ovarian complex. This inflammatory response is finally encapsulated by an abscess wall as the host seeks to localize and sequester the infection.[3]

Tubo-ovarian abscesses may rupture resulting in an acute abdomen. Depending on the host response and the organisms present sepsis and/or reconfinement may occur.

DIAGNOSIS

The diagnosis of tubo-ovarian abscesses is difficult. The majority of patients have pelvic/abdominal pain, fever > 38.5 C and leucocytosis. However, some patients are asymptomatic and have a normal physical examination and laboratory tests. The clinical presentation of tubo-ovarian abscesses may overlap with uncomplicated PID.

Useful nonspecific laboratory tests include erythrocyte sedimentation rate, white blood cell count and acute-phase serum C-reactive protein.

Ultrasound has been used to document the presence of an adnexal mass and to monitor therapy. Ultrasound diagnosis requires demonstration of a well defined complex adnexal mass > 4 cm in size with no apparently normal ipsilateral ovary.[4]

Radionucleotide scans using gallium - 67 and indium - 111 labelled white blood cells have been shown to be highly sensitive in the location of intra abdominal abscesses. However, the reliability of these tests remains in question.[3]

Computed tomography and magnetic resonance imaging are expensive and perhaps marginally superior to ultrasound.

Laparoscopy is the most accurate technique for diagnosing tubo-ovarian abscesses. It has a low surgical complication rate and allows for excellent visualization of the pelvis for diagnostic purposes. Reich[5] and Henry-Suchet[6] have advocated using the laparoscope therapeutically to drain or remove the abscess.

MICROBIOLOGY

A wide variety of organisms may be involved in the pathogenesis of tubo-ovarian abscess. It has been proposed that *N. gonorrhoea* or *C. trachomatis* initiates the process by gaining access to the upper genital tract producing tissue damage that allows for superinfection with anaerobes and aerobes from the cervix.[3] Cultures of tubo-ovarian abscesses rarely reveal *N. gonorrhoea* or *C. trachomatis.*

Tubo-ovarian abscess cultures usually yield a variety of organisms including *E. coli, B. fragilis,* aerobic streptococci and *Peptostreptococcus sp.* After a few days of treatment cultures are often negative.

TREATMENT

Historically, management consisted of drainage and ultimately extirpative surgery.[7] More recent first line therapy has been medical although rupture of a tubo-ovarian abscess has been generally accepted as an indication for operative intervention.[8,9] The standard practice for tubo-ovarian rupture has been to recommend hysterectomy plus bilateral salpingo-oophorectomy. Rivlin, however, demonstrated that with aggressive peritoneal lavage and drainage only 3% of patients needed hysterectomy.[10]

Kaplan pointed out that there is a high residual morbidity in the conservatively managed group and advocated early definitive surgery via total abdominal hysterectomy and bilateral salpingo-oophorectomy (TAH/BSO).[2] His results showed quick resolution with few operative sequelae. However, there have been concerns re radical surgery in this often young group of women.

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Landers and Sweet reviewed 232 patients with tubo-ovarian abscesses in whom 175 patients were treated with antibiotics alone.[1] Sixty-eight percent of patients treated with a clindamycin containing regimen had an appropriate response while only 36.5% of those patients with a non clindamycin regimen had a response. Metronidazole was not used in this review.

Reed compared a clindamycin plus aminoglycoside regimen to cefoxitin plus doxycycline, obtaining a similar response rate of approximately 75%.[4]

Thadepalli compared ciprofloxacin with a clindamycin plus gentamycin regimen showing equivalent efficacy with a response rate of 95% in acute pelvic infections.[11] A subgroup of five patients with tubo-ovarian abscess treated with ciprofloxacin all responded.

The guidelines for proceeding with surgical management in the face of failed medical management include persistent fever, increase in the size of the abscess, persistent ileus, suspicion of rupture, septic shock, and uncertainty of diagnosis.[2,7]

Franklin suggested medical therapy initially.[9] In cases where there was a fluctuant mass filling the true pelvis which was midline and dissecting the rectovaginal septum, he advocated colpotomy drainage as have others.[4,12] Ten percent of Franklin's group failed early treatment requiring TAH/BSO. Sixteen percent of Franklin's group that had initially responded to treatment ultimately came to definitive surgery.

Ginsburg reported on 160 patients who were treated initially medically.[8] One hundred and ten patients initially responded to medical treatment, but 34 patients in this group ultimately required surgery with 32 having TAH/BSO. There were 50 patients who immediately failed medical therapy with 40 requiring TAH/BSO.

Both Ginsburg[8] and Reed[4] have shown that the larger the tubo-ovarian mass is at diagnosis the greater the need for surgical intervention. In Reed's study, 18% of patients with masses < 6 cm needed surgery; 65% of patients with masses > 10 cm required surgical intervention.

Rivlin described 113 patients who failed conservative medical therapy.[10] At laparotomy all loculations of pus were opened and lavaged with adhesions freed. Three drains were left in place for suction and irrigation. Three per cent of patients had hysterectomies, 73.5% had menstrual function preserved and 42.5% had potential for pregnancy. 17.7% of patients required subsequent surgery with only one patient having a hysterectomy.

Laparoscopic treatment of tubo-ovarian abscess was first reported in 1972.[5] In 1984, Henry-Suchet, reported on fifty cases of tubo-ovarian abscesses where the treatment was done laparoscopically.[6] In thirty-two cases of recent onset, the friable adhesions between organs were broken up with a rod under direct vision. Purulent fluid was aspirated by suction and the pelvis irrigated with antibiotic in saline solution. Eighteen cases of long standing tubo-ovarian abscesses were encountered. The bowel could usually be separated from a dense adnexal mass. The mass was punctured and aspirated of purulent fluid, then irrigated without complete lysis of adhesions. No drains were left in these fifty patients. Intravenous antibiotics were commenced after cultures were obtained in the operating room. Ninety percent of cases had quick recovery with one patient needing a TAH/BSO and two patients having bilateral salpingectomies for treatment failures.

Reich in 1987 reported on twenty-five women who were treated laparoscopically for pelvic abscesses with lysis, drainage and irrigation.[15] Twenty-four had complete resolution of their abscesses. Of the twenty-one cases of tubo-ovarian abscesses one patient required a TAH/BSO one month after her laparoscopy. Six women became pregnant during the follow-up period for a pregnancy rate of 24%.

Worthen reported on the options of computed tomography or ultrasound guided drainage of intra abdominal abscess with an overall success rate of 77% when the procedure was done twenty-four hours after commencing intravenous antibiotics.[13]

ANTIBIOTICS

Clindamycin

This antibiotic provides excellent activity against Gram-negative anaerobes and is transported into abscesses by polymorphonuclear leukocytes. It also covers Gram-positive aerobes and is active against *C. trachomatis.*

Tetracyclines

These antibiotics are the treatment choice for *C. trachomatis* but are not recommended as a single agent as their spectrum is too narrow.

Aminoglycosides

Aminogycosides provide aerobic Gram-negative coverage but are not indicated as a single agent.

Cephamycins and Cephalosporins

Cefoxitin and Cefotetan have a wide spectrum of Gram-positive and Gram-negative coverage but are inactive against *C. trachomatis.*

Penicillins

Many Gram-negative anaerobes are resistant to the basic penicillins. Extended activity of penicillins (piperacillin, mezlocillin) have been designed to maintain wide coverage against Gram-positive anaerobic and aerobic cocci (including Group B Streptococci and enterococci) while giving good Gram-negative coverage. They are inadequate against *C. trachomatis.*

Penicillin/Beta-lactamase Inhibitor Combinations

These combinations have not been sufficiently evaluated in clinical trials to make current recommendations for use in treating tubo-ovarian abscess.

Fluorquinolones

These drugs have good activity against *N. gonorrhoea,* most Gram-negative and Gram-positive bacteria and most anaerobes. Ciprofloxacin is active against *C. trachomatis*

and has demonstrated a concentrating effect in abscesses.[3]

Metronidazole

This antibiotic is highly effective against Gram-negative anaerobic bacteria and has good abscess penetration.

Antibiotic therapy is usually given intravenously for at least seven days with at least a seven day course of oral agents post hospital discharge. The response to antibiotics may be slow. Patients should be given 72 hours to improve prior to their being considered treatment failures.

FOLLOW-UP

Patients who have responded to treatment and who seem to be doing well should have follow-up pelvic exams at two and six week intervals. Ultrasound evaluations should be done at six weeks to compare to the examination done on admission. Persistent pelvic masses need to be evaluated by laparoscopy or laparotomy to confirm a diagnosis.

SUMMARY

Tubo-ovarian abscesses are a common complication of PID or may occur postoperatively. Usual treatment is with intravenous antibiotics but, if there is no response in 72 hours, surgery is required. Some authors recommend early laparoscopy as diagnosis is often not clear cut and this gives an option for treatment as well. The role of aggressive surgery has been questioned as conservative surgery has yielded acceptable results with an approximate 10% preservation of fertility.[1] □

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The Importance of Sexually Transmitted Disease Control for the Prevention of Infertility

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This article describes the importance of sexually transmitted disease (STD) control as a key measure of preventing female infertility. The most important modifiable risk factor for infertility in Western society is pelvic inflammatory disease (PID) associated with infections with the sexually transmitted agents, *Neisseria gonorrhoea* and *Chlamydia trachomatis*. The prevention of STD-related infertility lies with primary and secondary preventive manoeuvres for gonorrhoea and chlamydia control and effective management of PID. Physicians can reduce preventable infertility among their female patients by suspecting and confirming the existence of STDs, using efficacious therapy, complying with reporting and contact tracing requirements, and ensuring patient compliance with treatment and follow-up.

