# THE NOVA SCOTIA MEDICAL BULLETIN




## A Light Look at "The Literature Jungle"

This month's *Bulletin* is a collection of articles on many subjects involving clinical aspects of medicine, epidemiology, laboratory medicine and even management concerns (from a consumer's perspective). Perhaps it is this diversity that may make this *Bulletin* interesting in contrast to many of the other 2800 journals received at the Kellogg Health Sciences Library on a regular basis.

A survey of the library's journals reveals the frightening level of specialization being achieved in many bodies of knowledge. Titles such as *Cell, Spine, EHP, (Environmental Health)* and *FEMS* (microbiology, not the subject that one might first think), makes the reader wonder at the level of ignorance he or she has maintained. It stretches the credibility that all 2800 journals are read, or even indexed for possible retrieval. More knowledgeable people than this editor, including Peter Morgan in the *Canadian Medical Association Journal*, Jan. 15, 1986, have discussed at length the problem of ever increasing numbers of journals.

Surely something must soon be attempted to solve this problem. Eliminating journals will not, of course, make all this "information overload" disappear, but pragmatic considerations alone will dictate some change eventually.

Meanwhile, the richness and variation of material is something not to be ignored. You are challenged to leave your piles of unread and unsolicited free journals in your office and browse. An undisciplined reading expedition leads to all kinds of discoveries. For instance, an old issue of *Hosiptals,*(May 1985) talks of rationing of health care in its editorial. "Hospitals cannot start making potentially life or death decisions without some guidance from society on such questions as whether rationing must occur, how it should be configured, whom it will affect, and when it should take place. It is true that hospitals have the issue forced upon them, but they must force the issue on everyone, providers, patients, taxpayers, politicians and society itself."

Under miscellaneous media in the same journal, we learn that Otis G. Bowen, Clinical Professor of Family Medicine, Indianna University, and Governor of Indiana from 1973 to 1981, has been sworn in as Secretary of the Department of Health and Human Services. It makes one feel a family physician may yet find his place in the medical system.

In the *American Medical Association Journal* a letter to the editor documents the already known fact that an insect bite on the wrist can cause carpal tunnel syndrome, sometimes needing surgery. If you have practised medicine for years and didn't know that, your level of ignorance will drive you to read on.

From the *Journal of Evaluation and the Health Professions:* "Some of the literature on quality of care implies that cognitive factors in educating health professionals and the appropriateness of their practice experience to present needs." That statement should make health educators stop and think.

And in the *South African Medical Journal* we find an interesting historical note. The Marquis de Sade (1740-1814) spoke against physicians and surgeons being nominated to administer hospitals. He wanted administrators who had, "no pretention to the art of medicine". It is further noted that he ended his life in a mental hospital. It seems that doctors in the past knew how to handle a threat to their authority.

All the above is just offered as a simple celebration of the diversity available in the journals in our libraries. It is interesting to note that in these libraries you can still find things called books, and these keep coming out at about the rate journals used to be published 50 years ago. A visit to the Kellogg Health Sciences Library reveals few faces above the age of 25 or 30, so most of our local physicians must read their books at home. It is a rather frightening commentary on our educational system that many, if not most, graduates might not darken the door of a major medical library throughout their whole career. For fun, education, satisfaction and curiosity, or to break routine, you are urged to be an exception to that scenario. Among other journals you may even find *The Nova Scotia Medical Bulletin.* Feel free to peruse it, but don't stop there. After all there are approximately 2800 other journals, and somebody has to read them. □

J.F.O'C.



**NOVA SCOTIA LUNG ASSOCIATION**

**Presents**

**THE THIRD ANNUAL SEMINAR IN ACUTE RESPIRATORY CARE**
**Apl. 25-26, 1986**

**Halifax Sheraton Hotel**

**Themes:** New Techniques in Mechanical Ventilation
Weaning from Mechanical Ventilators
Cardioplumonary Interactions

**Target Groups:** THE TEAM
Physicians, Respiratory Technologists
Nurses, Physiotherapists

**PRE-REGISTRATION**
$50.00 Physicians
$35.00 Allied Health Professionals

Fee includes luncheon on Saturday, April 26th.

Thomas J. Marrie, M.D., F.R.C.P.(C)

**"Acquired Immune Deficiency Syndrome"**

**For information/registration:**
RD SEMINAR
Nova Scotia Lung Association
17 Alma Crescent, Halifax, N.S. B3N 2C4

# Arboviruses in the Maritime Provinces: California Encephalitis in Humans

Earl G. Morrison,* B.Sc., Bonnie L. McFarlane,** B.Sc., M.Sc. and
Juan A. Embil,*** M.D., Ph.D., F.R.C.P.(C), F.A.C.T.M.,

*Halifax, N.S.*

**Serologic surveys of domestic and wild animal populations in the Maritime Provinces have shown the California group of arboviruses to be endemic to the region. Evidence suggestive of western equine encephalitis, eastern equine encephalitis, St. Louis encephalitis and Powassan virus activity has also been found. Infection of animals with these viruses implies that humans in similar ecological settings may also become infected. The first documented cases of California encephalitis in the Maritime Provinces occurred in Nova Scotia in 1981 and in New Brunswick in 1984.**

## INTRODUCTION

Arboviruses are viruses maintained in nature by cyclic transmission between susceptible vertebrate hosts and bloodsucking arthropods. They are grouped according to their antigenic relationship by complement-fixing, hemagglutination-inhibiting, or neutralizing antibodies. Only a small proportion of the 350 arboviruses known to occur in the world have been associated with human disease. Arboviral infection in humans ranges from unapparent infection, fevers and hemorrhagic fevers to encephalitis, which often results in death.

Most arboviruses belong to the families Togaviridae and Bunyaviridae. Group A and B viruses belong to the family Togaviridae. In North America, western equine encephalitis (WEE) and eastern equine encephalitis (EEE) are the most common group A viruses and St. Louis encephalitis (SLE) is the most common group B virus. Sporadic human cases caused by Powassan (POW) virus of the group B complex have been reported in North America. The California (CAL) group of Bunyaviridae consists of 12 types and has an extensive distribution in North America.[1] CAL is the most common arboviral cause of encephalitis in the United States.[2]

Human cases of arbovirus infection occur annually in North America. In Canada, from 1975 to 1979, there were 74 cases of SLE, 19 of WEE, 4 of POW and the first 4 reported of CAL.[3] In the United States in 1984, there were 33 cases of SLE, 2 of WEE, 4 of EEE, and 74 of CAL.[4] Such cases may result from arboviruses already present in the environment (i.e., WEE, EEE, SLE, POW, CAL) or from arboviruses imported into North America by travellers returning home from visiting areas where such arboviruses are endemic or by emigrants from such countries (i.e., yellow fever and dengue). Therefore, it is important that North American physicians be aware of their patients' travel histories or countries of origin when dealing with possible arboviral infections.

