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THE NOVA SCOTIA MEDICAL BULLETIN

The Canada Health Act

"It's not the principle of the thing, it's the money". Using this statement as her anchor, Monique Bégin has proposed The Canada Health Act to prevent "greedy doctors" from destroying Canada's health system. Using extra billing as her cover and the popular belief that doctors earn too much money, she plans to alter many of the basic principles of the health care system. In fact, she refuses even to discuss some of these very important principles, including doctor's basic rights, real accessibility problems due to lack of facilities, underfunding and the importance of the basic contract to the doctor-patient relationship.

We of course cannot deny our very real interest in money. Our training and effort, we believe, justify substantial income. The Toronto *Globe and Mail* of December 16, 1983 (speaking in favor of the Canada Health Act) states: "Certainly doctors should be well paid, even extremely well paid". The specifics of "well paid" are not addressed, and certainly eliminating extra billing does not help achieve this goal.

It is however an unfortunate fact that the citizens of Canada are just not concerned about doctors incomes or perceive them as too high; we would do well to concentrate on the other principles of The Canada Health Act, despite Bégin's refusal to talk, even though our views might be looked at with scepticism. This very scepticism is one of the things we mourn with the introduction of The Canada Health Act.

We are no longer believed when we tell the body politic what we think is best for its medical welfare. If an individual patient refused our prescription made for his welfare we might be justifiably upset. Usually, the patient seeks our advice and meets his part of the established contract by complying with our recommendations. Monique Bégin has not accepted our prescription for the best health care system for Canadians. It is the bureaucrats who will prescribe the system, and the very people who spend their life working in and understanding all the implications of good health care, will be ignored. The patient in the form of political bureaucracy is writing his on her own prescription.

Other than our contract with society as a whole, more directly our individual contract with our patient is compromised by The Canada Health Act. Monique Bégin in her letter to all Physicians in August of 1983 gave her "personal commitment that new Federal Health Legislation will not interfere either with the professional privileges and standards of medical practice, or with the relationship between doctors and provinces". Dr. Roland Saxon in his newsletter of December 19, 1983 explained clearly why that commitment cannot be met. "The physician's position as a self-employed professional, free to join in combat with hospital administration and government on behalf of his patient, free to speak out publically on health care issues, inadequate services or government, will be checked. With government as his only paymaster, the physician's ability to serve as the patient's advocate, to provide (or see that the patient is provided with) the best care possible will be seriously eroded".

It is also true that many provincial health departments which have related to provincial medical societies very well in the past may well see this good relationship end, especially in Nova Scotia. Monique Bégin's commitment to our professionalism and provincial autonomy then is either made out of stupidity or insincerity.

The Canada Health Act has other unfortunate ramifications for our profession as a whole. We are moved closer to being a mere union with all that unionism entails. Movement towards a union will mean more militancy, not less, and certainly less sense of responsibility. One would hope that this would not be so, but the anger and resentment that this legislation brings out in most doctors will certainly lead us in that direction. If one is treated like a state employee, one begins to act like one.

Country, province and individuals are showing a collective lack of faith in our professionalism. So be it. But we must let them know at least that this upsets us, alters the contract, and has many repercussions in the future. Our new role is different and not necessarily as they might desire it to be, and we must tell them so frequently, as the President of our Society has attempted to do. It is not money that upsets us, but the principle of the thing. While we are of course concerned about the money, we are more concerned about patient care itself. Unfortunately we no longer expect this to be believed. Perhaps the lack of faith is justified, but whether justified or not the country has shown very little understanding of our position. It is a difficult position to explain and may not effect the outcome of the legislative process. At least, in trying to be understood and having people see our position a little more clearly, we may ease the problems which many physicians think The Canada Health Act may cause in the near and distant future.

"A good lie can travel half around the world before the truth can get out of bed." — Mark Twain. □

J.F.O'C.

An Appreciation

DR. HAROLD J. DEVEREAUX

Harold Joseph Devereaux died on November 24, 1983 in Sydney where he had practised for over forty years.

Truly a man for all seasons, his contributions have been many and varied. While his interests included both provincial and medical politics and community organizations, as well as church and family, he will be best remembered for the warmth of his personality in his daily contact with people, as friend, counsellor or physician. His ability to brighten the day for others, to elicit a smile or a cheery word from those he met on the street, in the elevator or in the office, was clearly the mark of one who understood the art of medicine. His breadth of tenure spanned the years when house calls were routine, when maternity cases were called confinements and when compassion entered freely into fee arrangements.

Born at Kelly's Cross, P.E.I. in 1910, educated at St. Dunstans and Tufts in Massachusetts, he graduated from Dalhousie Medical School in 1936. Postgraduate training in cardiology at the Royal Victoria in Montreal and in obstetrics at The Boston Lying In preceded his start in practice at Sterling Mines. He soon joined Dr. M.G. Tompkins at Dominion, practising there until 1942 when he moved to Sydney.

In 1940, Harold married Margaret MacDonald, a young nurse who was his constant companion until his untimely death. They raised four sons, Greg, Brian, Dennis and Bruce and one daughter Diane, a practising psychiatrist.

His leadership appointments included president of St. Rita Hospital Staff, president of City Hospital Staff and Chief of Staff at both hospitals. He served as president of the Cape Breton Medical Society and The Medical Society of Nova Scotia. At other times he was on the executive of the Canadian Medical Association as well as the Hospital Insurance Commission and Blue Cross.

Dr. Devereau was honored with Senior Membership in The Canadian Medical Association in 1976 and The Medical Society of Nova Scotia in 1980. In 1981 he was named Alumnus of the Year by the Dalhousie Medical Alumni Association.

His many local interests included Home and School, United Appeal, Kiwanis and his church. He was a tireless supporter of the Conservative party in provincial and federal politics. In 1976 an amateur theatrical group, in appreciation, dramatized his life story on the stage of the Savoy Theater in Glace Bay.

One abiding passion was his love for the Mira River. He and Margie replaced their summer cottage there with a permanent home where he loved to garden, to work on the property, to entertain friends and enjoy the tranquillity of the country, an opiate which sustained his spirit when his health failed.

To his wife Margie and his children we extend our most sincere sympathy. Harold Devereaux left his mark on all of us, and we are the better for it. □

N. Kenneth MacLennan, M.D.
Sydney, N.S.

How Safe Is Diagnostic Ultrasound?

B. St. J. Brown,* M.B., B.S., F.R.C.P.(C)

Halifax, N.S.

Questions regarding the safety of diagnostic ultrasound in clinical practice are a genuine concern of patients, nurses, technologists and physicians. My purpose is to give an overview of published evidence to date of the immediate and possible long-term effects of diagnostic exposure to this form of energy.

Sound waves, both below and above the acoustic range of the human ear, are well-known to occur in nature. Low frequency sound waves are emitted by whales and can travel a distance of 30 miles through sea water. High frequency sound waves above the human acoustic range occur among mammals (the basis of the "silent" dog whistle) and are used as an echo device for target and range finding, and for aerial navigation by bats during flight. These forms of naturally occurring ultrasound are of low intensity and are harmless. On the other hand, if the frequency of the sound waves is increased, and the intensity brought to high levels, this form of energy can be a very powerful tool.

In clinical medicine, the *therapeutic* effects of high intensity ultrasound waves are well-known. They are used in the treatment of certain articular and periarticular conditions, and also in dentistry, where the "cavitron" is used to separate plaque, calcareous material and even tobacco stains from dental enamel. High intensity ultrasound waves have also been used to fragment objects such as large gallstones during removal. Certain ultrasonic devices are used to clean laboratory glassware. Workers in these fields have not been shown to suffer ill effects from the recommended technique of application.

Before considering the diagnostic application of ultrasound in clinical medicine, a brief description of the basic physics is in order. Ultrasound is a wave form of non-ionizing energy. In order to be transmitted through tissue, it depends on oscillation or "ripple effect" of one molecule on an adjacent molecule as in a row of dominoes. It has no direct relationship to x-rays which are damaging to tissues because of their ionizing effect upon the living cells.

Ultrasound is used in departments of radiology as an imaging method. Information is primarily obtained from the reflections or echoes produced from tissue planes or interfaces. Also the characteristics of the transmission of the beam may "characterize" the tissue through which it passes. Tissues of different density and "stiffness" produce a sudden change in the speed of transmission of the ultrasonic beam thus producing different echo patterns.

Water is an excellent conductor of ultrasound and transmits wave energy superlatively well. Tissues with a high water content and relatively homogeneous architecture will regularly produce a relatively homogeneous hypoechoic pattern. On the other hand, gas is a very poor conductor of ultrasound and almost totally blocks and obscures transmission, since the molecules in a gas do not easily transfer a ripple movement. Tissues with a hard or stiff consistency such as bone will transmit the ultrasound waves with greatly increased speed (compared to that of water) but also have a very high or dense reflectivity and therefore block the transmission of the beam.

These physical facts have an important bearing on the resolution or visualization power of ultrasound. The gallstone of about 2 mm diameter in a background of "translucent" echo-free bile will shine like a star in a clear sky, whereas a cyst of five times the diameter located deep in a background of high density echoes, e.g. a small pancreatic cyst, will be difficult to visualize because of the "foggy background".

Ultrasound has two physical forms which are currently in general use in diagnostic clinical practice:

a) ***The pulsed form,*** in which intermittent pulses of energy are used. The pulses are of microsecond duration and are repeated up to 1000 times per second. The transducer emits these pulsed signals intermittently and then receives the echoes or "listens" in the intervals between pulses. Modern equipment "listens" for 99.9% of the examination time, hence even in a thirty minute examination, the patient is exposed to the energy for only a few seconds. The peak energy of the pulse is similar in magnitude to that of therapeutic ultrasound.[1] Since the inception of this technique no deleterious effects have been reported in humans.[4]

b) ***The continuous beam*** relies on the Doppler shift of moving structures in the beam to produce information. Because of this principle, the Doppler technique exposes the patient to the ultrasound beam continuously, hence much longer exposures of the patient are inevitable.

Recently, a pulsed form of Doppler has been developed.

Biological Burden Implications

The physical effects derived from animal and other research can be summarized as follows:

i) ***Heat:*** the low average intensity used in diagnostic pulsed ultrasound (less than 33 mW/cm^2) produces no significant degree of heating.[3] This level of intensity is used in the fetal monitoring Doppler devices. However, other Doppler devices, especially those used for arterial studies, employ intensities that may produce significant heating. These Doppler devices are not suitable for fetal monitoring.

ii) ***Cavitation:*** This is a complex phenomenon in which gas-filled bubbles grow in an ultrasound field. At high intensity levels, these bubbles may collapse suddenly causing large but localized temperature rises, thermal decomposition of water, and release of free radicals. This phenomenon has been termed "transient cavitation".[3,4,5] At lower intensity levels, the gas bubbles may pulsate over an indefinite period, described as "static cavitation". If these stable gas bubbles are caused to vibrate by ultrasound even of low intensity, they can generate shearing stresses which can affect the structure as well as presumably the behaviour

*Director, Department of Radiology, Grace Maternity Hospital and Radiologist, I.W.K. Hospital for Children, Professor of Radiology, Dalhousie University, Halifax, N.S., B3H 1W3

of cells in their locality.[6] The threshold to produce this form of cavitation in cell suspensions in tissue fluid is 500 mW/cm^2 and presumably by lower intensities at lower frequencies.[7] This threshold is lowest when the pulse length is in the range of 1-30 m sec.[8] The converse is also true. The threshold for pulses of *microseconds* duration has not yet been established. So far, the brevity of the pulses used in clinical ultrasonography probably prevent the occurrence of cavitation at the intensities used for diagnostic purposes. However, one author claims that transient cavitation can occur with pulses as short as one cycle at intensities of 10 W/cm^2.[9]

The continuous nature of Doppler implies that the peak intensities should be retained below the cavitation threshold. For obstetric use, the Doppler output should be kept below 20 mW/cm^2, which appears to be below all known thresholds for the production of other biologic effects.[10]

During the transmission of an ultrasound beam, particles are subjected to oscillatory variations in pressure, velocity, acceleration and temperature. These forces include streaming, shear force, Bernoulli and Oseen forces, but since they act on a non-homogeneous medium, they give rise to time-averaged forces and do not self-reciprocate.[4]

Changes in Transport Across Membranes

Changes in membrane permeability have been shown in exposed liver cells with swelling of the endoplasmic reticulum and mitochondrial changes.[11] One possible mechanism for these changes is the "streaming" or movement of particles away from the origin of the ultrasound beam. Such stresses are facilitated by the presence of resonant cavities. Under these conditions, it has been calculated that significant stress could be induced by intensities as low as 0.5 mW/cm^2.[12] This is lower than that found for any reported bioeffects and it seems unlikely that such cavities exist in most animal tissues.

Alteration of Mitochondria

Swelling and disruption of the cristae may be a direct effect or part of a more widespread change associated with membrane permeability.[13,14]

Lysosomal Changes

In rodent liver, to produce lysosomal changes, peak intensities in the region of 25 W/cm^{-2} with pulse lengths of 10 m sec., separated by intervals of 90 m sec. for five minutes were required.[11] These pulse lengths were 10,000 times longer than those used in diagnostic ultrasound. Exposure of epidermal lysosomes to therapeutic levels of ultrasound (intensity 0.5 W/cm^{-2}, frequency 3 mHz; pulsed 2 m sec. on, 8 m sec. off for five minutes) showed increased permeability of exposed lysosomes.[15] This is not thought to be necessarily harmful and may even stimulate reparative processes indirectly by increasing the availability of low molecular weight precursors as raw material for repair.[16]

Production of Centrilobular Necrosis of the Liver

This appears to be dose dependent, the doses being greater than those used for diagnostic examinations.[17,18]

Paraplegia

This can be used as a dramatic end point of ultrasonic damage. The dose response curve has been elaborated by extrapolation,[19] the threshold intensity required for paraplegia in rats is as follows — a single pulse of microsecond duration at 10^{4}W/cm^2; repetitive pulses for 10 m sec. duration at peak intensity of only 25 W/cm^2; exposure time for production of hemorrhage increased with higher frequencies up to 5 MHz, at which frequency the lesion no longer occurred. Reducing arterial oxygen tension to 50 mm mercury while retaining normal CO_2 levels markedly decreased the exposure time required to produce hemorrhage. Further experiments to produce paraplegia in rats showed that the onset of damage was dependent only on the integrated dose time. By extrapolation to the levels of energy used in ultrasonic diagnosis, several hours of continued exposure would be required to produce damage, even allowing for hypoxia in the fetus. In these experiments, the shorter pulses similar to those used in diagnostic ultrasound were safer.

Production of Fetal Anomalies

In mouse pregnancies, intensities of up to 490 W/cm^2 showed no difference in mortality or congenital anomalies comparing exposed and control groups.[20] However, in chick embryos of 18 hours incubation, during active organogenesis a significant increase in incidence of congenital anomalies occurred at intensities of 25 W/cm^2 and above.[21,22] However, it should be noted that the pulse length used in these experiments was 20 times greater than that used with diagnostic techniques and the pulse repetition rate was 5 times greater. These experiments were repeated at a later stage of development after organogenesis was complete, corresponding to about 6 weeks of human development. At this stage of maturity, exposure at the same level of energy used previously did not produce any evidence of congenital anomalies. Also when the energies were increased to 100 W/cm^2 and pulse length increased to 10 m sec. on and 90 m sec. off for five minutes, there was no apparent damage to the embryos.

Stimulation of Regeneration and Wound Healing

In experimental animals, tissue regeneration and skin repair has been shown to be stimulated by ultrasound; also in man, the healing of pressure sores and varicose ulcers can be stimulated.[23-27]. For tissue regeneration, the most effective dosage was 0.5 W/cm^2 at 3 MHz, pulsed 2 m sec. on, 8 m sec. off for five minutes, three times weekly.[23] Peak stimulation occurred at 21 days following by a temporary decrease between 28 and 35 days when collagen fibre is maturing. Autoradiographic and electron microscopic evidence showed that cell proliferation was stimulated at these dose levels. In vitro work suggests that although fiber maturation is retarded by ultrasound, the production of fiber precursors is stimulated.[28] Pretreatment of the cells with cortisone, which stabilizes lysosomal membranes, blocked this ultrasonic stimulation effect. It was also found that the directionality of movement of human fibroblasts towards the injured site was improved after exposure to therapeutic ultrasound.