The establishment of the Royal Commission on New Reproductive Technologies has focused attention on infertility in Western society and on technological methods which assist childless couples to have biological offspring.[1] The need for attention to the preventable causes of infertility, especially among women, has been cited as an essential component of the Royal Commission report.[2] This paper discusses the role of sexually transmitted disease control in the prevention of female infertility.

THE EPIDEMIOLOGY OF INFERTILITY

The prevalence of infertility (failure to conceive after one year of regular intercourse), is a function of the age distribution of the population, the age at which childbearing is begun, the type of contraceptive used before attempting to conceive and the interval into which intended childbearing is compressed.[3] The prevalence of infertility in developed countries is often cited as up to 15% of all couples, although in Canada, infertility has been estimated to affect fewer than 7% of women surveyed. This discrepancy in estimates may be related to differences in wording of questionnaires between studies.[4]

Ovulatory dysfunction and seminal factors are the leading causes of infertility with tubal factors, endometriosis and idiopathic infertility accounting for the remaining causes.[5,6] Three clinical studies reported that male factors accounted for infertility in 18 to 30% of couples for whom a diagnosis was possible.[5,6] With regard to female factors, tubal defects were considered the primary cause in 12 to 20% of couples, ovulation defects in 15 to 30%, and endometriosis in 3 to 25%. Idiopathic infertility has been estimated to occur in 3 to 30% of couples.[5,6]

The sequelae of sexually transmitted diseases (STD) are the single greatest contributor to preventable infertility today.[7] Pelvic inflammatory disease (PID) and ectopic pregnancy are acute sequelae of infection with gonorrhoea and chlamydia. In some areas of the US, 20% of infertility is felt to be related to STD, mediated through PID.[7]

THE RELATIONSHIP OF GONORRHOEA AND CHLAMYDIA TO PELVIC INFLAMMATORY DISEASE

Pelvic inflammatory disease probably occurs in 10 to 20 percent of women with gonorrhoea, but in one study, 47 per cent of women recently exposed to gonorrhoea developed signs suggestive of upper genital tract infection.[8] The frequency of isolation of *N. gonorrhoeae* from women with PID is highly variable. The organism has been isolated from the cervix from 50 per cent or more of cases of PID in the United States,[9] yet from less than 20 percent of recent cases in Sweden.[10]

Pelvic inflammatory disease may also be caused by a variety of other organisms including *C. trachomatis*, vaginal anaerobes, Mycoplasma hominis, and facultative gram-negative rods.[9,10] *C. trachomatis* is the causative organism in 20 to 66% of cases of PID in studies done in northern Europe[11,12] and the infection results in an increased incidence of ectopic pregnancy[13,14] and infertility.[12,15,16]

Seroepidemiologic case-control studies of infertile women have shown that infertile women with tubal disease are two to four times more likely to have elevated antibodies to *C. trachomatis* than either infertile women with normal tubes or pregnant women. In three of six centres, more than half of the women with positive chlamydial serologies gave no history of pelvic inflammatory disease.[17-19] This suggests that chlamydial infection of the upper genital tract may often be subclinical and an insidious cause of eventual infertility.[20]

It is estimated that womens' risk of acquiring PID with either gonorrhoeal or chlamydia STDs ranges from about 12.5% in teenagers to 4% in women aged 30 to 34 years.[21] Teenagers are thus especially vulnerable to long-term complications of STD exposure. Current US figures estimate that 3% of all teenagers acquire PID in a given year.

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Both a past history of gonococcal infection and a past history of PID are risk factors for the development of subsequent PID.[22,23] In one study, 41.7% of PID cases versus 14% of control women had had previous PID and the risk from previous disease was independent.[22] The first episode of infection is said to permanently damage tubal defence mechanisms and render the adnexae susceptible to further damage through subsequent infections.[22]

THE RELATIONSHIP OF PID AND INFERTILITY

A single episode of gonococcal PID is reported to lead to a 6% chance of infertility. [24] One study found that approximately 10% of women who recover from a single episode of PID become infertile whatever the etiologic agent.[8]

Infertility is highly correlated with prior PID,[25] and PID contributes to infertility in more than 20% of women presenting to infertility clinics.[7] The sequelae of acute salpingitis are the most common cause of female infertility[26] and post-infectious tubal damage has been cited as the etiologic factor in 30 to 40% of cases of female infertility in one report.[27] Patients presenting to infertility clinics may not recall having had an illness consistent with PID. In one study, only one-third of patients reported that they had had PID although prior PID had been documented by hysterosalpingogram and/or laparoscopy.[25]

In a prospective study, women who had had one episode of tubal infection had a 12.8% rate of infertility; 35% of those with two infections were sterile and after three or more infections, infertility was found in 75% of women.[28] After one or more tubal infections 62.4% of women will go on to have an intrauterine pregnancy, 4.1% will have an ectopic pregnancy and 12.6% will be involuntarily infertile secondary to tubal occlusion.[27]

If the current rates of infection among teenagers continue, it can be estimated that about 20% of all young women will acquire PID before the age of 20.[29] If nearly a quarter of these become infertile, we can expect 4 to 5% of the next reproductive cohort of women to have tubal infertility.[29] Based on the current background infertility rate of 15%, roughly a 33% increase in infertility could occur.[29]

PREVENTION OF INFERTILITY

Most population-based programs directed towards true prevention of infertility attempt to deal with STD prevention. Interest in these programs has been augmented by concern over the transmission of HIV infection, especially among young people. Both STDs and the AIDS virus can be prevented through use of condoms during sexual intercourse. Barrier methods have the added benefit of pregnancy prevention.

When STDs are suspected, prompt, effective therapy must be initiated. The availability of treatment services is sometimes limited for young women who are fearful of parental disclosure, who may be unfamiliar with how to seek care, and who may face financial obstacles to STD treatment. Public health's involvement in STD control includes contact tracing to reduce carriers in the community. Contact tracing is limited by the poor reporting of STDs and by personnel and financial constraints.

Primary Prevention of Sexually Transmitted Diseases

Stone, Grimes and Magder have discussed the primary prevention of sexually transmitted diseases.[30] Primary preventive methods relevant to gonorrhoea and chlamydia include modifying the selection of sexual partners, avoiding certain sexual practices and using mechanical or chemical barriers during intercourse.

The major strategy for PID prevention has been the management of uncomplicated infections due to gonorrhoea and chlamydia as well as the investigation, diagnosis, contact tracing and treatment of those so infected.

Several studies have suggested that the use of barrier contraceptives (condom, diaphragm, or spermicide) lowers a woman's risk of acquiring cervical gonorrhoea,[31] PID,[32] and cervical dysplasia,[33] which behaves epidemiologically like an STD.[34] In one study, the risk of PID in current users of barrier methods (condom, diaphragm with jelly, spermicidal foams, jellies and creams) was 0.6 (95% C.L. (0.5-0.9).[35] Barrier users were 70% less likely to develop PID than their IUD using counterparts.[32]

The Control of Gonorrhoea and Chlamydia

In most developed countries, control of gonorrhoea currently focuses on early diagnosis and treatment, while emphasis on prevention of the disease varies. Clearly, early diagnosis and treatment are appropriate and potentially effective where techniques for diagnosis and therapy are advanced.

General measures of gonorrhoea control include: disease surveillance; directed case finding by contact tracing; direct screening of high-risk groups; therapy monitoring; and laboratory surveillance for penicillinase-producing *N. gonorrhoeae* and isolates from treatment failures.[36]

Case finding priorities for gonorrhoea include: contacts of women with pelvic inflammatory disease; contacts of men or women with penicillinase-producing *N. gonorrhoeae* or other resistant isolates; contacts of men with asymptomatic infection; contacts of disseminated gonococcal infection cases; and disease repeaters.[36]

Chlamydial infections require longer therapy to eradicate than gonococcal infections. Given a longer incubation period during which some patients may be infectious, a high rate of asymptomatic infection, and a persistent carrier state, a large reservoir of transmitters of *C. trachomatis* infection undoubtedly exists.[37]

On the other hand, the efficiency of transmission of *C. trachomatis* appears to be less than that of *N. gonorrhoeae*.[37] Since as many as 29 to 48 percent of women with a gonococcal infection may also have a concomitant chlamydial infection and most are treated for their gonococcal infection with either a single-dose penicillin regimen or five days of tetracycline, inadequate, tempo-

rarily suppressive treatment of chlamydial infection is undoubtedly common.[38] All these points have important implications for control programs.

Secondary Prevention of STDs

Screening and case-finding of STDs is not generally cost- effective although it is effective among high risk groups with a gonococcal prevalence rate of greater than 5%.[39] All at-risk hospitalized and prenatal women should be screened for gonorrhoea.[40] The role of the urban community hospital in gonococcal surveillance has been stressed by Rendtorff and Packer.[41] When large numbers of infected women are identified and treated for STD, a decline in PID hospitalization occurs.[23,41] Although, infections with STD, may be a behaviourial problem, a surveillance program for gonorrhoea in women may nevertheless stop lower genital infections from progressing to PID.