## EPIDEMIOLOGY IN NORTH AMERICA

Table I lists the mode of transmission of the various arboviruses.

WEE has been responsible for several epidemics and epizoötics in Canada's Prairie Provinces and in the western United States. Outbreaks in horses occurred in Manitoba and Saskatchewan as early as 1935.[7] Evidence for WEE activity in humans has been found as far east as Montreal[7] and along the eastern coast of the United States. There have been no epidemics or epizoötics of WEE in the eastern United States, however.

No epidemics of EEE have been reported in Canada. However, small equine outbreaks occurred in St. George, Ontario, during 1938 and in Quebec's Eastern Townships in 1972.[7] EEE is a more serious problem in the Atlantic United States, where epizoötics and epidemics have occurred as far north as Massachusetts.

The first reported Canadian epidemic caused by SLE was in Ontario during 1975.[7] Climatological data indicate a change in normal weather conditions contributed to the outbreak.[8] Human cases also occurred in Manitoba in 1941 and Quebec in 1975.[7] SLE has caused several epidemics in the eastern United States, ranging from Florida to New York State.

POW virus is the only arbovirus originally isolated in Canada. Human cases have originated in Ontario

*2nd year medical student, Dalhousie University, Halifax, N.S.; recipient of a Summer Research Project, School of Medicine, Dalhousie University.
**Department of Microbiology, Dalhousie University.
***Professor, Microbiology, Community Health and Epidemiology and Associate Professor, Pediatrics, Dalhousie University, Halifax, Nova Scotia; Consultant in tropical diseases, virology, and highly dangerous exotic Communicable Diseases, Maritime Region, Health and Welfare Canada; Resource person for reference in investigation, diagnosis, treatment and follow-up of tropical and parasitic diseases.

**Correspondence:** Dr. Juan A. Embil, Infection and Immunology Research Laboratory, Izaak Walton Killam Hospital for Children, 5850 University Avenue, Halifax, B3J 3G9.

and Quebec.[9] In the eastern U.S., human cases have been reported in Pennsylvania and New York.[9]

Three antigenic strains of CAL — snowshoe hare (SSH), Jamestown Canyon (JC) and Trivittatus (TVT) — have been isolated from mosquitoes in Canada.[7] CAL viruses are the most widely distributed arboviruses in Canada.[7] Their importance has increased since 1978 when the first human cases reported in Canada were diagnosed in the Gaspé Peninsula and in Ontario. A total of at least 13 cases have occurred.[1,10-12] CAL viruses have played a more significant role in human disease in the eastern United States than in Canada. Cases have been diagnosed in Florida and as far north as New York State.[13]

Dengue fever and urban yellow fever pose a danger to travellers visiting southern countries where the viruses are known to be endemic.

## ARBOVIRAL ACTIVITY IN THE MARITIME PROVINCES

The wide distribution of arviruses in North America prompted prospective serological surveys for CAL, WEE, EEE, POW and SLE antibodies in animal populations of the Maritime Provinces. Their purpose was to determine the presence of these viruses, to assess their potential as agents in human infection, and to clarify their transmission cycles.

Hemagglutination-inhibition (HI) and neutralization (NT) tests were used to detect these arboviral antibodies in wild and domestic animal sera collected from 1975 to 1979.

Evidence suggestive of group A arbovirus activity has been found in the Maritime Provinces. HI antibodies to WEE and/or to EEE have been found in horse and bird sera of Nova Scotia, horse and

**TABLE I**

**EPIDEMIOLOGY OF ARBOVIRUSES THAT MAY APPEAR IN NORTH AMERICA***

| Arbovirus Type | Vector | Primary Host | Humans Affected |
|---|---|---|---|
| WEE | mosquito (*Culex tarsalis, Culiseta melanura*) | small birds, small mammals | infants, children, older adults |
| EEE | mosquito (*Culiseta melanura, Aedes* spp.) | birds | infants, children, elderly |
| Dengue** | mosquito (*Aedes aegypti*) | humans, monkeys | infants, older adults |
| Yellow Fever** | mosquito (*Aedes aegypti*) | humans, monkeys | all ages |
| SLE | mosquito (*Culex nigripalpus, C. pipiens*) | birds | older adults |
| POW | ticks (*Ixodes* spp.) | small mammals (ground hog) | all ages |
| CAL | mosquito (*Aedes* spp.) | small mammals (snowshoe hare, chipmunk, squirrel) | children less than 15 years old |

*Data adapted from references 5 and 6. **These are not endemic to North America and are imported.

**TABLE II**

**CAL ARBOVIRUSES IN THE MARITIME PROVINCES**

| Area | Host | No. Tested | No. (%) Positive | Arbovirus Found | Type of Test | Ref. No. |
|---|---|---|---|---|---|---|
| N.S. | hares | 1003 | 113 (11.3) | SSH | HI | 13 |
| N.S. | moose | 280 | 189 (67.5) | SSH | NT | 14 |
| N.S. | moose | 280 | 6 (2.1) | JC | NT | 14 |
| N.S. | horses | 861 | 106 (12.3) | SSH | HI | 15 |
| P.E.I. | horses | 248 | 48 (19.4) | SSH | HI | 16 |
| P.E.I. | hares | 215 | 33 (15.4) | SSH | HI | 16 |
| P.E.I. | cattle | 40 | 1 (2.5) | SSH | HI | 16 |
| N.B. | moose | 116 | 72 (62.9) | SSH | NT | 17 |
| N.B. | horses | 204 | 54 (26.5) | SSH | HI | 17 |

snowshoe hare sera of Prince Edward Island, horse sera of New Brunswick (McFarlane BL, Artsob H, Embil JA: unpublished data). Inconclusive evidence for group B arbovirus activity has been found in pigs and chickens from Nova Scotia (McFarlane BL, Artsob H, Embil, JA: unpublished data). Further testing is needed before any conclusive statements regarding group A and B arbovirus activity in the Maritime Provinces can be made.