Doppler

An increased incidence of congenital abnormalities in mice after exposure to a commercially available Doppler device has been reported.[29] Also, an increased incidence in perinatal morbidity after three minutes of exposure of 125 mW/cm^{-2} and also decreased fetal weight in mice exposed to intensities of 500 mW/cm^{-2} for five minutes has been reported.[30,31]

The possibility of chromosomal aberrations has been the subject of conflicting reports. An initial report by McIntosh[32] showing increased incidence of chromosomal aberrations could not be repeated by other workers and the author himself failed to reproduce his own initial results.[33] Unfortunately, the first positive report obtained more widespread publicity than the subsequent retraction!

In chick embryo, red cell stasis could be produced by ultrasound in a standing wave field.[34] Intensity threshold for this phenomenon is approximately 500 mW/cm^2. In the uterine vessels of the mouse, temporary stasis could be produced only when the vessel lay between the transducer and an adequate reflecting surface.[35]

SUMMARY

Lowest levels of ultrasound at which bio-effects have been reported in tissues is shown in Nyborg's graph (Fig. 1). In view of extensive animal research and over 25 years diagnostic application in clinical medicine, the Bio-effects Committee of the American Institute of Ultrasound in Medicine released its statement on Mammalian in Vivo Ultrasonic Bioeffects in August 1976, (revised October 1982): This statement is reviewed annually and at the present time it is still deemed to be essentially correct[36]: "In low megahertz frequency range, there have been (as of this date) no independently confirmed significant biological effects in mammalian tissue exposed to intensities* below 100 mW/cm^2. Furthermore, for ultrasonic exposure times ess than 500 seconds and greater than 1 second, such effects have not been demonstrated even at higher intensities, when the product of intensity* and exposure time is less than 50 joules/cm^2."

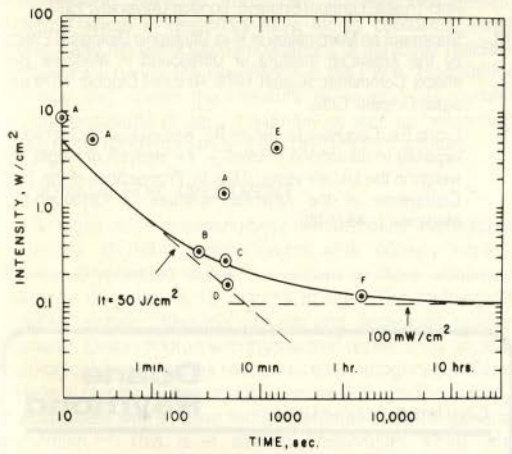

Fig. 1. Lowest levels at which bioeffects have been reported in tissues, as of March 1976. A, fetal weight reduction; B, postpartum mortality; C, wound healing; D, altered mitotic rate (variable results); E, genetic damage (negative); F, fetal abnormalities ("postulate"). (Reproduced from Nyborg WL: Physical mechanisms for biological effects of ultrasound. Bureau of Radiological Health, HEW publication 78-8062, 1978, p. 41)

*Spatial peak, temporal average as measured in a free field in water.
†Total time: this includes off-time as well as on-time for a repeated-pulse regime.

Up to the present time documentation has been orientated towards equipment output rather than to tissue exposure, e.g. fetus. This should be rectified in the near future.

A six year follow-up of 10,000 human fetuses (1970-1978) exposed to diagnostic ultrasound have shown no conclusive deleterious effect attributable to ultrasound as shown by the somatic growth of the resulting infants and children.[37]

CONCLUSION

To revert to the title "How Safe is Diagnostic Ultrasound?"

a) How Safe for the Human Race?

The question of long term biologic burden on exposed populations cannot yet be answered. Ongoing objective studies in different centres across the world are difficult to identify and are not well publicized. In view of our experience with ionizing radiation, radiologists are well aware of possible unforeseen effects of newer forms of energy in diagnostic medicine. Questions regarding ultrasound in this context are very similar to those posed for any new form of energy such as that emitted by a microwave appliance, radio frequency waves from CB transmittors and the energies emitted by television sets. The fact that such forms of energy are ubiquitous in the western world must not be allowed to interfere with their objective appraisal.

b) How Safe for the Patient?

Diagnostic ultrasound has been in active clinical use for over 25 years in obstetrics. This is the field of greatest tissue sensitivity where the early embryo in its formative stage is most likely to suffer damage. There has been no conclusive evidence of harmful effect ever recorded in humans following diagnostic application of ultrasound in clinical medicine.

This is a remarkable safety record. It should encourage the physician when making a selection of diagnostic imaging procedures for individual patients. This safety record should also be recognized when considering diagnostic screening programs in the delivery of health care. □

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On the Diagnosis of Cryptorchidism*

Robert D. Schwarz, M.D., F.R.C.S.(C)

Halifax, N.S.

In recent years, there have been advances in the understanding and management of undescended testes. Early scrotal positioning and brief hospitalization (even out-patient surgery) is recommended. At the I.W.K. Hospital for Children, boys are still being referred at an older age and hospitalized for a longer time than ideal.

The diagnosis is still based on physical examination which can be difficult. Questions about diagnosis can lead to confusion in interpreting therapeutic statistics.

The European literature suggests that Gonadotrophin Releasing Hormone (Gn. R.H. or L.H.R.H.) offers a therapeutic alternative to surgery and warrants a North American trial.

Baby boys are relatively easy to examine for testicular descent. In the neonatal period, the scrotal skin is lax and the cremaster muscle is usually not developed sufficiently to pull the testis up. About 3% of term infants are born without complete testicular descent.[1] Within the first few months of life, however, the majority of undescended testes will assume a scrotal position and, by 1 year of age, the incidence of true maldescent is about the same as the adult incidence — less than 1%.[1,2]

The difficulty in assessing a 1 or 2-year-old boy is differentiating retractile from truly undescended testes. Possible errors in diagnosis have led to confusion about therapy and the associated implications of testicular maldescent. We will review the experience at the I.W.K. Hospital for Children and review the literature to support our current recommendations of early treatment as well as presenting some of the controversies and possibilities for the future.

RATIONALE FOR TREATMENT

The major issue in treating boys with testicular maldescent is fertility. Bilateral cryptorchidism after puberty implies sterility. Cryptorchid testes are shown to have abnormal histology as early as 18 months of age. The histological changes include interstitial fibrosis and decreased tubular diameter. Older children with cryptorchid testes show greater histological abnormalities related to spermatogenesis. Even unilateral cryptorchidism treated successfully prior to puberty is associated with a lower fertility potential than the general population.[3,4] This is a striking observation when one considers that a boy who has lost a testis to trauma or torsion preserves a normal fertility potential.

Testicular malignancy associated with testicular maldescent is another concern. Although the chance of developing a malignancy is very low, the increased risk of testis tumor has been estimated between 25-40 times the general population. About 10% of the testicular malignancies associated with unilateral cryptorchidism occur in the contralateral normally-descended testis.[5]

The fact that unilateral maldescent implies reduced fertility and a risk of malignancy in the contralateral side suggests not only that a cryptorchid testis is a bit of a "dud", but also that the boy with unilateral cryptorchidism may have a congenital problem with **both** gonads. Alternately, the one abnormal testis may influence the normal contralateral testis. Recently, evidence showing that one "damaged" (ischemic) testis can cause degeneration in the normal contralateral testis in experimental animals has been presented and an immunological mechanism suggested.[6]

How much of the problem with fertility is related to testicular position, and how much is an expression of a congenital deficiency (one expression of which is testicular maldescent), are questions that keep recurring in any discussion of undescended testes. The answer is not yet known, partly because it is difficult to separate retractile and cryptorchid testes and partly because the results of early therapy will not be known for one generation. To the extent that the gonads are dysfunctional because of position, the dysfunction is potentially reversible. Fertility potential is improved by orchidopexy and epidemiological evidence suggests that earlier therapy may reduce the risk of malignancy.

Scrotal positioning of testes is, therefore, important for the boy's future. Technique and timing are important issues for the boy who has been accurately diagnosed. Two-year-old boys are unconcerned about their testes, and yet cryptorchid testes from children 12 months to 2 years of age show ultrastructural and histological changes.[7,8] The histological changes are progressively more marked the longer the testis remains cryptorchid. It is for these reasons that current recommendations are for early evaluation and treatment (12-18 months of age). Intervention before 1 year is not currently recommended because there may be normal testicular descent in the first months of life.

DIAGNOSIS

How then can one be certain to differentiate a boy with retractile (normal) testes from the child with true cryptorchidism? An observant mother may notice that retractile testes assume a scrotal position in a warm bath, although the failure to see testes then does not exclude retractile testes. Physical examination must be the critical differential evaluation. Every effort should be made to allow the child to be relaxed. The fingers of the left hand are used to express the testis down and medially from the level of the internal ring towards the scrotum while the index finger and thumb of the right hand are used to retract the scrotum towards the external ring in an effort to feel the testis and deliver it to the scrotum. Occasionally, the supine or squatting knee-chest position will express the testis so that it can be felt scrotally and brought down. It is a difficult examination and should be

*From the Department of Urology, Dalhousie University, I.W.K. Hospital for Children, Halifax, Nova Scotia.
Reprint requests to: Dr. Robert D. Schwarz, Dept. of Urology, I.W.K. Hospital for Children, 5850 University Avenue, Halifax, N.S., Canada B3J 3G9.

repeated if necessary. Even an expert can be mistaken and occasionally a second observer will be able to deliver testes which had been felt were truly cryptorchid.

The current recommendations are for scrotal assessment at birth and repeated examinations of those boys whose testes have not descended by 8-10 months. If the testes cannot be expressed into the scrotum by 1 year of age, the child should be evaluated for scrotal positioning, recognizing that some of the children who appeared to have cryptorchid testes may be shown on physical examination by others to have retractile testes.

It would be interesting, but impractical ethically, to obtain follow up testes biopsies after scrotal positioning. Unfortunately, the animal studies have not helped to determine whether the histological changes can be arrested or reversed.

TREATMENT

Traditional treatment for undescended testis is operative orchidopexy. Techniques have improved so that small, ischemic testes after orchidopexy are fortunately quite rare. One of the advances in recent years has been the use of a scrotal pouch technique which allows the testis to lie comfortably in the scrotum without tension on the cord vessels. Boys now can have orchidopexy done as outpatients or with one day of hospitalization.

A review of the last 100 boys treated at the I.W.K. Hospital for Children has shown that we are not living up to the current recommendations. Sixty percent of the boys came from Halifax County, the others from the rest of the province, P.E.I., and eastern New Brunswick. The period of hospitalization was no different for those who lived nearby or outside Halifax County. Both groups stayed in hospital an average of *5 days!* There was a difference in the ages of boys having orchidopexy. Those from Halifax County were seen and treated at 5 years while those from outside the County had surgery at 6 years of age (Table I). Ideally in either case, the children should be treated at a younger age.

TABLE I

	Halifax County	Outside Halifax County
Hospital stay (days ± S.D.)	5.0 ± 3.7	4.9 ± 3.8
Age at treatment (yrs. ± S.D.)	5.3 ± 3.7	6.1 ± 3.8

There are still important problem areas for the surgeon or urologist operating on these boys. Occasionally, one finds that the child had unilateral anorchia. In that case, there is no testis or only a fibrous remnant, and both the vas and the cord vessels are seen as remnants in the inguinal canal. It is suggested that the testis was exposed to pre-natal ischemia — probably torsion. Silicone testicular prostheses are life-like and help the boy through otherwise embarrassing times in the future.

A boy with bilateral impalpable testes may have had bilateral anorchia. He would be normally virilized because of early fetal testicular function. Since there is no testicular tissue, hormonal stimulation with Human Chorionic Gonadotrophin (HCG) would fail to show a normal testosterone response. Such a child need not undergo surgical exploration if diagnosed hormonally. Bilateral impalpable testes may also be found in a child with hypogonadotrophic hypogonadism. These boys need a longer course of gonadotrophin to show an equal androgen response. It may be necessary in some instances to perform laparoscopy to confirm the presence or absence of a gonad.

The presence of looped vas alone without cord vessels in the canal usually means that the epididymis failed to attach to the testis which may actually be found inside the internal ring or even higher in the peritoneal cavity.

A major technical problem for the urologist is dealing with the high testis which, even after extensive retroperitoneal dissection, cannot be delivered into the scrotum. Of the surgical options, the best chance appears to be a two-staged orchidopexy. Microvascular anastomosis to the inferior hypogastric vessels had sounded attractive but recent studies have shown damage to the germinal epithelium of experimental animals after 60 minutes of ischemia. Relying on vasal vascular anastomosis — dividing the cord — results in 20% atrophic testes. Older children with unilateral testicular maldescent and very high testes who come to surgery are best treated by orchiectomy and testicular prosthesis.

There has been work suggesting that gonadotrophins will induce testicular descent. Job *et al.* claims close to 30% descent using HCG in a population of boys over 5 years (but much less in younger boys).[9] At the I.W.K. Hospital for Children, Dr. Sonia Salisbury has given HCG to eighteen boys with undescended testes — five of them had bilateral undescent, six had unilateral cryptorchidism, three proved to be anorchic, and three showed hypogonadotrophic hypogonadism. One boy had been seen in early childhood and felt to have retractile testes. He returned at age 5 years and the testes could not be expressed from the level of the pubis into the scrotum. He responded well to HCG with bilateral testicular descent. None of the other boys treated yet has achieved testicular descent with HCG, although the hormonal stimulation produced an androgen response biochemically and somatically with some penile growth. Several children who were felt by one of us to be truly cryptorchid were found by a second examining physician to have retractile testes and were therefore excluded from treatment.

Hormonal influences on testicular descent is a subject of active investigation internationally. Investigators in Europe have treated boys with Gonadotrophin Releasing Hormone (Gn.R.H.) and its analogues by nasal instillation with great success.[10] Of course, the question of accurate diagnosis is still open. Because of the problem of diagnosis, a prospective study has to be done in North America comparing LHRH to placebo in a double blind trial.

FUTURE DIRECTIONS AND QUESTIONS REMAINING TO BE ANSWERED

As outlined above, many questions remain to be answered about testicular descent and the best management of a boy with cryptorchidism. Before one can define what errors in development have occurred, a better understanding of the normal mechanism of descent must be achieved. The role of the epididymis in testicular descent is as controversial as the role of Gn. R.H., D.H.T. (Dihydrotestosterone), and M.I.S. (Mullerian-Inhibiting Substance). There may even prove to be other locally-produced, locally-active substances that

stimulate the events which prepare for testicular descent (cord lengthening, gubernacular changes, invasion of the processus).

Correct therapy demands an understanding of normal descent and the extent to which the changes of cryptorchid testes are reversible by early scrotal positioning. Is the testis destined to be something of a "dud" even if its position is corrected or is it potentially normal if treated early? In unilateral cryptorchidism, how is the risk to the contralateral normally-descended testis mediated? Is that risk reversible by therapy? Accurate diagnosis is an essential first step in beginning to answer these questions.

RECOMMENDATIONS

Any boy who is born with a testicle which cannot be delivered into the scrotum should be followed closely, examined on several occasions, and if the testis cannot be brought down by 10-12 months of age, he should be selected for hormonal or surgical scrotal positioning. The role of hormonal therapy is still controversial. Endocrine testing, however, is essential for boys with bilateral impalpable testes or sexual ambiguity. Since physical examination is the only way to diagnose cryptorchidism, and since it is a difficult examination, we recommend repeated examinations and examinations by more than one physician if practical. Surgery should be done at an earlier age than currently practised and can be done with shorter hospitalization, even as an outpatient procedure. □

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The Six Diseases of The W.H.O.: Upon Our Threshold

A. K. Sharma* and J. A. Embill,** M.D., Ph.D., F.R.C.P.(C), F.A.C.T.M.

Halifax, N.S.

