Second Class Mail Registration No. 5363
Halifax, N.S.

THE NOVA SCOTIA MEDICAL JOURNAL



# Physicians and the Nursing Profession

The goals, aspirations and problems of the nursing profession seem to be misunderstood by the medical profession of Canada. In fact, ignorance of the nursing profession may be a better description of the situation. Most physicians are aware of the move to have all RNs achieve a Bachelor's Degree, and that nurses want to stop wearing "caps". However, there is much more to know.

If you are unaware of your level of ignorance, a look at the latest nursing journals may be what is needed. Suggested reading would be *Nursing 1989* or *The Canadian Nurse* the official Journal of the Canadian Nurses Association. You may be in for a shock.

A regular reader of *Nursing 1989* would tell you what you have been missing. Excellent clinical articles that are pertinent to what we as physicians do every day will be found in these pages. Articles are often written from the point of view that physicians do not really exist or, if present, are almost auxiliary personnel. Suggestions regarding comprehensive care, practical tips, description of new products and treatments are all to be found. Despite the range of medical journals available, the material here is useful and often not available to physicians in the usual literature. The fact that these articles deal with some of the finer aspects of diagnosis and treatment, which is often what is considered to be the physicians prerogative, should not surprise you. Increasingly, nursing journals over the years have been teaching more comprehensive ways of thinking to their audience. With more knowledge of what nurses are reading, you may be able to understand the challenges you encounter, the forceful suggestions and the need to "consult" the nursing staff more often and more meaningfully.

Further perusal, for instance, of the April Issue of *The Canadian Nurse* will lead you to political questions also. The lead editorial shows that the C.N.A. directors have approved a five-year plan that includes "steps to define and clarify the role of nurses in the practice of primary care, establish stable and adequate funding to facilitate demonstration projects for nursing practice in this area, a look at

implementation for educational programs in nursing and establish a lobby campaign to expedite the development of nursing practice in primary health care".

What does such a statement signify? These ideas could translate into many thoughts, including a criticism of the way we as physicians handle primary health care. It could mean a simple political move for power and status. It certainly will mean financial and economic consequences in a time of financial restraint. The idea of nurses providing cheaper care, while completely unproven and doubtful, will be tempting to health planners. There is no doubt, however, that it is possible for nurses to accomplish a primary health care role given proper training. In a time of nursing shortage, however, it is difficult to understand the C.N.A's drive in this direction, to "reinvent the wheel" as it were. It is difficult, as it has been said above, to understand the reasoning behind their agenda, but it is certainly important to try to do so.

Further examination of nursing journals also reveals national and international recruitment programs of many hospitals, demonstrating a real shortage of nurses in many locations. Advertisements are strong on promises of career *development,* assurances of *respect,* and invitations for nurses to become an equal partner with doctors in management. This is the atmosphere in which Nova Scotia and the Maritimes must function.

Our dialogue with the nursing profession leaves something to be desired for whatever reasons. Probably both medical and nursing professions should be making more efforts in this regard. More formal discussions collaboratative research and stronger medical/nursing liason committees would be a start. Nurses are increasingly aware that they too are being held legally responsible for patient care and justifiably cannot blindly obey the doctor.

Accepting this new reality has been very gradual. Understanding the aspirations of the nursing profession is becoming more than something of interest, or politeness. It is becoming a necessity.

J.F. O'C. □

---

# An Appreciation

## DR. CLENNEL EVELYN van ROOYEN

Clennel Evelyn van Rooyen, M.B., Ch.B., M.D., D.Sc., FRCP(Lond.), FRCP(C), FRC (Path), FRS(Can) died in Halifax on March 16, 1989 after a long illness. He was a distinguished medical scientist, author and physician. He was born in Ceylon, the son of a physician. Educated in Bracondale School in Norwich and Edinburgh University, he received his M.B. in 1931 and his M.D. in 1934, with high commendation.

Thereafter, he devoted his professional life to microbiology, virology and infectious diseases, and excelled in all. He was a pupil and later a colleague of some of the founders of modern microbiology and indeed did much fundamental early work on virus morphology and transmission. He isolated the prototype II poliovirus. He served in the RAMC in World War II in Europe and the Middle East, where he did important work in hepatitis, poliomyelitis, typhus and smallpox. He has written five text books and contributed chapters to many others. His classical textbooks *Virus Diseases of Man* and *A Textbook of Virology* were both in collaboration with his distinguished colleague and friend, Dr. Andrew Rhodes. He has written two hundred medical articles and served as a member of numerous prestigious advisory bodies.

In 1947 he became Professor and Head, Department of Virus Infections, School of Hygiene, University of Toronto and a Research Member of The Connaught Medical Research Laboratories. From 1956 to 1973 he was Professor and Head, Department of Microbiology, Dalhousie University and Director of The Nova Scotia Government Public Health Laboratories, and these he considered his happiest years of teaching, research and clinical medicine.

Although his outstanding career speaks for itself, it is as a teacher, colleague and friend that "Van" will be remembered by all who knew him and especially by his intensely loyal staff. His intellect, curiosity and devotion to duty were accompanied by a deep concern for the common good and compassion for those who were ill. His dedication as a consultant in infectious disease will never be forgotten by his colleagues and the Medical Officers of Health, to whom he was always available, without reservation. Thus, not only did he practise medicine but he also made permanent contributions to the scientific base that all physicians use.

"Van" was a noble, honourable man who bore his tragic blindness with grace and dignity and goodwill to all.

To Hilda, John and Jennifer, we express our deep sorrow in your great loss. You were always very close to his heart and mind as he carried the burdens of his distinguished career. We thank you for sharing his life with us and with mankind.

Dr. Alan J. MacLeod □

# Hip Fractures in the Elderly

David B. Hogan, MD, FRCPC, Michael Gross, MB, FRCS, FRCSC, Karen V. Mann, RN, MSc, PhD, Kathleen M. MacPherson, MD, MPH and Mary Gordon Rassmusson, RN, BSc, MSc

*Halifax, N.S.*

**Hip fractures have become an increasingly important health care problem. In this article we provide information regarding the risk factors, management and prognosis of this condition. In spite of the large amount currently known about hip fractures, much work remains to be done.**

## DEFINITIONS

For the purpose of this paper, *hip fractures* are open or closed fractures of the proximal femur (including femoral neck, trochanteric region, and the uppermost femoral shaft or subtrochanteric region). *Elderly* refers to people 65 and over except when stated otherwise.

In this paper we will not include discussion of pathological fractures due to localized bone destruction or weakness, such as is seen in secondary malignant disease, localized destructive tumors (both benign and malignant), and localized bone infection.

## TYPES OF FRACTURES

Evans divided fractures of the proximal femur into four categories, according to their stability:

1. two fragments undisplaced;
2. two fragments displaced but stable after reduction;
3. three fragments; and
4. four fragments — often termed comminuted.[1]

Fractures can also be classified by the fracture sites. The traditional categories are into femoral neck fractures, intertrochanteric fractures, and subtrochanteric fractures — see Figure 1.[1]

*Femoral neck fractures* occur between the trochanters of the femur and the femoral head. They can be further subdivided into *subcapital* (just below the femoral head), *intercapsular* (within the joint capsule), and *transcervical* (in the middle of the femoral neck). The blood supply of the femoral neck is derived from the femoral circumflex and retinacular arteries which travel along the neck of the femur, and it can be injured by fractures of the femoral neck. Interruption of the blood supply may lead to avascular necrosis of the femoral head. In addition fractures within the capsule may heal poorly.[1] Nondisplaced femoral neck fractures can be impacted and may present only with mild pain. This type of fracture — sometimes also termed a *stress fracture* — may have an initially negative x-ray. Classically, a displaced fracture is associated with moderate-severe pain, external rotation of the limb, and shortening of the leg.

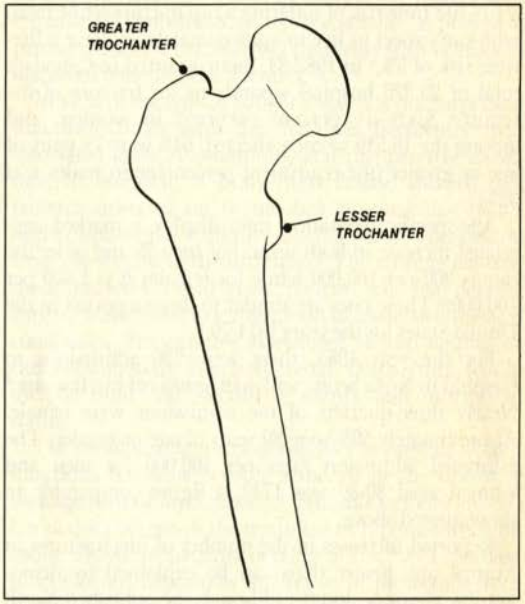


**Figure 1**

*Intertrochanteric fractures* occur between the greater and lesser trochanters at the base of the femoral neck, and severity is related to their instability. Individuals with this type of fracture typically are older, more disabled-diseased, and have a greater degree of osteoporosis than patients with femoral neck fractures.[1]

The least common type of hip fracture, constituting approximately 10% of events, is the *subtrochanteric fracture*. These can be quite difficult to treat, being associated with problems in reduction, fragmentation, and long-term complications.[2]

## EPIDEMIOLOGY/RISK FACTORS

Hip fractures occur most commonly in the elderly — in particular, the aging female. This was recognized as long ago as the early 1820s when Sir Astley Cooper wrote: "... the fracture of the neck of the thigh bone within the capsular ligament seldom happens but at an advanced period of life ... that regular decay of nature which is called old age is attended with changes which are easily detected in the dead body; and one of the principal of these is found in the bones, for they become thin in their shell and spongy in their texture ...

From the Dalhousie Working Group on Hip Fractures, Dalhousie University, Halifax, N.S.

Correspondence: Dr. David B. Hogan, Camp Hill Hospital, 1763 Robie Street, Halifax, N.S. B3H 3G2.

Women are much more liable to this species of fracture than men."[3]

The fracture rate doubles for each decade of life after the age of 50.[4] An American study found that by the age of 90, 32% of women and 17% of men have sustained a hip fracture.[4] It has been estimated that adult white women, who on average live to approximately 80, have a 15% life-time risk of suffering a hip fracture while men, who can expect to live to approximately 75, have a life-time risk of 5%.[5] In 1982-83, there occurred in Canada a total of 25,220 hospital separations for fracture of the femur.[6] Sixty-six percent occurred in women, and among the 16,539 women affected, 64% were 75 years of age or greater (the equivalent percentage in males was 32%).

Age-specific separation rates display a marked age-related increase in both sexes. For men 75 and older the rate is 800 per 100,000 while for females it is 1,800 per 100,000.[6] These rates are similar to those reported in the United States for the years 1974-79.[7]

For the year 1985, there were 790 admissions to hospital in Nova Scotia with a diagnosis of hip fracture.[8] Nearly three-quarters of the admissions were female. Approximately 50% were 80 years of age or greater. The estimated admission rates per 100,000 for men and women aged 80-89 was 1749, a figure comparable to those quoted above.

Reported increases in the number of hip fractures in Europe are greater than can be explained by demographic changes alone[9-16] although the published North American studies are not as conclusive.[17,18] A Canadian study showed an increase in the annual age-adjusted incidences.[19] As a society we will be facing an "epidemic" of hip fractures purely from demographic changes. An additional increase in the total number of fractures because of an increase in the age-adjusted incidence will compound the strain on our health care system.

A number of risk factors for hip fractures have been reported in the literature and are as follows: increasing age[1,5], female sex[1,5], race[5,6], endogenous hormonal status[6], exogenous hormonal status[20-26], cigarette smoking[6,25], dietary factors[6], physical activity[6], alcohol use[22,26], hemiplegia[27], medication use[28], osteoporosis[1], tendency to fall[1,5], decreased arm muscle area/triceps skinfold thickness[29], and low relative body weight.[5,20-22,25-26] The primary risk factors appear to be osteoporosis and the propensity for falling.[30] The other reported risk factors appear to have an impact on the development of either osteoporosis or increasing the likelihood of falling. As such one may anticipate that risk factors for either osteoporosis[31] or falls[32] could increase the likelihood for subsequent hip fractures.

Bone mineral density of the femoral neck decreases an estimated 58% in women and 39% in men between the ages of 20 and 90.[33] Bone mineral density is highly correlated with bone strength[34] so this decline is accompanied by a corresponding decrease in the strength of the proximal femur.[35] The utility of bone mineral density determinations in the prediction of hip fracture risk is debated.[36,37] It is felt that patients with hip fractures do not appear to be clearly more osteoporotic then similarly aged controls. Against this conclusion is the well-substantiated finding that post menopausal estrogen use is protective with regards to hip fractures, presumably because of estrogen's retardant effect on the development of osteoporosis.[20-26] It can be viewed that osteoporosis, which becomes essentially universal beyond a certain age in our society, provides the necessary predisposition to hip fractures in the elderly, but that an episode of trauma, typically a fall, is required for it to actually occur. A small proportion of patients with a hip fracture will have suffered a spontaneous fracture of their femur related to profound osteoporosis but they are a distinct minority.

The occurrence of hip fractures is closely linked to the incidence of falls which, like fractures, rises rapidly with age.[30,38] In institutions, falls are associated with skeletal fractures in between 2.8 and 6.1% of occurrences.[39]

The influence of dietary factors on the risk of hip fractures has been evaluated.[5] It is presumed that they would have their effect by promoting or retarding the development of osteoporosis. The most quoted "positive" study comes from Yogoslavia where residents of a district with a high intake of dairy products (and thereby calcium) were found to have a 50% lower incidence of hip fractures in comparison to a district with a much lower consumption.[40] Holbrook found that the age-adjusted risk of hip fracture was inversely associated with dietary calcium intake in a group of adult, Caucasian residents of an upper-middle class community.[41] Other studies have not confirmed this finding.[5] Additional dietary factors examined include phosphorus, protein, vitamin D, fluoride, and caffeine, but no definite relationship with hip fractures has been found.

While physical activity appears attractive as a method of primary prevention, there is no direct evidence that this is indeed so.[5,6] Physical activity may increase bone mineral content by mechanical stimulation[42] and may reduce the risk of falling.[38] A negative relationship between participation in outdoor games and the risk of a hip fracture has been reported.[22] Athletic team participation appeared to increase the age of first reported fractures in women 50 and older.[43] The first National Health and Nutrition Examination survey found that recreational exercise was associated with a decreased risk for a hip fracture.[29] Finally, participation in regular physical activity by elderly men and women was associated with a reduced risk of enduring a skeletal fracture.[44] Unfortunately all the above noted studies had important limitations and further research into this area is urgently needed.

With regards to medication use, the psychotropics have attracted the most interest recently.[28] In particular the neuroleptics, tricyclic antidepressants and long-acting benzodiazepines which may increase the risk of falling have been found to have a relationship with the occurrence of hip fractures.[28]

In view of the magnitude of the problem, prevention is the only cost-effective approach. To quote Oliver Wendall Holmes: "And lo! the starry folds reveal the blazoned truth we hold so dear: to guard is better than to heal, — the shield is nobler than the spear!"[45] Prevention would be aimed at retarding the onset of osteoporosis or eliminating the risk of falling. Measures of potential use in preventing osteoporosis include increasing dietary calcium intake, increasing physical activity, decreasing cigarette/alcohol consumption, and the post-menopausal use of estrogens.[31] Prevention of falls is more problematic but would include "safe-proofing" the environment and judicious use of medications.[39] It may be also possible to delineate a group at high risk for falling so that specific interventions may be targeted at them.[46]

## ACUTE MANAGEMENT

Unless there are pressing reasons otherwise, the management of the displaced hip fracture entails an operative procedure.[1] For patients with little or no chance to ambulate, non-surgical management may well be safer, more humane, and less expensive.[47] The aims of surgical treatment are: 1) to reduce the morbidity and mortality rate after injury; 2) to avoid a second operation in an elderly patient; 3) to return the patient rapidly to his pre-injury social and ambulatory status; and 4) to minimize the cost to the health care service.[30] The operative management of these fractures usually consists of either a sliding compression nail, long nails driven from the knee through the femoral head, or replacement of the entire femoral head with a prosthesis.

The most common operative intervention for hip fractures at present in North America is the use of a compression nail or a screw.[1] After the fracture has been aligned with a pin, the nail is driven into the femoral neck and head, holding the fractured elements in proper alignment. A plate which is attached to the nail is then screwed into the cortex of the proximal femur. The sliding feature of the nail allows the distal portion of the nail to telescope into the proximal part as the femoral head further impacts on the neck. This prevents the distal nail from pushing through the femoral head and irritating the acetabulum. An alternative extension device is the condylocephalic rod or nail. In this technique, after alignment is achieved, a single rod or a series of nails is (are) driven from the medial condyle of the femur at the knee through the entire femoral shaft and impacted into the femoral head.