In many settings, asymptomatic persons should be screened for *C. trachomatis.* When resources for testing for *C. trachomatis* are rationed, highest priority for testing should be for patients, especially women, in sexually transmitted disease clinics who otherwise would not be offered antichlamydial treatment.[42]

Where resources allow, priority for screening should be based on youth, low socioeconomic status, female gender, number of sexual partners, and absence of one of the indications of anti-chlamydial treatment noted above. At a minimum, pregnant women should be tested at the first prenatal visit if they are under 20 years old, unmarried, have other sexually transmitted diseases, have several sexual partners or have males partners who are not monogamous.

HOW EFFECTIVE WILL STD CONTROL BE IN INFERTILITY PREVENTION?

The reduction of infertility through the implementation of effective STD prevention programs is a logical and attractive conclusion from this review. Unfortunately, much is still unknown with respect to the effectiveness of such programs in preventing infertility.

We know that an efficacious STD-related infertility prevention program should include: vigorous contact tracing with appropriate therapy and epidemiological treatment of contacts;[43-45] use of mechanical and chemical barriers to STDs;[30,36,46] patient education to increase compliance with STD therapy;[36,45,47,48] screening for STDs in certain high risk groups;[45,48,49] and the administration of antimicrobial therapy according to standard protocols.[43,48] We are less certain, however, on how to implement these components.

While many components of a program that might reduce STD-related infertility operate in public health units,[45,50] in schools,[29,48] within physician offices[51-53] and in youth clinics,[29,48] there are no fully operational, coordinated community or province-wide programs. Provinces using model standards for STD control have gone some way, within the domain of public health, to establishing a programmatic approach to STDs.[42,43]

Research priorities for the prevention of STDs and STD-related infertility include: the development of effective screening programs for gonorrhoea and chlamydia; epidemiological studies of the prevalence of STDs in Canadian men and women, and of the occurrence of PID; evaluation of the impact of comprehensive contact tracing on the occurrence of STDs, PID and ectopic pregnancy; the efficacy of various preventive strategies; and basic laboratory research to help develop inexpensive, sensitive, and specific diagnostic tests to detect chlamydial infection.[54]

Until the answers to these questions are known, physicians should follow the principles of community effectiveness for STD control described by Tugwell and colleagues[55] and modified for practitioners: diagnose well, use efficacious therapy, comply with reporting and contact tracing requirements, and ensure patient compliance with treatment and follow-up. □

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Continued on page 196.

Epidemiology of STDs in Nova Scotia

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Halifax, N.S.

This paper will present a brief descriptive overview of the epidemiology of sexually transmitted diseases (STDs) in Nova Scotia. Studying their epidemiology assists us in assessing the health status of the population and ultimately, helps us set priorities for health. Ideally, one could use the data to assess specific effects of programs directed towards the control of STDs. Chlamydia, gonorrhoea, HIV, and hepatitis B will be discussed in terms of their change in incidence over time, as well as their age and sex distributions. These data are derived largely from the notifiable diseases database which compiles data received from laboratories and some physicians.

METHODS

Data are collected routinely by the health units on notifiable diseases identified in the regulations of the Health Act. Reporting is excellent for most laboratories but poor from physicians unless the public eye is focused on a particular disease such as with meningococcal disease in the winter and spring of 1992. Therefore, there is serious under-reporting for those diseases which do not require laboratory confirmation. As well, there are biases which affect testing and therefore reporting. For example, it is easier to test females than males for chlamydia and, although there is under-reporting of disease for both sexes, it is likely substantially greater for males. Reporting varies by geographic area and has varied over time. Minor changes in the data must therefore be ignored as they may be due to many of the above factors.

All of the ratios presented represent the number of events per population and not the number of individuals who have suffered one or more events. For example, the pregnancy ratio per 1,000 females will include women who have had more than one pregnancy per year, e.g., a spontaneous abortion followed by a delivery, and women who have had only one pregnancy per year. Data from the communicable disease surveillance system are based on unique identifiers and it is impossible to tell if a reported event represents a recurrent infection in someone previously reported, or a new infection in an individual not previously reported. The data presented here are therefore more accurately described as ratios rather than rates.

RESULTS

Figure 1 illustrates the number of reported cases of chlamydia and gonorrhoea from 1981 to 1991.[1] As has been observed elsewhere, ratios for gonorrhoea have dropped markedly. Chlamydia, on the other hand, has increased sharply, peaking in 1988 and then dropping slightly. Much of the increase in chlamydia over the decade does not represent a real increase in its incidence but improvement and increased availability of laboratory methods as well as better suspicion of the diagnosis. The peak in 1988 may well have represented the indiscriminate use of laboratory tests without confirmation, especially in asymptomatic women who have unnecessarily received a chlamydia test with every vaginal examination. On the other hand, there are many diagnoses made clinically which are probably correct but not reported to the Department of Health and therefore not reflected in these data.

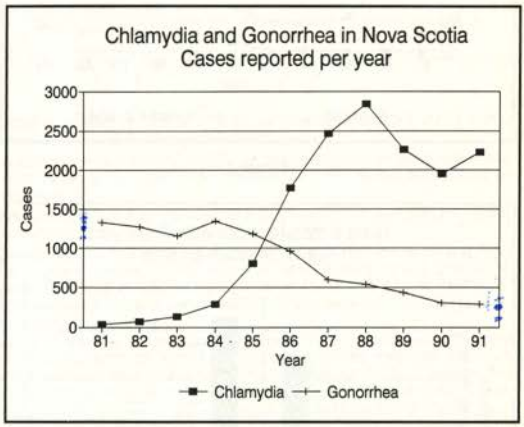

Figure 1

Figure 2 demonstrates increases in both acute hepatitis B and AIDS over the last 11 years.[1] Except for a peak in hepatitis B cases from 1987 to 1989, the cases have been increasing by approximately 10 per year. The peak is mostly explained by a large outbreak of hepatitis B among illicit injection-drug users in Cape Breton. The number of cases of AIDS has been increasing very slowly and has levelled off over the last few years, probably partly due to the use of AZT. Unlike chlamydia and gonorrhoea, these diseases require laboratory confirmation and are therefore considered to be fairly accurate.

Figures 3 and 4 describe chlamydia according to age and sex distributions.[1] In Figure 3, the years 1985 and 1986 as well 1990 and 1991 were combined to increase the sample size. There has been a shift in the age distribution for females. In 1985-86, females under the age of 20 represented only 30% of cases as opposed to 36% of cases in 1990-91. In the same age group, the number of cases has increased from 519 in 1985-86 to

*Provincial Epidemiologist, Nova Scotia Department of Health.

1146 in 1990 - 91. This, in combination with the shift in age distribution to younger females, suggests that there may be an increase in sexual activity in this age group. However, one cannot rule out the impact of changes in the degree of under-reporting or tendencies of physicians to order laboratory tests according to age. Such biases *may* account for the differences seen. Figure 4 indicates that males are diagnosed at a relatively older age than females.

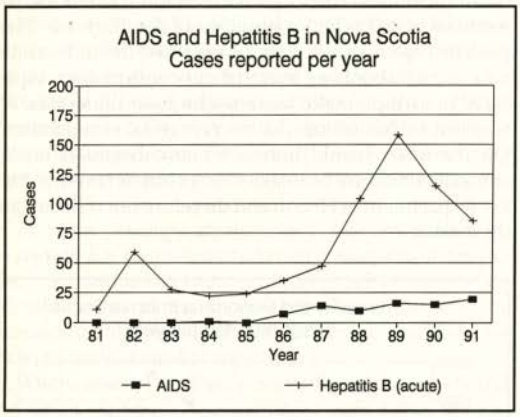

Figure 2

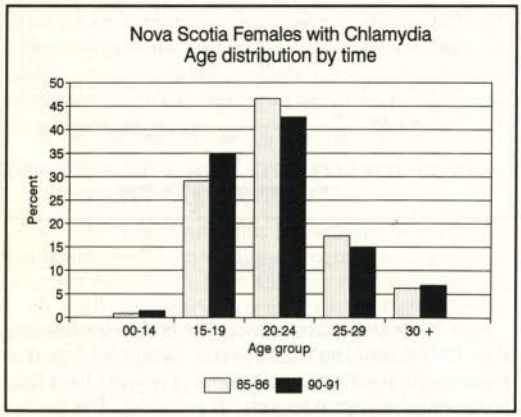

Figure 3

Figure 5 presents pregnancies, therapeutic abortions and total deliveries per 1000 women age 15 to 19 in Nova Scotia.[2] Pregnancies are defined as all events which represent a termination of pregnancy. These are called abortions if the gestational age is less than 22 weeks and deliveries if 22 weeks or greater. Abortions are either therapeutic (legally-induced), spontaneous or other miscellaneous conditions terminating the pregnancy before 22 weeks. Total deliveries comprise live births and still births. Live births represent the vast majority.

The total delivery ratio and pregnancy ratio declined gradually throughout the 1980s, reached a nadir in 1987, and then began to climb. From these data, one cannot determine whether this recent increase is due to an increase in the unintended or intended pregnancy ratio or a combination of the two. The therapeutic abortion ratio is relatively stable about 13 per 1000 women in ages 15 to 19. In other words, one out of every 77 adolescent girls aged 15 to 19 will undergo a therapeutic abortion each year.