"What seems to us more important, more painful, and more unendurable is really not what is more important, more painful and more unendurable, but merely that which is closer to home. Everything distant which for all its moans and muffled cries, its ruined lives and millions of victims, that does not threaten to come rolling up to our threshold today, we consider endurable and of tolerable dimensions."

— Alexander Solzhenitsyn

INTRODUCTION

Approximately one in every four people in the world is exposed to one or more of a group of six diseases that, when taken collectively, are responsible for more human morbidity and mortality than any other group of infectious diseases. The World Health Organization's Special Programme for Research and Training in Tropical Medicine has chosen to focus on these six diseases — filariases, leishmaniasis, leprosy, malaria, schistosomiasis and trypanosomiases — because of "their incidence, severity, their effect on capacity for work, and the lack of effective measures for their control."[1]

Although none of the six is endemic or transmissible in Canada, the possibility of one or more of them being imported into the country is always present. In 1979, almost 700,000 Canadians travelled to countries where these diseases are endemic, and 70,000 people immigrated to Canada from countries where they are prevalent.[2] Therefore, a Canadian physician may have a patient who has one of these six infections. This means that Canadian physicians need to be aware of how to diagnose and treat people with these diseases. In this article, we will briefly review the clinical picture, diagnosis and treatment of each of these infections.

All six of these diseases occur in tropical and subtropical areas (Figs. 1 and 2). Table I gives some general information about each disease. All of the six, except leprosy, which is caused by a bacillus, are parasitic infections.

THE CLINICAL PICTURE

Filariases

There are two main types of filariasis: onchocerciasis ("river blindness") and lymphatic filariasis. Both are slowly progressive diseases that develop as the parasitic filarial worms mature in the human host.

In onchocerciasis, adult worms live singly and in masses in the subcutaneous tissue, where they become surrounded by fibrous scar tissue forming nodules that tend to overlie bony prominences. Nodules are also found in lymphatic filariasis, but in tissue immediately associated with lymphatic vessels in which the worms reside; the nodules are composed of cellular granulation tissue. Also common to both forms of filariasis are skin lesions, distributed irregularly over the body, which are extremely itchy and can cause insomnia.

Fever is a common symptom of lymphatic filariasis, but may not appear in onchocerciasis. It may be associated with rigors and vigorous sweating and tends to decline after the first day or so. Lymphangitis is frequent, with the femoral and malleolar vessels being most commonly affected; the spermatic cord and testis are especially susceptible. The vessels are acutely tender and easily palpable. Sometimes abscesses form in the affected vessels and discharge on the surface. Untreated lymphatic filariasis can result in such manifestations as elephantiasis, chyluria or hydrocele.

In onchocerciasis, the main damage is not to the lymphatic vessels but to the eye.[3] The microfilariae can be found in the cornea, anterior chamber, retrolental space and vitreous humor. Early symptoms of ocular infection are conjunctivitis, lacrimation and photophobia. Untreated onchocerciasis can result in total blindness.

Leishmaniasis

There are three clinically important types of leishmaniasis: visceral (kala-azar), cutaneous (leishmaniasis recidiva, diffuse cutaneous leishmaniasis, and Oriental sore), and mucocutaneous leishmaniasis.

In visceral leishmaniasis (kala-azar), the outstanding physical signs are enlargement of the spleen, and secondarily, the liver. There is often a fever that has two daily peaks. Lung involvement results in an irritating cough. The skin pigment frequently darkens (thus "kala-azar", literally "black sickness") and diarrhea develops. If untreated, the patient may die, usually due to pulmonary or intestinal superinfection.[4]

In the cutaneous forms, the parasite causes skin nodules which may be self-limiting.[5] With the Oriental sore, they undergo necrosis and shallow ulcers develop. Multiple ulcers may be due to many bites by the transmitting vector, or to metastases from the original ulcer. In leishmaniasis recidiva, the initial sores either do not heal or may recur.[6] In diffuse cutaneous leishmaniasis, nodules 1-2 cm in diameter are very widespread, may cover the entire body and result from an insufficient or absent immune response in the host.[7]

*3rd Year Medical Student, Dalhousie University, Halifax, N.S. Based on a 2nd-year elective.

**Departments of Microbiology, Pediatrics and Community Health and Epidemiology, Dalhousie University, Halifax, N.S.

Address for reprints: Dr. J. A. Embil, Infectious Disease Research Laboratory, Izaak Walton Killam Hospital for Children, 5850 University Avenue, Halifax, N.S. B3J 3G9

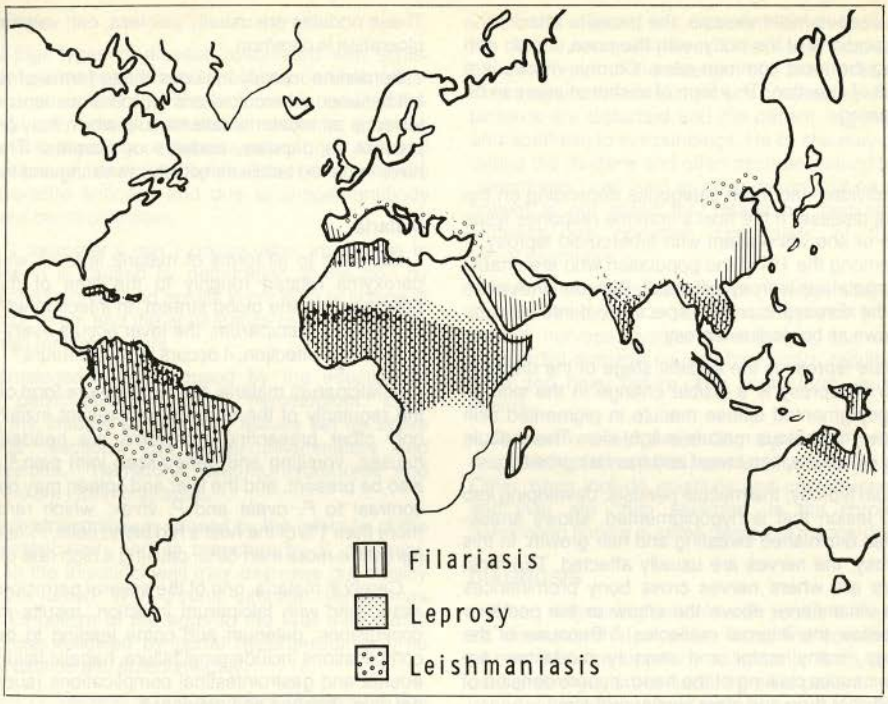

Fig. 1. Geographical distribution of filariasis, leprosy and leishmaniasis.

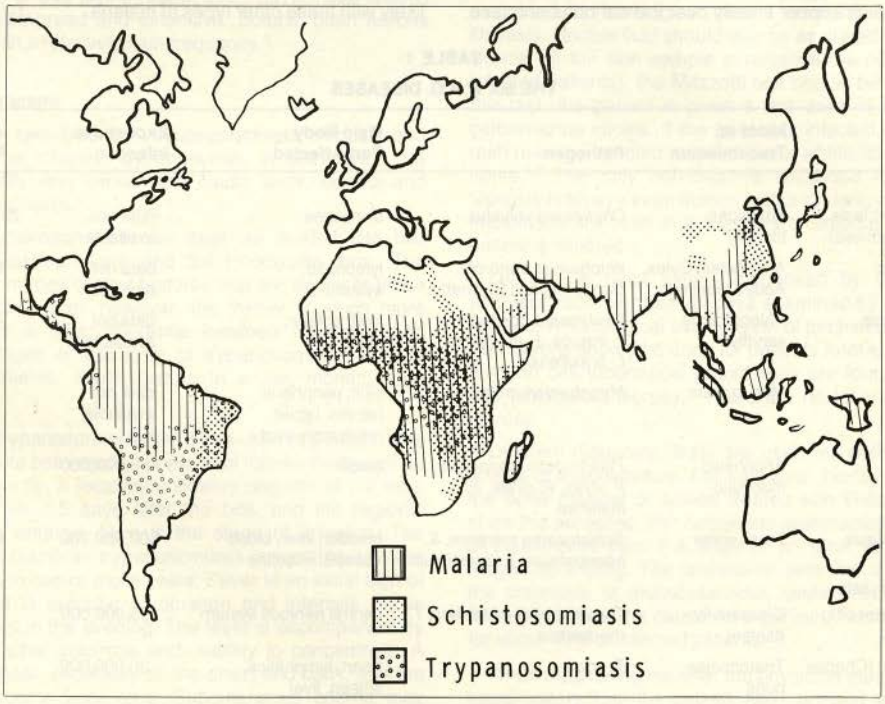

Fig. 2. Geographical distribution of malaria, schistosomiasis and trypanosomiasis.

In mucocutaneous leishmaniasis, the parasite attacks the cartilaginous portions of the body, with the nose, mouth and pharynx being the most common sites. Chronic rhinitis is a frequent result of infection. This form of leishmaniasis can be horribly disfiguring.

Leprosy

Leprosy is divided into three categories depending on the severity of the disease. If the host's immune response lyses the bacilli, he or she will present with tuberculoid leprosy. If he or she is among the 1% of the population who are unable to do this, lepromatous leprosy will result. Between these two extremes of the disease is a wide spectrum of intermediate infections known as borderline leprosy.

Indeterminate leprosy is the earliest stage of the disease. The first sign of leprosy is a visible change in the skin — usually a hypopigmented diffuse macule in pigmented skin and a slightly erythematous macule in light skin. This macule retains tactile sensitivity, can sweat and has hair growth.

In tuberculoid leprosy, the macule persists, developing into a tuberculoid lesion that is hypopigmented, shows anaesthesia, and has diminished sweating and hair growth. In this stage of leprosy, the nerves are usually affected. The most common sites are where nerves cross bony prominences (such as the ulnar nerve above the elbow or the posterior tibial nerve below the internal malleolus).[5] Because of the nerve damage, many motor and sensory modalities are affected. This causes clawing of the hand, hyperextension of the phalanges, foot drop and other similar problems.

Lepromatous leprosy has multiple erythematous or hypopigmented macules as its earliest dermal lesions.[5] These evolve into aggregations of lepromatous tissue known as nodules which appear initially over the earlobes and face. These nodules are usually painless, can vary in number and ulceration is common.

Borderline leprosy includes those forms of leprosy which fall between tuberculoid and lepromatous leprosy. The initial lesion is an indeterminate macule which may proliferate and present as papules, nodules or plaques. These macules have impaired tactile sensitivity, sweating and hair growth.[7]

Malaria

Common to all forms of malaria is fever which occurs in paroxyms related roughly to the time of the release of parasites into the blood stream. In infection with *P. vivax, P. ovale* and *P. falciparum,* the fever occurs every 48 hours.[5] In *P. malariae* infection, it occurs every 72 hours.[5]

In falciparum malaria, the most severe form of the disease, the regularity of the fever is not present initially. Often, the only other presenting symptoms are headache, malaise, nausea, vomiting and generalized joint pain.[8] Anemia may also be present, and the liver and spleen may be enlarged. In contrast to *P. ovale* and *P. vivax,* which rarely parasitize more than 1% of the host's red blood cells, *P. falciparum* may parasitize more than 50%, causing a high rate of mortality.[9]

Cerebral malaria, one of the several pernicious syndromes associated with falciparum infection, results in hemiplegia, convulsions, delerium and coma leading to death.[10] Other complications include renal failure, hepatic failure, pulmonary edema and gastrointestinal complications (such as abdominal pain, diarrhea and melena).[8]

Malaria caused by the other *Plasmodiae* are usually similar to falciparum malaria but less severe. Although there are fewer complications and less mortality, relapse is more likely with these other types of malaria.

TABLE I

THE SIX W.H.O. DISEASES

Diseases	Mode of Transmission	Pathogen	Main Body Part Affected	Exposed to Infection	Incidence/ Prevalence
1) Filariasis					
a) Onchocerciasis (river blindness)	Simulidae blackfly	*Onchocera volvulus*	skin, eyes	data not available	20-40,000,000
b) Lymphatic	Anopheles, Culex, Aedes mosquito	*Wuchereria bancrofti, Burgia malayi, B. timeri*	lymphatic system	data not available	250,000,000
2) Leishmaniasis	Phlebotomus sandfly	*Leishmania donavani, L. tropica, L. mexicana L. brasiliensis*	liver, spleen, skin	data not available	400,000/year
3) Leprosy	Man to man	*Mycobacterium leprae*	skin, peripheral nerves, upper respiratory tract	data not available	15,000,000
4) Malaria	Anopheles mosquito	*Plasmodium falciparum, P. vivax, P. ovale, P. malariae*	blood	1,468,000,000	12,000,000*
5) Schistosomiasis	Fresh water	*Schistosoma mansoni, S. hematobium, S. japonicium*	bladder, liver, blood vessels, intestine	500,000,000	200,000,000
6) Trypanosomiasis					
a) African (sleeping sickness)	Glossina fly (tsetse)	*Trypanosoma gambiens, T. rhodesiens*	central nervous system	35,000,000	10,000/year
b) American (Chagas' disease)	Triatomidiae bugs	*T. cruzi*	heart, lymphatics, spleen, liver	30,000,000	7,000,000

*in 1979

Schistosomiasis

There are four types of disease associated with schistosomae infection: dermatitis, vesical schistosomiasis, intestinal schistosomiasis and Far Eastern (or visceral) schistosomiasis. In all, except dermatitis, anemia (due to hematuria, rectal bleeding or hematemesis from ruptured esophageal varicies) and eosinophila (due to the host's reaction to parasite antigens and due to antigen-antibody complexes) are commonly seen.

Dermatitis ("swimmer's itch") occurs after immersion in infected water. It is caused by penetration of the skin by non-human schistosomes and results in prickling or itching of skin for an hour or so, and the appearance of small macules that may persist for some hours.

Vesical schistosomiasis is caused by the infection of vesical and pelvic plexuses by *S. hematobium.* Eggs deposited in the bladder and urethral tissues set up active granulomatous reactions that cause inflammatory and obstructive lesions.[11] Hematuria, dysuria, hydroureter and hydronephrosis are commonly seen.

Intestinal schistosomiasis is caused by the infection of the inferior mesenteric vein and its branches by *S. mansoni.* Granulomas in the intestinal wall may decrease gut motility and narrow the lumen. With chronic infection, carcinomas may occur.[5] Embolism of the eggs to the liver may cause periportal fibrosis followed by portal hypertension with its characteristic ascites and splenomegaly.

Far Eastern or visceral schistosomiasis (Katayama disease) results from *S. japonicium* infecting the superior and inferior mesenteric vessels. The mode of pathological lesions are the same as for other schistosomiases; however, due to the high rate of egg laying (about 3000/day), these lesions are more widespread and extensive. Ectopic brain lesions also occur with a relatively high frequency.[5]

Trypanosomiases

There are two types of trypanosomiasis: African and American. The infecting trypanosomes in both forms are morphologically very similar and cause fever, edema and meningoencephalitis.

African trypanosomiasis can itself be divided into two forms: the Gambian form and the Rhodesian form. The Rhodesian form has clinical features that are similar to those of the Gambian form; however, the former is much more acute. There is little difference between the early and advanced stages of this form of trypanosomiasis and, in untreated patients, death occurs in a few months after infection.

Gambian trypanosomiasis has three distinct phases. The first is the acute bite reaction phase that follows the bite of an infected tsetse fly. A local inflammatory reaction of 1-2 days duration occurs 3-5 days after the bite, and the regional lymph nodes enlarge. Next is the stage of invasion. The incubation of Gambian trypanosomiasis is variable, ranging from 10 days to two or more years. Fever is an initial sign of infection, and is irregular in duration and intensity but is usually highest in the evening. The fever is accompanied by severe headache, insomnia and inability to concentrate. A rash may appear, especially on the chest and back, and the skin may become very itchy. Subcutaneous edema may develop.[5]

The last stage of this disease is when the parasites enter the central nervous system. This usually occurs 6-12 months after the onset of the disease, but, like onset, this can vary. This stage is the classic picture of sleeping sickness. Sleep patterns are disturbed and the patient is generally fatigued and apathetic to surroundings. He or she may begin to sleep during the daytime and often must be roused to be fed. This "sleepiness" can lead to malnutrition and emaciation if the patient is not cared for. Headache, backache and neck stiffness are common complaints of this stage of trypanosomiasis.