The treatment of high femoral neck fractures is controversial. Because such fractures can interrupt the blood supply to the femoral head, avascular necrosis of the femoral head is a potential complication. In view of this, many surgeons elect to treat high femoral neck fractures with primary removal of the femoral head and replacement with a femoral prosthesis. The advantages of internal fixation in comparison to a prosthesis include low infection rate, no risk of dislocation, and lower mortality at six months.[30] Unfortunately, internal fixation is associated with some risk for nonunion and avascular necrosis. Reoperation is also more common. Primary prosthetic replacement is itself associated with a number of disadvantages — higher infection rate, risk of dislocation, increased mortality at six months, risk of late problems with pain from prosthesis and it does not avoid the possibility of a second operation which would be more difficult to revise, especially if the prosthesis is cemented.[30] Unfortunately, it is not possible at present to predict avascular necrosis and problems with healing of displaced femoral neck fractures.[30]

In view of the typical patient's age, it is not surprising that individuals with hip fractures frequently have associated medical conditions, and preoperative assessment is essential. A study from Boston showed that patients operated on in the first hospital day had a higher in-hospital mortality rate than those operated on from the second to the fifth day.[45] The authors felt that the improved survival in the patients who had delayed surgery was related to stabilization of associated medical conditions. Preoperative assessment should emphasize cardio-pulmonary function, renal status (this would include fluid and electrolyte status), and nutritional status.[1]

The prophylactic use of antibiotics to prevent wound infections has now become standard in the operative management of hip fractures.[1] Thromboembolic disease is a major concern in the treatment of hip fractures, and about 40-50% of patients develop a D.V.T.[51] Low dose subcutaneous heparin is ineffective in protecting from thromboembolic disease for patients undergoing surgery for hip fractures.[48] The N.I.H. recommends consideration of Dextran 70, pneumatic compression, or pressure-gradient stockings.[49]

Standard postoperative care would include early mobilization. This may help diminish problems such as venous thrombosis with pulmonary embolism, pneumonia, and decubitus ulceration. Ambulation, with at least partial weight bearing, usually starts within days of surgical repair of a fractured hip.

## PATIENT OUTCOMES

The most frequently measures outcome mentioned in the literature is mortality.[30] Early mortality which usually refers to death within three to six months of the fracture tends to be in the range of 10-15%. One-year mortality for individuals who have suffered a hip fracture appears to be in the range of 20%.[1,30] Risk factors for mortality include presence of additional diseases, increasing age, male sex, and increased dependency before the fracture.[30]

Another adverse patient outcome mentioned in the literature is institutionalization, and predisposing factors include increased age, female sex, poor general medical condition, living alone, and poor functional capabilities.[30] Historically after hip fracture, approximately one-quarter of survivors who are admitted from the community will require discharge to an institution. At one year after the hip fracture approximately one-

fifth of those who are admitted from their own homes at the time of fracture will be found living in an institution.[30]

In the United States, changes since the implementation of the Prospective Payment System have been noted.[50] In a large midwestern hospital the mean length hospitalization decreased (from 21.9 to 12.6 days), inpatient physical therapy sessions decreased (from 7.6 to 6.3 sessions) and the maximal distance walked before discharge fell (from 27 to 11 metres) for patients 65 years of age or older who were hospitalized with a new hip fracture. Concomitantly, the proportion of patients discharged to nursing homes rose (from 38 to 60%) as did the proportion remaining in nursing homes one year after hospitalization (from 9 to 33%). It was concluded that the implementation of the Prospective Payment System had reduced the amount of care given to hip fractures and had shifted much of the rehabilitative burden to nursing homes.

There has been an attempt to evaluate the effect on patient outcomes of specialized units designed to deal with individuals who have suffered a hip fracture. Lefroy, in a descriptive study, showed that in-hospital mortality rate for individuals admitted to an orthopedic-geriatric service was 6% with 67% of individuals admitted from home returning there.[57] Boyd showed that the in-hospital mortality rate decreased from 25% to 20% and the number of patients who were discharged home increased from 55% to 61% after the opening of an orthopedic-geriatric unit.[52] In contrast a study by Wilson could not show a need for specialized geriatric units to help care for elderly patients presenting with hip fractures.[53] At present all one can say is that the results of the reported studies have been inconclusive. There may very well be a subgroup of patients who would benefit from a specialized orthopedic-geriatric unit but further work is required to characterize this group.

Part of the problem of increased dependency after hip fracture relates to the question of change in mobility status. In community surveys of individuals suffering hip fractures, between 48 and 56% will show decreased mobility postevent. Twenty-two to thirty percent will be nonambulant postfracture. Significant risk factors for becoming nonambulant are residing in institution at the time of hip fracture and having prior problem with mobility.[30]

The two factors which best predict a patient's recovery from a hip fracture are mental status and preexisting functional state.[54-57] One study found a one year mortality of 47% for patients with dementia as opposed to 18% in individuals without a confusional state. Several studies have found that a patients ability to care for himself/herself before the fracture is the best predictor of outcome. Rapid return of ambulation (within two weeks of surgery) predicts a good outcome.

## CONCLUSION

In spite of the abundance of information concerning hip fractures in the elderly, a number of unanswered questions remain. Some of them are:

1. Is the age-adjusted incidences for hip fractures increasing?
2. Can a high risk group for hip fractures be delineated and can effective preventive measures be instituted to decrease their risk of subsequent fracture?
3. What is the most appropriate timing for surgical intervention? What is the most appropriate method of operative repair?
4. What is the most appropriate organization for postoperative care to minimize mortality and morbidity? Are there specific sub-groups of elderly patients with hip fractures who require a specific form of rehabilitation such as admission to a gerior thopedics service?

---

Over the last 25 years, the major problem of proximal femoral factors in the elderly has changed from mortality to morbidity. While there are several guides for "estimating" risk of major morbidity, they are not foolproof. More work in this area is urgently needed. □

References supplied upon request.

---

## THE EFFECT OF CHOLESTEROL ON RED BLOOD CELLS

**Continued from page 91.**

10. Oldfield E, Chapman D. Dynamics of lipids in membranes; Heterogeneity and the role of cholesterol. *Fed Eur Bioch Soc Lett* 1972; **23:** 285-297.
11. Darke A, Finer EG, Flook AG, Philips MC. Complex and cluster formation in mixed lecithin/cholesterol bilayers. Cooperativity of motion in lipid systems. *Fed Eur Bioch Soc Lett* 1971; **18:** 326-330.
12. Lange Y, Slayton JM. Interaction of cholesterol and lysophosphatidylcholine in determining red cell shape. *J Lipid Res.* 1982; **23:** 1121-27.
13. Lee SP, Scott AJ. Dihydrocholesterol induced gallstones in the rabbit: Evidence that bile acids cause gallbladder epithelial injury. *Brit J Exp Path. 1979;* **60:** 231-38.

## PATIENTS WHO "FAKE" ILLNESS A MEDICAL QUAGMIRE

**Continued from page 94.**

8. Ford CV and Zaner RM. Response to the Article "Ethical and Management Considerations in Factitious Illness: One and the Same" by Sadler JZ. *Gen Hosp Psychiatry* 1987; **9:** 37-39.
9. Meropol NJ *et al.* Factitious Illness: An Explanation in Ethics. *Perspect Biol Med* 1985; **28:** 269-281.
10. Bhugra D. Psychiatric Manchausen's Syndrome. *Acta Psychiatr Scand* 1988; **77:** 497-503.

# Unipolar Depression

Charles J. David,* MBBS, DPM (Lond), MRC Psy (Lond), FRCP C

*Halifax, N.S.*

The term *unipolar depression* is used by psychiatrists to describe an affective disorder or a depressive disorder which may be recurrent, but is not associated with hypomanic or manic phase. It is one of the most common psychiatric disorders, and it is estimated that 15% of all adults will experience a depressive episode at some point in their lives.[1] Depression is also common amongst medical and surgical patient populations.

**TABLE I**

**ORGANIC CAUSES OF DEPRESSION**

| | |
|---|---|
| **1. Infections** | |
| T.B. | Hepatitis |
| S.B.E. | Brucellosis |
| Lues | Encepalitis |
| Mononucleosis | Post encephalitic states |
| **2. Drugs and Poisons** | |
| Amphetamines | Sedatives (other) |
| Cocaine | Steroids |
| Methyldopa | Bromides |
| Alcohol | Lead poisoning |
| Antabuse | Oral contraceptives |
| Propanolol | Other heavy metal poisons |
| Opiates | Carbon disulphide |
| Barbiturates | |
| **3. Metabolic Disorders and Endocrine Disorders** | |
| Pellagra | Hypothyroidism |
| Pernicious Anemia | Hyponatremia |
| Severe Anemia (any cause) | Hypokalemia |
| Diabetes | Cushing's Disease |
| Uremia | Addison's Disease |
| Porphyria | Hypopituitarism |
| Hepatic Disease | Wernicke-Korsakoff Syndrome |
| Hypoparathyroidism | Wilson's Disease |
| Hyperthyroidism | |
| **4. Degenerative Disorders** | |
| Parkinson's Disease | M.S. |
| Huntington's Disease | Other C.N.S. degenerations |
| Alzheimer's Disease | |
| **5. Neoplastic** | |
| Carinomatosis | Primary cerebral tumor |
| Ca of Pancreas | Cerebral Metastasis |
| **6. Miscellaneous** | |
| Postpartum syndrome | Chronic pyelonephritis |
| Pancreatitis | Chronic subdural haematoma |
| Menière's Disease | Normal pressure |
| Lupus & other collagen diseases | hydrocephalus |
| | Etc., Etc. |

Depression, as an illness, represents a disturbance in the person's emotional state that affects and causes difficulties in cognitive and pscyhomotor functioning. This disrupts and may compromise interpersonal activities and ability to cope on a day-to-day basis. The condition may range from a sadness to dysthymia to depression to abject apathy. *Dysthymia* is often described as a chronic disturbance of mood which may be associated with irritability and it seems to be a consequence of preexisting, chronic non-mood disorders, e.g. as a feature of anorexia nervosa, somatization disorder, and/or others. There is often coexisting with dysthymia personality disturbance. Acute and/or chronic psycho/social stresses may trigger these disturbances. Depression, in a variety of settings, is a leading cause of suicide. There is an enormous range in the severity, duration and therapeutic response of these disturbances of mood.

**TABLE II**

**CRITERIA FOR MAJOR DEPRESSION (After DSM III-R)**

1. Depressed mood most of the day, nearly every day.
2. Diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day.
3. Significant weight loss or weight gain when not dieting, or decrease or increase in appetite nearly every day.
4. Insomnia or hypersomnia nearly every day.
5. Psychomotor agitation or retardation nearly every day.
6. Fatigue or loss of energy — lack of enthusiasm or feelings of enjoyment.
7. Diminished ability to think or concentrate, to make decisions, to study, to get things done.
8. Feelings of worthlessness or excessive or inappropriate guilt; feeling unwanted, sinful, sometimes leading to the thought that life is not worth living.
9. Recurrent thoughts of death, recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.
10. Somatic complaints that are real: may include any of the following: indigestion, constipation or diarrhea, chronic pain in the back, abdomen, or almost anywhere else, which may be quite severe.
11. Loss of sex drive, often complete.

At least five of the above symptoms (see Table II) have been present during the same two week period.

These criteria define depression as a clinical entity rather than a variation of mood experienced in relation to day-to-day living. At times, there is a blurring of the boundary between normal and pathological mood changes.

*Department of Psychiatry, Associate Professor, Dalhousie University, Halifax, N.S.

## EPIDEMIOLOGY

Unipolar depression occurs eight times as frequently as bipolar disorder and twelve times as frequently as schizophrenia.

If dysthymia or neurotic depression are included, the risk for depression in the course of a lifetime rises as high as 20 to 30%. The incidence ranges from 10 to 20 per 100,000 (0.01 to 0.02%.).[1] The prevalence (total number of cases existing in a population in a given year) ranges from 18 to 23% of all adult female population in the U.S., Canada and Europe, and from 8 to 11% of adult males so affected. Of those affected, 6% of females and 3% of males require hospitalization.[1]

Depression may occur at any age and is twice as common in women than in men. Admissions to hospital peak in age group 40 to 60.[2] Married women show higher rates than married men.

Between 5 and 15% of patients have one episode of depression only. However, recurrence is common in depressive disorders. The average duration of an episode varies from 4-8 months.[3] Chronicity is seen in 10 to 20% of cases — often in the older age group patient.[4]

Of patients with unipolar depression, 80 to 85% improve with a planned bio-psycho-social treatment plan. Treated depressions respond in weeks to months.

## CLINICAL COURSE

Depression may present in a variety of ways. In some cases, rather than a depression of the mood, there may be recurrent panic attacks, anxiety symptoms, phobic symptoms, or chronic pain. In others, alcoholism, drug abuse or delinquent behaviour may mask the underlying depression. Hypochondriacal preoccupation may be present in older people and in men who may perceive complaining of emotional distress as a weakness.

In general, as women grow older, the episodes tend to become longer; as men with depression grow older, they show a greater number of episodes. Follow up of depressed inpatients has shown that 13 to 15% of patients develop a mania.[5]

Recovery in untreated depression is often spontaneous. Even untreated chronic cases are often seen to recover in 10 years.

A severe form of depression called *melancholia* appears commonly in the 40 to 60 age group in women, and after age 60 years in men. Depression here is of psychotic proportions and treatment may require hospitalization and possibly electroconvulsive therapy.

Depression in the elderly may present with complaints of memory impairment, disorientation and confusion. This condition is referred to as pseudodementia. These symptoms invariably lift with the successful treatment of depression.

Depression associated with other medical and surgical conditions is referred to as *secondary depression.* Similarly, depression may be associated with alcohol and substance abuse or as a result of pharmacological therapy for other conditions (see Figure 1).

## THEORIES OF DEPRESSION

*Genetic factors* are seen to play an important role in certain affective disorders including depressions. The

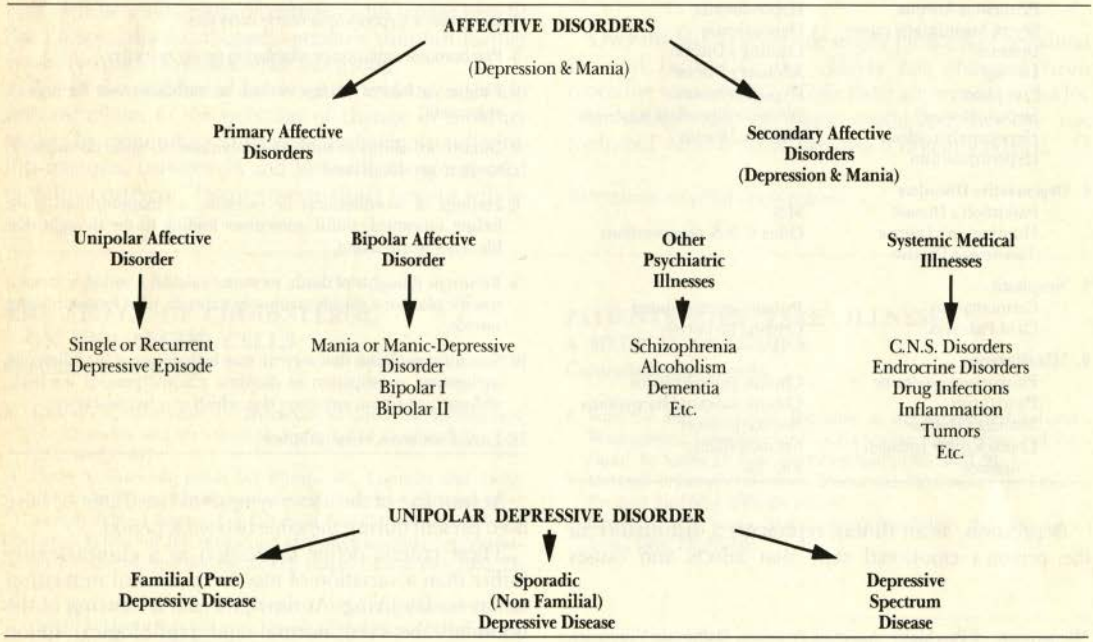


**Figure 1**

concordance rates for unipolar twin probands vary from 40 to 45% in MZ twins and from 22 to 24% in DZ twins. There is an increase of depressive disorders in the biological relatives of affectively ill patients.

In recent years, there has been exciting research in the area of genetic markers in relation to depressive disorders. These include: ABO blood types, detected on blood cells and HLA antigens, detected on leukocytes. A number of biological studies have been used in clarifying the diagnostic problems related to these disorders. These may be listed:

1. Dexamethasone suppression test (DST)
2. Platelet monoamine oxidase activity
3. Cerebrospinal monoamine metabolites (5HIAA) and homovanillic acid (HVA) and their relation to affective subtypes.
4. Cholinergic rapid eye movement induction.

Their usefulness for the clinician is still being evaluated.

### Psychodynamic Theories

These theories include a variety of points of view. Depressed patients, according to one view, come from a childhood characterized by losses, hypercritical parents, exploitation (either physical or emotional) and parental deprivation, etc. Painful disappointments and losses may trigger depression in susceptible individuals with poor self-esteem, associated with negative and distorted thinking. Depression is also viewed as *inner directed* anger and rage built up over the years towards significant others. These ideas will be pursued in detail in an article to follow.

### Neurotransmitter Hypothesis

Dysfunction of biogenic amines as neurotransmitters has been extensively studied as an etiological factor in affective disorders from mid 1960s. Some depressions are thought to be associated with a functional deficiency of either norepinephrine (ne) or serotonin at important synapses of the brain.

Adrenergic receptors have also been implicated and have led to the development of *challenge tests*, which have been useful in the diagnosis and therapy. Other neurotransmitters such as dopamine, and the role of plasma tryptophan have also been investigated. In 1972, Janowsky and colleagues studied the cholinergic system and its relationship to affective disturbances. The symptoms of depression in some patients are seen to be consistent with central cholinergic hyperactivity.[6]

These studies continue and are providing interesting and challenging information pertaining to the organic basis of depression.