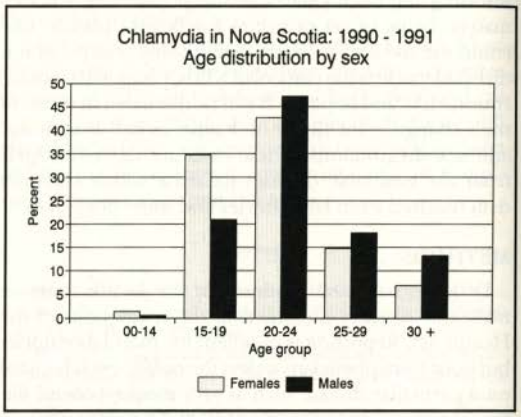

Figure 4

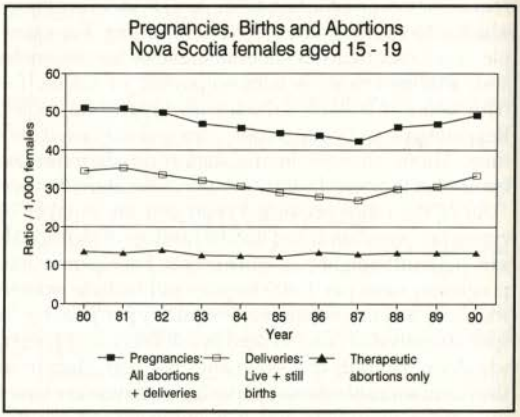

Figure 5

DISCUSSION

Sexually transmitted diseases are a major problem in Nova Scotia and have an impact especially on teenagers and young adults in our society. In addition to the morbidity resulting from the sexually transmitted diseases themselves, the sequelae of these diseases, such as infertility, abortion, and ectopic pregnancy for chlamydia, and chronic hepatitis or hepatocellular carcinoma from hepatitis B, represent serious consequences for those affected.

The communicable disease surveillance system is an example of a "passive surveillance" system, i.e., a system

in which no special effort is expended to collect disease information which has not been reported to the Department of Health. Therefore, descriptive data such as these are obtained simply and at relatively low cost. An "active surveillance" system is one in which human resources are dedicated to ensuring that data are reported. An example is a hospital infection control program with a dedicated infection control nurse who actively seeks out nosocomial infections. An example in public health is a sentinel physician program in which designated physicians are asked to provide more detailed information about certain diseases than their colleagues.

Although descriptive data from surveillance systems can provide insights into temporal patterns of disease, they are inadequate for investigating causal pathways, and do not capture the level of detail needed to understand physician and client behaviour. There will always be need for special surveys which assess knowledge, attitudes and behaviours and which assess the impact of public health interventions such as school-based clinics or sessions designed to increase decision-making skills in adolescents.

Under-reporting remains the greatest barrier to the utility of the data from the Nova Scotia Communicable Disease Surveillance System, especially for conditions not requiring laboratory confirmation. Although difficult in any case, without physician cooperation and consistent policies of follow-up and control across the province, it will be impossible to control sexually transmitted diseases, their sequelae, and other diseases which have consequences for the public health. □

ACKNOWLEDGEMENT

I would like to acknowledge the contribution of Heather Young, assistant Director of Nursing at the Department of Health, for compiling the data related to pregnancies.

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SEARLE PATIENT PROMISE

CYTOTEC® (misoprostol) 100 mcg 200 mcg

CYTOTEC BRIEF PRESCRIBING INFORMATION

Therapeutic Classification

Cytoprotective Agent

INDICATION:
CYTOTEC (misoprostol) is indicated in the treatment and prevention of NSAID-induced gastric ulcers (defined as ≥ 0.3 cm in diameter) and in the treatment of duodenal ulcers.

CONTRAINDICATIONS:
Known sensitivity to prostaglandins, prostaglandin analogues, or excipients (micro-crystalline and hydroxypropyl methylcellulose, sodium starch and hydrogenated castor oil).

Contraindicated in pregnancy.

Women should be advised not to become pregnant while taking CYTOTEC. If pregnancy is suspected, use of the product should be discontinued and the pregnancy followed very closely (weekly) for the next four weeks.

WARNINGS:
Women of childbearing potential should employ adequate contraception (i.e. oral or intrauterine devices) while receiving CYTOTEC. (See CONTRAINDICATIONS.)

Nursing Mothers: It is unlikely that CYTOTEC is excreted in human milk since it is rapidly metabolized throughout the body. However, it is not known if the active metabolite (misoprostol acid) is excreted in human milk. Therefore, CYTOTEC should not be administered to nursing mothers because the potential excretion of misoprostol acid could cause significant diarrhea in nursing infants.

Pediatric Use: Safety and effectiveness in patients below the age of 18 have not been established.

PRECAUTIONS:
Selection of Patients: Before treatment is undertaken, a positive diagnosis of duodenal ulcer or NSAID-induced gastric ulcer should be made. In addition, the general health of the patient should be considered. Misoprostol is rapidly metabolized by most body tissues to inactive metabolites. Nevertheless, caution should be exercised when patients have impairment of renal or hepatic function. Experience to date with such patients is limited.

Diarrhea: Rare instances of profound diarrhea leading to severe dehydration have been reported. Patients with an underlying condition such as irritable bowel disease, or those in whom dehydration were it to occur, would be dangerous, should be monitored carefully if CYTOTEC is prescribed.

ADVERSE REACTIONS:
Gastrointestinal: In subjects receiving CYTOTEC (misoprostol) 400 or 800 mcg daily in clinical trials, the most frequent gastrointestinal adverse events were diarrhea, abdominal pain and flatulence. The average incidences of these events were 11.4%, 6.8% and 2.9%, respectively. In clinical trials using a dosage regimen of 400 mcg bid, the incidence of diarrhea was 12.6%. The events were usually transient and mild to moderate in severity.

Diarrhea, when it occurred, usually developed early in the course of therapy, was self-limiting and required discontinuation of CYTOTEC in less than 2% of the patients. The incidence of diarrhea can be minimized by adjusting the dose of CYTOTEC, by administering after food and by avoiding coadministration of CYTOTEC with magnesium-containing antacids.

Gynecological: Women who received CYTOTEC during clinical trials reported the following gynecological disorders: spotting (0.7%), cramps (0.6%), hypermenorrhea (0.5%), menstral disorder (0.3%) and dysmenorrhea (0.1%).

Elderly: There were no significant difference in the safety profile of CYTOTEC in approximately 500 ulcer patients who were 65 years of age or older compared with younger patients.

Incidence greater than 1%: In clinical trials, the following adverse reactions were reported by more than 1% of the subjects receiving CYTOTEC and may be casually related to the drug: nausea (3.2%), headache (2.4%), dyspepsia (2%), vomiting (1.3%) and constipation (1.1%). However, there were no clinically significant differences between the incidences of these events for CYTOTEC and placebo.

DOSAGE AND ADMINISTRATION:
Treatment and Prevention of NSAID-Induced Gastric Ulcers: The recommended adult oral dosage of CYTOTEC for the prevention and treatment of NSAID-induced gastric ulcer is 400 to 800 mcg a day in divided doses. NSAIDs should be taken according to the schedule prescribed by the physician. When appropriate CYTOTEC and NSAIDs are to be taken simultaneously. CYTOTEC should be taken after food.

Duodenal Ulcer: The recommended adult oral dosage of CYTOTEC (misoprostol) for duodenal ulcer is 800mcg per day for 4 weeks in two or four equally divided doses (i.e. 200 mcg qid or 400 mcg bid). The last dose should be taken at bedtime. Antacids (aluminum based) may be used as needed for relief of pain. Treatment should be continued for a total of 4 weeks unless healing in less time has been documented by endoscopic examination. In the small number of patients who may not have fully healed after 4 weeks, therapy with CYTOTEC may be continued for a further 4 weeks.

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CYTOTEC 200 mcg tablets are white to off-white – scored, hexagonal with SEARLE 1461 engraved on one side.

CYTOTEC 100 mcg tablets are white to off-white, round tablets with SEARLE engraved on one side and CYTOTEC on the other.

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THE IMPORTANCE OF STD CONTROL FOR THE PREVENTION OF INFERTILITY

Continued from page 192.

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THE MEDICAL SOCIETY OF NOVA SCOTIA

1992 NOTICE OF MOTION
RE:
BY-LAW AMENDMENTS

The following proposed By-Laws Amendments will be presented at Council in November.

EXISTING	PROPOSED
	8.4 Attendance At Meetings
	8.4.1 Committee members are required to attend two thirds of all meetings and not to miss more than two meetings consecutively. Failure to meet these requirements may be cause for the Executive Committee to replace a committee member.
10 OFFICERS and OFFICIALS	**10 OFFICERS and OFFICIALS**
10.1.1. No full time appointed Official shall have a vote, by virtue of his appointment at any meetings of The Society or of its Commitees. This shall not prevent such an Official from exercising his right to vote as an Ordinary Member of The Society.	**10.1.1.** With the exception of section 12.6.1 of these By-Laws, no full time appointed Official shall have a vote, by virtue of his appointment at any meetings of The Society or of its Committees. This shall not prevent such an Official from exercising his right to vote as an Ordinary Member of The Society.
11.7.1 The Executive Director	**11.7.1 The Executive Director**
a. The Executive Director shall be appointed by the Executive Committee on recommendation of the Officers. A committee of the President, President-Elect and Past President shall determine the salary and benefits of the Executive Director.	a. The Executive Director shall be appointed by the Executive Committee on recommendation of the Officers.
b. The Executive Director shall be accountable to the President and the Chairman of the Executive Committee. He may delegate portions of Society staff functions to members of the staff, but may not delegate his overall responsibility or accountability for the proper conduct of Society affairs.	b. The Executive Director shall be accountable to to the Staffing Committee. He may delegate portions of Society staff functions to members of the staff, but may not delegate his overall responsibility or accountability for the proper conduct of Society affairs.