Table II presents evidence of CAL activity in the Maritime Provinces. Antibodies to SSH in wild and domestic animal populations have been found in all 18 counties of Nova Scotia.[13-15] HI positive hare and horse sera were collected from all 3 counties of Prince Edward Island, indicating a wide geographical distribution.[16] Survey findings in the moose population of New Brunswick are similar to those of Nova Scotia; a large percentage had neutralizing antibodies to SSH.[17] Since SSH positive animal sera were found in southcentral New Brunswick and 2 human cases were reported from the bordering Gaspé Peninsula[1], it seems highly likely that SSH is endemic to the entire province.

Figure 1 presents a possible transmission cycle for CAL in the Maritime Provinces, based on these sero-epidemiological data and from host-vector studies in other areas of North America.[18]

In addition to indigenous arbovirus activity in the Maritime Provinces, there is also the possibility of arboviruses being introduced from other areas. A human case of POW, believed to have originated in New York State, was diagnosed in Halifax during 1979.[19] Human cases of dengue have been brought into Canada by travellers returning to Canada from the Caribbean and Central America. At least 26 travellers returning to Canada during 1976-1979 were reported as having dengue.[3] In Nova Scotia, one case was imported from Jamaica during 1977.[20]

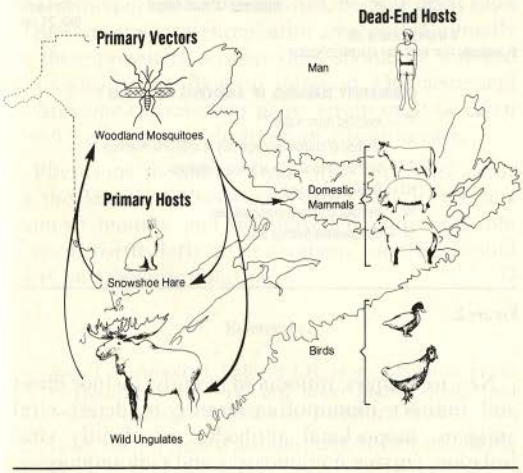



**Fig. 1** Possible transmission cycle for CAL in the Maritime Provinces.

Urban yellow fever poses a danger in regard to visitors to, or emigrants from, Central and South America, the Caribbean and Africa. Historical accounts of yellow fever in Nova Scotia have been documented among people arriving on boats during the 18th and 19th centuries when yellow fever was common in Bermuda, the West Indies and the eastern United States. Reports suggest that some cases were acquired within the province.[21]

## HUMAN CASES OF CALIFORNIA ENCEPHALITIS IN THE MARITIMES

Animal findings indicate that at least one strain of CAL, SSH, is endemic to the Maritime Provinces and that another strain, JC, may be endemic at least to Nova Scotia. Infection of domestic animals implies that humans, living in close proximity to these animals or in a similar ecological setting, could become infected with these viruses. The endemicity of CAL in the Maritime Provinces indicates that a potential for human cases exists in these provinces.

CAL serogroup virus activity in Canada, in association with human diseases, had only been reported in Quebec and Ontario.[22] The first case in the Maritimes occurred in 1981 and involved a 5-year-old boy from Halifax County, N.S., who had not travelled more than 40 km from home before his illness (SSH serotype) during the summer of 1981.[22] High SSH antibody levels had been found in the snowshoe hare in Halifax County.[13] During the next summer, 2 of 5 sentinel rabbits placed in the boy's yard developed SSH antibody levels demonstrating the virus to be persistent.[23]

In August 1984, the second reported Maritime case occurred in an 11-year-old boy from Chatham, Northumberland County, New Brunswick, who had not travelled outside that county for at least one month prior to his illness (also SSH serotype).[11] A 1979 Northumberland County survey showed 18 of 20 moose had SSH antibody and 2 of 2 horses in 1977.[7]

## CLINICAL MANIFESTATIONS

Many arbovirus infections are subclinical and manifest no evidence of disease. When disease does occur, it can range in severity from a flu-like illness to a fatal encephalitis. Manifestations usually follow an incubation period of 4 to 21 days and are characterized by sudden onset of fever, headache, disorientation and sometimes nausea, vomiting, stiff neck and Kernig's sign. Local and generalized seizures are common. The cerebral spinal fluid may contain up to 1000 leukocytes per cubic mm and may have increased protein as high as 100 mg per 100 ml.[24] Table III summarizes the characteristics of arboviral

TABLE III

CLINICAL CHARACTERISTICS OF ARBOVIRAL INFECTIONS*

| Type | Characteristics | Mortality | Sequelae | Comments |
|---|---|---|---|---|
| WEE | convulsions, coma, vomiting, excitement, meningeal signs, paralysis | 3-7% | only severe in infants; physical and mental handicaps | — |
| EEE | fever, lethargy, coma, convulsions, meningeal irritation | 50-70% | especially severe in children; central nervous system damage | — |
| Dengue** | high fever, headache, pain in muscles and joints, muscular rash; remission followed by recurrence | rare | rare | prolonged convalescence |
| Yellow Fever** | fever, chills, severe headache, vomiting; remission followed by high fever, jaundice; severe cases have proteinuria, hemorrhagic manifestations, black vomitus | high | recovery is complete | — |
| SLE | flu-like, aches in muscles, headache, tremors, abnormal coordination and motor cranial function | 5-10% | mental and emotional | — |
| POW | fever, headache, seizures, coma, paralysis | not stated | permanent neurologic damage | very uncommon |
| CAL | headache, vomiting, lethargy, disorientation, seizures, focal neurologic signs | <1% | rare; emotional disturbance | — |

*Data adapted from references 5 and 6.
**These are not endemic to North America and are imported.

infections that are common in North Amereica. For further information, the authors recommend reference 5.

Because clinical symptoms of arbovirus encephalitis are similar to those of viral encephalitis, exact diagnosis can only be made by laboratory tests.

## LABORATORY METHODS AND PATHOLOGY

Arboviral infections can be serious and life-threatening; as such, rapid diagnosis has become very important in many cases.[6]

Epidemiological and clinical study of arboviruses involves virus isolation and antibody measurement (Figure 2). Suckling mice and cell cultures are used for virus isolation. Blood or tissue specimens from the infected vertebrate and suspensions of homogenized arthropods are inoculated intracerebrally either into suckling mice (in which arboviruses are neurotropic) or into cell cultures (where they produce cytopathic effects).