### Neurohormonal Hypothesis

Many depressed patients do not have the normal nocturnal reduction in plasma cortisol. Dexamethasone, which usually suppresses plasma cortisol, fails to do so in 50% of patients with a diagnosis of major depression.

### Biological Rhythm and Depression

The biological symptoms of depression consist of alterations in hypothalamic centers that regulate food intake, libido, circadian rhythms, and endocrine abnormalities such as hypercortisolism. Hypersomnia and hyperphagia may also be manifest.

### Sleep Disturbance and Depression

Recent EEG sleep studies have demonstrated specific abnormalities in the sleep of most depressed patients.

Depressed patients enter the first REM phase period usually early — called *shortened REM latency*. They have reduced delta sleep — referred to as *shallow sleep*. The sleep is inefficient, leading to frequent awakening during the night and early morning. Some 15 to 20% of depressed patients have hypersomnia.[7] Sleep and REM studies have been used in the diagnosis of depression and in predicting response to antidepressant therapy.

## DEPRESSION AND SUICIDE

Suicide is a major complication of depression. It is a major health problem. Some 40 to 50% of all suicides are committed by depressed patients.[8] Retrospective studies show that it occurs in association with a diagnosable psychiatric disorder. Many of these patients are under treatment or have recently been discharged from hospital. Hence, the close follow up of discharged patients is recommended.[9] Prescribed medications should be limited in quantity and monitored closely during the acute phase and during the post-hospital phase of this disorder. Risk of suicide is higher in those depressives who are single, separated, divorced or widowed, and particularly in older individuals and those with problems with alcohol and other substance abuse histories.[9] Suicide is also high amongst adolescents, young adults and native Canadian Indians. Establishing a close relationship is a tremendous help since this enables the patient to turn to you in such a crisis.

## TREATMENT OF DEPRESSION

An accurate diagnosis based on a complete history and mental status examination is the first step, since this will provide clues to management.[10] Patients with psychotic depression may require hospitalization as will patients with severe depression, and those with poor or absent familial, social, environmental support who need supervision. An ongoing supportive relationship is critical in the management. The duration and interval of contact will depend on the severity and response to therapy if social and other supports are adequate. Explanation to the patient and his family of depression as an illness, the nature of therapy and the expected response to medication, will all enhance compliance. Offering realistic hope and making oneself available is essential in the early stages of therapy. Specific psychotherapeutic and cognitive techniques are best left

in the hands of the experts. In any case, these methods are most useful in the less severe and nonpsychotic depression — notably mild cases and dysphoric and dysthymic states.

The choice of the antidepressant will depend on several factors such as:

1. The past history and response to previous antidepressant drugs.
2. The patient's cardiovascular status and cardiovascular side effects which occur with various tricyclic drugs.
3. The degree of sedation required.
4. The possibility of weight gain associated with some antidepressants.
5. Most antidepressants (particularly tricyclics) take 3 to 4 weeks to produce a therapeutic response. Concomitant use of other medications may be required (benzodiazepines, sedatives, hypnotics, phenothiazines) for specific symptom relief in this early stage. However, medication options should be limited to one or at most, two drugs.

Table III provides a quick reference to the various antidepressants in current use. It also provides a range of therapeutic doses for each drug and the pathway of the activity in producing antidepressant response.

Fluoxetine, a new antidepressant, has just become available. It is claimed to have less side effects and can be given in a single daily dose of 20 mg.

Most of these medications are associated with side effects. One should become as familiar as possible with a number of antidepressants so that one can use them comfortably and confidently. Their specific effects can likewise be used to advantage in particular subtypes of depression. M.A.O.I.s, for example, are frequently effective in dysphoric states.[11]

If a particular antidepressant fails to produce any response in 4 to 5 weeks, a change to another type is indicated.

Monoamine oxidase inhibitors in general are more stimulating and less sedating than the tricyclics. They also have less anticholinergic properties. Their common side effects are orthostatic hypotension, anorexia and insomnia. M.A.O.I.s interact with other medications, certain foods and wines that contain tyramine (a pressor amine), to cause hypertensive crises. A list of drugs, foods and substances which must be avoided by the patient while on this therapy is essential. M.A.O. inhibitors may take up to 6 weeks to show any improvement.

An overview of psychotherapeutic methods in the management of depression and dysphoria is beyond the scope of this publication. This will be described in detail in a forthcoming paper.

**TABLE III**

**ANTIDEPRESSANT DOSES***

| Drug | Therapeutic Dose Range (mg) | Neurotransmitter Affected | | |
|---|---|---|---|---|
| TRICYCLIC | | | | |
| Amitriptyline (Elavil) | 75-300 | NE(++) | 5-HT(+++) | DA(+) |
| Clomipramine (Anafranil) | 75-225 | NE(+) | 5-HT(++) | |
| Desipramine (Norpramin) | 75-300 | NE(++++) | 5-HT(+++) | |
| Doxepin (Sinequan) | 75-300 | NE(+) | 5-HT(++) | |
| Imipramine (Tofranil) | 75-300 | NE(+++) | 5-HT(+++) | |
| Nortriptyline (Aventyl) | 40-200 | NE(+++) | 5-HT(+) | |
| Protriptyline (Triptil) | 30-60 | NE(++++) | 5-HT(++) | |
| Trimipramine (Surmontil) | 75-300 | NE(++) | 5-HT(+) | DA(+) |
| DIBENZOXAZEPINE | | | | |
| Amoxapine (Asendin) | 100-400 | NE(+++) | 5-HT(++) | DA(++) |
| TETRACYCLIC | | | | |
| Maprotiline (Ludiomil) | 100-225 | NE(+++) | 5-HT(+) | |
| TRIAZOLOPYRIDINE | | | | |
| Trazodone (Desyrel) | 150-400 | | 5-HT(++) | |
| MAO INHIBITORS | | | | |
| Isocarboxazid (Marplan) | 30-50 | — Different Mechanisms | | |
| Phenelzine (Nardil) | 45-90 | | | |
| Tranylcypromine (Parnate) | 20-60 | | | |
| BICYCLIC | | | | |
| Fluoxetine (Prozac) | 20-80 | 5-HT(+++) | | |

5-HT — Serotonin NE — Norepinephrine DA — Dopamine

* Adapted from: *Clinical Handbook of Psychotropic Drugs.* (eds.) Beachlibnyk-Butler, K.Z. and Jeffries, J. Joel. Toronto: Hans Huber Publishers, 1989. □

**References**

1. Sederer I, Lloyd, ed. *Inpatient Psychiatry Diagnosis and Treatment.* Baltimore: Williams and Wilkins, 1983, Chapter 1.
2. Klerman GL. Affective Disorders. In: Nicholi AM, eds. *Harvard Guide to Modern Psychiatry.* Cambridge: Bilknep Press, 1978, 253-282.
3. Clayton PJ. *The Prevalence and Course of Affective Disorders.* In *The Affective Disorders. Area 12.* Davis JM, Maas JW, Eds. *Am Psych Press, Inc.,* 1983.
4. Murphy GE *et al.* Variability of the Clinical Course of Primary Affective Disorder. *Arch Gen Psychiatry* 1974; **30:** 757-61.
5. Clayton PJ. The Epidemiology of Bipolar Affective Disorder. *Compr Psychiatry* 1981; **22:** 31-43.
6. Janowsky DS, El-Yousef MK, Davis JM, *et al.* A Cholinergic-Adrenergic Hypothesis of Mania and Depression. *Lancet* 1972; **2:** 6732-6735.
7. Kupfer DJ. *Application of the Sleep E.E.G. in Affective Disorder.* In *The Affective Disorders. Area 7.* Davis JM, Maas JW, eds. *Am Psych Press, Inc.,* 1983.
8. Ghadirian A-M: Making a Diagnosis of an Affective Disorder. *Diagnosis.* December 1988.
9. Hirschfeld RMA, Davidson L. *Risk Factors for Suicide* In *Review of Psychiatry,* Vol. 7, Chapter 14 Frances, AV and Hales RE eds. *Am Psych Press,* 1988.
10. Robin EH. Depression Rx: Which Way is Right for your Patient? *Modern Medicine of Canada.* 1985; **40(2).**
11. Flynn P. — Department of Psychiatry: Handouts and personal communication, 1989.

# Carcinoid Tumor in Nova Scotia

## A TEN YEAR REVIEW

Michael Yoon,* BSc, and Ron MacCormick,** MD, FRCPC

*Halifax, N.S.*

Carcinoid tumors are relatively uncommon neoplasms of enterochromaffin cells. They have been reported in most tissue derived from the primitive endoderm, most commonly in the appendix and small bowel. Carcinoids comprise 77% and 47% of all malignancies for these sites, respectively.[1] The third most frequent site is the rectum, but carcinoid tumors of the esophagus, stomach, lung and bronchus, ovary, pancreas, biliary track and Meckel's diverticulum have also been reported.[2]

Characteristically, carcinoids are very slow growing neoplasms. They are considered members of the family of neuroendocrine or APUD (amine precursor uptake and decarboxylation) series of tumors. Consequently, metastatic carcinoids are associated with the release of several biologically active hormones (including serotonin and bradykinin) that may manifest as chronic diarrhea and/or cutaneous flushing. These symptoms, along with an elevated urinary 5-HIAA (5-hydroxyindoleacetic acid), are referred to as the carcinoid syndrome.

Early symptoms of the disease are related to the mass effect of the tumor but the carcinoid syndrome itself is usually indicative of liver metastases.[3] The tendency of carcinoids to metastasize is related to the site and size of the tumor; small carcinoids (less than 2 cm in diameter) and those localized to the appendix rarely metastasize. Metastatic disease is primarily to the liver but bone, lung and other organs may also be affected.

Since these tumors are slow to grow, therapy is directed at reduction of tumor bulk or palliation of symptoms of carcinoid syndrome.

We present a review of 10 years experience with carcinoid tumor in Nova Scotia.

## MATERIALS AND METHODS

Cases of carcinoid tumor in Nova Scotia were analysed retrospectively. Data were obtained from the Cancer Treatment Research Foundation (CTRF) of Nova Scotia, which houses the registry of all patients diagnosed and/or treated for neoplasms in the province. Cases were identified through a search of these files based upon the following criteria:

1. Microscopic confirmation of histology; and
2. Diagnosis between the years 1979 and 1988, inclusive.

Cases also presenting with carcinoid syndrome were identified. The data was analysed as to age, sex, geographic distribution and primary site of the tumor.

## RESULTS

In the ten year period from 1979 to mid-1988, 101 cases of carcinoid tumor were diagnosed and registered within the province of Nova Scotia. The case load was 10.1 per year on average, ranging from 4 cases in 1981 to 16 cases in 1983. The majority of patients were females, outnumbering the males by a ratio of 1.5:1. (61 females and 40 males). The median age for females was 62 with the age of diagnosis ranging from 11 to 87 years. The males were between 12 and 72 years of age with a median age of 57.5.

Cases of carcinoid were reported in all counties of Nova Scotia except for Digby and Victoria. Thirty-two of the patients lived in Halifax County, 2 in New Brunswick and 1 of unknown residence. The rest were distributed among other regions of the province.

**TABLE I**

**DISTRIBUTION OF CARCINOID TUMORS WITHIN NOVA SCOTIA BY COUNTY AND SEX**

| County | Male | Female | Total |
|---|---|---|---|
| Annapolis | 2 | 0 | 2 |
| Antigonish | 2 | 1 | 3 |
| Cape Breton | 4 | 8 | 12 |
| Colchester | 1 | 4 | 5 |
| Cumberland | 2 | 0 | 2 |
| Digby | 0 | 0 | 0 |
| Guysborough | 2 | 3 | 5 |
| Halifax | 12 | 20 | 32 |
| Hants | 3 | 3 | 6 |
| Inverness | 2 | 2 | 4 |
| Kings | 1 | 3 | 4 |
| Lunenburg | 1 | 2 | 3 |
| Pictou | 2 | 2 | 4 |
| Queens | 0 | 4 | 4 |
| Richmond | 2 | 3 | 5 |
| Shelburne | 2 | 2 | 4 |
| Victoria | 0 | 0 | 0 |
| Yarmouth | 3 | 0 | 3 |
| New Brunswick | 1 | 1 | 2 |
| Unknown | 0 | 1 | 1 |
| TOTAL | 42 | 59 | 101 |

The distribution of the tumor as to site is demonstrated in Table II. The largest number of patients had carcinoid of the lung (36), followed by appendix (19) and ileum (13).

Of the 101 patients on record, 11 had symptomatology in keeping with carcinoid syndrome. All had facial

*Second Year Medical Student, Faculty of Medicine, Dalhousie University, Halifax, N.S.

**Medical Oncologist, Cancer Treatment and Research Foundation of Nova Scotia, 5820 University Avenue, Halifax, N.S. B3H 1V7

flushing and diarrhea, with or without an elevated urinary 5-HIAA. Primary tumor sites were the lung, small bowel and stomach.

**TABLE II**

**DISTRIBUTION OF CARCINOID TUMORS BY SITE**

| Site | Total |
|---|---|
| Appendix | 19 |
| Breast | 1 |
| Cecum | 3 |
| Cervix | 1 |
| Colon | 3 |
| Ileum* | 13 |
| Jejenum | 2 |
| Small Bowel | 9 |
| Lung | 36 |
| Rectum | 5 |
| Stomach | 6 |
| Thymus | 1 |
| Unknown | 2 |
| TOTAL | 101 |

*Including Meckel's diverticulum

## DISCUSSION

Carcinoid tumors represent a challenge in both diagnosis and management. In most cases, the tumor is malignant in nature and, due to its slow growing characteristic, is either not diagnosed until late stages (metastatic disease) or found incidentally upon examination. The disease affects members of all age groups and roughly follows population densities in the province. In addition, carcinoids may manifest in several organ systems as a result of their selectivity for tissue derived from the embryonic foregut.

Typically, organ distribution of carcinoid is appendix, small bowel, rectum and lung in decreasing numbers.[2] The high prevalence of lung carcinoid in Nova Scotia is unusual, reflecting either a true excess of occurrence at this site or under reporting of the incidence of tumor at other sites.

The sex ratio of females to males of 1.5:1 is in keeping with other reported results[2], as is the age distribution. Younger age groups appear to be more susceptible to appendiceal carcinoids while females and older age groups are more likely to have one of the other sites affected.

Carcinoid syndrome was diagnosed in only a small number of people.[9] The manifestation of the symptoms is dependent upon the release of gut hormones and is usually related to metatases in the liver. The release of endogenous hormones (bradykinin, serotonin, prostaglandins) by the tumor, mediate the symptoms of the syndrome: cutaneous flushing and teleglectasia, chronic diarrhea and right valvular heart deposition of fibrous tissue. An elevated urinary 5-HIAA is indicative of the syndrome but may not be present in all patients.

The occurrence of metastatic disease is dependent upon the site and size of the tumor. Small (less than 2 cm in diameter) tumors and those localized in the appendix rarely metastasize, while those that are larger preferentially metastasize to the liver.[3]

Excessive vasodilation can lead to cardiac failure while the massive liver involvement may lead to hepatic failure, both of which will cause death. Therefore, current treatment involves measures to control hormonal release and growth. Surgical removal of the primary tumor is used for small and localized growths. With liver involvement, embolization of the hepatic artery and chemotherapy with 5-Fluorouracil and Streptozocin (33% response rate) are used.[4] A new treatment involves the use of Somatostatin Analogue (SMS 201-995). This analogue of the endogenous hormone, Somatostatin, is more effective in supressing all GI hormonal release. While the drug appears to be ineffective in slowing the progression of the disease, it does offer effective palliative care for the diarrhea and flushing.[5] □

### References


1. Jager, R.M., Pock, H.C.: Carcinoid apudomas. *Curr Probl Cancer* 1977;1: 11.
2. Godwin, J.D., III: Carcinoid tumors — an analysis of 2837 cases. *Cancer* 1975; 36: 560-569.
3. Mengel, C.e.: The carcinoid symdrome. In: Holland, J.F. and Frei, E., III (eds.), *Cancer Medicine*, 1982: 1818-1827.
4. Moertel, C.G.: Treatment of the carcinoid tumor and the malignant carcinoid syndrome. *J Clin Oncology* 1983; 11: 727-740.
5. Kvols, L.K., Moertel, C.G., O'Connell, M.J., Schutt, A.J., Rubin, J., Hahn, R.G.: Treatment of the malignant carcinoid syndrome — evaluation of long acting Somatostatin Analogue. *New Engl J Med* 1986; 315: 663-666.

# Clinical Trials Research at the Nova Scotia Cancer Treatment and Research Foundation

Clinical Trials Unit. CTRF*

The Clinical Trials Unit has been part of the Nova Scotia Cancer Treatment and Research Foundation for the last 3.5 years. It is a vital and growing part of cancer care in the Province. The function of the Unit is to aid investigators in the conduct of clinical trials research by providing administrative, nursing and data managerial assistance.

The rationale for clinical trials has been well established. It is currently the major method used for scientific study of the outcome of therapeutic interventions in man. Therefore, it is a valid method for evaluation of a new cancer treatment. This paper will outline the research to date, as well as all active clinical trials in solid tumors.