EXISTING	PROPOSED
d. It shall be the responsibility of the Officers Committee to develop the terms of reference for the position of Executive Director. These shall encompass the conditions set out above and shall constitute part of the terms of his appontment.	d. It shall be the responsibility of the Staffing Committee to develop the terms of reference for the position of Executive Director. These shall encompass the conditions set out above and shall constitute part of the terms of his appontment.
12..2 Appointment of Statutory Committees:	**12..2 Appointment of Statutory Committees:**
Statutory Committees shall be: The Nominating Committee The Executive Committee The Finance Committee The Officers' Committee	Statutory Committees shall be: The Nominating Committee The Executive Committee The Finance Commitee The Officers' Committee The Staffing Committee
12.5 The Finance Committee:	**12.5 The Finance Committee**
12.5.1 Terms of Reference	**12.5.1 Terms of Reference**
The Committee shall consist of the Treasurer as Chairman and four voting members of The Society approved by the Executive Committee. The Committee shall meet at least twice yearly. Two voting members of the Committee and the Chairman shall constitute a quorum. The Committee shall be charged with a special duty of studying the immediate and long term financial needs of The Society, including the establishing of reserve funds, and shall present in its Annual Report to Council appropriate fiscal policies to meet these needs. The Committee shall follow Society Investment Policy in managing Society investments, and may seek professional investment counsel in directing the policy. When directed the Committee shall assess the financial implications of programs and projects and make recommendations to the Executive Committee. The Committee shall prepare for approval by the Executive Committee a budget for the ensuing year indicating distribution of monies to be made available for all purposes. This Committee shall report regularly to The Society's Executive Committee. The Committee shall report annually to Council. Its report shall cover all financial activities of The Society during the previous calendar year and shall indicate the revenues necessary for	Delete last sentence. "The Committee shall be responsible to the Executive Committee for classification of positions, salaries, management and general welfare of the staff of The Society."

EXISTING	PROPOSED
12.5.1 continued	**12.5.1 continued**
the subsequent year. The adoption by Council of the annual reports of the Finance Committee and of the Auditors shall validate all financial transactions of The Executive Committee, the Finance Committee and other Committees of The Society during the year covered. The Committee shall recommend the appointment of Auditors to the Council annually. The Committee shall check all expenditures of funds to ascertain that they are properly made, and bring to the attention of the Executive Committee any likely over-expenditures, misappropriation, misuse or discrepancy in The Society's funds. The Committee shall be responsible to the Executive Committee for classification of positions, salaries, management and general welfare of the staff of The Society.	
12.6 Mediation Committee	**12.6 Mediation Committee** -renumber 12.7
12.7 Standing Committees	**12.7 Standing Committees** - renumber 12.8
12.8 Special Committees	**12.8 Special Committees** - renumber 12.9
12.9 Reports of Committees	**12.9 Reports of Committees** - renumber 12.10
12.10 Limitations of Committees Re: Finances	**12.10 Limitations of Committees Re: Finances** - renumber 12.11
	12.6 Staffing Committee
	12.6.1 The Staffing Committee shall consist of the President as Chairman, the Past President, the President-Elect, the Treasurer and the Executive Director, who shall be a voting member except when in the opinion of the Chairman, there is conflict of interest. It shall meet at least twice a year.
	12.6.2 The Staffing Committee shall be responsible for the classification of positions, salaries, and the general welfare of the staff of The Society.
	12.6.3 The Staffing Committee shall, after due consultation with the Finance Committee, be responsible for recommending appropriate changes in staffing levels and overall salaries, and for determining and reporting the financial effects of such changes.

EXISTING	PROPOSED
	12.6 continued
	12.6.4 The Staffing Committee shall be responsible for the resolution of any grievance that may arise between the Executive Director and any member of the staff. In such matters the Executive Director shall not vote. The decision of the Committee is final.
	12.6.5. The Staffing Committee shall be responsible for all matters relating to the employment of the Executive Director, including his salary and benefits. It shall also be responsible for an annual written evaluation, the results of which shall be given to the Executive Director, and the results shall be communicated to the Executive Committee.
	12.6.6 The Staffing Committee shall report to the Officers' Committee.

The Finance Committee has requested that the definitions of the various types of membership in The Society be removed from the body of the By-Laws and incorporated into the Rules and Regulations. The Executive Committee at its meeting of September 26, 1992 considered this matter and asked the By-Laws Committee to draw up the necessary amendments, which are presented here. Please note that section 6.1.4 of the By-Laws states that "All membership dues shall be set at the Annual Meeting." The Executive Committee will be empowered to adjust the definitions of membership categories but will not be able to add or subtract membership categories, or to change dues or voting rights.

6.2 Ordinary Members	**6.2 Voting**
6.2.1 A member in good standing who is licensed to practice medicine in Nova Scotia and who is resident in the province at the time of application	**6.2.1** The membership categories holding full voting power within The Society shall be: First Year of Practice, Ordinary, Senior, Post Graduate, Intern/Resident, Retired, Non-Practising Scientist, and Over Sixty-Five.

Sections 6.3 through 6.15.1 which contain the definition of membership categories should be deleted from the By-Laws and moved to Rules and Regulations as 5 through 5.15.1, and sections 6.16 through 6.17.3 must be renumbered downward.

6.16 Resignation from Membership:	**Renumber 6.3**
6.16.1 Resignation may be effected by giving notice to the Executive Director of the Society.	**Renumber 6.3.1**

EXISTING	PROPOSED
6.17 Suspension of Membership	**Renumber 6.4**
6.17.1 Any member whose license to practice medicine is revoked by the Provincial Medical Board of Nova Scotia under provisions of the Medical Act loses ipso facto his right to membership in the Society and thereby forfeits all his rights and privileges pertaining to membership in the Society.	**Renumber 6.4.1**
6.17.2 Any member whose license is temporarily suspended by the Provincial Medical Board of Nova Scotia for reasons of abuse of alcohol, drugs or other substances may retain membership in the Society as a Courtesy Member in accordance with section 6.5 of these By-Laws.	**Renumber 6.4.2**
6.17.3 Any member losing his membership in the Society pursuant to section 6.17 shall, subject to conditions imposed by the Executive Committee and provided that he has regained his license to practice medicine, be restored to membership upon resolution of the Executive Committee.	**Renumber 6.4.3**

An editorial amendment to 6.4.2 and 6.4.3 is necessary because of the renumbering.

6.4.2 (Previously 6.17.2)	**6.4.2** Any member whose license is temporarily suspended by the Provincial Medical Board of Nova Scotia for reasons of abuse of alcohol, drugs or other substances may retain membership in The Society as a Courtesy member.
6.4.3 (Previously 6.17.3)	**6.4.3** The same as the previous 6.17.3 except change Section 6.17 to read Section 6.4.

The Rules and Regulations need to be appended with the definitions of the membership categories (previously in the By-Laws as 6.3 through 6.15.1). The By-Laws Committee has taken the liberty of making editorial adjustments to the original text of the definitions where necessary.

EXISTING	PROPOSED
	Addition to Rules and Regulations
	5. MEMBERSHIP
	5.1 The categories of membership shall be defined as: -
	5.2 Ordinary Members
	5.2.1 Members in good standing who are licensed to practice medicine in Nova Scotia and who are resident in the Province at the time of application. Ordinary members may enjoy all rights and privileges of The Society.
6.3 Senior Members:	**5.3 Senior Members**
6.3.1 Any member of The Society in good standing for the immediately preceding ten year period who has attained the age of sixty-five years is eligible to be nominated for Senior Membership by an Ordinary Member or by any Branch of The Society. He may be elected only by unanimous approval of the members of the Executive Committee in session present and voting. No more than four may be elected in any one year. Senior Members shall enjoy all the rights and privileges of The Society but shall not be required to pay any fee. Senior Membership so approved shall be conferred by the President of The Society at the time of the Annual Meeting or at any Special Meeting, subject always to the rules affecting Special Meetings.	**5.3.1** Members of The Society in good standing for the immediately preceding ten year period who have attained the age of sixty-five years are eligible to be nominated for Senior Membership by an Ordinary Member or by any Branch of The Society. They may be elected only by unanimous approval of the members of the Executive Committee in session present and voting. No more than four may be elected in any one year. Senior Memers shall enjoy all the rights and privileges of The Society but shall not be required to pay any fee. Senior Membership so approved shall be conferred by the President of The Society at the time of the Annual Meeting or at any Special Meeting, subject always to the rules affecting Special Meetings.
6.4 Honorary Members:	**5.4 Honorary Members:**
6.4.1 Honorary Members shall be members of the profession or others who have distinguished themselves by their attainments in medical or allied sciences who have rendered signal service to this Society. Recommendations for election to Honorary Membership shall come solely from the Executive Committee. Honorary Members shall enjoy all the privileges of The Society with the exception of the power to vote or hold office and shall not be required to pay any fee.	**5.4.1** Honorary Members shall be members of the profession or others who have distinguished themselves by their attainments in medical or allied sciences who have rendered signal service to this Society. Recommendations for election to Honorary Membership shall come solely from the Executive Committee. Honorary Members shall enjoy all the privileges of The Society with the exception of the power to vote or hold office and shall not be required to pay any fee.