Serum antibody studies are essential in patient diagnosis. For diagnosis, one acute and at least one convalescent phase serum must be taken at 2- to 3-week intervals. Serological tests should include a battery of arbovirus antigens common or suspected in the geographical location where the infection was acquired. A fourfold rise or fall in antibody titre between acute and convalescent sera indicates current viral infection. If antibody is detected without change in antibody titre, a current infection is not necessarily in progress.

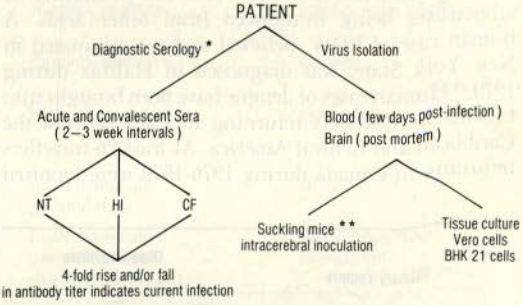



Figure 2.

New techniques, introduced recently, include direct and indirect immunofluorescence to detect viral antigens, monoclonal antibodies to identify viral isolation, enzyme immunoassay and radioimmunoassay and biochemical analysis of viral proteins and nucleic acids.[6]

Seroepidemiologic studies are used to determine the vertebrate species involved in the transmission cycle, specific human populations that may be infected and the geographic distribution of the arboviruses.[5]

Pathological manifestations of arbovirus encephalitis are usually confined to the central nervous system. Inflammatory foci of the brain show necrosis of neurons and glial cells, perivascular cuffing, capillary thrombosis, infiltration of lymphocytes, and varying degrees of meningitis. (Perivascular cuffing is also characteristic of other viral encephalitides: poliomyelitis, rabies and subacute panencephalitis).

## PREVENTION AND TREATMENT

Because several members within a group have common antigens, infection with one virus may confer immunity to others. Immunity is long-lasting and may be permanent.

No effective vaccines for WEE, EEE, SLE, POW and CAL are currently available for general use in humans. There is, however a live, attenuated vaccine to protect humans against yellow fever which is safe, effective and offers long protection.[5] Vaccines are also available for WEE and EEE infections in horses and in humans who are considered at high risk of infection (e.g., laboratory workers).

There are no therapeutic agents for specific treatment of arbovirus infections except supportive care.[6] Breaking the transmission cycle by vector control has proven effective during some epidemics of SLE and urban yellow fever.[5]

## SUMMARY

The endemicity of CAL and evidence suggestive of group A and B arbovirus activity in the Maritime Provinces indicate that human infection could occur in this area. By 1984, the first two Maritime cases (both SSH serotype) had been diagnosed. Because many cases of presumptive viral encephalitis are reported annually in these provinces, suspect cases should be screened serologically for arbovirus infection. One acute and at least one convalescent phase serum must be taken 2 to 3 weeks apart to diagnose an arbovirus case.

Physicians should be aware that CAL may occur in the Maritime Provinces, particularly in the late summer months, and that travellers returning from abroad, particularly from southern countries, could carry an arbovirus infection. □

### References

1. Fauvel M, Artsob H, Calisher CH, *et al.*: California group encephalitis in three children from Quebec: clinical and serologic findings. *Can Med Assoc J* 1980; **122:** 60-64.
2. Gregg MB (ed): Annual summary, 1983. *MMWR* 1984; **32(54):** 21.
3. Artsob H, Spence L: Human arbovirus infection in Canada, 1976-1979. *Canada Diseases Weekly Report* 1980; **6(2):** 5-6.
4. Gregg MB (ed): Arboviral infections of the central nervous system — United States, 1984. *MMWR* 1985; **34:** 283-286, 291-294.
5. Forthingham TE: Arboviruses, In: *Zinsser Microbiology*, 18th ed., Joklik WK, Willett HP, Amos DB (eds). Norwalk, CT: Appleton-Century-Crofts 1984; pp. 1071-1081.
6. WHO: Arthropod-borne and rodent-borne viral diseases. *WHO Tech Rep Ser* 1985; **719:** 1-116.
7. Artsob H, Spence L: Arboviruses in Canada, In: *Arctic and tropical arboviruses*. Kurstak E (ed). New York: Academic Press 1979; pp. 36-65.
8. Bristow GE: Climatological conditions in southwestern Ontario in 1975. Their possible impact on the outbreak of St. Louis encephalitis, In: *Arboviral encephalitides in Ontario with special reference to St. Louis encephalitis*. Mahdy MS, Spence L, Joshua JM (eds). Toronto: Ontario Ministry of Health 1979; pp. 70-77.
9. Wilson MS, Wherrett BA, Mahdy MS: Powassan virus meningeoencephalitis: a case report. *Can Med Assoc J* 1979; **121:** 320-323.
10. Artsob H, Spence L: Arbovirus activity in Canada, 1983. *Canada Diseases Weekly Report* 1984; **10(24):** 93-94.
11. Embil JA, Camfield PR, Artsob H: California encephalitis in New Brunswick. *Can Med Assoc J* 1985; **132:** 1166.
12. Artsob H: Distribution of California serogroup viruses and virus infections in Canada, In: *California serogroup viruses*. Calisher CH, Thompson WH (eds). New York: Alan R. Liss, Inc. 1983; pp. 277-290.
13. Embil JA, Embree JE, Artsob H, *et al.*: Antibodies to snowshoe hare virus of the California group in the snowshoe hare (*Lepus americanus*) population of Nova Scotia. *Am J Trop Med Hyg* 1978; **27:** 843-845.
14. McFarlane BL, Embil JA, Artsob H, *et al.*: Antibodies to the California group of arboviruses in the moose (*Alces alces americana* Clinton) population of Nova Scotia. *Can J Microbiol* 1981; **27:** 1219-1223.
15. McFarlane BL, Embree JE, Embil JA, *et al.*: Antibodies to snowshoe hare virus of the California group in the horse population of Nova Scotia. *Can J Microbiol* 1980; **26:** 654-657.
16. McFarlane BL, Embree JE, Embil JA, *et al.*: Antibodies to showshoe hare virus of the California group in the snowshoe hare (*Lepus americanus*) and domestic animal populations of Prince Edward Island. *Can J Microbiol* 1981; **27:** 1224-1227.
17. McFarlane BL, Embree JE, Embil JA, *et al.*: Antibodies to the California group of arboviruses in animal populations of New Brunswick. *Can J Microbiol* 1982; **28:** 200-204.
18. Sudia WD, Newhouse VF, Calisher CH, *et al.*: California group arboviruses: isolations from mosquitoes in North America. *Mosquito News* 1971; **31:** 576-600.
19. Embil JA, Camfield P, Artsob H, *et al.*: Powassan virus encephalitis resembling Herpes simplex encephalitis. *Arch Intern Med* 1983; **143:** 341-343.
20. Haldane EV, Marrie T: Dengue fever — Nova Scotia. *Canada Diseases Weekly Report* 1978; **4(5):** 19-20.
21. Grant MHL: Historical sketches of old hospitals and alms houses in Halifax, Nova Scotia, 1749 to 1859. *NS Med Bull* 1983; **17:** 491-512.
22. Embil JA, Camfield PR, Artsob H, Rozee KR: California encephalitis in Nova Scotia. *Can Med Assoc J* 1982; **127:** 958-959.