## RESEARCH TO DATE

In the spring of 1985, the staff of Clinical Trials in Oncology was transferred from the Department of Surgery, Dalhousie University, to the Nova Scotia Cancer Treatment and Research Foundation. This transfer was to better serve the mandate of the Foundation to "research into the problems related to cancer". Currently, the Clinical Trials Unit is involved with 18 studies — 7 active and 11 follow-up studies (approximately 280 patients are being followed). There are studies in all major disease sites, some of which are Phase II investigational new drug, while others are Phase III randomized control trials. The Division is currently staffed by one Administrative Coordinator, who is also the Clinical Trials Nurse and one Data Manager, who is a Health Records Administrator.

The conduct of a clinical trial consists of various steps:

1. Development and acceptance of a protocol by a group of physicians interested in a specific disease site.
2. Submission of the protocol to a Committee for Ethics and Research Review. Until approval is forthcoming, no trial can begin.
3. Finally, submission of the protocol to the Clinical Trials Advisory Committee, to ensure the Nova Scotia Cancer Treatment and Research Foundation's ability to conduct a study.

Each trial has pre-set eligibility criteria and pre-trial investigations. These must be met prior to allocation of a patient to a study. Every patient must give written informed consent, and once enrolled in a study, investigations and evaluations are done according to the protocol. All aspects of toxicity of treatment are evaluated, since some of the therapies employed are investigational new drugs.

In many cases, where local participation occurs in national cooperative studies, the Cancer Treatment and Research Foundation of Nova Scotia is given credit and recognition in any publication generated from the trial.

The following is a list of current active studies.

BREAST

C. Metastatic: CMF vs. CEF.

Eligibility: Patients with metastatic breast cancer off any other treatment for 4 weeks. Patients must have no prior treatment with an anthracycline.

Objectives: To assess response rate, time to progression, and severity of toxicity in patients with metastatic breast cancer (no prior anthracycline chemotherapy) following random allocation to Cyclophosphamide, Methotrexate and 5-FU OR Cyclophosphamide, Epirubicin and 5-FU.

Principal Investigator: Dr. R. MacCormick

D. Metastatic: IND. 45 NCIC Phase II Study of Oral Menogaril.

Eligibility: Patients with measurable metastatic breast cancer who have received only adjuvant hormonal and/or non-anthracycline containing chemotherapy.

Objectives:
1. To determine the efficacy of Menogaril as a first line therapy.
2. To determine the qualitative and quantitative toxicity of Menogaril in patients with measurable breast cancer. (No prior anthracycline).

Oral Menogaril 275 mg per square metre weekly.

Principal Investigator: Dr. R. MacCormick

E. Metastatic: Standard Dose Epirubicin vs. High Dose Epirubicin for Women with Metastatic Breast Cancer.

Objectives:
1. To determine whether high dose Epirubicin produces: a) significant prolongation of median survival; and b) increased response rate compared to standard dose Epirubicin.
2. To compare the response duration, time to progression, acute toxicities, and cardiac toxicities of both regimens.

* G. Hirsch, R.N., MSN, Administrative Coordinator, M. Nolan, H.R.A., Data Manager, Medical Advisory Group, Dr. A.J. Bodurtha, Dr. P. Joseph and Dr. R. MacCormick

Correspondence: CTRF Nova Scotia, 5820 University Ave., Halifax, N.S. B3H 1V7

3. To compare the quality of life of both groups of patients and relate any changes in quality to toxicity, response and survival in women who have failed CMF chemotherapy.

Patients are randomly allocated to: Conventional dose Epirubicin 75 mg per square metre vs. High dose Epirubicin 135 mg per square metre every 3 weeks.
Principal Investigator: Dr. R. MacCormick.

LUNG

A. Limited Non-Small Cell Lung Cancer: Study of Radio-sensitization in inoperable non-small lung cancer.

Objectives:
1. To determine efficacy of combination of Lonidamine and Radiation therapy as compared with radiation therapy alone, in terms of the response rate, local disease control and survival, in patients with limited disease non-small cell carcinoma.
2. To determine the qualitative and quantitative toxicity of radiotherapy alone or in combination with Lonidamine.

Principal Investigator: Dr. P. Joseph.

SARCOMA

A. Adjuvant: NCIC Randomized Trial of a single agent (High Dose Methotrexate) compared with a multi-drug regimen.

Objectives: To assess two programs of chemotherapy in patients with osteosarcoma of long bones. The endpoints of the study being survival, relapse-free interval and response to pre-operative chemotherapy.
Principal Investigator: Dr. R. MacCormick.

COLO-RECTAL

A. Adjuvant: Colon NCIC CO3 Study.

Objectives: To determine whether adjuvant therapy with 5-FU/Folinic Acid is effective in increasing survival and prolonging the disease-free interval in patients undergoing curative resection of colonic adenocarcinoma (Dukes B + C). Patients are randomly allocated to Six courses of 5-FU/Folinic Acid OR No additional treatment.
Principal Investigator: Dr. A.J. Bodurtha

B. Adjuvant: Rectal NCIC CO.4 Study.

Objectives: To determine whether adjuvant therapy with 5-FU/Folinic Acid is effective in increasing survival of patients who had a curative resection of carcinoma of the rectum (Stages T2N0; T2N1; T3N0; T3N1; T3N2) and who will receive post-operative pelvic irradiation.
Random allocation to: Pelvic radiation or 5-FU/Folinic Acid — one course + Pelvic radiation followed by five courses of 5-FU/Folinic Acid.
Principal Investigators: Drs. A.J. Bodurtha, O.S. Wong and R. MacCormick.

MELANOMA

A. Adjuvant NCIC ME7. Study of biologic response modifiers (Levamisole vs. Interferon) in melanoma.

Objectives:
1. To compare duration of overall and relapse-free survival among patients who are at high risk for recurrence of melanoma.
2. To estimate rate of toxicities of the treatment.
3. To compare the quality of life among patients who are randomly allocated to either gamma-Interferon or Levamisole in patients who have Stage I or II malignant melanoma (complete resection).

Principal Investigator: Dr. A.J. Bodurtha.

HODGKIN'S DISEASE

A. HD4 — NCIC Stage III-IV for advanced and recurrent Hodgkin's Disease.

Objectives:
1. To determine effectiveness of MOPP/ABV Hybrid and alternating MOPP/ABVD combination.
2. To analyze and compare the toxicity and tolerance of MOPP/ABV Hybrid vs. alternating MOPP/ABVD.

Principal Investigator: Dr. R. MacCormick.

PANCREAS

A. A Phase II Study of 5-FU/Folinic Acid for the treatment of Stages 2, 3, or 4 Pancreatic Cancer.

Objectives:
1. To determine if there is a survival difference in patients with unresectable pancreatic cancer.
2. To determine the qualitative and quantitative toxicity of this regimen. 5-FU/Folinic Acid × 5 days every 28 days.

Principal Investigator: Dr. R. MacCormick.

## CONCLUSION

The Nova Scotia Cancer Treatment and Research Foundation is mandated to research into problems related to cancer. Clinical Trials remain one of the valid methods for evaluating new therapies in cancer. To provide patients in Nova Scotia with up-to-date treatment, clinical trials research is integral to this and any input of patients to the trials would aid in fulfilling a major role of the NSCTRF. □

# The Effect of Cholesterol on Red Blood Cells

P.F. Leahy, MB, MCh, FRCSI

*Windsor, N.S.*

**Cholesterol-rich membranes are the hallmark of "spur" red cells. Spur cells accumulate cholesterol from cholesterol-rich serum lipoproteins. Previous studies suggested that this added cholesterol is responsible for the altered morphology of red blood cells. To examine this process, 2% cholesterol was added to the diet of ground squirrels. The animals were sacrificed at 3 days, 1, 2, 3, 10 and 20 weeks, and the influence of this lithogenic diet upon red blood cell morphology was studied.**

**Red blood cell morphological examination was performed with light microscopy and scanning electron microscopy. The alteration in serum cholesterol concentration was monitored. Changes in red blood cell membrane shapes coincided with a rise in serum cholesterol concentration. It is concluded that excess serum cholesterol probably does influence the structure of red blood cell membranes.**

When ground squirrels are fed a 2% cholesterol diet for more than seven days, 80% form cholesterol stones. The histological examination of gallbladders from these animals show features of cholecystitis at a time before stones are formed.[1] The serendipitous discovery of a high incidence of crenated red blood cells in blood smears of these animals, in whom gallbladder disease was induced, prompted the investigation of the effects of altered serum cholesterol concentration on red blood cell morphology.

In recent years, a vast amount of knowledge has been accumulated about the normal mechanisms responsible for the shape of red cells and of the abnormalities that engender the shapes of target cells and echinocytes. Cholesterol is confined to the membrane of the red cell[2] where it exists almost entirely in the free unesterified form, comprising about 30% of the membrane lipids.[3] Unlike the several phospholipids which turn over or exchange with serum lipids slowly or not at all[4], red cell cholesterol is in rapid exchange equilibrium with the free cholesterol of serum, wherein it exists bound to lipoproteins.[5] Cooper and Arner have provided evidence that the distribution of free cholesterol in the cell membrane may be responsible for the cells biconcave shape.[6]

The present study was designed to elucidate further the association of red blood cell membrane defects with an alteration in serum cholesterol concentrations. The results support the concepts a) that red cells may serve as a repository for cholesterol loosely bound to serum lipoproteins; and b) that concomitant with the acquisition of cholesterol, red blood cell membrane architecture is distorted.

Department of Surgery, University of Alberta, Edmonton, Canada T6G 2N8

Correspondence: Dr. Patrick F. Leahy, P.O. Box 2320, Windsor, N.S. B0N 2T0

## MATERIALS AND METHODS

Ninety young Richardson ground squirrels *(Spermophilus Richardsoni)* captured in their wild state were the animals of choice for this experimental study. Their weight, both males and females, ranged from 220 g to 700 g. Each was caged individually in a thermoregulated room (23°C) on a 12 hour: 12 hour day/night light cycle. Control animals were fed a standard laboratory rat pelleted chow (Wayne Lablox, Allied Mills, Inc. Chicago, Illinois). The experimental animals were fed a laboratory diet similar to the above but fortified with 2% cholesterol (United States Biochemical Corporation, Cleveland, Ohio).

The animals were matched and sacrificed by cervical dislocation at 3 days, 1 week, 2, 3, 10 and 20 weeks. Blood was aspirated from the right and left ventricles of the heart and blood smears were prepared immediately and stained with Wright's stain and examined under light microscopy. Further haematological analysis was performed with a Coulter counter.

Red blood cells were prepared for SEM studies by the following steps: approximately 1 ml of blood was centrifuged at 160 × g for 3 minutes and the plasma and buffy layer removed with a Pasteur pipette. The erythrocytes were then diluted with 2 ml of 0.1% gluteraldehyde in physiological saline at room temperature and allowed to fix for half an hour. The cells were then centrifuged at 100 × g for 3 minutes and the supernatant discarded. They were then resuspended in 2 ml of 2% gluteraldehyde and allowed to fix for 30 minutes; then centrifuged at 50 × g for 2 minutes and the supernatant removed; and then washed with 2 ml of the saline solution for 5 minutes to remove the gluteraldehyde and centrifuged at 50 × g for 2 minutes.

After removal of the supernatant, the cells were ready for dehydration by washing with increasing concentrations of ethanol (10, 20, 40, 50, 70, 80, 90 and 95% in that order). They were exposed to each alcohol solution for 2 minutes and centrifuged at 50 × g for 2 minutes in each case. Once the cells had been resuspended in 95% ethanol, they were ready for examination in the scanning electron microscope. One millilitre of the final suspension was air dried on a glass coverslip glued to an aluminum scanning electron microscope stub. A coating of gold was applied before the cells were observed in the microscope at a beam voltage of 20 KV.

## RESULTS

Fifty nine per cent of the Wright stained smears from the lithogenic diet group were observed to have cells with a regular pattern of spiculation around the periphery. In contrast, only 12% of the control group displayed this irregularity of red cell membranes. (Fig. 1).

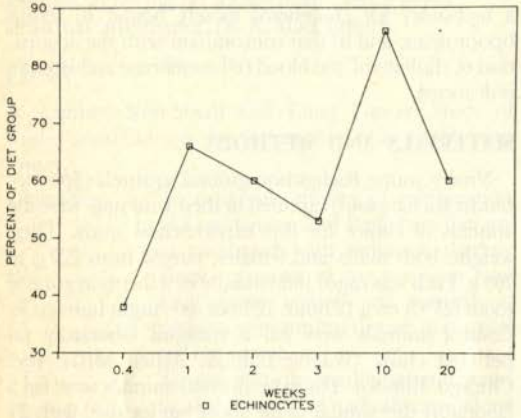


**Fig. 1** Line graph representing percentage of diet group observed to have irregular blood cells. (N-56). Smears were deemed irregular if deformed red cells occupied greater than 50% of field of vision.

Fourteen blood samples were prepared for scanning electron microscopy. In these studies an array of stereotypic (shape changes) were observed ranging from discocytes (biconcave) to the three grades of echinocytes (crenated or spiculated).[7] The most bizarre crenated forms (echinocyte 3) were observed in the experimental group on lithogenic diet. In two cases from the control group, however, early stereological changes characteristic of the echinocyte 1 group were observed. (Figs. 2 and 3).

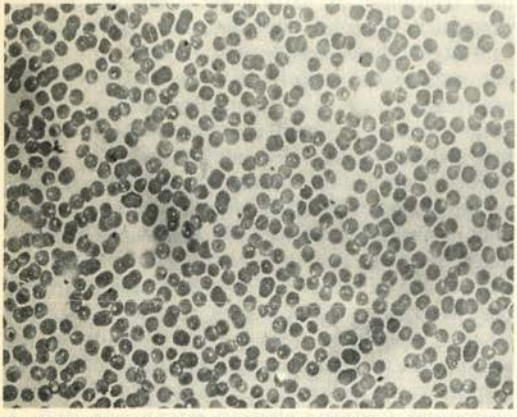

**Fig. 2** Photomicrograph of Wright stained blood smear, illustrating spiculated membranes of red blood cells.
Light microscopy. Magnification × 400

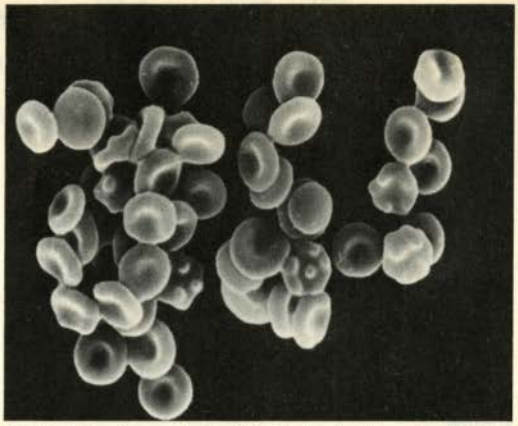

**Fig. 3.1** Photomicrograph of red blood cells, showing early echinocyte (1) formation.
SEM. Magnification × 2000

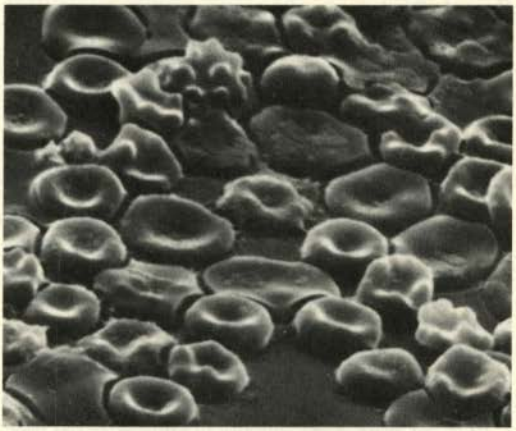

**Fig. 3.2** Photomicrograph of red blood cells, showing severe crenation of cell membranes.
SEM. Magnification × 2000

There was no abnormality detected in mean haemoglobin concentration or red blood cell volume at the various stages of dietary manipulation. Table I.

Serum cholesterol concentration was markedly elevated as early as day three of lithogenic diet. (Fig. 4).

## DISCUSSION

The red blood cells in the treated animals showed a striking picture of deformed membranes. Most of these consisted of marked crenation of cell membranes and thorny projections picturesquely described as echinocytes.[8] The time course of the appearance of the abnormal echinocytes at 3 days would correspond to the rate of acquisition of membrane cholesterol by red cells. Cooper *et al.* have shown that the cholesterol content of red cells incubated in the presence of cholesterol-rich serum increased by 150% after 24 hours.[6]

The observation of isolated echinocytes in 12% of the control group was difficult to explain initially, however,

recent studies using the same pelleted control diet have demonstrated similar echinocyte 1 formation and have attributed these changes to 18:2 fatty acid deficiency.[9]

**TABLE I**

**MEAN VALUES FOR HAEMOGLOBIN CONCENTRATIONS, HAEMATOCRIT, RED BLOOD CELL VOLUME AND WHITE CELL COUNTS.**

| Table | Haematological Indices [Mean, SEM] | | | |
|---|---|---|---|---|
| | Controls | 3 Days | 1 Week | |
| Hb. | 16.6 ± 0.2 | 16.7 ± 0.3 | 16.5 ± 0.8 | grms% |
| RBC. | 10.0 ± 0.1 | 10.2 ± 0.3 | 10.8 ± 0.4 | ×10⁶cmm |
| WCC. | 6.9 ± 0.8 | 11.4 ± 1.5 | 7.5 ± 2.2 | ×10³ |
| Hct. | 51.4 ± 0.7 | 50.7 ± 1.6 | 49.8 ± 3.1 | % |
| | 2 Week | 10 Week | 20 Week | |
| Hb. | 15.7 ± 0.1 | 16.2 ± 0.9 | 17.2 ± 0.5 | grms% |
| RBC. | 10.5 ± 0.07 | 10.2 ± 0.5 | 11.6 ± 0.4 | ×10⁶cmm |
| WCC. | 8.7 ± 0.8 | 7.6 ± 2.1 | 4.6 ± 0.5 | ×10³ |
| Hct. | 50.7 ± 0.4 | 51.4 ± 3.3 | 52.6 ± 1.5 | % |
| | 2 Week | 10 Week | 20 Week | |
| Hb. | 15.7 ± 0.1 | 16.2 ± 0.9 | 17.2 ± 0.5 | grms% |
| RBC. | 10.5 ± 0.07 | 10.2 ± 0.5 | 11.6 ± 0.4 | ×10⁶cmm |
| WCC. | 8.7 ± 0.8 | 7.6 ± 2.1 | 4.6 ± 0.5 | ×10³ |
| Hct. | 50.7 ± 0.4 | 51.4 ± 3.3 | 52.6 ± 1.5 | % |

The reason for the irregular contour of the cholesterol rich blood cells remains unclear. It has been suggested that biologic membranes are not uniform but rather that they have rigid as well as fluid domains within the bilayer.[10] It has also been suggested that with very high cholesterol concentrations within membranes, lecithin-cholesterol complex formation may occur.[11] Either of these phenomena would create a non-uniformity within the cholesterol-rich membrane that may account for the irregularities of contour.