In American trypanosomiasis (Chagas' disease) the acute form can also result in death in a very short time, due, not to central nervous system involvement, but usually to the myocardial damage caused by *T. cruzi* resulting in arrhythmias and heart failure. The acute form usually occurs in infants.

The commonest presenting signs are unilateral edema (especially of the eyelids) and locally involved lymph nodes.[5] Other signs include epistaxis and convulsions. The spleen and liver are often enlarged. In the chronic form, the dominating finding is some degree of myocardial fibrosis.

DIAGNOSIS

When diagnosing patients with possible infection by these six diseases, the physician must first consider the clinical picture and then perform diagnostic tests to recover the causative organism or bacilli from the patient. In all cases it is important to take a travel history from the patient. This may show that the patient was in an area where one or more of these diseases is endemic.

A finding of filarial worms or eggs in a skin sample examined by histological means confirms a diagnosis of *filariasis.* Nodule fluid should also be aspirated to find eggs or worms.[5] If the skin sample is negative (as occurs in lightly infected patients), the Mazzotti test should be performed. In this test, the patient is given a test dose of 50 mg diethyl carbamazine citrate. If the patient is infected, itching and a rash over the infected area will occur within 30 minutes to 24 hours.[12] The only non-invasive technique for diagnosing filariasis is an eye examination with a slit-lamp microscope. If microfilaria are seen in the cornea or anterior chamber, the patient is infected.

Leprosy is most clearly diagnosed by the finding of acid-fast bacilli in nasal mucosa examined by the slit-smear method. A histological examination of persistent, anaesthetic skin lesions should be done for patients from endemic areas. If round cell tuberculoid granulomas are found, the patient has tuberculoid leprosy.[5] There are no laboratory tests for leprosy.

Different diagnostic tests are used for each of the three strains of *leishmaniasis.* For kala-azar, biopsy material from the bone marrow or spleen treated with Giemsa stain can show the parasites. For cutaneous leishmaniasis, the finding of *Leishmania* from the edge of an ulcer is an important diagnostic finding. The leishmanin skin test is important for the diagnosis of mucocutaneous leishmaniasis since it is difficult to isolate the causative organism. This test is positive for about 90% of infected patients.[13]

When diagnosing *malaria,* the physician must first obtain a travel history from the patient. Next, a blood sample should be taken and thick film smears (to find the parasite) and thin

film smears (to determine the species) made from it. The thick film smears should be made and examined daily by a competent technician, since the parasite is periodically released and may not be present initially.[14] If any indication of cerebral malaria appears, chemotherapy should be immediately begun.

The finding of *Schistosoma* eggs in the urine or stool indicates a diagnosis of *schistosomiasis*. The nucleospore filtration method[15] and the Kato stool smear[16] will provide information on the intensity of the infection.

African trypanosomiasis is best diagnosed by finding trypanosomes in the blood. A thick blood slide is used for *T. rhodesians* while node aspirations are better for *T. gambiens*.[17] The blood concentration method of Woo[18] is best for detecting low levels of parasitemia and the anion exchange technique (usually feasible only in large centres) is the most sensitive, detecting as few as five parasites per ml of blood.[19]

In *American trypanosomiasis*, the trypanosomes are found by wet and stained blood films, by blood culture, or cerebral spinal fluid examination.[5] Animal inoculation is also used to determine the diagnosis.

TREATMENT

The most common treatment of the six WHO diseases is chemotherapy, although work is currently underway for the development of vaccines against leprosy, leishmaniasis, malaria and schistosomiasis.

The two most commonly used agents in *filariasis* treatment are diethyl carbamazine citrate and Suramin. Diethyl carbamazine citrate is only effective in killing the microfiliariae and can cause severe allergic reactions.[20] In such cases, it is given carefully with beta-methasone to reduce the reactions. Suramin is the only clinically available compound which kills the adult *O. volvulus* worms. Ivermectin is also used. This drug has a good anti-microfilarial activity.[21] Surgery is also a mode of treatment for filariasis. The intent of surgery is to excise all nodules thus diminishing the number of adult worms.

Dapsone (dimethyl diphenyl sulphate), despite increasing bacterial resistance, is the mainstay of *leprosy* treatment. However, Rifampin is a valuable alternative and renders patients non-infective within two weeks.[22] Treatment with these drugs should be continued for at least two years and until the skin smear results are negative.

Leishmaniasis is usually treated by antimonals, and pentavalent compounds (the commonest being Pentostam and Glucantime) are the most common and effective drugs in all forms of the disease.[13] Because these drugs have an arsenic-like toxicity, they must be carefully monitored. Amphothericin B, an antibiotic, is a potent anti-leishmanial drug but, due to its toxicity, it is restricted to mucocutaneous leishmaniasis cases that are resistant to antimonals.

The most commonly used drug for *malaria* is chloroquine (also used as prophylaxis). Primaquine is also used, sometimes in combination with chloroquine.[23] For treatment of chloroquine-resistant malaria, a combination of pyramethamine and sulfadoxine (Fansidar) is recommended. Unfortunately, Fansidar is not available in Canada.

Praziquantel (Biltricide) is a drug that is effective against all three major types of *schistosomiasis*.[24] In addition, metrofonate (Bilarcil), is commonly used against *S. hematobium*, and Oxamniquine is commonly used against *S. mansoni*.

The most common drugs used for early *African typanosomiasis* (i.e. before the central nervous system is involved) are Suramin for *T. rhodesiens* and Pentamide isethionate for *T. gambiens*.[25] Melarsopol is the most effective compound in treating the last stage of trypanosomiasis.

Drugs used to treat African trypanosomiasis are ineffective against *American trypanosomiasis*. However, Nitrofurazone is sometimes effective.[5]

CONCLUSION

These six diseases have detrimental, often devastating, effects on many millions of people, constituting a major stumbling block to development. At present, control of these diseases is slight: there are no effective vaccines available, and mass chemotherapy is limited by the ineffectiveness and toxicity of some agents. However, research into the causes and treatments of these diseases continues.

Although none of the six diseases are endemic in Canada, we need to be aware of the possibility that our patients who travel to tropical and subtropical countries or who have immigrated to Canada from such countries may be infected by these "exotic" diseases. We must be able to recognize and treat such illnesses when they appear, for they are already "rolling up to our threshold." □

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The Role of The Canadian Heart Foundations in Public and Professional Education

Frances M. Gregor,* R.N., M.N. and Helen Greenough,** B.Sc.

Halifax, N.S.

February is Heart Month and across Canada, volunteer canvassers will ask Canadians to donate to the Heart Fund. In 1983, over 19 million dollars were raised through business and personal donations to the annual fund-raising campaign of the Canadian Heart Foundation. In Nova Scotia, contributions from our population of some 825,000 persons permitted gross spending of over $850,000.00 by the local Heart Foundation. The Canadian Heart Foundation is the only voluntary health agency in the country solely devoted to the study, control and prevention of heart and blood vessel diseases. It is a federation of eight provincial Foundations and two Divisions.

Most physicians know that the Canadian Heart Foundation supports medical research. Seventy cents of every dollar contributed to the Heart Fund in 1982 was spent on research. Since 1955, over 125 million dollars has been allocated to support projects in cardiovascular and stroke research.[1]

A second major role is in public education which has been defined by the Foundation as "a process whereby people are encouraged to use information provided to them."[2] It is more than the simple dissemination of facts about the heart and heart disease. Public education is a form of health education, defined by Green *et al.* as "any combination of learning experiences designed to facilitate voluntary adaptations of behavior conducive to health."[3] Its activities occur in clinical settings, schools, the home, the community and the workplace. The methods include mass media, group work, audio-visual methods, patient teaching programs, health fairs and exhibits as well as routine health provider-consumer encounters.

The objectives of the Foundation in public education are:
— to prevent or reduce the occurence of cardiovascular disease in healthy individuals;
— to reduce premature death and disability from cardiovascular disease; and
— to improve the quality of life for those individuals afflicted with cardiovascular disease.[2]

Public education programs are designed to achieve these objectives. In 1982-83, the national public education program focused on the youth of Canada. Over 125,000 children in 1,350 schools participated in *Jump Rope for Heart,* a program to teach precision rope skipping and facts related to cardiovascular health. In 1983-84, the target for educational programs is the adult population, and will focus on signals and actions of heart attack. The objective of the *Signals and Actions* program is to decrease pre-hospital morality following heart attack. The program has three components: education of the public about signals of impending heart attack and the appropriate response to these signals; training in cardiopulmonary resuscitation (C.P.R.); and raising community awareness about the need for emergency medical services.

Provincial Foundations work cooperatively in the implementation of national programs and independently in response to provincial needs and conditions. In Nova Scotia, some recent projects in the area of public education include: 1) The development and evaluation of a junior high school curriculum guide entitled *Eating and Exercising for Heart Health*; 2) The design and evaluation of an activity resource book for elementary school classroom teachers called "Cardiovascular Activities for Classroom Teachers"; 3) A "Signals and Actions" media project was conducted to determine the effectiveness of the use of the media, — T.V., radio — during prime time, on the denial time of heart attack victims; 4) A self-teaching program for post heart attack victims called *Learn About Your Heart* was developed and evaluated; and 5) Currently, an *Instructional Program for Patients with Occlusive Arterial Disease of the Lower Limb* is being developed.

Support is also provided for service oriented projects. This is granted in the form of "seed money", providing opportunity for the role and/or need to be demonstrated. For example, a program called *Heart Talk,* which is an educational lecture/discussion series for post cardiac patients and their families, received start up support. In addition, the Nova Scotia Heart Foundation has been instrumental in starting several exercise rehabilitation programs for post heart attack patients across the province.

The provincial office stocks and supplies educational materials related to risk factors, structure and function of the cardiovascular system, treatment, patient education, family education, general public information, and information appropriate for use in the school system. Most of these materials are purchased from the American Heart Association, although some have been produced by Canadian Heart Foundations. More Canadian publications are introduced yearly. For example, Nova Scotia produced the junior high school curriculum guide *Eating and Exercising for Heart Health* and recently has assisted in the development of the *Risk Factor Statement by the Medical Advisory Committee of the Canadian Heart Foundation.* In addition, Nova Scotia initiated an examination of the reading levels of all Canadian Heart Foundation materials. This led to a general policy that reading levels of target populations be reflected in the content of future materials.

Through the provincial office liaison is maintained with groups who have mutual concerns and interests, for example, the Nova Scotia Council on Smoking and Health (N.S.C.S.H.). Its aim is to promote nonsmoking as a healthy lifestyle and this is accomplished through cooperative

*Chairman of the Nova Scotia Heart Foundation Public Education Committee.

**Public Education Coordinator of the Nova Scotia Heart Foundation.

delivery of public education projects, and encouragement and support of legislation aimed at regulating tobacco use and tobacco advertising. One current project in which the Heart Foundation is participating through the N.S.C.S.H. involves encouraging restauranteurs to provide non-smoking sections for their patrons, and those that do are presented with recognition certificates. The Heart Foundation was involved in organizing the newly formed group *Association for the Advancement of School Health,* whose main objectives are to promote comprehensive school health education and to provide a forum to collaborate in making contributions to school health education.

The third major role of the Foundation is in professional education through which new knowledge, discovered by research, is disseminated to physicians, nurses and paramedical professional personnel. This is done through a year-round program of scientific meetings, symposia, teaching sessions, in-hospital training programs, films, etc.

The Professional education activities supported by the Nova Scotia Heart Foundation include the distribution of current medical journals to doctors and nurses on a select mailing list. These publications are: *Current Concepts of Cerebrovascular Disease; Modern Concepts of Cardiovascular Disease;* and *Cardiovascular Nursing.*

The Don MacLeod Memorial Lecture is an annually sponsored event in which expenses are covered for a visiting lecturer to speak on cerebro or cardiovascular research at the "Friday at Four" lecture series organized by Continuing Medical Education (C.M.E.). C.M.E. at Dalhousie University has also been the recipient of yearly grants from the Nova Scotia Heart Foundation over the last several years, the amount of which now stands at $15,000.00.

One area in professional education that the Nova Scotia Heart Foundation plays a major role is Emergency Cardiac Care (E.C.C.) and C.P.R. The Foundation is responsible for the training of Instructors and Instructor-Trainers in ECC/CPR. A number of these Instructors are health professionals teaching both on the job (i.e. in hospitals) and in the community. A significant role is also played in the design, promotion and implementation of both advanced cardiac life support (A.C.L.S.) instructor and provider courses, as they are overseen by a regional planning committee made up of Heart Foundation representatives from the four Atlantic provinces. The planning committee works with Dalhousie University C.M.E. which administers these courses. Heart Foundations from the Atlantic provinces provided funding to purchase the equipment needed to carry out these courses.

Research projects in the area of professional education are also supported. For example, assistance was provided over a three-year period to a nurse researcher looking at the role of the nurse in hypertension control. Recently, two physiotherapists received support to develop and evaluate *Recovery Maximized. Nursing the Acute Stroke Patient to Maximize Functional Recovery.* The booklet is designed to improve the acute care management of those who have suffered a stroke. A grant was provided to print *A Handbook of Hypertension* for family physicians. This handbook was distributed to general practitioners in the province.

The mortality rate for all cardiovascular diseases for people under 65 years of age has decreased by 31% since 1955. It is obvious that the objectives of the Canadian Heart Foundations are being met through co-operative activities with international agencies, professional groups and the many medical and lay volunteers associated with the organization. The continued evidence of public confidence in the Heart Fund is demonstrated by large increases in contributions every year since its inception. This is tangible proof that Canadians in general and Nova Scotians in particular are aware of and willing to support the research and educational activities of the Canadian Heart Foundations. The same awareness and support is needed from all elements of the medical care groups and our politicians to meet the ultimate objective, i.e. eradication of cardiovascular diseases by the end of this century. □

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Cancer of the Cervix in Nova Scotia*

W.R. Carl Tupper, M.D., F.R.C.S.(C), M. Gregory Tompkins, M.D., F.R.C.S.(C) and Mary Nolan, H.R.A.

Halifax, N.S.

A review of the incidence and mortality of invasive carcinoma of the cervix in Nova Scotia over the years 1965 to 1978 showed a decrease in both, in keeping with results in other centres. In attempting to correlate the incidence with Papanicolaou (Pap) smear screening it was revealed that there was great variation in the incidence of this disease throughout the Province, with a marked increase in three counties. It is interesting to note that in 1981 less than 50% of the women in these counties were screened for cervical cancer.

To provide a survival curve for this disease during the same time period, a chart review of 1068 cases was carried out, revealing a five-year survival of 62.5%. A comparison of survival curves within three time periods showed no real improvement in survival over the fourteen years studied, suggesting that with our present treatment modules no real change in improvement can be expected, and that our efforts should now be directed toward prevention and early case finding.

For many years, gynecological malignancy has been of primary interest to the Department of Obstetrics and Gynecology, Dalhousie University. A multifaceted educational approach by the profession, medical students and the public at large, combined with an extensive on-site consultation with recognized world authorities in oncology, has been carried out in an effort to formulate routines. An updating of the necessary hardware to deliver appropriate control dosages of radiation and the establishment of a multidisciplinary Oncology Tumour Clinic, which provides a disposition service to all physicians and cancer patients in Nova Scotia, has been established. A Dysplasia Clinic, with colposcopy, has been offered to all physicians and patients within the Province. More recently, a Nova Scotia Trophoblastic Disease Registry has been established, registering all molar pregnancies in Nova Scotia.

Cancer of the cervix appears to offer the greatest challenge in gynecological malignancy, not only in treatment and quality of life post-treatment, but also in its prevention. We are concerned with the impact of this effort on the incidence and survival of patients with this disease, and thus have undertaken to review our experience with cancer of the cervix in Nova Scotia.