EXISTING	PROPOSED
6.5 Courtesy Members:	**5.5 Courtesy Members:**
6.5.1 A member in good standing in The Society whose license has been temporarily revoked by the Provincial Medical Board pending rehabilitation may retain membership in the Society as a Courtesy Member. Courtesy members may attend meetings but may not vote.	**5..5.1** Members in good standing in The Society whose licenses have been temporarily revoked by the Provincial Medical Board pending rehabilitation may retain membership in The Society as a Courtesy Members. Courtesy members may attend meetings but may not vote.
6.6 Medical Student Members:	**5.6 Medical Student Members:**
6.6.1 All medical students enrolled in the Faculty of Medicine, Dalhousie University are recognized as Medical Student Members of The Medical Society. They may enjoy the rights and privileges of the Medical Society but may not hold office or vote except as set out in Chapters 9 and 12 of the By-Laws.	**5.6.1** All medical students enrolled in the Faculty of Medicine, Dalhousie University are recognized as Medical Student Members of the Medical Society. They may enjoy the rights and privileges of The Medical Society but may not hold office or vote except as set out in sections 9.2.1(9) and 12.3.2.1 (ii) of the By-Laws.
6.7 First Year Practice Members:	**5.7 First Year Practice Members:**
The following definitions apply:	
6.7.1 An Ordinary Member in his/her first year of practice following initial licensing in Canada.	**5.7.1** Ordinary Members in their first year of practice following initial licensing in Canada.
6.8 Post Graduate Members:	**5.8 Post Graduate Members:**
6.8.1 An Ordinary Member undertaking post-graduate training program of not less than one year, either through the Dalhousie Program but externally funded or through another program.	**5.8.1** Ordinary Members undertaking post-graduate training program of not less than one year, either through the Dalhousie Program but externally funded or through another program.
6.9 Interne/Resident Members:	**5.9 Interne/Resident Members:**
6.9.1 An Ordinary Member undertaking post graduate training in the Dalhousie Program.	**5.9.1** Ordinary Members undertaking post graduate training in the Dalhousie Program.
6.10 Non Resident Members (Nova Scotia - only):	**5.10** Non Resident Members (Nova Scotia - only):
6.10.1 A member residing in a province of Canada other than Nova Scotia, or outside Canada and not a C.M.A. member.	**5.10.1** Members residing in a province of Canada other than Nova Scotia, or outside Canada and not a Canadian Medical Association member.

EXISTING	PROPOSED
6.11 Non Resident Members (Conjoint):	**5.11 Non Resident Members (Conjoint):**
6.11.1 A member residing outside Canada or Canadian Territory beyond the jurisdiction of any Division.	**5.11.1** Members residing outside Canada or Canadian Territory beyond the jurisdiction of any Division of The Canadian Medical Association.
6.12 Retired Members:	**5.12 Retired Members:**
6.12.1 An Ordinary Member no longer in active practice, defined as less than ten hours/week in the practice of medicine.	**5.12.1** Ordinary Members no longer in active practice, defined as less than ten hours per week in the practice of medicine.
6.13 Non-Practising Scientist Members:	**5.13 Non-Practising Scientist Members:**
6.13.1 An Ordinary Member employed as a basic scientist in Dalhousie University and not practising medicine except on an emergency basis.	**5.13.1** Ordinary Members employed as basic scientists in Dalhousie University and not practising medicine except on an emergency basis.
6.14 Over Sixty-Five Members:	**5.14 Over Sixty-Five Members:**
6.14.1 An Ordinary Member not retired; effective the fiscal year following the fiscal year in which the age of 65 was attained, the dues for this category to be 50 percent of ordinary dues.	**5.14.1** Ordinary Members not retired; effective the fiscal year following the fiscal year in which the age of sixty-five was attained.
6.15 Temporary Members	**5.15 Temporary Members**
6.15.1 Temporary Membership in the Society may be granted to physicians temporarily licensed to practice in Nova Scotia for a pre-defined purpose. Such members may attend meetings of the Society but may not vote.	**5.15.1** Temporary Membership in The Society may be granted to physicians temporarily licensed to practice in Nova Scotia for a pre-defined purpose. Such members may attend meetings of the Society but may not vote.

The Society's By-Laws will be further amended to coincide with changes to The Society's Executive Committee resulting from Council's decision November 1992.

An Appreciation

DR. TADEUSZ K. KRZYSKI

Dr. Tadeusz K. Krzyski was born in Warsaw, Poland in 1919, the son of an Army Doctor. He started his medical studies in 1938 in Cracow, Poland. When the Second World War was starting, he was conscripted and served in the Polish Army in Poland, France and Great Britain. After the war, he finished his medical studies at the Polish School of Medicine at Edinburgh University, Scotland in 1949. In 1953 he came to Canada and started his work as a Staff Physician and Assistant Medical Superintendent at Point Edward Hospital until its closure in 1973. He then practised General Medicine in the Gaum Clinic in Sydney until his retirement in 1991.

He was active in the Sydney Militia Medical Company. In 1978 he was appointed Honorary Lieutenant-Colonel of the 35th Medical Company in Sydney. For his service in the Militia he was awarded the Canada Decoration.

A soft-spoken unassuming man he was loved by his patients for his gentleness, graceful bed manners, dedication and compassion. During his long professional life as a TB Physician and General Practitioner, he earned the well deserved-esteem of the citizens of Sydney and industrial Cape Breton. Tadeusz loved his work and had the admiration and respect of his patients and colleagues alike.

Ted, as he was known, was a devoted husband and family man. He managed to find time for varied hobbies as woodworking, gardening, music, reading and travel.

He died unexpectedly in July. He is survived by his wife, Halina Krystyna, now residing in Bedford, N.S., two sons, four grandchildren and one sister.

Dr. Janusz Wawrzyszyn and Dr. A. Freeman □

OBITUARIES

Dr. Ahmet Slomic, (67) of Amherst, Nova Scotia died on August 5, 1992. Born in Yugoslavia, he received his medical degree from Sarajevo University in 1954. He later received his specialization in Radiology and Neurophysiology from the University of Paris. In 1970 he came to Canada, serving on the faculty of Laval University and in 1979, moved to Amherst, Nova Scotia where he became radiologist at the Highland View Regional Hospital. He is survived by his wife, a daughter and a son. The *Journal* extends sincere sympathy to his family.

Dr. Mary Jane Sullivan, (41) of Bridgewater, Nova Scotia died on September 16, 1992. Born in Saint John, New Brunswick she received her medical degree from Memorial University, in 1979; she practised in Newfoundland and completed her postgraduate work in psychiatry in Newfoundland in 1988. Having spent many summers in Chester, Lunenburg County, she decided to settle in Bridgewater. She is survived by her husband and three daughters to whom the *Journal* extends sincere sympathy.

Dr. John B. McGregor Shaw, (47) of Halifax, Nova Scotia died on September 29, 1992. Born in Montreal, he received his medical degree from Dalhousie University in 1975. He was a member of The Medical Society of Nova Scotia and the Canadian Medical Association, He practised medicine in the Clayton Park area with the Northwest Medical Group. He is survived by his wife and three daughters. We offer sincere sympathy to his family. □

LOCUM REQUIRED

MID-JANUARY TO MID-APRIL 1993

Please Contact:

Dr. H.G. Khalsa
Glace Bay, N.S.

Tel. 902-849-2929

1992 Annual Meeting Reminder

The 1992 Annual Meeting of The Medical Society of Nova Scotia will be held November 19-21, 1992. This year, this event will be held at the Prince George Hotel and the World Trade & Convention Centre in Halifax.

NON-SYSTEMIC

PSULCRATE®

sucralfate/NORDIC

PRESCRIBING INFORMATION

THERAPEUTIC CLASSIFICATION

Gastroduodenal Cytoprotective Agent

ACTIONS AND CLINICAL PHARMACOLOGY

SULCRATE® (sucralfate) exerts a generalized gastric cytoprotective effect by enhancing natural mucosal defence mechanisms. Studies conducted in animals and clinical trials in humans have demonstrated that sucralfate can protect the gastric mucosa against various irritants such as alcohol, ASA, hydrochloric acid, sodium hydroxide or sodium taurocholate.

In addition, sucralfate has been demonstrated to have a greater affinity for ulcerated gastric or duodenal mucosa than for non-ulcerated mucosa.

Sucralfate produces an adherent and cytoprotective barrier at the ulcer site. This barrier protects the ulcer site from the potential ulcerogenic properties of acid, pepsin and bile. Furthermore, sucralfate blocks acid diffusion across the sucralfate protein barrier and also complexes directly with pepsin and bile.

The action of sucralfate is non-systemic as the drug is only minimally absorbed from the gastrointestinal tract. The minute amounts of the sulfated disaccharide which are absorbed are primarily excreted in the urine.

Each gram of sucralfate contains approximately 200 mg of aluminum. The aluminum moiety can dissociate at low pH and aluminum release in the stomach can be expected; however, aluminum is poorly absorbed from the intact gastrointestinal tract. Following administration of 1 g of sucralfate (tablets or suspension) four times a day to individuals with normal renal function, approximately 0.001% to 0.017% of sucralfate's aluminum content is absorbed and excreted in the urine. This results in an aluminum load of between 0.008 mg and 0.136 mg following a 4 g daily dose. Individuals with normal renal function excrete absorbed aluminum and can respond to an increased aluminum load by increasing urinary excretion. These values were determined in individuals with intact gastrointestinal mucosa. Available evidence does not indicate that absorption of aluminum would be different in individuals with ulcerated gastrointestinal mucosa.

Experiments have shown that sucralfate is not an antacid.