Other investigators have implicated factors such as bile acids as important in the genesis of morphologic abnormalities of red cells.[13] However, by utilising an artificial system, Lange has reproduced these morphologic abnormalities in the absence of bile acids.[12] Lee and Scott have shown that these deformed red blood cells were more resistant to osmotic haemolysis and demonstrated an increased reticulocyte count suggesting an overall shortened red cell survival.[13] Similarly, the observation of structural changes in erythrocytes after short term dietary manipulation, indicate that these changes are not related to the normal life span of red cells.

These morphological observations and the association of raised serum cholesterol substantiate the recent observations that cholesterol is an important determinant of red cell shape. Presumably, any process or disorder affecting cholesterol exchange *in vivo* is capable of critically modifying the shape and behaviour of red blood cells. □

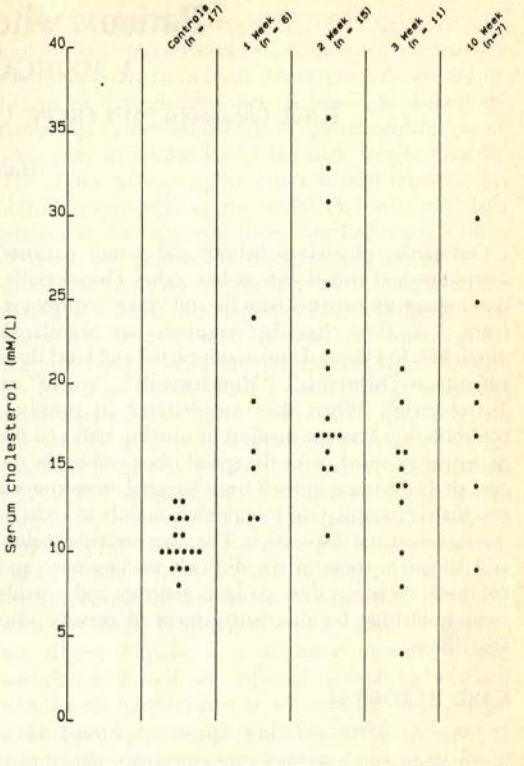


**Fig. 4** Frequency array diagram showing changes in serum cholesterol concentrations.

**References**


1. Freston JW, Bouchier IAD, Newman J. Biliary mucous substances in dihydrocholesterol-induced cholelithiasis. *Gastroenterol* 1969; **57:** 670.
2. Dodge JT, Mitchell C, Hanahan DJ. The preparation and chemical characteristics of hemeglobin-free ghosts of human erythrocytes. *Arch Biochem Biophys* 1963; **100:** 119.
3. van Deenen LLM, de Gier J. Chemical composition and metabolism of lipids in red cells of various animal species. In *Red Blood Cell.* CW Bishop and DM Surgenor, editors. New York: Academic Press, Inc., 1964, pg. 243.
4. Weed RI, Reed CF. Membrane alterations leading to red cell destruction. *Am J Med* 1966; **41:** 681.
5. Lange Y, Dolde J, Steck TL. The rate of transmembrane movement of cholesterol in the human erythrocyte. *J Biol Chem.* 1981; **256:** 5321-5323.
6. Cooper RA, Arner EC, Wiley JS, Shatil SJ. Modification of red cell membrane structure by cholesterol-rich lipid dispersion. A model for primary spur cell defect. *J Clin Inv* 1975; **55:** 115-126.
7. Bessis M, Weed RI. Preparation of red blood cells for SEM: a survey of various artifacts. *SEM/IITRI* 1972; **5:** 290-96.
8. Lessin LS, Klug PP, Jensen WN. Clinical implications of red cell shape. *Advanc Int Med,* 1976; **21:** 451.
9. Rao GA, Siler K, Larkin EC. Diet induced alterations in discoid shape and fatty acid compositions of rat erythrocytes. *Lipids,* 1979; **14:** 30-39.


**Continued on page 78.**

# Patients who "Fake" Illness

## A MEDICAL QUAGMIRE

K.N.R. Chengappa,* MD, FRCPC, U. Jain,* MD and P. Flynn, ** MD, FRCPC

*Halifax, N.S.*

**Ordinarily, physicians believe and accept patients' symptoms and complaints at face value. Occasionally, they encounter patients who lie and "fake" symptoms. Once it is clear that the symptoms are simulated, physicians feel cheated and manipulated and label these patients as "histrionic", "Munchausens", "crocks" or "psychiatric". When they are detected in hospital, confrontation between medical or nursing staff and the patient may ensue, with the spiral often ending in the patient discharging himself from hospital. Sometimes a psychiatric consultation is requested, mainly to assist in management and disposition. The cases presented below will illustrate some of the difficult management and treatment decisions these patients generate and provide some guidelines for the management of persons who fake illness.**

### CASE REPORTS

1. Ms. A., a 30 year old, single, registered nurse presented to a tertiary care emergency department twice during the same day. She claimed to have "syncopal attacks" which she believed had led to injuries of her right elbow and knee, and she insisted that she had sustained a fracture of the right femur. She used medical jargon freely. Comprehensive assessment, including orthopedic consultation and x-rays, revealed soft tissue injury around the elbow and knee. She was told that she had no fracture of the femur and was discharged on mild analgesics. Within days, she presented to a neurologist's office claiming that she had "shadows in the left field of vision". She was admitted to the neurology service of the same tertiary care hospital. Her medications on admission included phenobarbital, 90 mg daily, and phenytoin, 300 mg daily, for a 15 year history of a bonafide seizure disorder. While in hospital, she frequently demanded pain medications, including narcotic drugs. She badgered nursing and medical staff, the hospital administrator, the hospital lawyer and the hospital patient advocacy representative, claiming she was receiving inadequate medical and nursing care and she threatened to sue the hospital. Careful examinations by the neurology service, including relevant investigations, failed to substantiate evidence for any physical illness. At this point the psychiatric consultation service was asked to assist in her management.

   The psychiatric consultant obtained accurate collateral history from a sibling. It was ascertained that she had been dismissed from her nursing post at a U.S. hospital for alleged narcotic drug theft and that, during the past year before admission, the family had received numerous phone calls from different U.S. hospitals regarding the same pattern of demanding and disruptive behaviour. The U.S. police had phoned the family a few months earlier questioning whether wounds that she claimed were a result of a sexual assault were, in fact, self-inflicted. As an adolescent, even when good control of her seizure disorder had been achieved, she was known to feign "pseudoseizures" at school and at home to gain attention in times of stress. She had been unsuccessful in two recent litigation suits against U.S. doctors.

   Based on collateral history and mental state examinations and the negative physical findings, the following psychiatric diagnoses were entertained: a) factitious disorder with physical symptoms; b) severe underlying personality disorder with borderline and histrionic traits; and c) well controlled seizure disorder. She was advised to accept a transfer to the psychiatric inpatient facility within the same tertiary care hospital, which she did rather reluctantly. Interestingly, on the psychiatric service, she no longer complained of the initial physical symptoms but insisted her illness was not 'psychological' but 'physical'! Fairly soon thereafter, she left the hospital wishing to make her own follow-up arrangements. It has since come to our attention that she has presented to walk-in clinics and emergency departments of other hospitals with complaints that could not be explained purely on a physical basis.

2. Mr. B., a divorced, 50 year-old male with borderline intelligence was admitted to a general hospital having swallowed items of cutlery. This was his 54th admission to hospital! Over the years he had swallowed butter and dinner knives, spoons, trimmed down forks, etc. Numerous abdominal operations had left him with scars resembling a grid iron abdomen. As well, on previous occasions, while waiting to be transferred from the surgical

From the Division of Consultation/Liaison Psychiatry, Victoria General Hospital, Halifax, NS B3H 2Y9

* Residents in Psychiatry, Dalhousie University, Halifax, N.S.
** Professor of Psychiatry, Dalhousie University, Halifax, N.S.
(K.N.R. Chengappa — Currently: NIMH Research Fellow, Psychiatric Institute and Clinic, Pittsburgh, PA)

service to a psychiatric facility, he had managed to break a coat hanger to smaller pieces and ingest them, necessitating surgery again!! He had also been admitted for overdoses with pills and 'lysol spray'. He abused alcohol and his notoriety had spread beyond the health system to involve social service agencies, the police, alcohol and drug dependency counsellors, etc. Some of his 14 admissions to a psychiatric facility resulted from transfers from a surgical service and others were direct admissions.

The first contact ever with psychiatric services was for a forensic assessment on a court order remand for a charge of bestiality. He had been convicted earlier for sexually assaulting a teenage girl. Over the years, psychiatric diagnoses had included: borderline intelligence, antisocial and immature personality disorder, alcoholism, affective disorder and factitious disorder. He also had a 30-year history of well controlled seizure disorder. Phenytoin had been the mainstay of treatment for his seizure disorder.

On the last two occasions, some of the objects that were accessible to endoscopy were removed and surgery was not undertaken. Psychiatric consultation was requested and he was transferred to a psychiatric facility yet again, with a view to attempting behaviour modification that might prevent him swallowing cutlery when under stress.

## DISCUSSION

These cases sample the spectrum of factitious illnesses and illustrate the difficult problems that characterize the maladaptive, 'hospital-admission-seeking' behaviour. Unlike 'real' patients who become sick and take on the responsibility for getting better, people who fake symptoms do not have any investment in getting 'better'! The differential diagnoses of this group of patients includes, amongst others, factitious disorders, malingering, conversion/dissociative hysteria, somatization disorders and hypochondriasis.[1,2,3,4,5] Table I attempts to distinguish among the first three categories of patients.[10]

The first case illustrates one end of the spectrum, with multiple factitious physical symptoms which resembles the traditional description of Munchausen's syndrome. Inspite of limited data, there is reason to suspect the patient had conversion hysteria (pseudoseizures) as an adolescent in addition to a bonafide seizure disorder. The choice of nursing as career helped reinforce her abilities to manipulate the health care system in later admissions for factitious illness. She had many features of borderline personality disorder and this made for additional problems during her stay in hospitals. She signed herself out from different hospitals. In time she learnt that 'illness' was one way she could obtain attention from nursing and medical colleagues. As well, her concerned family spent a small fortune when she made long-distance collect telephone calls. She then discovered that her medical disability insurance paid her more when she was 'ill' then she could earn when she was 'well'! This also freed her from work obligations. In summary, it seems likely she progressed from a diagnoses of 'conversion disorder' to a 'factitious disorder' and finally to 'malingering', as monetary benefit became the motive. As her notoriety grew, she went to different hospitals to validate what she claimed was illness. Finally, in a desperate attempt to draw attention to herself, she inflicted wounds to her chest which were later verified as self-inflicted. As the legal and financial pressures grew she returned to this country only to start the same patterns here. As far as we know she has not shown up recently at hospitals in the Halifax metropolitan area.

Mr. B. was introduced to psychiatric services through the legal system. Even as a child, as one of 26 full and half-siblings, he had learnt to swallow coins to gain attention within the family. During a seizure/?automatism as a young adult, he had swallowed nails unintentionally, and had learnt the value of the 'sick role' in terms of the nursing and medical attention he received. In periods of stress, interpersonal conflict and loneliness, he swallowed items of cutlery and 'thrived' on the hospital ward milieu whilst recovering from surgery. This reinforcement set up a vicious cycle of

**TABLE I**

**DIFFERENTIAL DIAGNOSIS OF THE PATIENT**

| Classification | Conscious (Psychological defences) | Voluntary | Other Features |
|---|---|---|---|
| 1. Hysteria (Conversion/dissociative | Unconscious | Involuntary | Clear psychological precipitant with primary gain and possibly secondary gain |
| 2. Factitious disorder (Including Munchausen's dermatitis artefacta, factitious fevers, etc.) | Unconscious | Voluntary | May have psychological precipitant but there is a *Repetitive Need* to maintain the "Sick Role". Major gain is to be a "Patient". |
| 3. Malingering | Conscious | Voluntary | Obvious environmental precipitant. (Trouble with law, military setting, financial problems, etc.) |

unhealthy swallowing behaviour. We wondered whether alcohol, directly or indirectly through its effect on seizure threshold, served to reinforce this behaviour. His 'needs' seem to be better met by a 'sick-role' and he has little investment in getting 'well'! This makes the task of rehabilitation very difficult.

There is a considerable literature regarding the description and management of people who simulate illness and these may be reviewed for comprehensive advice.[1,2,3,4,5]

The following is a summary of important points to bear in mind when dealing with these people:

1. *Early Recognition:* Any person, and especially any health professional whose clinical presentation is 'dramatic' with free use of medical jargon, with no verifiable physical evidence to account for the clinical features and whose history includes visits to multiple hospitals, should raise the possibility of a factitious disorder. Additional clues may include borderline and dependent personality traits, threats of litigation, familiarity with the medical system, pathological lying (including fantastic tales — "pseudologia fantastica"), multiple operations, multiple names (aliases) and a history of drug abuse.
2. *Collateral History:* This is of the utmost importance and cannot be neglected. A patient refusing permission to phone relatives or friends may suggest the diagnosis. Physicians who have dealt with the patient before may provide valuable information.
3. A thorough *history and physical examination* is essential to rule out organic pathology. Investigations must be pertinent to objective medical evaluation rather than being based on patient demands.
4. *A second opinion,* preferably from a senior medical colleague, may help rule out organic pathology, especially in situations where junior house-staff find themselves manipulated by the experienced pseudo-patient.
5. Once it is clear that the diagnosis is 'factitious disorder' or 'malingering', then the *diagnosis must be presented* matter of factly and in a professional manner to the patient, preferably with other medical and nursing staff as witnesses.
6. These people are notorious in splitting the ward staff into those who ally with them and those who do not. To prevent a difficult situation from becoming worse, a consistent approach must be taken by the entire treatment team. A consensus by the entire ward staff of the approach to be taken may prevent some of the problems that arise once the patient is told the diagnosis.
7. In an *emergency room* setting, the prototypical Munchausen's patient will probably leave the hospital when confronted. On the other hand, in an in-patient setting, when patients are confronted with their diagnoses of factitious disorder, they may actually appear more relaxed.[3] This is sometimes the case when evidence for haematuria or diarrhea (e.g., cutting one's finger and adding blood to urine, or adding water to dilute faeces) is discovered and presented to the patient. Other patients may never acknowledge the factitious nature of the illness in spite of evidence to the contrary.
8. Co-operative patients who have personality disorders (borderline, dependent, etc.) may benefit from a psychiatric consultation and may accept psychiatric investigation and treatment. The sociopathic and criminal-type Munchausen's patient is unlikely to accept any form of treatment and will likely move to another city and seek admission to a hospital there.
9. In the case of malingerers, where the manipulation and environmental stress is obvious, health care services have little to offer. They may be more appropriately dealt with by a social worker, a drug dependency counsellor, or by the legal/police system.

There has been a lively discussion in recent literature regarding the costs[6] and ethical considerations[7,8,9] of factitious illness. These articles have raised such issues as whether or not these people are 'patients', whether or not they have the same rights as patients and whether or not insurance companies should pay the bill for "non-illness", etc. These issues are beyond the scope of this paper and references will provide the reader with good reviews.[6,7,8,9]

In conclusion, these patients were among several such patients seen recently by the psychiatric consultation service of a major teaching hospital. This article attempts to illustrate some of the problems these patients cause and to provide some practical management guidelines for the practising physician. □

**ACKNOWLEDGEMENT**

Thanks are due to Shirley Taylor for her secretarial assistance.

**References**

1. Smith RC. A Clinical Approach to the Somatizing Patient. *J Fam Pract* 1985; **4:** 294-301.
2. Nadelson T. False Patients/Real Patients: A Spectrum of Disease Presentation. *Psychother Psychosom* 1985; **44:** 175-184.
3. Reich P and Gottfried LA. Factitious Disorder in a Teaching Hospital. *Ann Intern Med* 1983; **99:** 240-247.
4. Carney WPC. Antefactual Illness to Attract Medical Attention. *Brit J Psychiat* 1980; **136:** 542-547.
5. Stevens H. Is it Organic or is it Functional. *Psychiatric Clin North America.* 1986; **9:** 2:241-255.
6. Donovan DM. Costs of Factitious Illness. *Hosp Comm Psychiatry* 1987; **38:** 571-572.
7. Sadler JZ. Ethical and Management Considerations in Factitious Illness: One and the Same. *Gen Hosp Psychiatry* 1987; **9:** 31-36.