The Nova Scotia Tumour Clinic was established in 1953. Ten years later the Tumour Registry was set up under the Department of Health and, from this time onward, cancer became a notifiable disease in Nova Scotia. Registration of all patients was required. Doctors, clinics, hospital record departments, radiotherapy and pathology departments were asked to complete confidential primary reports of neoplasms. In practice, most of the information came from hospital records, radiotherapy and pathological department records, with a few reports received from specialized physicians such as dermatologists, whose patients were diagnosed and treated in an office environment.

The figures obtained from this registry are used to indicate trends in the incidence of various forms of cancer, including cancer of the cervix. Most published reports and data on incidence of this disease are based on individual hospital records, and often are coded as "uterus unspecified". There are very few reports representing the incidence data from a well defined homogeneous population, such as in Nova Scotia. Population figures remain fairly stationary; most of the population is of Caucasian origin; there is a fairly stable distribution of racial groups; and there are only two treatment centres located in the city, where practically all cases are treated. Our follow-up is comparatively good.

With the above in mind then, an attempt will be made to answer the following questions:

1. What has happened to the incidence and mortality of this disease in Nova Scotia, and how do they compare with other centres?
2. Is there any area in Nova Scotia with higher than average incidence?
3. What is the comparison of the incidence of cancer of the cervix with the incidence of corpus uteri in Nova Scotia; and
4. What is the outcome of treatment? How does survival compare with other centres?

Material

In the years 1965 to 1978 inclusive, 1068 cases of invasive carcinoma of the cervix were registered in Nova Scotia. Figure 1 shows the incidence each year per 100,000 women of twenty years and over. It is obvious that there is a decrease in incidence from 46/100,000 in 1965 to 21 in 1978, and in 1979 the incidence fell to 15.2, a relative decline of approximately fifty-seven percent. This decreasing incidence is similar to that found in other reports, and Figure 2 illustrates how this disease declined in two other centres, Louisville, Kentucky and Toledo, Ohio.[1]

During this same period, 2,139 cases of in-situ carcinoma of the cervix were registered. The incidence increased from 1965 to 1972, but from 1972 onward there is a decrease, which has continued well into the 1980s (Figure 3). In 1979 the incidence was 39.7 per 100,000 women over age 20.

Figure 4 compares the incidence of in-situ and invasive carcinoma of the cervix during the same period of time. Again one sees an almost parallel decrease in both diseases. This finding is different from published data from other centres, and particularly from British Columbia, where the decrease in invasive disease is accompanied by a rise in the in-situ lesions.[2]

*From the Division of Gynecological Oncology, Department of Obstetrics and Gynecology, Faculty of Medicine, Dalhousie University, Halifax, N.S.

Funded from a grant from the Nova Scotia Division, Canadian Cancer Society.

Reprint requests to: Dr. W.R.C. Tupper, Department of Obstetrics and Gynecology, 5821 University Avenue, Halifax, N.S., Canada B3H 1W3

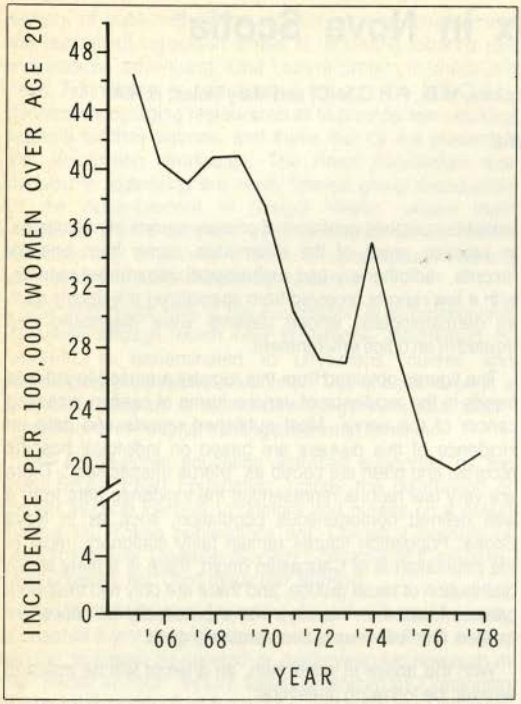

Fig. 1. Incidence rates per 100,000 women over age 20 in Nova Scotia for the years 1965-1978.

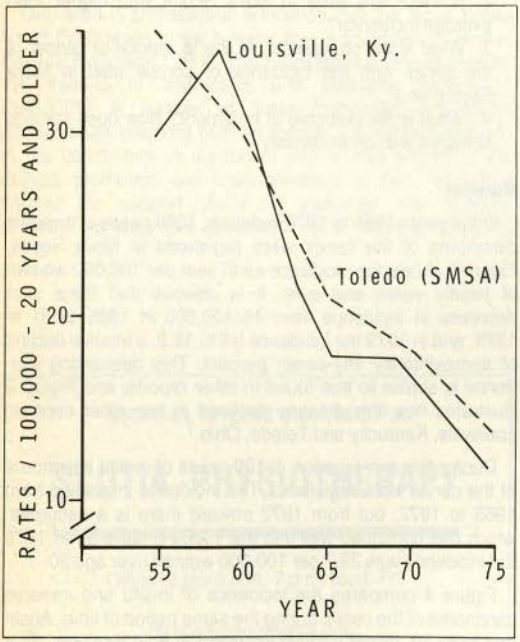

Fig. 2. Trend in the average crude age-adjusted incidence rates for invasive squamous carcinoma of the cervix in women 20 years and older: Louisville, Kentucky and Toledo (SMSA), Ohio.

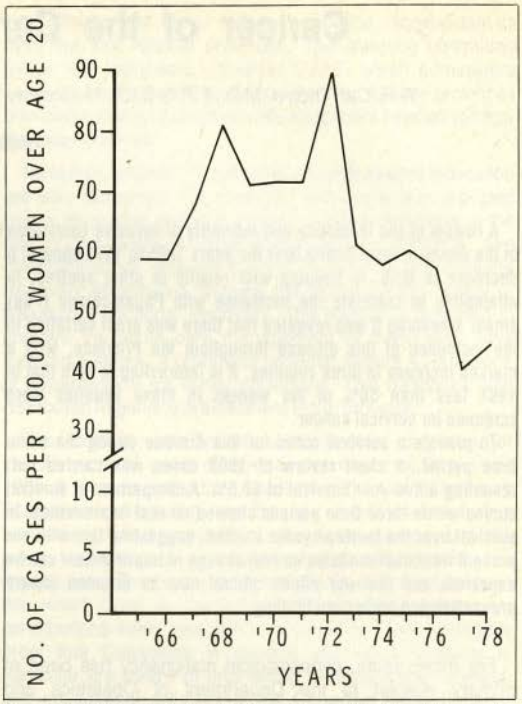

Fig. 3. Incidence rate trends in carcinoma-in-situ of the cervix in women over age 20 in Nova Scotia for the years 1965-1978.

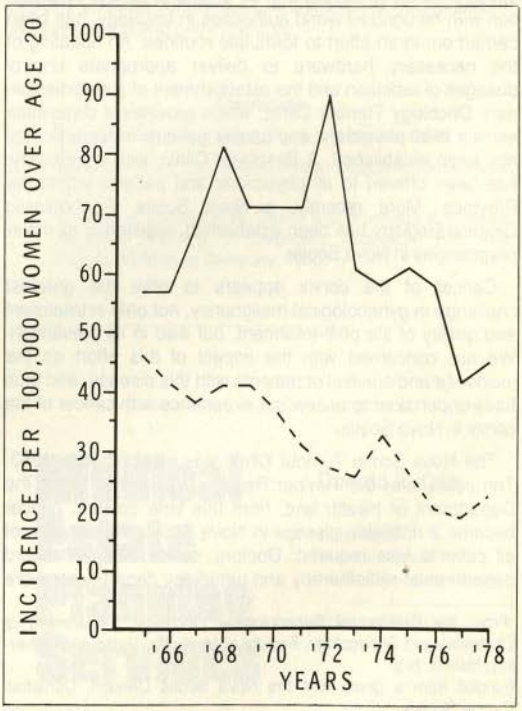

Fig. 4. Rate trends for carcinoma in situ (solid line) and invasive squamous carcinoma of the cervix (broken line) for the years 1965-1978.

The question is often asked, "Is the decrease in the incidence of invasive disease occurring in any special age group?" Figure 5 would tend to support the general feeling that there is no difference; all age groups are benefiting from the decreased incidence, although the decline is less rapid for those over 50 years of age.

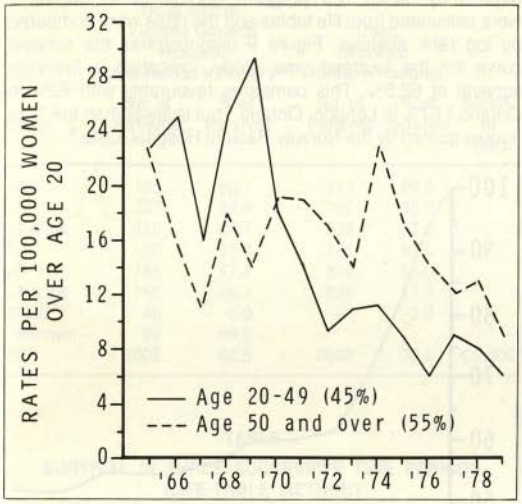

Fig. 5. Average annual incidence rates for invasive carcinoma of the cervix in women ages 50 and older vs. women ages 49 and under. Solid line, ages 20-49 (45%); broken line, ages 50 and over (55%).

It is interesting to note that as the incidence of carcinoma of the cervix decreases, the incidence of carcinoma of the corpus uteri increases (Figure 6). An increase in the incidence of carcinoma of the corpus uteri has been observed in the early seventies in other reports.[3,4]

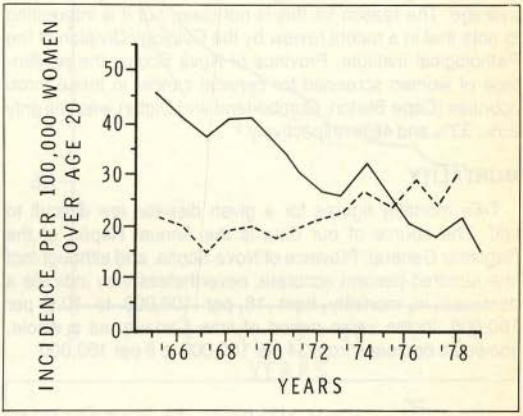

Fig. 6. Incidence rate trends for invasive carcinoma of the cervix and corpus uteri in Nova Scotia for the years 1965-1978.

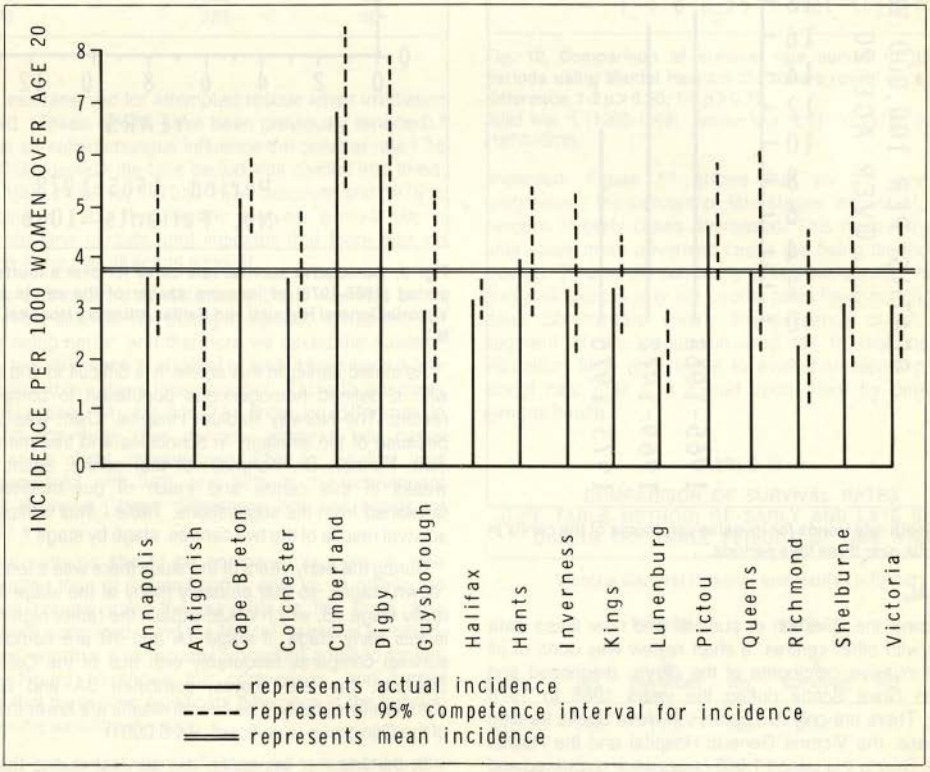

Fig. 7. Incidence of invasive carcinoma of the cervix in eighteen counties in Nova Scotia — 1965-1978. Solid line, actual incidence; broken line, 95% confidence interval; double solid line, mean incidence.

Over the years observations have been made by individuals and interested groups that some areas of Nova Scotia may have had an increased incidence of this disease. The data (Figure 7) for each county per 1,000 women over the age of 20 reveal considerable variables in the incidence. A chi-square test rejects the hypothesis of equal incidence in Nova Scotia ($\chi^2 = 82.29$ or $p < 0.001$), implying that the incidence in Cape Breton, Cumberland and Digby counties is above average, but that Antigonish and Lunenburg are below average. The reason for this is not clear, *but* it is interesting to note that in a recent review by the Cytology Division of the Pathological Institute, Province of Nova Scotia, the percentage of women screened for cervical cancer in these three counties (Cape Breton, Cumberland and Digby) was still only 25%, 33% and 46% respectively.[5]

MORTALITY

True mortality figures for a given disease are difficult to find. The source of our data is the Annual Report of the Registrar General, Province of Nova Scotia, and although not one hundred percent accurate, nevertheless they indicate a decrease in mortality from 18 per 100,000 to 10.5 per 100,000. In the same period of time Canada, as a whole, showed a decrease from 34 per 100,000 to 8 per 100,000.

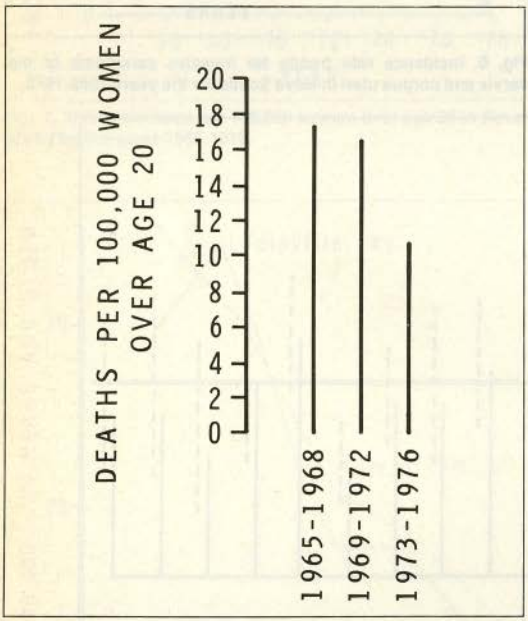

Fig. 8. Death rate trends for invasive carcinoma of the cervix in Nova Scotia over three time periods.

SURVIVAL

To answer the question of survival and how these data compare with other centres, a chart review was done of all cases of invasive carcinoma of the cervix, diagnosed and treated in Nova Scotia during the years 1965 to 1978 inclusive. There are only two centres in Nova Scotia treating this disease, the Victoria General Hospital and the Halifax Infirmary. During this period 1,068 cases were registered and 63 cases either received no treatment or went elsewhere for their therapy, leaving 1,005 cases for analysis.