INDICATIONS AND CLINICAL USE

1. Tablets SULCRATE® (sucralfate) tablets are indicated for the treatment of duodenal and non-malignant gastric ulcer.

SULCRATE® tablets are also indicated for the prophylaxis of duodenal ulcer recurrence.

2. Suspension SULCRATE® suspension and SULCRATE® SUSPENSION PLUS are indicated for the treatment of duodenal ulcer.

CONTRAINDICATIONS

There are no known contraindications to the use of SULCRATE® (sucralfate). However, the physician should read the "WARNINGS" section when considering the use of this drug in pregnant or pediatric patients, or patients of childbearing potential.

WARNINGS

Use in Pregnancy There has been no experience to date with the use of SULCRATE® (sucralfate) in pregnant women. Therefore, sucralfate should not be used in pregnant women or women of childbearing potential unless, in the judgment of the physician, the anticipated benefits outweigh the potential risk.

Pediatric Use Clinical experience in children is limited. Therefore, sucralfate therapy cannot be recommended for children under 18 unless, in the judgment of the physician, anticipated benefits outweigh the potential risk.

PRECAUTIONS

General The following should be taken into account before treating patients with SULCRATE® (sucralfate):

Recurrence may be observed in patients after a successful course of treatment for gastric or duodenal ulcers. While the treatment with sucralfate can result in complete healing of the ulcer, a successful course of treatment with sucralfate should not be expected to alter the underlying cause of ulcer disease.

Proper diagnosis is important since symptomatic response to sucralfate therapy does not rule out the presence of a gastric malignancy.

Drug Interactions Antacids should not be taken within half an hour before or after sucralfate intake because of the possibility of decreased binding of sucralfate with the gastro-duodenal mucosa as a consequence of a change of intra-gastric pH.

Animal studies have shown that simultaneous administration of sucralfate with tetracycline, phenytoin or cimetidine results in a statistically significant reduction in the bioavailability of these agents. Cimetidine absorption was not reduced in humans. In clinical trials, the concomitant administration of sucralfate reduced the bioavailability of digoxin.

These interactions appear to be non-systemic and to result from the binding of sucralfate to the concomitantly administered drug in the gastrointestinal tract. In all cases, complete bioavailability was restored by separating the administration of sucralfate from that of the other agent by 2 hours.

Sucralfate, administered respectively 30 and 60 minutes before ASA or ibuprofen did not alter the bioavailability of these agents. In a study comparing the prior administration of a single dose of sucralfate tablets on the bioavailability of naproxen, indomethacin or ketoprofen versus administration in the absence of sucralfate, it was shown that the total amount of these drugs absorbed was not altered; however, the peak concentration of each was reduced, and the time to reach peak concentration was delayed. A single dose of SULCRATE® SUSPENSION PLUS administered one-half hour before naproxen had a similar effect on the bioavailability of naproxen.

The physician should consider the possible clinical implications of these interactions. It is recommended to separate the administration of any drug from that of sucralfate when the potential for altered bioavailability is felt to be critical to the effectiveness of that drug.

Unless specified, the above data are based on studies carried out with SULCRATE® tablets.

Chronic Renal Failure

Dialyzed Patients – Sucralfate should be used with caution in patients with chronic renal failure. When sucralfate is administered orally, small amounts of aluminum are absorbed from the gastrointestinal tract (see ACTIONS AND CLINICAL PHARMACOLOGY). Existing evidence indicates that patients with normal renal function receiving the recommended doses of sucralfate adequately excrete aluminum in the urine; however, patients with chronic renal failure or those receiving dialysis have impaired excretion of absorbed aluminum, and in these individuals, aluminum is known to accumulate in serum and in tissues. In particular, dialysis patients are at greater risk as aluminum does not cross dialysis membranes of the dialysis machine since it is bound to plasma proteins, most notably albumin and transferrin.

In patients with chronic renal failure undergoing dialysis, aluminum-related toxicity (encephalopathy and aluminum-related bone disease), associated with the administration of sucralfate and/or other sources of aluminum has been reported. Consideration should therefore be given to the total daily load of aluminum before administering sucralfate in combination with other aluminum-containing medications, such as aluminum-containing antacids.

Nondialyzed Patients – In a study of six nondialyzed chronic renal failure patients with glomerular filtration rates ranging from approximately 10 to 40% of normal, sucralfate administered at a dose of 1 g QID for three weeks resulted in elevated serum aluminum concentrations which plateaued at approximately 23 μg/L after one week of treatment from a pretreatment level of 3 μg/L. Renal aluminum clearance increased in relation to the increase in serum levels and returned to baseline within two weeks following discontinuation of sucralfate as did serum aluminum concentrations. No adverse events were reported in these patients.

These data indicate that the use of sucralfate in nondialysed chronic renal failure patients requires physician discretion since the excretion of absorbed aluminum may be impaired in these individuals.

ADVERSE REACTIONS

1. SULCRATE® Tablets and Suspension Very few side effects have been reported with SULCRATE® (sucralfate) tablets and suspension. They are mild in nature and have only exceptionally led to discontinuation of therapy.

The main complaint has been constipation ranging from 1.7% to 3.3% of patients.

Other side effects reported included diarrhea, nausea, gastric discomfort, indigestion, dry mouth, skin rash, pruritus, back pain, dizziness, sleepiness and vertigo.

2. SULCRATE® SUSPENSION PLUS In a placebo-controlled clinical trial involving 184 patients, the adverse event rates for SULCRATE® SUSPENSION PLUS was similar to that seen in the placebo group (SULCRATE® SUSPENSION PLUS 10.2% vs placebo 7.4%). The most common adverse event was headache (3.4%) followed by nausea (2.3%), abdominal pain (2.3%), constipation (1.1%), diarrhea (1.1%), and urticaria (1.1%). Only headache, abdominal pain and nausea had a higher incidence in the SULCRATE® SUSPENSION PLUS group relative to placebo.

See the PRECAUTIONS section for information on the potential for aluminum toxicity in dialyzed chronic renal failure patients.

SYMPTOMS AND TREATMENT OF OVERDOSAGE

Overdosage has never been observed with SULCRATE® (sucralfate) and appears to be unlikely since, using maximal doses of up to 12 g/kg/body weight in a variety of animal species, a lethal dose could not be established.

Overdosage is likely to be associated with symptoms similar to those described in the ADVERSE REACTION section, such as constipation. These should be treated symptomatically.

DOSAGE AND ADMINISTRATION

1. Tablets The recommended adult oral dosage of SULCRATE® (sucralfate) for duodenal and gastric ulcer is one tablet of 1 g four times a day, one hour before meals and at bedtime, on an empty stomach. For duodenal ulcer, SULCRATE® may also be administered as two 1 g tablets twice daily, on waking and at bedtime on an empty stomach.

In duodenal ulcers, while healing with SULCRATE® often occurs within two to four weeks, treatment should be continued for a maximum of 8 to 12 weeks unless healing has been demonstrated by X-Ray and/or endoscopic examination.

In the case of gastric ulcers, an alternative treatment should be considered if no objective improvement is observed following 6 weeks of SULCRATE® therapy. However, patients with a large gastric ulcer that has demonstrated a progressive healing tendency may require an additional 6 weeks of treatment.

For the prophylaxis of duodenal ulcer recurrence, the recommended dosage is one tablet of 1 g twice daily, on an empty stomach. Treatment may be continued for up to one year.

For relief of pain, antacids may be added to the treatment. However, antacids should not be taken within ½-hour before or after SULCRATE® intake.

2. Suspensions *a. SULCRATE® Suspension (500 mg/5 mL)* The recommended adult oral dosage of SULCRATE® (sucralfate) suspension for the treatment of (acute) duodenal ulcer is 1 g (10 mL) four times a day on an empty stomach before meals and at bedtime, or 2 g (20 mL) twice a day on waking and at bedtime on an empty stomach.

b. SULCRATE® SUSPENSION PLUS (1 g/5 mL) The recommended adult dose of SULCRATE® SUSPENSION PLUS for the treatment of (acute) duodenal ulcer is 2 g (10 mL) twice a day on waking and at bedtime on an empty stomach.

Duration of continuous treatment in patients with chronic renal failure receiving dialysis should be evaluated by periodic monitoring of serum aluminum levels, due to the possibility of aluminum accumulation in these patients (see PRECAUTIONS). According to information widely available in the literature, patients with serum aluminum concentrations that approach 100 μg/L should be carefully monitored for symptoms of aluminum toxicity and treatment should be discontinued if such symptoms appear.

There is no evidence to indicate that patients with chronic renal failure, who do not require dialysis, are at risk of developing aluminum toxicity while receiving the recommended doses of sucralfate. Physician discretion should be exercised when considering the duration of treatment (see PRECAUTIONS).

AVAILABILITY

1. Tablets Each white, capsule-shaped, compressed tablet, monogrammed "SULCRATE®" on one side and "NORDIC" on the other side, contains 1 g of sucralfate. To be kept and dispensed in a well-closed container. Bottles of 100 and 500 tablets.

2. Suspensions *a. SULCRATE® Suspension* Each 5 mL of pink suspension contains 500 mg of sucralfate. Supplied in bottles of 400 mL. Shake well before using. Store at room temperature. Avoid freezing.

b. SULCRATE® SUSPENSION PLUS Each 5 mL of white, creamy, caramel-flavoured suspension contains 1 g of sucralfate. Supplied in bottles of 500 mL. Shake well before using. Store at room temperature. Avoid freezing.