**Continued on page 78.**

# Rectus Sheath Hematoma

## A CASE REPORT

Dane N. Abbott,* MD and Arthur W. Zilbert,** MD, FRCSC

*Dartmouth, N.S.*

Spontaneous rupture of the rectus muscle with rectus sheath hematoma is an uncommon and often misdiagnosed condition. It can mimic other serious intra-abdominal conditions and should be included in the differential diagnosis of any patient who presents with the acute onset of lower abdominal pain.

### CASE REPORT

A forty-three year old Gravida 1 Para 1 female presented to the Emergency Department after she awoke at 0200 hours with severe lower abdominal pain. The pain was steady and non-radiating. She had nausea but no vomiting, bowel or urinary symptoms. She had a respiratory tract infection for several days and had fits of violent coughing. Her LMP was three weeks previously and was normal. She had a tubal ligation a few years previously. She was on no medications.

On examination, she was afebrile with stable vital signs but distressed with pain. Lung fields were clear and heart sounds were normal. Her lower abdomen was slightly distended with a tender fullness. Bowel sounds were normal. Pelvic examination revealed tenderness and a more thorough examination could not be performed because of the pain.

Hemoglobin was 14.3; WBC 19.1 with 86% granulocytes; electrolytes, serum amylase and urinalysis normal. An abdominal series showed no free air, a normal distribution of gas and fecal matter throughout the colon, a few slightly distended loops of small bowel and a rounded mass in the pelvis, mainly on the left but extending across the midline. A pelvic ultrasound revealed a 15 centimetre cystic mass in the left hemipelvis. This was interpreted as being an ovarian cyst and the patient was prepared for laparotomy.

At surgery, approximately 500 ml of blood and clots was found between rectus muscle and peritoneum. The inferior epigastric vessels were identified and ligated. No obvious bleeding points were seen. Abdominal cavity was explored and pelvic organs were normal. The patient was discharged on the fifth post operative day.

*Third Year Resident, Department of Obstetrics and Gynecology, Dalhousie University.

**Lecturer, Department of Obstetrics and Gynecology, Dalhousie University, Halifax, N.S. Consultant, Dartmouth General Hospital, Dartmouth, N.S.

### DISCUSSION

The source of bleeding in rectus sheath hematomas is the rupture of the inferior epigastric artery or tear of the rectus muscle, or both. Minor trauma such as coughing or twisting can cause the damage. It has been experienced during pregnancy, labor and the puerperium. It is associated with collagen vascular disease, hypertension, arteriosclerosis, blood dyscrasias and anticoagulation therapy.

The true incidence of rectus sheath hematomas is unknown and it is felt to be far more common than is generally recognized. It occurs three times more often in females and usually occurs in the fifth decade of life.

Rectus sheath hematoma can present as an acute abdomen. Fothergill sign may aid in the diagnosis: upon contracting the abdominal rectus wall muscle by sitting up, a mass in the abdominal wall remains fixed on it, while an intra-abdominal mass disappears. Lateral decubitus x-rays and transverse sonograms are useful to locate a mass in the anterior abdominal wall, if the diagnosis is suspected.

Usually, management can be conservative with pain control, ice packs and bed rest. The pain gradually lessens over several days and the mass resolves over several weeks. However, hematomas which compromise the patient's hemodynamic status or cause peritoneal signs with marked pain and gastro-intestinal or urinary symptoms, usually require evacuation with careful hemostasis of epigastric vessels. The prognosis is good with a low mortality rate. There are no reports of recurrence. □

#### References

1. Zainea GG, Jordan F: Rectus Sheath Hematomas: Their Pathogenesis, Diagnosis, and Management. *Am Surg* 1988; **54:** 630-3.
2. Simon BC, Rosen P: Abdominal Wall Hematoma: Case Report and Subject review. *J Emerg Med* 1984; **1:** 219-22.
3. Ducatman BS, Ludwig J, Hunt RD: Fatal Rectus Sheath Hematoma *JAMA* 1983; **249:** 924-925.
4. Cervantes J, Sanchez-Cortazar J, Ponte RJ, Manzo M: Ultrasound Diagnosis of Rectus Sheath Hematoma. *Am Surg* 1983; **49:** 542-5.
5. Sybrandy R, Engels PHC: Ultrasonographic Diagnosis of Spontaneous Hematoma of the Abdominal Wall. *Neth J Surg* 1983; **35:** 110.

***A man is a small thing, and the night is very large and full of wonders.***

Edward, Lord Dunsany (1878-1958), Irish poet, dramatist and novelist.

# A Pathologist's Viewpoint

Annette Foyle,* MDCM, FRCP C

*Halifax, N.S.*

**Fig. 1** A young live woman as seen through my eyes. What is the tissue?

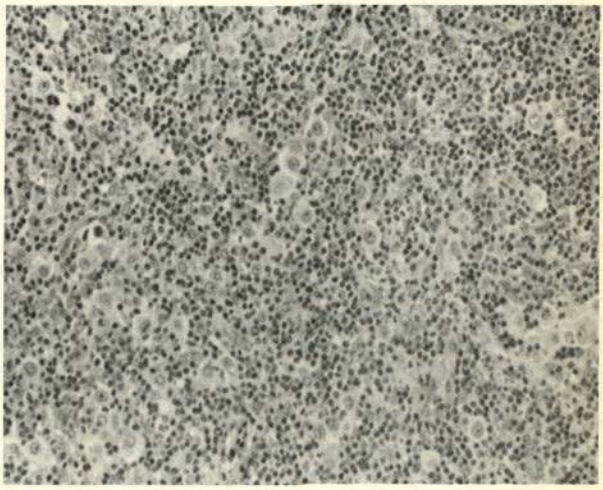

**Fig. 2** What are these large objects in the tissue?

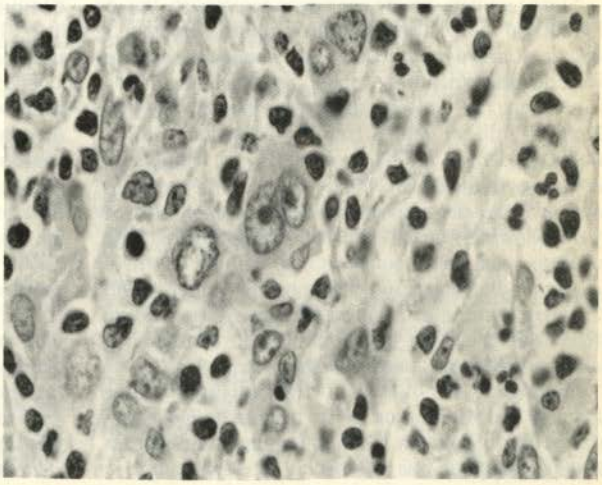

**Fig. 3** What is this very large diagnostic cell? What is the diagnosis?

From the Department of Pathology, Dalhousie University and Victoria General Hospital, Halifax, N.S.
* Assistant Professor, Department of Pathology, Dalhousie University.

The patient is a 20 year old female university student who was well until 9 months previously, when she began feeling constantly fatigued to the extent of missing classes. She also experienced night sweats, anorexia, mild pruritus of the legs, and had lost 20 lb. in weight over a 4 month period.

Physical examination revealed a pale, thin woman, with enlarged lymph nodes in the right neck including a large 4 cm lymph node in the right supraclavicular region. The spleen tip was palpable. The remainder of the exam was normal.

Laboratory investigation revealed the following: Hgb: 115, MCV 73, wbc 12.0, platelets 584,000; atypical lymphocytes in the peripheral blood; polyclonal gammopathy with elevated IgG and IgA; Albumin 29 (N 38-50g/L), ALKPase 287 (N 30-104U/L), SGOT 73 (N 8-29U/L), LDH 396 (N 117-250 U/L). The chest X-Ray disclosed a large anterior mediastinal mass.

The patient then underwent a biopsy of a node in the right neck under local anaesthesia. The histology of this node, however, was inconclusive, so that a second biopsy was necessary, and the largest lymph node (Fig. 1) in the supraclavicular area was biopsied under general anaesthetic. This lymph node showed total effacement of the normal architecture, a fundamental requirement for the diagnosis of a lymphoid neoplasm, and contained many large abnormal Hodgkin's cells (Fig. 2) and lacunar cells. Diagnostic Reed-Sternberg cells (Fig. 3) were also present and the diagnosis of Hodgkin's disease was thus established.

How is Hodgkin's disease classified histologically, and why, in any case, do pathologists subclassify such tumors? When one observes the long lists of categories and subcategories of malignant neoplasms for a single organ, one may well ask this question. For a classification to be useful, it should be relatively simple, easily understood by pathologists and clinicians alike, reproducible by pathologists using it, and provide some useful clinical information in terms of prognosis and therapy.[1]

These requirements are not easy to accomplish, but in the 60s and 70s the histologic classification of Hodgkin's disease fulfilled all these goals. This was the Rye classification which was adopted at a conference in 1965 in New York.[2] Still in use today, this divides the Hodgkin's disease into 1) lymphocyte predominant; 2) nodular sclerosis 3) mixed cellularity; 4) lymphocyte depletion. In the 60s and 70s, lymphocyte predominant and nodular sclerosis vied for the best prognosis and were followed by mixed cellularity, whereas lymphocyte depletion had a very poor prognosis. With the advent of modern radiotherapy and chemotherapy, patients with Hodgkin's disease have a greatly improved survival and recent studies have suggested considerable narrowing of differences between these histologic subtypes.[3]

Staging of the disease involves quantifying the amount and distribution of its lesions. In Hodgkin's disease this is probably the most important measure for determining prognosis and therapy. At a symposium in Ann Arbor, Michigan in 1971, the following were defined.[4]

| Stage | Features |
|---|---|
| I | Involvement of a single lymph node region |
| II | Involvement of 2 or more lymph node regions on the same side of the diaphragm alone or with involvement of limited, contiguous extralymphatic organ or tissue |
| III | Involvement of lymph node regions on both sides of the diaphragm which may include the spleen and/or limited contiguous extralymphatic organ or site |
| IV | Multiple foci of involvement of one or more extralymphatic organs or tissues |

The stages are further divided to indicate the absence (A) or presence (B) of the systemic symptoms: fever, night sweats and/or unexplained weight loss of greater than 10% of the body weight. The term clinical stage implies the stage as determined by a single diagnostic biopsy and diagnostic examinations. In some instances, however, a staging laparotomy is performed, whereby the surgeon biopsies the celiac, splenic hilar, portal, mesenteric and para-aortic nodes and any other suspect nodes as well as performing liver biopsies and splenectomy.[5] The stage of the disease assessed in this way is referred to as the pathologic stage.

For this case, after the diagnosis of Hodgkin's disease was established, the patient underwent the additional clinical investigation. A lymphangiogram was interpreted as normal. A gallium scan disclosed hepatosplenomegaly, multiple areas of uptake in the right neck, mediastinum, aorto-pulmonary window just below the medial aspect of the spine of the left scapula. A CT scan of the chest, abdomen and pelvis revealed a large superior mediastinal mass, hepatosplenomegaly and para-aortic adenopathy. The patient was considered to have stage 4B Hodgkin's disease. In this case, it was classified histologically as nodular sclerosis but the case also illustrates that favorable histology does not necessarily mean early stage.

The therapeutic regimen planned was that of 3 courses of chemotherapy, followed by intensive radiotherapy to the mediastinal mass followed by 3 more courses of chemotherapy. The discussion of all the therapeutic modalities is better left to the experts in this field, but the strides that have been made in this disease are truly remarkable. Therapy has transformed an almost uniformly lethal disease to one where cure is possible if not expected.

**The presentation depicts the pathologist's role in diagnosis, classification and staging of a hematologic malignancy using the model case of a 20 year old woman with Stage 4B Hodgkin's disease and a large mediastinal mass. This required aggressive combination chemotherapy and radiotherapy to the mass. While the**

**prognosis of Hodgkin's disease as a whole is excellent, this patient's outlook is somewhat guarded because of the advanced disease at presentation.** □

### References


1. Rappaport H, Braylan RC. Changing Concepts in the Classification of Malignant Neoplasms of the Hematopoietic System in: *The Reticuloendothelial Systems.* Edited by John W. Rebuck. Monographs in Pathology. Baltimore: The Williams and Wilkins Company. 1975, p. 1.
2. Lukes RJ, Craver LF, Hall TC, Rappaport H, Ruben P. Report of the nomenclature committee. *Cancer Res* **26:** 1311, 1966.
3. Colby TV, Hoppe RT, Warnke RA. Hodgkin's disease: A Clinicopathologic Study of 659 Cases. *Cancer* **49:** 1848-1858, 1981.
4. Carbone PP, Kaplan HS, Musshoff K, Smithers DW, Tubiana M. Report of the committee on Hodgkin's disease staging. *Cancer Res* **31:** 1860-1861, 1971.
5. Beutler E, Ersler AS, Lichtman MA, Williams WJ. *Hematology.* (3rd Ed.) New York: McGraw Hill Book Company, 1983.


## CORRESPONDENCE

**Continued from page 107.**

the above trials, etc, should, at least, give the practising doctor some reservations about intervening in every person with a marginally raised serum cholesterol. It would seem that there is an obvious need for further basic research into the accuracy of cholesterol testing, the amount of biological variations and the fundamental mechanisms of pathogenesis before vast resources are committed to treatment of a problem that may not exist.

Yours truly,

P. Loveridge, M.D.
Department of Family Medicine
Yarmouth Regional Hospital □

### References


1. Langille, D.B. *NS Med J,* 1989; **68:** 39-40.
2. *Lancet* — 1989; **i:** 473-474.
3. Anderson, Castelli and Levy, *JAMA* 1987; **257:** 217-80.
4. Forette, Tortratz and Wolmark *Lancet* 1989; **i:** 868-70.
5. Isles, Hole, Gillis, Hawthorne and Lever *Brit Med J* 1989; **298:** 920-4.
6. McCormick, Skrabenek *Lancet* 1988; **ii:** 839-41.
7. Oliver, M.F. *Circulation,* 1986; **73:** 1-9.
8. Oliver, M.F. *Lancet,* 1981; **ii:** 1090-95.
9. Tan, M. Personal communications. 1988.

# Leprosy in Cape Breton (1852 - 1907)

K.R. Murray,* MD, CCFP

*Neil's Harbour, N.S.*

Although most common in tropical climates, leprosy is not purely a tropical disease. In the nineteenth century, leprosy was prevalant in several northern European countries (including Norway and Iceland), and in North America. In Canada, localized outbreaks occured in several areas including Cape Breton Island, N.S.

The earliest and most significant leprosy outbreak in Canada began in 1815 in the Miramichi and Bay of Chaleur areas of New Brunswick. By the 1840s, leprosy had become a significant public health problem in these areas. As a result in 1844, the New Brunswick Government opened a lazaretto (a hospital for the treatment of leprosy) at Tracadie, N.B. Over the next 120 years this lazaretto treated over 300 patients with the disease.

In 1865, Dr. Alfred C. Smith of Bathurst, N.B. was appointed Medical Superintendent of the lazarreto. During his 40 years in this position, he made several trips to Cape Breton to examine patients suspected of having leprosy. He also played an important role in the eventual control of the disease in Cape Breton.

Two different foci of leprosy appeared in Cape Breton in the early 1850s — one at Lake of Law and the other around Lake Ainslie in Inverness County. It was not until after 1880, however, that any medical researchers visited the area to investigate reports of the disease. The first was Mr. William Fletcher, a medical student, in 1880, followed by Dr. Smith in 1885 and 1889, and by Dr. John Cameron of Port Hood N.S. in 1888.

## CHARACTERISTICS OF LEPROSY

Leprosy (from Greek *lepros* — scaly) is a mildly contagious chronic infectious disease caused by *Mycobacterium leprae* (Hansen's bacillus). This bacillus is bacteriologically related to and shares some of the epidemiological features of tuberculosis.

Spread of leprosy appears to be person-to-person without intermediate hosts. Prolonged contact in squalid living conditions favors transmission. Males are more susceptible than females (ratio 2:1) and children more susceptible than adults (peak incidence of infection 10-20 years). Although not an hereditary disease, hereditary factors affect susceptibility and the course of the disease in a given host. Occasionally, transmission can occur after brief contact in a susceptible individual. Climate, diet and race are not factors in the spread of the disease. Incubation varies widely from 2 to 20 years after exposure.

Currently, four somewhat overlapping clinical stages of leprosy are recognized. *Indeterminate* or early leprosy is characterized by patchy skin discoloration without sensory disturbance. It may regress spontaneously or progress to more severe forms. *Tuberculoid* leprosy runs a chronic stable course, is relatively non-infectious and usually not fatal. This type indicates adequate immune response on the part of the host and arrest of the disease. Clinical features include localized skin lesions, sensory disturbance, deformities and ulceration of the extremities and diffuse cutaneous lumps (tubercles). *Lepromatous* leprosy is unstable and progressive, indicating failed immune response. Features include generalized nodular swelling and thickening of the skin, loss of eyebrows and hair, sensory loss and deformity of the extremities, blindness and facial disfiguration. It is contagious and usually fatal if untreated. *Borderline* leprosy is an unstable intermediate form with features of both the tuberculoid and lepromatous forms. It may regress or progress to other forms.