A registered medical record librarian, supervised by one of the authors, reviewed each chart and abstracted 86 variables. The data were key-punched and stored for retrieval by the S.P.S.S. program. Cumulative survival rates were calculated from life tables and the rates were compared by log rank analysis. Figure 9 demonstrates the survival curve for the fourteen year study, indicating a five-year survival of 62.5%. This compares favourably with 62% in Ontario,[6] 67% in London, Ontario,[7] but is lower than the 70% figures quoted by the Norway Radium Hospital, Oslo.[8]

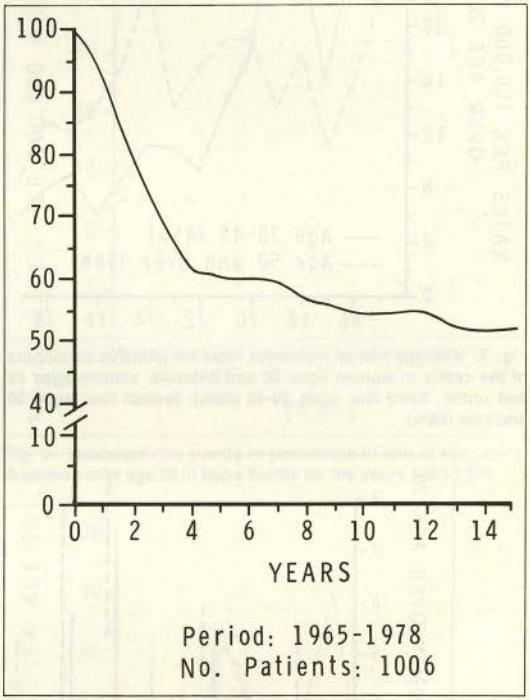

Fig. 9. Cumulative survival rate curve for over a fourteen year period (1965-1978) of invasive cancer of the cervix from the Victoria General Hospital and Halifax Infirmary Hospital, Halifax, N.S.

As stated earlier in this article, it is difficult to find a centre with a defined homogeneous population to compare our results. The Norway Radium Hospital, Oslo, was selected because of the similarity in schedules and treatment to our own. Further, Dr. Kottmeir, of that centre, spent several weeks in this centre and much of our treatment was fashioned from his suggestions. Table I thus compares the survival results of the two centres, stage by stage.[8]

During the early years of the study there was a tendency to "down stage", so that probably many of the stage 1A were really stage 1B, which would explain the rather high mortality in this early stage. If stage 1A and 1B are combined the survival compares favourably with that of the Oslo group. Similarly, the late stages, combined 3A and B, again compare favourably. The overall results are lower than those of the Oso group (significant; $P < 0.0001$).

In the hope of improving the survival during the period studied, various changes in treatment were made. However, the major treatment modality continued to be irradiation, and

TABLE I

INVASIVE CARCINONA OF THE CERVIX SURVIVAL COMPARISON (LIFE-TABLE METHOD)

Stage	Nova Scotia-Victoria General Hospital and Halifax Infirmary		Oslo, Norway, Radium Hospital		
	No. of Patients	% of Survival	No. of Patients	% of Survival	Signif. of Diff.
1A	105	82.1	147	99.0	
1B	307	87.4	792	85.0	
1A & 1B	412	84.7	939	87.0	
2A	190	61.8	371	69.0	
2B	166	47.2	374	55.0	
3A & 3B	162	24.7	224	27.0	
4ABC	46	0.0	94	3.0	
Unknown	29	66.0			
All	1005	62.5	2002	70.0	<0.0001

TABLE II

SURVIVAL IN THREE SUCCESSIVE TIME PERIODS (LIFE-TABLE METHOD)

Period	Number	% Survival
1965-1969	398	63
1970-1975	350	62
1975-1978	228	58

surgery was reserved for attempted rescue when irradiation had failed. (These results have been previously reported).[9] Did these so-called changes influence the survival rate? To answer this question the time period was divided into three: 1965 to 1969 inclusive, 1970 to 1974 inclusive, and 1975 to 1978 inclusive. Table II shows the five-year survival rate in these three time periods, and indicates that there was no difference in the overall actual survival.

In spite of the fact that statistically there was little or no change in the survival rate during this period, it was felt that we were doing better, and therefore we asked the question "Is there any difference in survival of early cases versus late cases in the above three time periods?" Table III illustrates that early cases (1A, 1B and 2A) show no difference in survival in the three time periods, whereas late cases indicate some slight improvement (25% in the first time period compared with 34% and 37% in the subsequent periods). However, these differences are not significant ($P < 0.05$).

Survival patterns should be compared properly in their entirety rather than at isolated points only so, in addition to the above comparisons, the survivals in the three time periods were compared using the Mantel Haenszel summary chi-square, which is a chi-square procedure adapted for this purpose. Figure 10 shows this comparison, and further indicates that there is no significant difference in the survival rates over the past fifteen years.

Although the overall survival rates did not change, the question was asked whether the type of patient could have changed, thus explaining why the overall survival has not

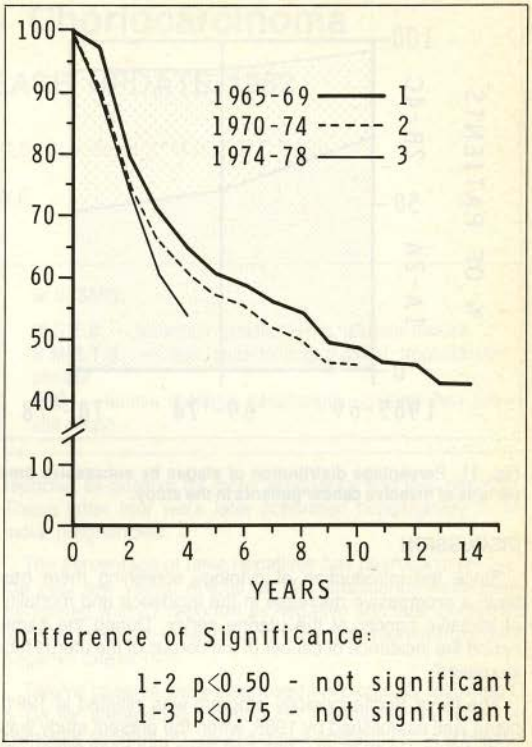

Fig. 10. Comparison of survival rate curves in three time periods using Mantel Haenzel chi square reveal no significant difference. 1-2 $p > 0.50$; 1-3 $p > 0.75$.
Solid line, **1** (1965-1969); broken line, **2** (1970-1974); thin line, **3** (1975-1978).

improved. Figure 11 shows that as the time period progressed, the percent of late stages increased, and the percent of early cases decreased. This means that in the later years more advanced cases are being diagnosed and treated. This would adversely affect our survival rates and may well explain why our overall rates have not changed. It does demonstrate clearly the presence of an isolated segment of the population who fail to respond to the education blitz, and refuse to avail themselves of professional help until it is forced upon them by deteriorating general health.

TABLE III

COMPARISON OF SURVIVAL RATES (LIFE TABLE METHOD) OF EARLY AND LATE STAGES DURING THE THREE PERIODS OF TIME STUDY

Victoria General Hospital and Halifax Infirmary

Stage	1965-69 No.	1965-69 % Survived	1970-74 No.	1970-74 % Survived	1975-78 No.	1975-78 % Survived
A1, 1B 2A	286	77.0	199	79.7	117	79.2
2B, 3AB, 4ABC	112	25.2	151	34.0	111	36.6

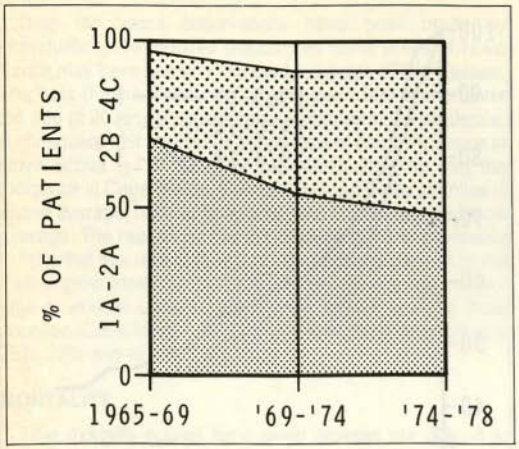

Fig. 11. Percentage distribution of stages by successive time periods of invasive cancer patients in the study.

DISCUSSION

Since the introduction of cytology screening there has been a progressive decrease in the incidence and mortality of invasive cancer of the uterine cervix. During the same period the incidence of cancer of the corpus of the uterus has increased.

The Nova Scotia cytology program was initiated in 1961, being well established by 1965, when the present study was undertaken. By 1976 over 985,510 Paps had been taken; yet 80% of cases of invasive cancer of the cervix had never had a previous Pap smear. (Results of uterine screening in the Atlantic Provinces: S.C. Robinson).[10] This suggests failure to screen patients at risk, and that the screening program is far from complete. Recently the data on all Pap smears has been computerized, and differentiation between primary and secondary screening will be possible — thus permitting a more accurate assessment of the impact that this simple test has had on the incidence and mortality of cervical disease.

With the media reporting from time to time the influence of environmental factors in the etiology of cancer, it is natural when several cancers occur in an isolated area that the question then arises, "Is there an epidemic of cancer in our area?" An attempt was made to answer this inquiry with the incidence of invasive cancer of the cervix in various counties, realizing that there may be many factors influencing the findings, such as the number of women in a specific age group, the overlapping of county populations, the individual care provided to the particular area, and many others. Nevertheless the information provided does indeed indicate a higher than average incidence in three counties. This finding is being investigated further. It is interesting to note that less than a third of the women at risk in these counties have been screened for cervical cancer.

The rise in incidence of in situ carcinoma of the cervix from 1965 to 1972 could well be explained by suggesting that the Pap smear program, initiated by the Department in the sixties, uncovered a large accumulated population thus leading to an apparent but spurious rise in incidence which was only temporary. The recent decline could, in fact, reflect treatment of the precursor dysplasia as initiated by the oncology group early in the seventies.

The results in treatment of invasive cancer of the cervix are the same, state by stage, in Halifax and Oslo, and also are no better than they were fourteen years ago. It is interesting that in the report of the Task Force (Cervical Cancer Screening Programs, 1982) recently published, the same conclusion was drawn for all of Canada. This would suggest that with the present tools at hand, one can expect very little further salvage of identified cases.

It has been suggested that invasive carcinoma is a sequential disease. The process begins with the induction of cellular changes within the nucleus of normal squamous epithelium of the cervix. These changes progress through cellular dysplasia, carcinoma in situ, micro-invasion, and eventually invasive disease. During the early stages of this process in many people the changes are reversible, and do indeed reverse. However as the dysplastic changes become less mild, fewer are reversible and most of the severe cases do go on to invasive cancer. The assumption is that changes identified before becoming freshly invasive can be treated successfully by one means or another.

The above concept has been readily accepted by the medical profession of Nova Scotia, and now all cases with abnormal Pap smears are recommended to be examined by colposcopy.

This will undoubtedly *reduce the incidence of invasive carcinoma*, and will lead to early diagnosis and treatment of a few cases, but *it will not* change the overall mortality of the disease. Since most of the invasive cases come from unscreened population, the obvious front for attack should be in the direction of early case finding.

CONCLUSIONS

1. Both the incidence and the mortality of invasive cancer of the cervix in Nova Scotia have continuously decreased over the study period, 1965-1978.
2. There are three counties in Nova Scotia with an incidence above the normal average.
3. The results in treating invasive cancer of the cervix are much the same, stage by stage, in Oslo and Halifax, and are no better than they were twenty years ago.
4. Since it is doubtful that salvage will be increased by further improvement in treatment, the obvious answer is better early case finding. To date most of the efforts have been *towards already identified cases.* The new approach must *concentrate on early diagnosis* by increasing our screening program to cover *all* women in Nova Scotia. □

ACKNOWLEDGEMENT

Acknowledgement is made to the Department of Community Health and Epidemiology (Dr. Aden C. Irwin and H. Jean Thiebaux) for help in the statistical analysis of this paper and to Helen Snow, R.N.

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Continued on page 32

Molar Pregnancy — Choriocarcinoma

TROPHOBLASTIC DISEASE UPDATE, 1982

B. Pierce,* R.N., R.C. Fraser,** M.D., F.R.C.S.(C), I. Zayid,*** M.D., F.R.C.P.(C)

Halifax, N.S.

This is the third annual report from the Nova Scotia Gestational Trophoblastic Disease Registry and Surveillance Clinic.

This year, a total of 35 new patients were registered of which 32 were diagnosed initially as benign hydatidiform molar pregnancies. Five of them subsequently developed non-metastatic disease, and two developed metastatic disease. Two patients were diagnosed as "questionable" molar pregnancies, and one patient was found to have choriocarcinoma following a salpingectomy for an ectopic pregnancy. (Table I)

TABLE I

THE EXPERIENCE OF THE REGISTRY IN 1982

Total number of patients registered	**35**	
Benign Mole	25	
N.M.G.T.D.	5	
M.G.T.D. (post M. preg)	2	
M.G.T.D. (post ectopic)	1	(choriocarcionma)
"Questionable Mole"	2	
TOTAL	35	

The two patients falling into the category of the "questionable" mole [*N S Med Bull* 1981; **61:**151] had uneventful follow-up with both having titres which returned to normal within 15 weeks.

The study regarding the "questionable mole" was begun in May of 1981 and to date, a total of 11 patients have been entered. This study continues and your support is appreciated.

The geographical breakdown of patients registered in 1982 is as follows: Nova Scotia 19(3), New Brunswick 7(3), Newfoundland 9(2). The numbers in parenthesis indicate the patients who developed N.M.G.T.D. or M.G.T.D. (invasive mole or choriocarcinoma) and required adjunctive chemotherapy and/or hysterectomy to eradicate their disease completely.

As in past years, the majority of patients with molar pregnancies were diagnosed by ultrasound. A total of 24 ultrasound examinations were performed with 20 being reported as positive for molar pregnancy and four negative. These latter four were later confirmed histologically to be molar pregnancies.

GLOSSARY:

M.G.T.D. — Metastatic gestational trophoblastic disease
N.M.G.T.D. — Non metastatic gestational trophoblastic disease
HCG — Human chorionic gonadotropin (serum) Beta sub unit assay)

The percentage of false negatives has been constant over the past few years. A total of 116 ultrasounds have been performed with 17 reported as negative and later confirmed to be hydatidiform molar pregnancies. This represents a false negative rate of 14%.

Twelve patients were diagnosed histologically following D & Cs for therapeutic abortion (1); incomplete abortion (1); complete abortion (4); spontaneous abortion (1); and missed abortion (5). One patient was diagnosed following salpingectomy for ectopic pregnancy.

Approximately 16% of the patients who have had a molar pregnancy will require chemotherapy and/or hysterectomy to completely eradicate their disease. For this reason follow-up with HCG titres is *essential* and can not be too greatly emphasized.

The follow-up protocol for patients with gestational trophoblastic disease as outlined by the Nova Scotia Gestational Trophoblastic Disease Registry is as follows:

After hospital discharge:

a) HCG weekly until three consecutive normal levels are achieved. Then. . . . . .

b) HCG monthly for one year. Pregnancy is permissible after 6 months of normal titres. If pregnancy is suspected an ultrasound is indicated for early confirmation.

If chemotherapy was required then follow-up is as follows:

a) HCG weekly until three consecutive normal levels are achieved. Then. . . . . .

b) HCG monthly for one year. Then. . . . . .

c) HCG once every three months for one year. Pregnancy is permissible after 12 months of normal titres. If pregnancy is suspected an ultrasound is indicated for early confirmation.

If chemotherapy was administered for high risk trophoblastic disease follow-up is as follows:

a) HCG weekly until three consecutive normal levels are achieved. Then. . . . . .

Nova Scotia Gestational Trophoblastic Disease Registry, Department of Obstetrics and Gynecology, Dalhousie University, Halifax, Nova Scotia, *Co-ordinator GTD Registry, **Director Gynecology Oncology, ***Professor of Pathology, Department of Pathology, D.J. MacKenzie Diagnostic Centre.
The Nova Scotia Registry is located on the 5th Floor, Ambulatory Care Center, 5820 University Avenue, Halifax, N.S. B3H 1V7.

b) HCG once a month for *two* years. Pregnancy is permissible after two years of normal titres. Once again if pregnancy is suspected an ultrasound is indicated.

c) HCG once every three months for the third year.

d) HCG once every six months for the fourth year.

e) Yearly thereafter.