**References continued on page 9.**

# Q-Fever
## A CASE REPORT

David P. Cudmore,* M.D., C.C.F.P. and Olding C. MacIntosh,** M.D., C.R.C.P.,
*Antigonish, N.S.*

**A case of Q-Fever in a part time sheep farmer in Nova Scotia is presented. The infection followed a post mortem examination conducted by the patient on the body of a pregnant ewe.**

### INTRODUCTION

Q-Fever is an acute illness caused by *Coxiella Burnetii*. The disease is not common in man although it has recently been recognized as endemic in Ontario[1] and the Maritime Provinces.[2]

*Coxiella Burnetii* causes widespread infection in wild and domestic animals. Sheep, cattle and goats are commonly infected. In these animals the organism appears to multiply in the placenta and mammary glands, and is present in large numbers in post-partum discharges, milk and feces. It is capable of surviving prolonged drying in dust and excreta and it can remain viable for several months in water and milk. Humans are often infected from these sources.

### CASE HISTORY

A 32 year old part-time sheep farmer presented on March 29, 1984 with a five day history of general malaise and weakness, severe headache and mild dry cough. He appeared toxic and had a temperature of 40.5°C. Examination of the head, neck and chest was unremarkable.

Upon further questioning it was found that about two weeks prior to the onset of symptoms, the patient had carried out a post mortem examination on a neighbor's pregnant ewe. As part of his examination the patient had cut through placental tissue. Subsequent cultures from the animal grew pasteurella hemolytica which was assumed to be the cause of the ewe's death.

The patient's laboratory findings were as follows: Hgb 14.0 gm%, WBC 7.1 x $10^3$/$mm^3$, Polys 52%, Bands 17%, Lymphs 15%, Monos 6%. Sed Rate 52 mm/hr. A chest x-ray showed mottled opacities in the left lower lung field, right upper lung field and right lung base.

Q-Fever was suspected at this time on the basis of the above findings. The patient was started on Tetracycline 500 mg po, qid, which was continued for three weeks. Serology specimens were drawn, and a test for cold agglutinins was negative.

After 24 hours the patient reported that his fever was down and he was feeling much better. Ten days later he was further improved but complained of shortness of breath with minimal exertion, and he was unable to do any work around the farm. A chest x-ray showed resolving opacities in the left lower lobe and was otherwise clear. On the 18th day blood findings were as follows: Hgb 14.0 gm%, WBC 5.5 x $10^3$/$mm^3$, Polys 59%, Lymphs 33%, Monos 7% and Sed Rate 6 mm/hr.

At 3½ weeks he was able to do light farm chores, and at 5½ weeks he returned to full time work. However, at 8 weeks he still had mild dyspnea on exertion. Blood gases and pulmonary function tests were normal. A repeat chest x-ray was normall.

Serology reports confirmed the diagnosis of Q-Fever. The multiple phase antigen complement fixation titre for *Coxiella Burnetii* antibodies rose from less than 1:8 on April 2, 1984 to 1:128 on April 16, 1984. Haemagglutination tests on the same sample showed a rise in phase II antigen (acute phase) from 1:8 to 1:256 while phase I antigen (chronic phase) showed an insignificant rise from 1:8 to 1:16. The mycoplasma pneumonia titre remained unchanged at 1:32.

### DISCUSSION

This case illustrates the danger of acquiring Q-Fever from the placental tissue and birth fluids of domestic animals. The organism can be acquired both from direct contact and by airborn transmission.[3] Welch and his co-workers found the organism in the air within a few minutes after parturition and up to twelve days later.[3] The same authors found viable *Coxiella Burnetii* in the soil at a lambing site 150 days after parturition.[4] The uncomplicated infection ordinarily does not kill the animal. In this instance the flock involved was decimated by a pasturella infection and the *Coxiella Burnetii* infection was an incidental finding.

Symptoms in humans are intitially non-specific, including headache, chills and fever, myalgias, anorexia and malaise. High fever and headache tend to predominate. Cough, chest pain and inspiratory rales usually develop after 4 to 5 days. The illness typically lasts for one to two weeks and complications are rare.

Granulomatous hepatitis can occur and is associated with a more protracted illness.[5] Endocarditis has also been reported and usually affects the aortic valve.

*Department of General Practice, St. Martha's Hospital.
**Department of Pathology, St. Martha's Hospital, Antigonish, N.S.

Laboratory findings may include an elevated ESR, abnormal liver enzyme tests, thrombocytopenia and positive RA factor tests.

Chest x-ray findings are non specific and are described by Marie as "multiple rounded segmental consolidations of the density of ground glass."[2] "Other findings include linear atalectases, lobar or partial lobar consolidations and often slight pleural reactions." These findings may take up to seventy days to resolve.

Serological testing is the most reliable method of confirming the diagnosis of Q-Fever. The complement fixation and haemagglutination methods are used and a positive result is based on a four fold rise between an acute and convalscent titre. The complement fixation procedure for multiple antigens serves as a screening procedure. Haemagglutination tests include separate titres for phase I and phase II antigens. Phase I antigen titres usually indicate chronic disease while phase II antigens indicate acute disease.