Subclinical infections occur. Diagnosis is very difficult in the early stages but easier as the disease advances. Transmission within communities and individual families is sporadic with some people never acquiring the disease even after prolonged close contact.

## LEPROSY AT LAKE OF LAW

The first reported case of leprosy at Lake of Law was Betsy (Hardy) McArthy, a native of Yorkshire, England, who first developed symptoms in 1852. She died in 1864 after an illness of 12 years. Her husband, Justin McArthy, born in Newfoundland of Irish parents, lived to age 97 without becoming infected. Between 1852 and 1880, four of five McArthy sons and one of three daughters became infected and died after illnesses ranging from 10 to 20 years. Three McArthy sons-in-law and two grandchildren became infected as did Joseph Brown, a neighbor, who tended one of the leprous McArthy children. Mary Rachel Harris, a niece of Joseph Brown, developed the disease later. The thirteen persons at Lake of Law, reported as having leprosy, are summarized in Table I.

Medical documentation of these early cases is scarce. Leprosy was first suspected by a local physician and by a parish priest who formerly served at Tracadie and tended patients of the lazaretto. By the time William Fletcher visited the area in 1880, only James Cameron (son-in-law of the McArthy's) and Mary Rachel Harris (whose disease had not yet fully developed), were still living.

* Family Physician, Highlands Medical Clinic, Neil's Harbour, N.S. B0C 1N0

Fletcher interviewed and examined James Cameron and subsequently made a detailed report describing the onset of the disease in 1870 and the progress of his symptoms over the next 10 years. He was hampered somewhat by the fact that Cameron, the son of Scottish immigrants, spoke only Gaelic and had to be interviewed through an interpreter. Fletcher's case report is summarized as follows:

**TABLE I**

**LEPROSY AT LAKE OF LAW**
**(WILLIAM FLETCHER — 1881)**

**(Note: Italicized names developed leprosy)**

Justin McArthy m. ***Betsy Hardy McArthy***

| | |
|---|---|
| 1. ***Richard*** | — wife and six children all well |
| 2. ***John*** | — wife and three children all well |
| 3. ***Michael*** | — unmarried |
| 4. ***William*** | — unmarried |
| 5. Henry | — wife and children well |
| 6. ***Mary*** m. ***John Doyle*** | Two children with leprosy<br>Five children well |
| 7. Susannah m. ***James Cameron*** | Two children well |
| 8. Catherine m. ***John O'Connor*** | Children all well |

***Joseph Brown***

***Mary Rachel Harris***

***James Cameron*** — Northeast Margaree, Inverness Co.
Age 42 — Farmer. Married Susannah McArthy — 2 children, both well. Examination — Yellowish discharge from mouth; tongue and mucous membranes thick; skin thick, brownish, scaly with purple spots; nose flattened with septum visible; generalized, small, hard immoveable nodules; a few whiskers on the chin, but otherwise no hair; loss of fingernails and distal phalanges; diminished sensibility of hands and feet; mentally alert; very despondent.[1]

Cameron's symptoms suggest borderline or lepromatous leprosy. He died of his disease a few years later.

## LEPROSY AT LAKE AINSLIE

When Fletcher visited Lake Ainslie, he found that six people had already died of "a disease exhibiting the characteristic symptoms of leprosy". He also examined two sisters, Christine and Margaret MacLean, and their brother, Neil MacLean, who he felt had the disease.

In 1888, Dr. John Cameron of Port Hood visited Lake Ainslie at the request of Mr. W. S. Fielding, the Provincial Secretary. In addition to the persons who had already died, he found six people in four communities (East Lake Ainslie, Egypt, Little Narrows and Lake of Law) who he felt had leprosy. Those cases are summarized in Table II. Dr. Cameron also visited Justin McArthy who was now 92 and still in good health, to review the history of the disease in his family. Mary Rachel Harris refused to be examined but relatives provided him with a detailed description of her disease. In his report to Mr. Fielding, Dr. Cameron included case histories on six persons, two of whom are summarized as follows:

***John Gillis*** — Egypt, Inverness Co.
Age 50. Ill for over 30 years; Married, wife and 10 children all well. Examination — face tubercular and irregular; ulceration and flattening of the nose; eyelashes destroyed; ears elongated; husky, hoarse voice; pale throat, uvula destroyed; hands blue and tubercular; legs atrophic and wasted; feet anaesthetic with ulcers.[2]

***Duncan MacKinnon*** — East Lake Ainslie, Inverness Co.
Age 52. Shoemaker. Unmarried. Examination — face swollen and tubercular; nose enlarged; eyelashes destroyed; hair falling out; ears tubercular, elongated; inflamed ulcerated throat; hoarse voice; severe shiverings.

John Gillis had symptoms and a clinical course compatible with tuberculoid leprosy. Duncan MacKinnon on the other hand almost certainly had the lepromatous form. He was removed to Tracadie in 1889 and died in 1890.

As far as is known, there was no relationship between any of the five Lake Ainslie families. Also, according to Fletcher, there was no traceable connection between the Lake Ainslie and Lake of Law cases. It is interesting to note that at Lake Ainslie, infection did not pass to second and third generations as at Lake of Law.

**TABLE II**

**LEPROSY AT LAKE AINSLIE**

**(Note: Italicized names — patients examined by Dr. John Cameron 1888)**

| | |
|---|---|
| **East Lake Ainslie** | |
| John MacLean | Died of leprosy before 1880 |
| Archie MacKinnon<br>Donald MacKinnon<br>Sarah MacKinnon | All died of leprosy before 1880 |
| Archie MacLean | Died of Leprosy age 40 after illness of 20 years |
| ***Margaret MacLean*** | (40) Ill with leprosy since age 18 |
| ***Christine MacLean*** | (49) Ill with leprosy since age 20 |
| Neil MacLean | Died of leprosy |
| ***Duncan MacKinnon*** | (42) Ill with advanced leprosy<br>Removed to Tracadie 1889 |
| **Egypt** | |
| ***John Gillis*** | (50) Ill with leprosy for 30 years<br>Wife and 10 children well |
| Donald Gillis | Died of leprosy |
| Angus Gillis | Ill with leprosy |
| **Little Narrows** | |
| ***Flora Matheson*** | (33) Ill with leprosy (mild type) for 8 years<br>Sister of Duncan MacKinnon |

## WAS IT LEPROSY?

Initially, some physicians were sceptical of the diagnosis of leprosy. Dr. R. G. Gunn of Strathlorne, Inverness County, felt it was not leprosy but a "hereditary disease". He wrote, "leprosy would have overspread the country before now. Nor is the disease so fatal or so loathsome as leprosy is represented to be."[3] In 1885, Dr. Smith, following his first visit to Cape Breton, reported he found only one person with symptoms suggestive of leprosy. He concluded it was "not leprosy but another disease. Leprosy no longer exists, if it ever did exist, in Cape Breton."[4]

A number of infectious disease can mimic leprosy, especially in the early stages. The only other chronic infectious diseases prevalent in Canada at that time that might cause some of the symptoms described are tuberculosis and syphilis. Tuberculosis would not have posed a diagnostic problem for most physicians at the time but syphilis might have. Certainly, the pattern of sporadic spread through families and districts, the male/female ratio of infection, the peak incidence of infection before age 20, together with the chronic progressive mutilating, often fatal course are very typical of leprosy.

Following the admission of Duncan MacKinnon to the lazaretto in 1889, Dr. Smith confirmed the presence of leprosy in Cape Breton. By this time, it was possible to carry out microscopic analyses on tissue specimens to confirm the diagnosis, and some Cape Breton victims had the benefit of this examination. Dr. Smith concluded, "that leprosy exists in Nova Scotia, there is no manner of doubt. In addition to the general symptoms, the march of the disease etc., the microscope shows the sections filled with leprosy bacilli, and there is the histological structure of the disease".[5]

## ISOLATION AND CONTROL

Dr. Smith complained of considerable difficulty dealing with leprosy victims in Cape Breton. Some refused examination while others, once diagnosed, refused to be removed to the lazaretto. Only one was taken to Tracadie while the others were isolated in their homes under threat of forceable removal. Dr. Smith petitioned the Government of Nova Scotia for more power to deal with these cases. His request was granted but he did not need to use any forceable measures, as he had successfully isolated all remaining cases in their homes. To ensure compliance, Dr. Smith sometimes appointed neighbors to keep watch on the affected families and report to him any breaches in his instructions. The already wary neighbors were usually more than willing to help out.

Dr. Smith advised employers to dismiss any employee he suspected of having leprosy or face "public disclosure and ruin". Leprosy victims and their families, once the diagnosis was made, were doomed to a life of impoverished isolation. The John Gillis family of Egypt were in such a circumstance. Outsiders would not approach the house or assist with chores. Even charitible neighbors left offerings in a barrel at the roadside.

Public alarm over the presence of leprosy in Cape Breton sometimes reached the press. The *Island Reporter* in 1891 criticized Dr. Smith for not placing two suspected leprosy cases at Englishtown in the lazatetto. Following an investigation, Dr. Smith stated he did not find evidence of leprosy at Englishtown and that any public concern was unwarranted.

By the 1890s, leprosy was declining in Cape Breton and in New Brunswick. The policy of enforced isolation in the home seemed to have some effect. The last surviving leprosy victims were Margaret and Christine MacLean who lived out their time in a decrepit hut at Lake Ainslie, supported by the charity of neighbors and a small municipal grant. Margaret died in 1899 and Christine in 1907.

## HOW WAS LEPROSY INTRODUCED?

As almost 30 years passed between the appearance of leprosy in Cape Breton and the first visits by medical researchers, many of the early victims had died and clues to the origin of the disease were lost. As far as can be determined, none of the affected families had any family history or known contact with leprosy. How exactly leprosy was introduced is not known although several theories have been proposed.

One researcher, Dr. A. S. Ashmead, felt leprosy may have been present in North American Indians before European settlement but this was later disproved.[6] Others felt it may have been imported from Scotland by early immigrants to Cape Breton. The last case of endogenous leprosy in Scotland died in 1798 — more than 50 years before the appearance of the disease in Cape Breton. This makes it very unlikely that Cape Breton leprosy was imported directly from Scotland.

Acadian families returning from exile in Louisiana, where leprosy was prevalent, may have introduced some leprosy to New Brunswick. It is tempting to think that they may have introduced leprosy to Nova Scotia but this is most unlikely. No cases of leprosy appeared in the Acadian districts of the Province, and there is no evidence of the existence of leprosy in the Acadian population of the Maritimes, prior to the expulsion in the mid-eighteenth century.

Not infrequently, escapees from lazarettos were at large in Europe and North America. Other persons with leprosy sometimes concealed their disease or were hidden by their families to avoid incarceration. It is possible that some of the immigrants to Cape Breton may have unwittingly come in contact with such persons on the voyage across the Atlantic or after their arrival in North America. Some of the Cape Breton settlers first lived in New Brunswick or Prince Edward Island and may have contacted the disease there.

The people of Lake Ainslie themselves believed that leprosy was introduced to their area about 1825 by men who had fought as soldiers on the Plains of Egypt and later settled in Cape Breton. The 25 year time lapse before the appearance of leprosy in Cape Breton argues against but does not exclude this possibility.

It is not unusual for leprosy to appear unexpectedly in areas where it has not occured before as it did in Cape Breton. Some writers thus describe leprosy as appearing *de novo* without external source. More than anything else, this explanation indicates that there are still significant gaps in our knowledge of the transmission of leprosy.

Which if any of these theories explain the introduction of leprosy to Cape Breton will probably remain unanswered.

## CONCLUSION

In all, 26 cases of leprosy were recorded at Lake of Law and Lake Ainslie between 1850 and 1907. There were some other cases mentioned in Dr. Cameron's report but names were not given. A few isolated cases appeared elsewhere in Nova Scotia but the disease did not spread. Leprosy is now a rarity in Canada but interest continues through the work of organizations such as the Order of St. Lazarus and the Institute Armand Frappier in Montreal.

The stigma of leprosy, which dates back to the Bible, still endures. In 1904, Dr. Smith wrote "of Cape Breton leprosy I shall say little. I wish to respect a request made by the Honorable Mr. Fielding when premier of Nova Scotia that I would refrain from giving any publicity to the existence of leprosy on the island. Besides many of the decendants of those who were lepers now occupy important positions in society and are very sensitive to any reference concerning the disease of their forefathers".[7] □


## ACKNOWLEDGEMENTS

I would like to thank Linda Murray and Marjorie Murray for their assistance in preparing this article.


### Bibliography


1. McPhedran, A. Leprosy in Cape Breton, N.S. *Can Med Science.* 1882; **VII:** 15-16.
2. *Ibid,* **VI:** 271-274.
3. Ashmead, Albert S. Introduction of Leprosy into Nova Scotia and the Province of New Brunswick Micmacs Immune. *JAMA* 1896; **26:** 202-208.
4. *Ibid,* 204.
5. Losier, M.J. and Pinet, C. *Children of Lazarus.* Fredericton, N.B.: Goose Lane Publications, 105.
6. Ashmead, Albert S. *op cit.,* 207.
7. Losier, M.J. and Pinet C. *op. cit.,* 108.
8. Public Archives of New Brunswick. Microfilm. *Notes on the Tracadie Lazaretto.* F-140.
9. La lépre dans les Provinces Maratimes. *L'Evangéline* (Weymouth, N.S.) Jan. 15, 1891.
10. Cochrane, R.G. *Leprosy in Theory and Practice.* Baltimore: Williams & Wilkins: 1964.
11. Thangaraj, R.H., Yawalkar, S.J. *Leprosy for Medical Practitioners and Paramedical Workers.* CIBA-Geigy Ltd.: 1986.

# Current Topics in Community Health

Selected by: Dr. Karim H. Kurji
Department of Community Health & Epidemiology
Dalhousie University, Halifax, N.S.

## PUBLIC HEALTH PROGRAMS IN ONTARIO (New Guidelines)

In April 1989, the Ontario Ministry of Health released new guidelines for boards of health on mandatory health programs and services. The purpose of the standards is to set out the requirements for fundamental public health programs and services targeted at prevention of disease, health promotion and health protection. The following components have been specified for each standard: statutory authority, goal, objectives, requirements, staffing, and monitoring and evaluation. It is expected that programs and services shall be developed with extensive community partnership and that their implementation shall include intersectoral cooperation and coordination. The successful development and delivery of these programs and services will require boards of health to engage in ongoing planning, program evaluation, priority setting and needs assessment to ensure that programs are adapted to and effectively address local needs. Boards of health are expected to employ the services of appropriately trained professionals with skills in the following areas:

- community needs assessment
- program planning
- program evaluation
- data management
- health promotion (community development, social marketing, health education, adult education, behaviour change education)
- case management
- counselling
- immunization practices
- infection control
- health hazard investigation and assessment
- emergency planning

Public Health goals have been stated for four major areas as hereunder:

1. *Healthy Growth and Development:* All people in the community will have the opportunity to attain an optimal level of physical, mental, emotional and social development appropriate to their life stage.
2. *Healthy Lifestyles:* All people in the community will have the opportunity to adopt and maintain health promoting practices for themselves, their family and the community.
3. *Communicable Disease Control:* Communicable disease will be reduced or eliminated.
4. *Healthy Environments:* The community will be a health-supporting environment in which people will be protected from adverse health consequences of exposure to toxic hazardous substances and conditions in homes, public places and the workplace.

General standards have been established for the areas pertaining to equal access for mandatory public health programs and the area of Community Health Status Information. Program standards for each of the following areas have been established:

- healthy children
- healthy adolescents
- healthy adults
- healthy elderly
- tobacco use prevention
- substance abuse prevention
- nutrition promotion
- physical activity promotion
- reproductive health
- sexual health
- sexually transmitted diseases
- vaccine-preventable diseases
- tuberculosis control
- infection control in institutions
- food safety
- water quality
- rabies control
- emergency response
- non-communicable disease investigation

For each of the above programs, the statutory authority, goals, objectives, program requirements and standards have been identified. As an illustration, the "Healthy Children Program" has been selected here. The goal for this program is stated as: "To enable all children (newborn to nine years of age) in the community to attain their optimal level of physical, mental, emotional and social development". The objectives for this program include:

1. To increase the percentage of children practising health-enhancing behaviours.
2. To increase health knowledge and skills in parents and caregivers of children.
3. To increase the percentage of breastfed infants at hospital discharge and at four months of age.
4. To reduce the prevalence of dental disease in children.
5. To increase the early identification of vision and communication disorders significant enough to interfere with a child's education.

The guidelines make it incumbent upon boards of health to target as a priority, subgroups of parents

identified as most in need, such as parents of first babies, adolescent and single mothers, low socio-economic groups, and developmentally handicapped parents. The delivery approaches outlined include postnatal home visits, group sessions to parents and caregivers and consultation to and referral of parents and caregivers, as appropriate. As a minimum, the content of health education shall include:

a) parameters of normal physical development (including vision, hearing, and speech/language), emotional and mental development, and child behaviour;
b) management of breast feeding;
c) development of sound nutrition practices in infants and children;
d) preventive dental health and oral hygiene;
e) benefits of immunization;
f) health benefits of physical activity;
g) prevention of home and motor vehicle accidents, including appropriate use of child safety seats and seatbelts;
h) prevention of child abuse;
i) coping skills and stress management;
j) dynamics of family relationships and supportive family environments; and
k) use of the health care system.