Because a future pregnancy occasionally reactivates trophoblastic tissue, *EVERY* subsequent pregnancy must be followed by an HCG blood test six weeks after delivery or abortion.

TOTAL EXPERIENCE OF REGISTRY

The Nova Scotia Gestational Trophoblastic Disease Registry and Surveillance Clinic now has a total of 277 patients registered. (Table II) Two hundred and twenty-eight were confirmed to be benign hydatidiform moles and required no treatment other than the original D & C. Forty-nine, or 18%,* required adjunctive chemotherapy and/or hysterectomy; thirty-five for non-metastatic trophoblastic disease, and fourteen for metastatic disease. (Table III)

TABLE II

TOTAL EXPERIENCE

	1965-1970	1971-1975	1976-1980	1981-1982
Nova Scotia	13(4)	27(8)	105(13)	31(5)
New Brunswick		8(6)	30(2)	17(4)
Prince Edward Island			4(0)	5(0)
Newfoundland			15(4)	21(3)
St. Pierre			1(0)	
TOTAL	13	35	155	74
Total patients registered			277	
Total Patients Requiring Rx			49(18%)	

TABLE III

1. Benign Mole	228
2. N.M.G.T.D.	35
3. M.G.T.D. (post molar pregnancy — 9) (post ectopic pregnancy — 1) (post normal pregnancy — 4)	14
TOTAL	277

Thirty-four of the thirty-five patients with NMGTD were successfully treated with 1-4 courses of chemotherapy. One patient required a hysterectomy in addition to combined agent chemotherapy in order to remove drug resistant disease. All thirty-five patients remain in remission.

Nine of the fourteen patients with MGTD developed metastatic disease following a molar pregnancy. All were successfully treated with 1-8 courses of chemotherapy and all remain in remission.

*16% of patients with hydatidiform mole required adjunctive therapy. The 18% mentioned above includes choriocarcinoma post ectopic pregnancy (1), and post normal pregnancy (4).

One patient was diagnosed as metastatic disease (choriocarcinoma) following a left salpingectomy for an ectopic pregnancy. She required hysterectomy, omentectomy, and left oöphorectomy in addition to chemotherapy to erradicate her disease completely. She also remains in remission.

Four patients were found to have metastatic disease following a normal pregnancy. Only one patient responded to treatment and remains well and free of disease. The remaining three patients died; two from drug resistant choriocarcinoma, and one due to pulmonary embolus. One had a hydatidiform mole five years prior to her last delivery.

The diagnostic possibility of choriocarcinoma following a normal pregnancy must be considered in all female patients demonstrating bizarre signs and symtoms (e.g. neurological symptoms and/or persistent bleeding). These patients should be screened for the presence of chorionic gonadatrophin hormone in the hope that earlier diagnosis and treatment of this rare condition will increase the percentage of curable patients.

In addition to the normal functions of the registry (follow-up and surveillance), two other projects were undertaken and completed this year.

The first was the creation of a "normal regression corridor" for HCG titres based on the experience of this clinic. Fifty-six patients whose titres had fallen to within normal limits within 15 weeks of uterine evacuation were selected, and the regression curves of their Beta HCG titres were analyzed.

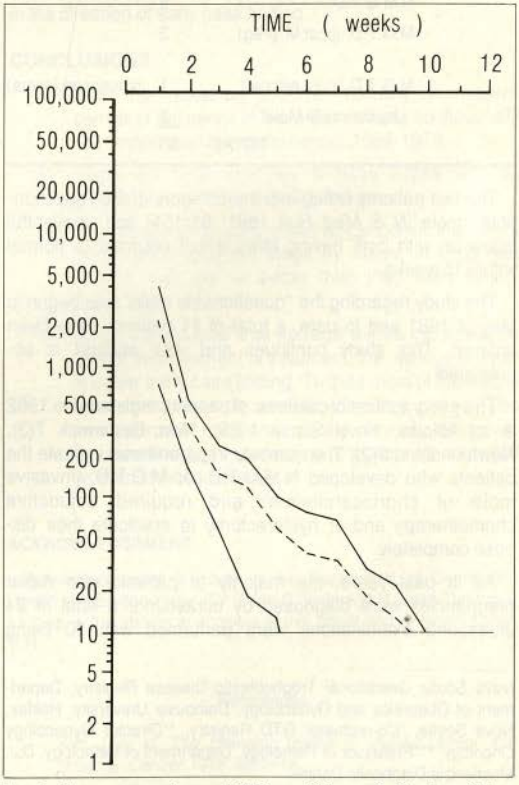

Fig. 1. The mean value and 95% confidence limits describing the normal post-molar B-HCG regression curve

By establishing the 95% confidence limits of the weekly mean HCG titre values of these patients (Figure 1), one sees demonstrated a spontaneous progressive fall in serum HCG titres providing us with a "normal" regression corridor. By then comparing the curves of nine patients with non-metastatic or metastatic disease with the normal regression corridor, it becomes obvious that the "abnormal" group very quickly diviates from the normal regression corridor. (Figure 2)

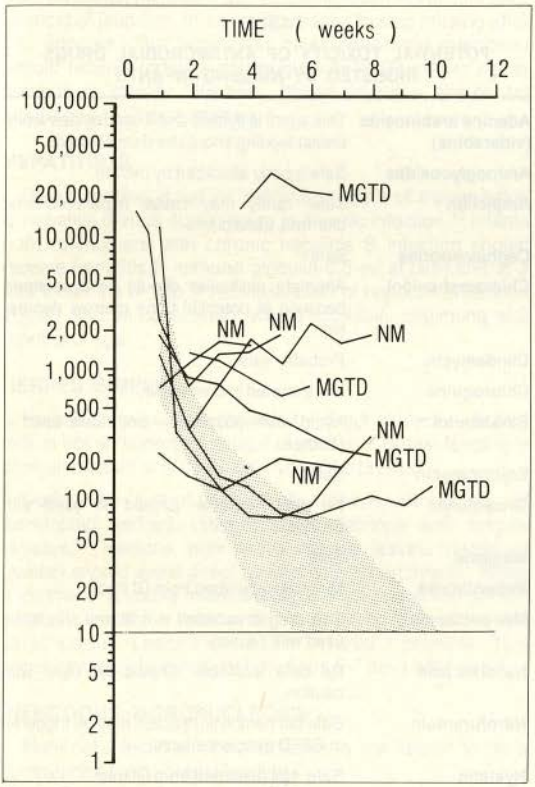

Fig. 2. The individual serum B-HCG titre curves of a patient with NMGTD or MGTD compared with the normal regression corridor

This has proved to be a useful tool in the assessment of HCG titres of patients with trophoblastic disease. It has been helpful also in more accurately assessing the unreliable patient whose specimens are not obtained on a regular basis.

The second was the design and printing of a pamphlet for patient education. It briefly explains molar pregnancy, attempts to answer a few of the commonly asked questions, and stresses the necessity of regular follow-up with Beta Subunit HCG titres. A copy of this pamphlet is sent automatically to the referring physician, to be given to the patient once she has been identified and registered. Additional copies are available on request.

Once again, we would like to extend our sincere thanks to the patients, physicians, and pathologists for their continued interest and support. □

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Breast Feeding by Infected Mothers*

The Canadian Paediatric Society has taken a formal stand in support of breast feeding, recognizing its many advantages over bottle feeding.[1-3] There are occasions when the advisability of breast feeding is questioned, such as when a nursing mother becomes ill with infection. In this situation, the infant might be harmed by microbes or antibiotics present in the milk or in blood ingested from cracked nipples. The closeness between mother and infant required for nursing may enhance spread of certain agents, such as respiratory viruses. This review discusses some common infections which may be acquired by nursing mothers at home and indicates their significance for the infant. In general, most nonserious maternal infections should not interfere with breast feeding.

ANTIBIOTICS

Most systemically distributed antimicrobial drugs penetrate to some extent into breast milk. The total amount of drug ingested by the infant rarely exceeds 1-2% of the maternal dose or 10-20% of the usual infant dosage. Whether the nursing infant will be harmed by such exposure depends upon the drug and the duration of exposure. Drugs usually contraindicated for infants should be avoided in the nursing mother ie tetracycline, sulfonamides (< 2 weeks of age). Similarly, use of drugs with potentially serious adverse effects, such as chloramphenicol, should be avoided. Table I indicates the potential toxicity of antibiotics ingested by nursing infants.[4-8]

BREAST INFECTION

About 1-5% of nursing mothers experience infection of the milk ducts (mastitis) in a portion of one or both breasts, usually within the first few weeks postpartum. Mastitis is associated with localized tenderness, redness and mild swelling. Bacteria responsible for infection (usually *Staphylococcus aureus*) are usually introduced into the milk ducts from the infants mouth. This infection improves quickly with antibiotic therapy and continued nursing from the affected breast is generally advised.[9] No illness has been reported in infants as a result of this practice and continued milk removal from the infected breast hastens recovery and diminishes the risk of abscess formation.[10]

Breast abscess, characterized by fluctuant swelling, usually develops after several days of untreated mastitis. Milk in this situation may be heavily contaminated with bacteria and infants nursing from abscessed breasts have developed fatal lung infections.[11] Use of the involved breast should be avoided until infection has cleared.

FEVER

Fever, by itself, should not be a contraindication to breast feeding. The cause of fever should be sought and considered separately. Antipyretic medications (acetylsalicylic acid, acetaminophen) are excreted in breast milk but short-term exposure of healthy infants in generally harmless.

GASTROENTERITIS

Microbial agents causing gastroenteritis are not transmitted in breast milk. Careful handwashing is necessary to avoid transmitting fecal bacteria and viruses to the infant.

TABLE I

POTENTIAL TOXICITY OF ANTIMICROBIAL DRUGS INGESTED BY NURSING INFANTS

Adenine arabinoside (vidarabine)	This agent is cytoxic and if used systemically breast feeding should be discontinued.
Aminoglycosides	Safe (poorly absorbed by mouth)
Ampicillin	Safe; rarely may cause hypersensitivity, diarrhea, candidiasis
Cephalosporins	Safe
Chloramphenicol	Alternate antibiotics should be considered because of potential bone marrow depression.
Clindamycin	Probably safe
Chloroquine	Not excreted in breast milk.
Ethambutol	Avoid when possible — not safely used in children.
Erythromycin	Safe
Griseofulvin	No data available. Should be used with caution.
Isoniazid	Safe
Mebendazole	Minimally absorbed from GI tract.
Metronidazole	This drug is excreted in milk and should be used with caution.
Nalidixic acid	No data available. Should be used with caution.
Nitrofurantoin	Safe but hemolytic reaction may be triggered in G6PD deficient infants.
Nystatin	Safe. Not absorbed from GI tract.
Para aminosalicylic acid	Probably safe.
Penicillin	Safe, rarely may cause hypersensitivity.
Pyrvinium pamoate	Safe
Rifampin	Safe
Sulfonamides	Avoid during first two weeks of life because of interference with bilirubin binding. Can cause hemolytic anemia in infants with G6PD deficiency.
Tetracycline	Theoretically may cause staining of teeth. Alternate antibiotics usually available.
Trimethoprim-sulfamethoxazole	See precautions for sulfonamides.
Vancomycin	Not absorbed from maternal intestine when give p.o.

"Safe", used in this table, means that adverse effects are unlikely to be observed in normal infants exposed to drug for short periods. All drugs absorbed by the infant can rarely cause hypersensitivity reactions. Broad spectrum antibiotics ingested in milk can alter the infants GI flora, resulting occasionally in diarrhea.

*A statement issued by The Canadian Paediatric Society. (*CPS News Bulletin Supplement;* Vol. XIV, No. 3)

GONORRHEA

Neisseria gonorrhea is usually acquired by the infant during birth from infected maternal genital secretions. Breast feeding is not contraindicated in infected mothers but the mother and newborn infant should be treated with systemic antibiotics.[12]

HEPATITIS A

The route of spread of hepatitis A virus is fecal-oral rather than via breast milk. As maximal contagiousness occurs prior to onset of jaundice, there is little reason to stop nursing after this appears. The baby (and other household members) should receive immune serum globulin (0.02 ml per kg) to prevent or modify infection. Proper hygiene should be emphasized to these mothers.

HEPATITIS B

Breast feeding is not an important means of transmission of hepatitis B virus during acute or chronic infection.[13] Infants born to mothers with chronic hepatitis B infection should receive hepatitis B immune globulin 0.5 ml at birth and at 3 and 6 months of age.[14] Administration of hepatitis B vaccine should also be considered, where available, beginning at 3 months of age.

HERPES SIMPLEX

Herpes simplex virus spreads by direct contact — breast milk is not an important source of infection. Breast feeding is contraindicated only if lesions involve the breast.

During the first month of life, infants are at risk of developing serious, disseminated infections with herpes simplex.[15] Persons with active herpes lesions (facial or genital) should avoid direct contact with young infants. When a mother of a young infant develops a cold sore, she should carefully wash her hands before touching the infant and avoid kissing. Lesions should be covered if possible. Cold sores generally become noninfectious 5-7 days after onset.

INFECTIOUS MONONUCLEOSIS

Maternal infectious mononucleosis is not known to be a contraindication to breast feeding.

MEASLES

If a mother develops measles, the baby should receive passive immunization with immune serum globulin (0.25 ml per kg). Breast feeding may continue. Active measles immunization should be given 3 months afterward, if the infant is 12 months or older.

MUMPS

This infection in unlikely to cause serious disease in an infant. Breast feeding may continue.

PERTUSSIS

If a mother has pertussis both she and her infant (and possibly other household contacts) should be treated with oral erythromycin. Breast feeding need not be discontinued.

RESPIRATORY TRACT INFECTIONS

Respiratory viruses spread as aerosols and by direct contact, not in breast milk. Handwashing prior to handling the infant helps to reduce spread of common cold viruses. Maternal over-use of oral decongestants may result in mild sedation of the infant but use of nasal sprays or drops generally has no effect on the nursing infant.

RUBELLA

This infection is unlikely to cause serious disease in an infant: breast feeding is not contraindicated. Administration of live, attenuated rubella virus vaccine to nursing mothers has resulted in no harm to their infants. Rarely, vaccine virus may spread to the infant in breast milk and stimulate immunity.

SCABIES

Scabies is communicable between mother and infant by direct contact but it should not interfere with breast feeding. Care should be taken to avoid contamination of the nipples with agents used to treat scabies. Lindane (gamma benzene hexachloride) is measurable in milk following cutaneous application: safer alternatives in the nursing mother are crotamiton (Eurax ®), benzyl benzoate and sulfur.

TUBERCULOSIS

An infant born to a woman with active, untreated pulmonary tuberculosis should be separated from the mother until treatment renders her infection non-contagious. Breast feeding may be started at this time. The infant should be placed on isoniazid preventive therapy.

An infant born to a mother who is being treated for tuberculosis which is considered non-contagious may breast feed. When treatment includes drugs not recommended for use in children (such as ethambutol), treatment should be altered or, if this is not feasible, breast feeding should be re-considered.

While breast feeding by mothers receiving TB chemotherapy is thought generally to be safe, it does involve chronic, unnecessary exposure of infants to multiple drugs. The advisability of such exposure needs to be weighed against the potential benefits of breast feeding in individual cases.

VARICELLA INFECTIONS

The infant of a mother who develops chickenpox will likely become infected, regardless of how it is fed. Varicella in infants is generally mild. A rare exception is the infant whose mother develops chickenpox 5 days before to 2 days after delivery: such infants may develop severe infection and should be given varicella-zoster immune globulin (VZIG).

Infants whose mothers develop shingles (herpes zoster) have a lower risk of subsequent chickenpox, as less virus is present and residual passive antibody may attenuate infection. Precautions to be observed to protect the infant include careful handwashing and covering involved areas. Breast feeding is contraindicated when breast skin is involved, until all lesions have crusted. □

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Continued on page 32.

The Old Potted Plant Game

An Allegory from the Days of Private Practice

Robert W. Napier,* M.D.

Halifax, N.S.

At one time the small island of San Remia was completely uninhabited. This seemed hard to believe. It was a beautiful island whose soil encouraged all manner of healthy growth. And further, it was located on the current of a natural ocean stream which, in its cruising, picked up other currents which influenced its course in such a way that without giving up any of its naturalness, it tended to carry all the way around the world. But hard in its folklore was the idea that at one time it was completely uninhabited.