Tetracycline 500 mg orally every six hours is the treatment of choice and usually results in a prompt recovery, as it did in this case. Some infections also respond to Erythromycin. In cases of Q-Fever endocarditis the patient may require valve replacement in addition to a long course of antibiotics to eradicate the infection.

Prevention requires education of the public about the proper handling of potentially infected livestock. Vaccines are available for high risk individuals. □

**References**


1. Simor A.D., Brunton I.L., Salit I.E., Vellend H., Ford-Jones L., Spence L.P., Q-Fever: Hazard From Sheep Used In Research. *Can Med Assoc J:* 1984; **130:** 1013-1061.
2. Marrie T.J., Haldane E.V., Noble M.A., Faulbner R.S., Lee S.M.S., Gough A., Meyers S., Stewart J., Q-Fever In Maritime Canada. *Can Med Assoc J:* 1982; **126:** 1295-1300.
3. Welch H.H., Lennett E.H., Abinanti F.R., et al. Airborne Transmission of Q-Fever: The role of paturition in the generation of infective aerosols. *An N Y Acad Sci:* 1958; **70:** 528-1958.
4. Welch H.H., Lennett E.H., Abinanti F.R., et al. Q-Fever Studies XXI "The Recovery of Coxiella Burnetii from the soil and surface water of premises harbouring infected sheep." *Am J Hyg:* 1959; **70:** 14.
5. Qizil A.H., The Pathology of Q-Fever As Seen On Liver Biopsy. *Arch Path Lab Med:* 1983; **107:** 364-367.


**ORGAN DONOR AWARENESS WEEK**

April 20-26, 1986

## ARBOVIRUSES IN THE MARITIME PROVINCES: CALIFORNIA ENCEPHALITIS IN HUMANS

**Reference continued from page 7.**


23. Embil JA, Artsob H, Hamilton DA: Persistence of snowshoe hare virus in Halifax County, N.S. *Can Med Assoc J* 1984; **130:** 1105-1106.
24. Heffner RR Jr, Strano AJ: Arbovirus encephalitis, In: *Pathology of tropical and extraordinary diseases.* Vol. 1. Binford CH, Connors DH (eds). Washington: Armed Forces Institute of Pathology 1976; pp. 36-40.
25. Kalter SS: *Procedures for routine laboratory diagnosis of virus and rickettsial diseases.* Minneapolis: Burgess Publishing Company 1963; p. 67.


**NOVA SCOTIA SAFETY COUNCIL**

4th Annual Safety Conference

Wednesday March 26th and
Thursday March 27th, 1986

Keddy's Motor Inn, 437 Prince Street, Truro, N.S.

To address the three disciplines of safety TRAFFIC, OCCUPATIONAL AND PUBLIC and to provide a forum for discussion of these topics with something new added — a Youth Symposium on the theme of Young Adult Involvement inTraffic Fatalities.

Cost — $65.00 per delegate and $25.00 per spouse.

For additional information contact:

Jackie A. Yule, Administrative Assistant
Nova Scotia Safety Council
2745 Dutch Village Rd., Suite 106,
Halifax, N.S. B3L 4G7
Telephone 454-9621 or 453-5525

# Therapy in Multiple Sclerosis: A Brief Overview

T.J. Murray,* M.D., F.R.C.P.(C), F.A.C.P.,

*Halifax, N.S.*

## INTRODUCTION

It is often frustrating to patients with multiple sclerosis (MS), and the physicians who care for them, to hear the statement that MS is "untreatable". Like diabetes, most cardiac disease and hypertension, all of which have a greater impact on life expectancy than MS, multiple sclerosis is as yet not curable, but there are many important treatment and management advances for patients with this disease.

In this very brief review, I will give an approach to treatment aspects of multiple sclerosis, used in the Dalhousie Multiple Sclerosis Research Unit,a recognizing that in an uncertain field, with many experimental treatments, other specialists in MS may have different opinions. It is hoped that this overview will give a practical guide to the physician entrusted with the care of the patient with MS, and give some understanding of the newer concepts of treatment that are under study but often mentioned in the popular press prematurely as "advances" or "breakthroughs".

## MANAGEMENT OF THE PATIENT

Initially, patients seek an explanation for the neurological symptoms they have been experiencing. Increasingly in recent years, patients with MS have suspected their own diagnosis because it is becoming a disease familiar to the public. The diagnosis is still a traumatic one, and patients require a lot of reassurance, explanation and advice. I spend a rather long sesion with patients initially and then have them return for a subsequent visit, asking them to make a note of any questions they have. We often suggest that they bring the spouse or another family member if they wish. Patients require a supportive but frank and honest discussion of the disease, with an indication of what we know and what we do not know and cannot effectively predict.

Physicians frequently disguise the diagnosis in obscure language, or give it another name that does not convey any information. Patients almost always come to resent this later, and are grateful if they receive honest and frank discussion of the diagnosis, even though it may be disturbing and difficult to accept. It is not a kindness to keep patients ignorant of a diagnosis and its implications when it will affect their lives and future decisions. I suspect doctors are reluctant to impart such a serious diagnosis, and are unwilling to spend the time with patients who are upset, and who suddenly have lots of pointed questions about a disease the physician does not understand well.

## TREATMENT OF THE DISEASE PROCESS

Unfortunately, therapy related to the underlying demvelinating process is inadequate at present because we have insufficient knowledge of the cause of the disease, and the mechanisms involved in the recurrent and ongoing process. There are methods of therapy, however, which can improve the patient's symptoms, and there are a number of other promising therapies under active investigation.

Treatments can be classified as those that manage acute attacks, those that are aimed at preventing recurrences or progression of the disease, and symptomatic therapies that are helpful for the problems and complications that may result from the disease.

### Treatment of Acute Attacks

Most recurrent symptoms in the disease require only assessment, reassurance and explanation, and a period of rest. Many of the symptoms and problems will settle down in time, and require no specific treatment or therapy. Most minor symptoms begin to settle down quite adequately in days or weeks.

Patients who have more severe attacks, or who continue to progress despite rest, can be treated with steroids. The usual treatment of acute attacks is with ACTH and a brief treatment is over two weeks, beginning with 80 units per day for four days, then decreasing progressively by 20 units every four days. Oral steroids are commonly used because of their convenience, but the value of this treatment is uncertain. In the last few years we have been treating our patients with high dose, pulsed methylprednisolone, giving 1,000 mg intravenously over thirty minutes, every second day for five doses. This is well tolerated and appears to be as useful as ACTH. We are currently carrying out a double blind study comparing ACTH with methylprednislone, but the results will not be known for another year.