Delivery approaches outlined include assistance in school curriculum development, education in community settings other than schools, and counselling to and referral of children and their parents, as appropriate. In addition, guidelines are provided for boards of health to encourage community-initiated efforts in specific areas as well as the provision of dental health programs and selected screening services for children.

In conclusion, the guidelines provide a useful basis for standardizing the delivery of services across a province while allowing further development of programs according to local needs. Much of the information contained is relevant to the development of public health programs across Canada. □

*Source:* Caplan E, Minister: *Mandatory Health Programs and Services Guidelines.* Ministry of Health, Ontario. April 1989.

# Notice Board

## FETAL CHROMOSOMAL ABNORMALITY AND SERUM ALPHAFETOPROTEIN LEVELS

Low levels of maternal serum alphafetoprotein have a weak association with abnormal fetal chromosomes. The screening of a population at low risk for Down Syndrome utilizing maternal serum AFP will detect 20 to 30% of those carrying an afflicted fetus.

However, universal AFP screening for Down Syndrome is not recommended because the false/positive rate is so high. One in 20 of those undergoing blood testing would require subsequent amniocentesis for diagnostic confirmation, and only one in 100 of this "amniocentesis group" will have abnormal chromosomes.

Evaluation of serum AFP may be useful, however, in women in the "borderline age group" who would not normally be subject to amniocentesis, i.e. in women age 33 to 34 years at time of delivery. Screening *may* also be useful in women 35 years of age or older who are reticent about proceeding to amniocentesis, as it can be used to identify those whose risk is higher than that attributable solely to their age alone. For example, a 38 year old pregnant woman carries a risk of a fetus with Down Syndrome, of approximately 1% on the basis of her age. If she is found to have lower than average maternal serum AFP level, the chance of a fetus with Down Syndrome may increase several fold.

It is important that the evaluation be made at 16 weeks gestation and other factors, such as maternal weight, may influence the serum level. Such testing is coordinated by the Prenatal Diagnosis Clinic. For further information call Carole Smith, R.N., Prenatal Diagnosis Coordinator, Atlantic Research Centre for Mental Retardation, telephone number 424-6491. □

# Advertisers' Index

# Correspondence

**To the Editor:**

I was very interested to read an article on Vertebral Artery Dissection in the February 1989 issue of *The Nova Scotia Medical Journal*. It is well known and accepted by members of the chiropractic profession that vascular accidents can result from manipulation. This is an infrequent but, recognized, complication. I was rather astounded however by the article trying to tenuously associate a stroke, nine days after manipulation as being somehow related. Long delays between the manipulation and the occurrence such as is alluded to here are not supported by the literature or in actual practice. I find the article stretches the limit of credibility and science by trying to tie together manipulation and stroke in these circumstances.

I note in the article itself that it refers to this being an "atypical feature", I would suggest a more accurate comment is that it is impossible to connect the two given the facts as presented.

I must also comment on the fact that chiropractic has a range of techniques and manipulations that it employs. Nowhere in the article is there a reference to what manipulation was or was not done, or in the interest of science or good professional relations was any attempt made to find out what role the chiropractor played in the treatment of this particular individual.

Yours truly,

Paul F. Carey, DC
President
Canadian Chiropractic Protective Association
Toronto, Ontario.

---

**To the Editor:**

The issues raised by Doctor Carey in the first paragraph of his letter are addressed in the discussion section of our paper.[1] Mas *et al* (reference 8 in our paper) reported a patient in whom one month elapsed between neck manipulation and brainstem infarction due to bilateral extracranial vertebral artery dissection.[2]

We acknowledge that chiropractic employs a range of techniques and manipulations. The patient told us that he had his neck manipulated by a chiropractor. We question how further knowledge of the technique used in this patient could help resolve the issues being debated here because vertebral artery dissection has been associated with several different types of blunt cervical trauma.[3]

We accept that a causal relationship between cervical chiropractic manipulation and stroke has not been proven beyond all doubt.[1,4] However, factors other than hard scientific evidence influence decisions about treatment. Benefits and hazards must be carefully weighed; stroke is much more serious than benign musculoskeletal disorders of the cervical spine.

We hope that this dialogue with Doctor Carey helps promote communication between physicians and chiropractors, and leads to better patient care.[5]

Stephen J. Phillips, MBBS, FRCP(C),
Department of Medicine, Camp Hill Hospital.

Jean Gray, MD, FRCP(C),
Department of Medicine, Camp Hill Hospital.

William J. Maloney, MD, FRCP(C),
Department of Radiology, Victoria General Hospital

Dalhousie University, Halifax, Nova Scotia

**References**

1. Gray J, Phillips SJ, Maloney WJ. Vertebral artery dissection following cervical chiropractic manipulation. *N S Med J* 1989; **68:** 30-32.
2. Mas J-L, Bousser M-G, Hasboun D, Laplane D. Extracranial vertebral artery dissections: a review of 13 cases. *Stroke* 1987; **18:** 1037-1047.
3. Hart RG, Easton JD. Dissections and trauma of cervico-cerebral arteries. In: Barnett HJM, Mohr JP, Stein BM, Yatsu FM, eds. *Stroke: Pathophysiology, Diagnosis, and Management*. Edinburgh: Churchill Livingstone, 1986: pg 775-788.
4. Mas J-L, Henin D, Bousser MG, Chain F, Hauw JJ. Dissecting aneurysm of the vertebral artery and cervical manipulation: a case report with autopsy. *Neurology* 1989; **39:** 512-515.
5. Milne M. Are physicians' opinions about chiropractors changing? *Can Med Assoc J* 1988; **138:** 1052-1055.

---

**To the Editor:**

In response to a recently published article in the October 1988 *Journal* (Nova Scotia Cholesterol Results for Nova Scotia Labs), we wish to comment further on some of the issues raised in this paper.

The importance of serum cholesterol in the pathogenesis of atherosclerosis and its associated cardiovascular complications is receiving increased attention. As the primary providers of laboratory data for the Halifax-Dartmouth teaching hospitals, we wish to comment further on one of the issues raised in this symposium, namely the laboratory's role in cholesterol analysis.

First, it is a primary objective of all clinical laboratories to supply a result that is both precise and accurate. That some hospital laboratories display a large bias is not unexpected and, indeed, the range of bias seen in Nova Scotia is not significantly different from that seen for the United States in general. That a large bias requires corrective action by the affected laboratory is not in doubt either, but it should be obvious that laboratories with large biases are likely to be those that handle the fewest specimens. Based on data for 102 external quality checks, the mean bias over a 3-year period has varied between 1.1% and 6.2%. These numbers

should be interpreted in the context of an individual physiological variation that amounts to at least 10%.

**We therefore recommend that physicians consider biological as well as analytical variation in interpreting sample-to-sample variation of serum cholesterol, and to review results of a complete lipid profile before categorizing patients as to their relative risk and need for therapeutic intervention.**

Second, the Nova Scotia Society of Clinical Chemists regards the laboratory targets for accuracy of 5% now and 3% within 5 years as laudable. The differences between laboratories are mostly due to differences in methodology and not to the relative care taken during analysis. Specifically, laboratory accuracy in cholesterol testing depends on the availability of inexpensive, stable, serum standards that cover a wide range of concentrations and have been assayed in a way that gives results that everyone can agree upon. Moreover, if we are to meet the guidelines set up by the CDC, all methods currently in use must be standardized against the method used to establish those guidelines. Given the magnitude of the hypercholesterolemia problem in Nova Scotia and its cost in terms of health dollars to the public treasury, it would be negligent not to examine cost-effective ways of meeting this need.

**For these reasons, we also recommend development of a regional Reference Lipid Laboratory responsible for establishing and maintaining standards of lipid analysis.**

Programs are being developed in areas of Canada where hypercholesterolemia is much less prevalent. There is reason to believe that Maritimers would benefit from a similar program here.

In summary, advances in the diagnosis of hypercholesterolemia require a co-ordinated approach with increased commitments from physicians, laboratories, and public policy-makers. Only in this way will the at-risk individual be accurately identified early enough to benefit significantly from treatment.

Yours sincerely,

Michael A. Moss, M.D., M.Sc., FRCP(C)
President, Section for Laboratory Medicine, Nova Scotia Medical Society, Director, Division of Clinical Chemistry at Dalhousie University and Victoria General Hospital, Halifax

Albert D. Fraser, Ph.D.
President, Nova Scotia Society of Clinical Chemists

---

**To the Editor:**

Although Dr. Langille[1] states that it is his belief that a "population based dietary approach" is the best way to deal with the cholesterol problem, it is by no means certain whether this would be effective and, in any case, is not followed, I feel, by most of the province's physicians who, in attempting to follow the various consensus reports on lipid levels, perform blood lipid analysis on demand, give instructions on diet for those people whose cholesterol levels are between 6 and 8 and may consider drug therapy for those with total cholesterol of >8. Drug companies are particularily active in stressing the latter.

Numerous recent studies suggest this may be a mistake. An editorial in *The Lancet*[2] calls attention to the observation that cholesterol levels were not related to CHD mortality in men over 50, and that a falling cholesterol was associated with a higher death rate,[3] and stated that there is no evidence that lowering plasma cholesterol is beneficial.

Forette *et al*,[4] in one of the few studies not directed at middle age men, show that, in women over 60, the mortality was lowest at a total serum cholesterol of 5.5 to 7.0, that mortality was over five times higher at a cholesterol level of <4.0 and 1.8 times higher at a cholesterol level of >8.8. They do not profer an explanation of this, though they rule out poor nutritional status as a cause. They conclude that "a reduction of cholesterol, either by drugs or polyunsaturated fats should not be advisable, at least if the cholesterol level is under 7".

Another large study, with a 12 year follow up showed that the "positive relation between cholesterol was balanced by an inverse relationship between cholesterol and cancer . . and other causes of death".[5] This study states that "it may be a mistake to assume that dietary advice given to the general population will reduce mortality. CHD may decrease but other risks might increase". They conclude that concentrating on those with the highest levels is likely to be the wisest strategy until more is known of the mechanisms underlying the risks of cholesterol values. This particular study was conducted under the auspices of the MRC hypertension unit in Glasgow, an auspicious organisation with a world renowned reputation.

Other eminent investigators[6,7] share this scepticism about a simple relationship between cholesterol levels and CHD; one postulates a biological mechanism whereby a rise in cholesterol level may be an adaptive, protective mechanism.[8] This contradicts Dr. Langille's statement about the lack of "biological plausibility" though, of course, as will all aspects of this controversy remains unproven. Dr. Langille's final point relates to the prevalence of hyperlipidaemia in Western Nova Scotia. I would point out that these are similar to those in the study at Paisley[5] and other UK and European surveys from which ethnic origins 95% of our population are descended, so this is not surprising, and can be confirmed by those specialising in lipid disorders in the province.[9]

Massive population intervention simply cannot be justified in the absence of clear benefit; study of some of

**Continued on page 99.**

# THE MEDICAL SOCIETY OF NOVA SCOTIA

NOVA SCOTIA DIVISION OF THE CANADIAN MEDICAL ASSOCIATION

## MEMBERS OF EXECUTIVE COMMITTEE

### OFFICERS

*President* ..... V.P. Audain
*President-Elect* ..... P.D. Jackson
*Past President (Immediate)* ..... J.D.A. Henshaw
*Chairman of the Executive Committee* ..... G.A. Ferrier
*Vice-Chairman* ..... S.M.T. Leahey
*Treasurer* ..... R. Brewer Auld
*Honorary Secretary* ..... E.R. Sperker
*Executive Director* ..... A.A. Schellinck

### OBSERVERS AND STAFF

*Economics Committee Chairman* ..... R.J. Gibson
*Director, Economics Department* ..... R.J. Dyke
*Director of Communications* ..... Bill Martin

### BRANCH SOCIETY REPRESENTATIVES

*Antigonish-Guysborough* ..... M.A. MacKenzie
*Bedford-Sackville* ..... J.V. Ramanauskas
*Cape Breton* ..... P.W. Littlejohn
*Colchester-East Hants* ..... D.E. Ryan-Sheridan
*Cumberland* ..... J.P. Donachie
*Dartmouth* ..... G.G.R. Stewart & W.D. Canham
*Eastern Shore* ..... D.R. Barnard
*Halifax* ..... J.J.P. Patil & D.C.S. Browne
*Inverness-Victoria* ..... G.W. Thomas
*Lunenburg-Queens* ..... D.V. Wright
*Pictou* ..... F.A.C. Galvon
*Shelburne* ..... Mark Riley
*Sydney* ..... R.H. Bedard
*Valley* ..... C.L. Smith & P.D. MacLean
*Western* ..... C. MacKinnon
*Student Members* ..... Sandeep Kapur, Sarah Kerr & Camille Sehmebey
*I.R.A. Representatives* ..... Daniel Vaughan, Leo Pereira

### OBSERVERS

*Editor-in-Chief — The Nova Scotia Medical Journal* ..... J.F. O'Connor
*Representative to Provincial Medical Board* ..... H.S. MacDonald
*Medical Director M.M.C. Inc.* ..... A.C. Watson
*President & C.E.O. M.M.C. Inc.* ..... D.L. McAvoy
*C.M.A. Board of Directors* ..... J.C. Kazimirski
*C.M.A. Council on Health Care* ..... A.H. Patterson
*C.M.A. Council on Economics* ..... P.D. Muirhead
*C.M.A. Council on Medical Education* ..... G.L. Myatt
*M.D. Management Limited* ..... G.A. Sapp

### STANDING COMMITTEES

| | Chairman |
|---|---|
| *Allied Health Disciplines* | A.H. Shears |
| *Archives* | D.B. Hogan |
| *Building* | J.F. Hamm |
| *By-Laws* | P.W. Littlejohn |
| *Child Health* | N.P. Kenny |
| *Community Health* | D.B. Langille |
| *Environment* | D.B. Shires |
| *Ethics* | D.M. Rippey |
| *Gynecological Cancer (Screening Program)* | R.C. Fraser |
| *Home Care* | D.P. Cudmore |
| *Hospital & Emergency Services* | D.M. Maxwell |
| *Liaison Committees:* | President |
| *(i) Minister of Health* | |
| *(ii) Workers' Compensation Board* | |
| *(iii) Registered Nurses' Association* | |
| *(iv) Faculty of Medicine* | |
| *Maternal & Perinatal Health* | E.R. Luther |
| *Mediation* | President |
| *Medical Education* | A.H. Murray |
| *Membership Services* | G.L. Myatt |
| *Mortality Review (Appointed by and Reports Through Section of Anaesthesia)* | K.W. Fairhurst |
| *Occupational Health* | A.D. Doucet |
| *Pharmacy* | S.G Carruthers |
| *Professionals' Support Program* | W.G. Gill |
| *Risk Management* | H. Yazer |
| *Senior Advisory Committee* | W.C. Acker |

**The Nova Scotia Medical Journal**
**6080 Young Street,**
**Suite 305,**
**Halifax, N.S. B3N 1Y2**
**902-453-0205**

### BRANCH SOCIETIES

| | President | Secretary |
|---|---|---|
| *Antigonish-Guysborough* | M.C. Gorman | J.R. Hamilton |
| *Bedford-Sackville* | C.A. Forbes | J. Fraser |
| *Cape Breton* | D.P. Hickey | P.W. Littlejohn |
| *Colchester-East Hants* | G.A. Corbett | D.B. Langille |
| *Cumberland* | A. DeCroos | W. Smolonski |
| *Dartmouth* | M.F. Moriarty | G.G.R. Stewart |
| *Eastern Shore* | D.P. Sinha | R.J. Fraser |
| *Halifax* | J.J.P. Patil | D.A. Stinson |
| *Inverness Victoria* | K.R. Murray | C.S. Chow |
| *Lunenburg-Queens* | G.P. Ernest | K.R. Crawford |
| *Pictou* | A.K. Clark | M.N. MacDonald |
| *Shelburne* | I.C. Cree | Mark Riley |
| *Sydney* | S.G. MacDougall | M.A. Campbell |
| *Valley* | R.G. Bustin | R.C. Jung |
| *Western* | W.M. Gorman | D.G. Johnston |

### SECTIONS

| | | |
|---|---|---|
| *Anaesthesia* | A.J.M. Clark | H.J.C. Taylor |
| *Emergency Medicine* | D.E. Sinclair | L.M. Costello |
| *General Practice* | M.E. Lynk | S.A. Canham |
| *Internal Medicine* | J.L. Sapp | N.L. MacDonald |
| *Internes & Residents* | R. Walter | R. Grimshaw |
| *Obstetrics and Gynaecology* | R.H. Lea | |
| *Ophthalmology* | D.M. Keating | F.J. Carpenter |
| *Orthopaedic Surgery* | J.D. Amirault | J.L. Leahey |
| *Otolaryngology* | J.D. Donaldson | G.F. Martin |
| *Paediatrics* | A.F. Pysemany | A.E. Hawkins |
| *Laboratory Medicine* | M.A. Moss | D.F. Glasgow |
| *Psychiatry* | I.M. Slayter | U. Jha |
| *Radiology* | W. F. Barton | M.D. Riding |
| *Surgery* | M.E. Delory | P.D. Roy |
| *Urology* | S.G. Lannon | R.W. Norman |
| *Vascular Surgery* | J.A.P. Sullivan | R.S. Dunn |