The early colonization of the sunny island could not have been difficult, since its original settlers were a band from the tribe of Santus. The Santus knew like a creed the idea of helping each other. And so, with an abundance of fertile land and hands to help, it was a natural thing for the island to become developed in an orderly and happy way.

Some from other tribes and from their own tribe came to visit the pioneer Santus, and saw the beauty and convenience of such a life; but felt they needed more diversity or were themselves unable to live comfortably in such a small and familiar setting. These went back to their own lands, or perhaps pushed on in the search for other lands.

The San Remians welcomed all who came; and adopted ideas that contributed to the conviviality of the lush island. And they were amazed by the others who came and chose not to stay, since they felt San Remia was complete. If they tried to say why of their island they would have called it a stable, value-based homeland having a happy blend of structure and freedom.

After many generations, the personality of the island was set. Some rickety or unsafe huts had been replaced or improved, wharves and moorings had been established that made for easy contact with neighbouring islands; and, most importantly, the upbringing of the children was such that they continued the pattern of cooperative improvement. Those who chose to go away travelled far and well because of the fineness of their training and the magic of their initiative.

And then the great storm came. And but for a brief calm that made the whole ocean seem like a mirror, there was no other warning. In a single day the forces of nature destroyed all building on the island; but the people stood firm and were not surprised. And while they had never before seen a typhoon, they suspected such a thing was possible and had gone into a great valley of the island and remained there until the storm was spent.

On resuming island life, the San Remians rethought the arrangement of their needs. . .but constructed essentially the same homes again, and again were happy. They replaced their wharves and built new boats and their industry was a model to their young.

*Family Physician, 5513 Spring Garden Rd., Halifax, N.S.

The same storm that destroyed San Remia's homes capsized a large ocean liner — a pleasure boat — and strewed passengers and wreckage over a large area. One survivor, a gambler, was left clinging to the wooden casing of a door. After many days the young man reached a small, wooded island. From the resources there and by his own skill he was able to make a crude raft. And this, in the stream of the ocean, carried him to another island whose people cared little for outsiders. They had no food to spare but were impressed by the gambler's determination; and helped construct for him a mast and sail. With these he navigated the current cunningly and swiftly, and soon came to another, more hospitable island whose natural springs and vegetation restored his health and strength, and whose people saw in him a great will to live and to accomplish many things. By these assistances the young man became both strong and keen of mind and filled with a zeal to change things for the better. Why? Because these helpers and admirers were sure he would do great deeds.

Modifications were made to his raft to store provisions and, with such sophistication, he easily cruised the ocean's current to yet another island. Here, tales of his fervor and messianic bent had gone before him. The inhabitants were intrigued by his coming and captivated on contact. He lived and moved among them, a hero in ascendancy. On leaving, it was a spontaneous and communal act of participation that gave to the young man a power launch and the services of a flawless navigator for as long as he should wish.

And so the young gambler reached San Remia. . .and fell fastly in love with the life he felt was the most natural and happy he had ever known.

Among all the San Remians he sensed their conviction that he would make many things better in the years of his life. And so he resolved in a genuine way to do good. He taught the children to be artful and shrewd, and assisted the parents in planting and planning and conserving in a hundred ways. The people loved him for his industry; but mostly for his sincerity and fervor.

In his resolve to make the little island perfect, the young gambler conceived of a great and sturdy tower that would house every San Remian in an equal and convivial way, free much of the fertile land for more and richer produce and remove any trace of material need. So happy were the Santus with the young man's fervent dream that, over their misgivings, they allowed themselves to be convinced that they could undergo a major structural change without jeopardy to their old happiness. They put a price on their rich produce and agreed to import steel and the means of constructing a concrete tower embedded deeply into the rich and sunny soil. The Santus now became very involved in change and, while it was exciting, there was no time left for the old ways.

Over many months the great tower climbed to thirty-eight gray storeys of concrete and steel. It impressed all of the gentle San Remians. They could not bring themselves to speak of its dominance. Also, it altered the sunniness and forever-sweepingness of the island days. Were these gone? The young gambler explained that his dream would work economies in time and eventually all would be better than they had ever known. To live with nature is fine, he professed, but to improve on nature is the fulfillment of man.

In their industry, the San Remians saw but hardly read the mirror of the water. It came wildly out of the east — a giant typhoon, raging and relentless — and hurled itself at the giant tower with cataclysmic force. The great implacable tower strained mightily at its foundations in the fury of the storm before it toppled massively to the west upturning the green island in the roiling sea like a potted plant.

The Santus easily got off of the island. They were natural swimmers. But they had nowhere to go. The gambler grimly, doggedly climbed to the upper surface of the capsized tower. He felt bewilderment; and, as his senses cleared, he remembered that through all of his life he had been clever. He rested a long moment on a twisted steel girder, fascinated, as the San Remians, adults and children, moved about their uprooted island adapting, preparing to make a new home along the current of the ocean stream. And gradually he felt it, blowing in the trade winds that waked the storm, nothing of hostility: a tremendous authority, a revelation that he was within a greater wisdom.

The Santus ached with a sadness of what was; and yearned to tell the young man of the magical chance he had had to work a great thing simply by his example. But they sensed he may not ever have understood the chance in their way. And, if he did not, there was little to be said. □

NEW MEMBERS

The Physicians listed below have joined the Medical Society of Nova Scotia between December 1, 1983 and January 31, 1984. A most cordial welcome is extended by the Society.

P.L. Allan	Halifax
J.W. Branson	Dartmouth
T.P. Corkum	Halifax
E.W. Collins	Halifax
E.G. Cooper-Rosen	Halifax
F.S.S. Crombie	Halifax
D.W. Himmelman	Pleasantville
B.L. Janes	Halifax
D.W.B. Keating	Halifax
J.L. Leahey	Halifax
S.E. Orrell	Sydney
P.S. Renault	Halifax
R.T. Tanton	Halifax
C.G.W. Turner	Truro

Correspondence

To the Editor:

RE: NUCLEAR WAR

When I saw the six title symposium in the October, 1983 issue of the *Bulletin* under the theme: "Medical Aspects of Nuclear War", I wondered how that topic could justify so many pages. And, assuming your editorial staff wanted something controversial, I asked myself: wouldn't *Bulletin* readers be better served by a symposium on the tendency of some physicians to order a lot of special tests or a lot of medications in situations where these services are not easily justified? And a further thought: was I about to be exposed to six biased and over-simplified presentations on a complex and multifaceted topic [as indeed I was 3 years ago when reading an article by M. Korcok about the "threat of a nuclear epidemic". *Can Med Assoc J* 1980; **123:**418-423].

Actually your non-medical article by Arthur J. Andrews, "The Cost of Nuclear Peace", was particularly well done and I wish to compliment the *Bulletin* staff on presenting more than one viewpoint on socio-political aspects of the nuclear dilemma.

I sensed an anti-American bias in Paul Cappon's article: "Socio-Political Aspects of the Nuclear Arms Race". It called to mind Prime Minister Trudeau's "open letter" to Canadians about testing the cruise missile in Canada. He says of cruise critics that: "Having convinced themselves that it is useless to denounce the SS-20s, people find it easier, I suppose, to forget about them". And: "The strange result. . . . is that it somehow becomes possible to portray the Soviet Union not as the aggressor but as an innocent target" [*The Chronicle-Herald,* May 10, 1983].

The medical articles in the symposium were informative but provided more detail than I wanted. There seems to be enough evidence to conclude, with M. Michael Cohen, that "In the aftermath of a nuclear attack, our capacity to respond as health professionals would be so severely limited that even first aid would be impossible — only last aid". This simple message should be shared widely with the general public — while acknowledging, with Arthur Andrews: (1) that nuclear weapons are staving off another world war; and (2) their elimination is not a realistic option. I also share Dr. Cohen's view that: "The medical dimensions of nuclear war are so staggering that to dwell on them seems almost absurd". To dwell at length on nuclear horrors can also serve the purposes of anti-American fear propagandists. [See article by E. Luttwak, reprinted from *The London Telegraph* by *The Chronicle-Herald,* December 29, 1983.]

Sincerely,

William I. Morse, M.D., F.R.C.P.(C)
Paradise,
Annapolis County, N.S. □

"An expert is a man who has made all the mistakes, which can be made, in a very narrow field."

— Niels Henrik David Bohr (1885-1962)

Current Topics in Community Health

Prepared by: Dr. Frank M.M. White,
Department of Community Health and Epidemiology
Dalhousie University, Halifax, N.S.

COMPUTERIZED RECORD LINKAGE: A MORE EFFICIENT TECHNIQUE FOR CARRYING OUT LARGE SCALE EPIDEMIOLOGICAL STUDIES

One major focus of epidemiological studies is the determination of factors which may be associated with increased risk of diseases. Two main approaches to identifying such factors are case-control and cohort studies.

In a cohort study one begins with a group of individuals who have been exposed to a particular risk factor and ascertains their subsequent morbidity or mortality experience. This process sometimes involves following a group for many years by personal contact, telephone and mail enquiries. Such studies are very time consuming and expensive.

A procedure has recently been established by Statistics Canada, Ottawa, which, it is hoped, will make more cohort studies possible, especially in the field of chronic disease research. It involves computer linkage of exposure files to national data bases of mortality and morbidity information. Both the mortality and morbidity files have been prepared from data that were being stored already in machine-readable form for other purposes.

The mortality file, the National Mortality Data Base, contains some four million records dating back to 1950. It is prepared from provincial death registrations and the records are sequenced using a phonetic coding system.

The National Cancer Reporting System, the morbidity file, was started in 1969 and contains about 330,000 records. It is not quite as accessible for record linkage, as some provinces are not yet contributing data to the file. There is difficulty also in having this information released because the individuals involved are still living.

The linkage of an exposure file to one of the national data bases involves two steps:

1. Searching: the bringing together of the two files for comparison; and
2. Linking: calculating agreements and disagreements of various identifiers and the odds in favor of the persons represented by the two files being the same.

A unique identifier for all Canadians such as the social insurance number would make record searching relatively simple, but this is not available on most records. A series of identifiers are used instead and a search will be most cost-effective when a sufficient number of such identifiers are present to finely divide the national data file. This is achieved through use of a phonetically coded form of the surname. A record from the exposure file will be matched only against those records in the national file that have the same phonetically coded surname.

In the linking step, weights are assigned for each comparison that takes place between the two records. If a certain minimum total weight is achieved during the comparison, the records are considered to represent the same individual and a "link" is made; if not achieved, the result is a "non-link" or a "possible link". In case of the latter, additional identifying information would be sought from manual records held at Statistics Canada and/or from source records for the exposure file in order to resolve the problem. The speed of such searches is high and the cost low compared to methods used in traditional cohort studies.

Most of the studies undertaken up to the present have been concerned with long-term consequences of various occupations, medical treatments and diagnostic procedures, reproductive problems, lifestyle and other environmental factors. There are certain procedures however that Statistics Canada must follow with regard to the release of national mortality and morbidity data because it carries responsibility for the confidentiality of the records entrusted to it. Consultation would be required for each anticipated study.

The recent developments at Statistics Canada should render many more long range epidemiological studies feasible than has been the case in the past.

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Source: Elva Heyge, Research Officer, Department of Community Health and Epidemiology, Dalhousie University.

Selected Abstract

USE OF STATISTICAL ANALYSIS IN THE NEW ENGLAND JOURNAL OF MEDICINE

A sorting of the statistical methods used by authors of the 760 research and review articles in Volumes 298 to 301 of *The New England Journal of Medicine* indicates that a reader who is conversant with descriptive statistics (percentages, means, and standard deviations) has statistical access to 58 per cent of the articles. Understanding *t*-tests

increases this access to 67 per cent. The addition of contingency tables gives statistical access to 73 per cent of the articles. Familiarity with each additional statistical method gradually increases the percentage of accessible articles.

Original Articles use statistical techniques more extensively than other articles in the *Journal*. Research studies based on a longitudinal design make heavier use of statistics than do those using a cross-sectional design. The tabulations in this study should aid clinicians and medical investigators who are planning their continuing education in statistical methods, and faculty who design or teach courses in quantitative methods for medical and health professionals.

Source: Emerson JD, Colity GA, *Use of Statistical Analysis in THE NEW ENGLAND JOURNAL OF MEDICINE. N Eng J Med* 1983; **309:**709-713.

SAFETY TIPS FOR FESTIVE OCCASIONS

Champagne-Cork Injury to the Eye*

Many eye injuries from flying champagne corks have occurred since the invention of the champagne cork at the end of the 17th century. It is interesting that the inventor, a Benedictine monk by the name of Dom Perignon, was himself blind, although the cause is not known.

A 1 ounce cork shooting from an upright bottle can reach a height of 40 ft. Quite often the cork is ejected spontaneously on removing the wire around the bottle neck. It has been recommended that champagne be served at a temperature of 8.3° C (47° F), and at this temperature the pressure in the bottle is about 90 lb. per square inch. At room temperature or higher the bottle pressure is even greater, and it may be further increased by shaking the bottle.

From this knowledge, it can be calculated that the cork strikes the eye at a velocity of about 45 ft. per second. At this speed a cork could fly to the eye from the held bottle in less than 0.05 second, before the blink reflex (which takes about 0.1 second) can exert its protective effect. The cornea could therefore receive the full impact. Considering the small area of impact and the distortion of the globe, the force exerted on the eye is estimated to be in the region of 100 atmospheres — similar to that in a blast injury.

These injuries can be avoided by care in opening the bottle. The Comité Interprofessional du Vin de Champagne has recommended how this should be done. A napkin or towel should be held over the cork and the neck of the bottle while the wire is being undone, and the cork is then to be gently eased off with the bottle pointing away from the face and other persons in the room. There should be no "pop", just a sigh. "White gloves may be worn but are not essential."

Patron Flambé**

Flaming foods and drinks add a festive touch to a meal. Patrons of restaurants enjoy the showmanship of the server and fully expect that the flaming shows are done safely. This, unfortunately, is not always the case. The University of California Irvine Burn Center has reported treating 8 patients for burns as a result of flaming meals or drinks in restaurants. Many of the patients (half were employees) suffered full thickness burns and required skin grafting. Three of the accidents occurred when the liquor in the bottle caught fire, producing a flame thrower effect.

As a result of these accidents, the Newport Beach Fire Department established the following requirements for serving flaming food or drinks: (1) a special permit for flaming food or drinks; (2) a maximum of 30 ml (1 oz) of liquor or brandy by laws when preparing the dish; (3) preparation of the dishes at the patron's table and no transport of the food or drink while ignited; (4) a wet towel be available in the preparation area in case of accident; (5) no flame higher than 20 cm. (8 in.); and (6) a spill-stop pouring device to be put on all bottles used for flambé.

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OBITUARIES

Dr. Robert C. Dickson, (75) died on February 19, 1984 in Vancouver. Born in St. Mary's, Ontario, he graduated from The University of Toronto in 1934 with an M.D. degree. In 1956 he was appointed Professor and Head of the Department of Medicine, Dalhousie University. He resigned from Dalhousie in 1983 and moved to Vancouver. The Robert Clark Dickson Centre named in his honour was opened in 1983. Located at the Victoria General Hospital; Halifax, it houses one of the world's finest cancer treatment centres. Our sympathy is extended to his family.

Dr. Edward T. Granville, (84) died at home on January 13, 1984. Born in Halifax, he graduated from Dalhousie Medical School in 1922. Before his retirement he practised in Elmsdale, Bedford and Halifax, N.S. Dr. Granville was recently awarded the Pro Ecclesia et Pontifice Cross by Pope John Paul II. The Society offers sincere sympathy to his wife and family.

Dr. G. Murray Smith, (67) of Windsor, N.S. died on December 24, 1983. A graduate of Liverpool High School, N.S., he obtained his pre-medical education from Mount Allison University and his medical degree from Dalhousie University. He retired as director of the Fundy Health Unit in 1980. He is survived by his wife, two daughters and a son. We extend our sincere sympathy. □

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