Treatment of the accute attack may also involve the specific treatment of the symptoms, as indicated in a later section.

*Director, Dalhousie Multiple Sclerosis Unit, Camp Hill Hospital, Halifax, N.S.

**Correspondence:** Dr. T.J. Murray, Clinical Research Centre, 5849 University Ave., Halifax, N.S., B3H 4H7.

**Treatment and Prevention of Chronic Disease**

A number of therapies are under study for their effect on the disease process, but none has as yet, an accepted place in treatment.

*Diet*

Dietary management of multiple sclerosis has a long history, but only in the last decade has there been a renewed interest in the use of a low animal fat diet with linoleic acid supplement. This diet may be beneficial to younger, less disabled patients, particulary early in their disease. As it is a healthy diet, it is probably wise to recommend this to all patients when they are diagnosed, even though the complete answers on the effectiveness of the diet will take some time.

*Cyclophosphamide*

There is some indication that this treatment, despite its significant side effects, may be the most useful current treatment for progressive multiple sclerosis. Experience with this drug has allowed us to control many of the complications such as bladder inflammation, infection and untoward hematological effects, but transient hair loss remains the most annoying side effecct to the patient. There is growing information that this treatment may be able to stop the progression of the disease in many patients, at least for a time. Some patients are being retreated with the hope that occasional retreatments may continue to keep the patient from the progressive and disabling stages of the disease. Our experience has been that the side effects and complications are too great to consider this treatment in most cases and patients themselves will stop long term therapy because of the complications.

*Azothioprine*

Although there is long experience in Europe suggesting that it may reduce long-term disability, the place of this immunosuppressant drug is uncertain. It is much easier to use than cyclophosphamide, as it requires only daily oaral doses, and repeated blood counts. The worry about long-term complications such as malignancy, is a real one and its place in the treatment of MS patients is uncertain.

*Plasmapheresis*

There are few promising reports of this treatment, and some negative reports. It is an expensive treatment requiring complex technology and skilled personnel, with some complications. Unfortunately, it is not clearly understood what we should be doing when we fractionate and remove plasma or cells from the circulation of MS patients.

*Hperbaric Oxygen*

This form of treatment has gained a lot of publicity in the past few years, and has the potential to be very strong, but expensive placebo. Whether it has any specific effect, or any lasting or useful benefit is still uncertain. A number of trials are underway but our ancedotal experience has been negative. We are currently attempting to measure psychophysicalogical changes during this form of treatment, but have not seen enough logical or promising signs in this therapy to recommend it for the treatment of our patients.

*Interferon*

There are currently a number of studies underway examining the effect of interferon administered by various routes, but its place in treatment is uncertain. Side effects are frequent and this remains an experimental treatment.

*Cyclosporin*

Because of its dramatic effect in transplatation rejection, cyclosporin has been of interest in multiple sclerosis, but it is too early to judge whether it is of any value.

*Copolymer 1*

This synthetic protein resembles myelin basic protein, and is currently undergoing further study. The preliminary results suggest that it is well tolerated, and may be of benefit in some patients, but large studies are required. The Dalhousie MS Research Unit will be involved in a large international cooperative study of this substance.

## SYMPTOMATIC TREATMENT

There are many symptoms that may occur in multiple sclerosis. Some of the symptoms are easily managed, although they may not be so frequent or disabling. The more disabling and frequent symptoms are less well managed. Symptoms have to be evaluated carefully in the disease, however, as it is common to assume that any symptom is due to multiple sclerosis, when these patients experience all the problems and diseases that any other person might experience.

**Fatigue**

Most patients with multiple sclerosis complain of excessive fatigue. It is described as a very abnormal overwhelming fatigue that is only partly related to work or exercise, and inadequately relieved by rest. It seems to us that this is a unique and pathological fatigue, greater than fatigue seen in other neurological diseases, but the central neurochemical or metobolic basis is unclear. In a study of this symptom, we found that 60% of the patients felt they had some limitation of activity because of their fatigue, and in 40% it was their chief complaint. It is thus one of the commonest complaints of MS patients.

In two studies we found that amantadine hydrochloride (Symmetrel ®) was successful in reducing fatigue

in 62% of the patients. In about one-third of the patients it was of no value, but in one-third it had a dramatic effect, which persisted long term if the drug was continued. There are significant side effects in many patients, although most continued to take the drug, sometimes in a reduced dosage. The dosage is 100 mg twice a day. But many patients tolerate 200 mg better in the morning. Common side effects include insomnia, hyperactivity, irritability, constipation and mild ankle swelling. In a few, more serious side effects such as confusion and hallucinations may result. We have had some milder improvement in patients treated with Cylert®. In some patients minor modifications of daily activities to compensate for this increased fatigue may be all that is required.

**Stress**

Everyone experiences stress in his or her life, but MS patients often have more difficulties to overcome. Most patients note that acute attacks, recurrent symptoms of worsening of the disease often occurs at the time of stress, and patients sometimes require advice on how to minimize and cope with manageable stress in their lives. The mechanism whereby stress induces worsening of the disease is unclear but it may relate to immunological changes that occur during periods of stress.

**Trigeminal Neuralgia**

If clear cut trigeminal neuralgia occur in a young person, it usually indicates underlying multiple sclerosis. When it does occur in MS patients it is treatable and manageable like "idopathic" trigenimal neuralgia in the older age group. Most patients respond well to carbamazepine (Tegretol/Reg), 400-800 mg per day. Patients who cannot tolerate this drug or who break through the response, usually respond to baclofen (Liorisal/Reg) 40-80 mmg per day in divided doses. Surgical procedures are very successful, but usually not required.

**Pain**

It is not often recognized that pain is a relatively frequent problem in MS patients, and one in ten may relate to muscle aching, secondary low back pain, trigeminal neuralgia, lancinating pains, dysesthesiae (described by the patient as pain) or painful muscle spasms and cramps. Although it is best evaluated and treated specifically, many of the undifferentiated or unclear pains respond to carbamazepin (Tegretol/Reg), baclofen (Liorisal/Reg) or amitriptyline (Elavil/Reg.)