Product Monograph available on request.

®Sulcrate is a registered trademark of Nordic Laboratories Inc.

PAAB

NORDIC LABORATORIES
Subsidiary of Marion Merrell Dow Canada
Laval, QC, Canada H7L 4A8

SU9201

Atrovent®

(ipratropium bromide)

INHALATION SOLUTION 250 µg/mL

THERAPEUTIC CLASSIFICATION
Bronchodilator

INDICATIONS AND CLINICAL USES
Atrovent (ipratropium bromide) solution is indicated for the therapy of acute exacerbations of chronic bronchitis. Atrovent solution, when used in conjunction with a β_2-adrenergic stimulant solution such as fenoterol or salbutamol, is indicated for acute asthmatic attacks. It is to be administered by compressed air or oxygen driven nebulizers.

CONTRAINDICATIONS
Known hypersensitivity to Atrovent (ipratropium bromide), to any of the product ingredients, or to atropinics.

WARNINGS
Atrovent (ipratropium bromide) solution in the 20 mL multidose bottle contains preservatives (benzalkonium chloride and disodium ethylene diamine tetraacetic acid - EDTA-disodium). It has been reported that these preservatives may cause bronchoconstriction in some patients with hyperreactive airways.

The 2 mL unit dose vial does not contain preservatives.

Atrovent should not be used alone for the abatement of an acute asthmatic attack since the drug has a slower onset of effect than that of an adrenergic β_2 agonist.

Care should be taken to ensure that the nebulizer mask fits the patient's face properly and that nebulized solution does not escape into the eyes. There have been isolated reports of ocular complications (i.e., mydriasis, increased intraocular pressure, angle closure glaucoma) when nebulized ipratropium bromide either alone or in combination with an adrenergic β_2 agonist solution has escaped into the eyes. In the event that glaucoma is precipitated or worsened, treatment should include standard measures for this condition.

PRECAUTIONS
General:
-Patients should be instructed in the proper use of the nebulizer.
-Caution is advised against accidental release of the solution into the eyes.
-In patients with glaucoma, prostatic hypertrophy or urinary retention, Atrovent (ipratropium bromide) should be used with caution.
-If a reduced response to Atrovent becomes apparent, the patient should seek medical advice.
-Atrovent solution, when administered to patients with acute severe asthma, should be used with concomitant β_2-adrenergic stimulant therapy.

Use in Pregnancy:
The safety of Atrovent in pregnancy has not been established. The benefits of using Atrovent when pregnancy is confirmed or suspected must be weighed against possible hazards to the fetus. Studies in rats, mice and rabbits showed no embryotoxic nor teratogenic effects.

Use During Lactation:
No specific studies have been conducted on excretion of this drug in breast milk. Benefits of Atrovent use during lactation should therefore be weighed against the possible effects on the infant.

Use in Children:
The efficacy and safety of Atrovent in children younger than 5 years has not been established.

Use with Other Drugs:
In patients receiving other anticholinergic drugs, Atrovent should be used with caution because of possible additive effects.
In patients with glaucoma or narrow anterior chambers, the administration by nebulizer of combined Atrovent-β_2 agonist solution should be avoided unless measures (e.g., use of swimming goggles) are taken to ensure that nebulized solution does not reach the eye. Exposure of the eyes of such patients to a nebulized combination of Atrovent and a β_2 agonist solution has been reported to result in increased intraocular pressure and/or acute angle closure.
Atrovent solution with preservatives (i.e. from the 20 mL multidose bottle) should not be mixed with sodium cromoglycate, as this produces a cloudy solution caused by complexation between the preservatives and sodium cromoglycate. If the patient's condition requires the administration of sodium cromoglycate, it should be given in combination with Atrovent solution without preservatives (i.e., from the unit dose vial).

ADVERSE REACTIONS
The frequency of adverse reactions recorded in 214 patients receiving Atrovent (ipratropium bromide) solution was as follows, given by percentage of patients reporting: Dry mouth or throat, 9.3; Bad taste, 5.1; Tremor, 4.2; Exacerbation of symptoms, 4.2; Burning eyes, 0.9; Nausea, 0.9; Sweating, 0.9; Cough, 0.9; Headache, 0.5; Palpatations, 0.5.
The adverse effect judged to be most severe was exacerbation of symptoms. This occurred in 8 patients treated with Atrovent solution alone, 6 of whom withdrew from the clinical studies.
Bronchospasm occurred in 3 patients with acute severe asthma who received Atrovent solution alone. In two patients, this was reversed after therapy with β_2 sympathomimetic solution. The third patient received no other therapy.
The following table compares the incidence of adverse effects of the combination of Atrovent and a β_2 agonist (either fenoterol or salbutamol) solution with that of the β_2 agonist alone.

ADVERSE EFFECT	ATROVENT + β_2 AGONIST (% of 94 patients)	β_2 AGONIST (% of 96 patients)
Tremor	31.9	26.0
Dry mouth	16.0	28.1
Bad taste	16.0	13.5
Vomiting	2.1	2.1
Palpitations	2.1	1.0
Headache	1.1	2.1
Cough	1.1	0.0
Flushing	1.1	0.0
Dizziness	0.0	1.0
Numbness in leg	0.0	1.0

There have been isolated reports of ocular effects such as mydriasis, increased intraocular pressure, and acute glaucoma associated with the escape of nebulized ipratropium bromide-alone or in combination with a β_2 agonist solution into the eyes.

DOSAGE AND ADMINISTRATION
In adults, the average single dose is 1-2 mL of Atrovent (ipratropium bromide) solution, containing 250-500 µg of ipratropium. In children, aged 5-12 years, the recommended dose is 0.5-1 mL (125-250 µg of ipratropium). This should be diluted to 3-5 mL with preservative free sterile Normal Saline [Sodium Chloride Inhalation Solution, USP 0.9%] or with a bacteriostatic sodium chloride solution, 0.9% preserved with benzalkonium chloride (see PHARMACEUTICAL INFORMATION).
Nebulization should take place using a gas flow (oxygen or compressed air) of 6-10 L/minutes and the solution nebulized over a 10-15 minute period. The Hudson Updraft™, Bennett Twin Jet® and Inspiron Mini-Neb® nebulizers, with facemask or mouthpiece have been used. The manufacturers' instructions concerning cleaning and maintenance of the nebulizer should be strictly followed.
Treatment with Atrovent solution may be repeated every 4-6 hours as necessary.

PHARMACEUTICAL INFORMATION
Stability and Storage Recommendation:
20 mL Bottle: Unopened bottles of Atrovent (ipratropium bromide) solution should be stored at controlled room temperature (below 30°C).Solutions diluted with preservative free sterile Sodium Chloride Inhalation Solution, USP 0.9% should be used within 24 hours from time of dilution when stored at room temperature and within 48 hours when stored in the refrigerator.
Dilutions may also be made with a bacteriostatic sodium chloride solution 0.9% which contains benzalkonium chloride as the bacteriostatic agent (see WARNINGS). This diluted solution may be stored at room temperature and used within 7 days.
Controlled laboratory experiments using mixtures of Atrovent solution with Alupent® (orciprenaline sulfate), Berotec® (fenoterol hydrobromide) or salbutamol sulfate (6mg/mL preserved with benzalkonium chloride) solutions and diluted with a sterile bacteriostatic sodium chloride solution 0.9% (i.e. normal saline), preserved with benzalkonium chloride, indicated that such mixtures were stable for 7 days at room temperature. For the preparation of such mixtures, it is recommended that only sterile solutions of bacteriostatic sodium chloride 0.9% preserved with 0.01% benzalkonium chloride be used to maintain the level of preservative in the mixture.
The safety of preservatives other than benzalkonium chloride has not been established.

Incompatibilities: Atrovent solution with preservatives (i.e. from the 20 mL multidose bottle) should not be mixed with sodium cromoglygate solution, as this produces a cloudy solution caused by complexation between the preservatives and sodium cromoglycate. If the patient's condition requires the administration of sodium cromoglycate, it should be given in combination with Atrovent solution without preservatives (i.e., from the unit dose vial).

2 mL Unit Dose Vial: Unopened unit dose vials of Atrovent solution should be stored at controlled room temperature (below 30°C) and protected from light. If required, the solution should be diluted with a preservative free sterile sodium chloride solution 0.9% and used immediately. Any solution remaining in the vial must be discarded.
The solution is physically compatible with Alupent® (orciprenaline sulfate), Berotec® (fenoterol hydrobromide) or salbutamol sulfate (6 mg/mL) solutions. If such mixtures are prepared, they should be diluted with preservative free sterile sodium chloride solution 0.9% and used immediately. Any unused portion of such combined solutions must be discarded.

AVAILABILITY
20 mL Bottle: Atrovent (ipratropium bromide) solution is provided as 20 mL clear, colourless or almost colourless solution containing 250 µg/mL (0.025%) Atrovent in isotonic solution. This solution is preserved with benzalkonium chloride 250 µg/mL and EDTA-disodium 500 µg/mL at pH 3.4 in an amber glass bottle with screwcap.
2 mL Unit Dose Vial: Atrovent solution is also provided as 2mL of clear, colourless solution containing 250 µg/mL (0.025%) ipratropium bromide in isotonic solution, presented in a plastic single use vial. One vial contains a total of 500 µg of ipratropium bromide.

The complete Product Monograph for Atrovent (ipratropium bromide) Inhalation Solution is available to health professionals on request. Patient Information/ Instructions are provided with the solution.

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