**Seizures**

Seizures occur in about 5% of the patients with multiple sclerosis. again, this is not a commonly recognized complication in the disease, but when one looks at NMR pictures or pathological specimens, it is not surprising that seizures might occur in the numerous scarred and demyelinated areas in the cerebral hemispheres. These seizures respond well to anticonvulsants, such as phenytoin (Dilatin/Reg) and carbamazepin (Tegretol/Reg). It has been our pattern to wait until the second seizure before treating the patient with long term anticonvulsants, although circumstances may dictate that initial treatment is necessary.

**Tremor**

Tremor, usually from cerebellar involvement, can be a serious disabling feature of the disease. When it is mild the patient can often compensate for the incoordination and unsteadines, but when greater, it is a very difficult problem. Some patients will respond to isoniazid (INH) in high doses, sometimes as high as 1200 mg per day, but our experience has shown that few patients benefit substantially, and many experience serious side effects of this new treatment. A few will respond to clonazzepam, and it is worth trying patients on this drug in dooes of 6-8 mg per day. The tremor can often be reduced to some degree by propranolol in doses of 120-240 mg per day.

**Muscle spasm**

Many patients with spasticity complain of sudden involuntary muscle spasms, often at night or at rest. These are sometimes painful. They respond well to baclofen 20-60 mg per day. Many patients cannot tolerate higher doses of baclofen because it makes them feel generally weak. However, this drug, disappointing in the treatment of spasticity, is very useful in the management of spasms.

**Fever**

Patients with MS are often sensitive to heat, particularly any circumstance in which their body temperature is raised, even a fraction of a degree. They may experience specfic symptoms such as diplopia or numbness, but often experience a generalized weakness and malaise. Any symptomatic bacterial infection should be treated, and even mild respiratory infections require anti-pyreticc medication to reduce the generalized effects of fever.

**Spasticity**

One of the most disabling and common problems of multiple sclerosis is the development of spasticity. Certainly this is one of the poor prognostic indicators. Unfortunately, the current management of spasticity is woefully inadequate. We no longer use dantrolene sodium because of its inadequate response and common side effects, and find that baclofen also gives only a mild response, often causing marked weakness in patients at therapeutic dosages. Although spasticity can be measurably altered, there is often no functional improvement in the patient.

### Ataxia

Ataxia is another serious and disabling feature when it develops. It may respond to high doses of isoniazid (INH), but we have had poor results using this, and the side effects are frequent. Some patients respond to clonazepam 4.8 mg per day, and some respond mildly to propranolol 120-240 mg per day.

### Numbness and Paresthesiae

Many patients complain of recurrent episodes of numbness, paresthesiae and dysesthesia. Often they require only reassurance as the numbness tends to be annoying but not disabling. An occasional patient will have such marked numbness that it does produce serious dysfunction in position sense and thus disability in the hands or imbalance in the legs. In such cases we treat this as an acute attack of the disease, particularly if it is sudden in onset. Usually the dysesthesia will respond to short-term steroids, or to the use of tricyclic anti-depressant medication.

### Lhermitte's sign

Is a sudden electric shock-like sensation, usually down the back or in the limbs, associated with flexion of the neck. This often frightens patients, but usually only requires some explanation or reassurance. It indicates inflammation or disruption of posterior column fibers.

### Impotence and Frigidity

Impotence is a common problem in male patients who have had the disease for many years, particularly if they have spasticity, and it is a very distrubing and serious problem for them. We attempt to differentiate those that have a significant psychological component, as this is often a manageable component of the problem. Others, we refer to a urologist for advice and management. In a few patients, we have recommended penile implants, but the success and satisfaction with these is usually moderate, at best.

This type of problem requires a lot of discussion and explanation with both partners and we have found that this discussion has allowed the couple to verbalize a lot of tensions and conflicts that they have not been able to bring into the open before.

### Diplopia

Double vision is a very annoying and sometimes disabling symptom if it occurs. It is usually transient and in most instances requires only reassurance and explanation. In a few cases, when it is striking or persistent, we have treated them as an acute attack and the response is usually good.

### Vertigo

This is also usually a transient symptom requiring only symptomatic treatment. We are not impressed that any of the so-called anti-vertigo drugs are very useful, and have had better results with diazepam, which reduces the vestibular reflex. In some cases, if the problem is marked or prolonged, we have treated the patient with methylprednisolone.

### Contractures

Patients with long-standing spasticity often begin to develop flexion contractures. Patients with dropped feet often develop Achilles tendon shortening. We take care to examine the range and motion of patient's joints, particularly if they are limited in their use of the limbs, and indicate gentle stretching exercises that they can carry out, sometimes with the help of a family member. In more dramatic cases a course of physiotherapy may be required.

### Memory Loss and Dementia

Some patients complain of memory loss, or intellectual change. Many of the patients who initially complain of memory loss have anxiety or depression, and should be reassured about the problem. Other patients have more long-standing disease with cerebral involvement and do develop intellectual and personality alteration. Euphoria is less common than stated in the older literature, but is often dramatic in the patients who demonstrate it. Depression is common in patients with MS and is treated with tricyclic drugs quite effectively. In the patients who do develop some intellectual change, late in the disease, we have not been able to alter this pattern, although reassurance, explanation and support for the family members if very important in appropriately coping with and managing the patient.

## ADVICE

MS patients require long term support, advice and encouragement, and need to know they have a medical advisor who will be available when problems arise. They come to know and understand the limitations of our current therapies but need the support of an understanding physician who can manage problems or help obtain the assistance of a consultant neurologist if this is deemed necessary. Many feel abandoned by the medical profession as patients often say "my doctor tells me he doesn't know such about MS". What they want is only the feeling that the physician is interested in them and their problem and will help and learn and suffer along with them.

## PHYSIOTHERAPY

Most patients do not require physiotherapy but those who have spasticity, ataxia, gait difficulty and other disability should be assessed to see if a course of therapy would be helpful.

**Continued on page 17.**

# Treatment of Depression in Parkinson's Disease

Douglas Maynes,* B.Sc., M.D., F.R.C.P.(C) and
Patrick Flynn,** M.D., F.R.C.P.(C), F.A.C.P.,

*Halifax, N.S.*

**Here, where men sit and hear each other groan,**
**Where palsy shakes a few sad last gray hairs,**
**Where but to think is to be full of sorrow,**
**And leaden-eyed despairs.**

***Ode to a Nightingale***
**by John Keats**

Depression is a common symptom in patients with P.D. (Parkinson's Disease). James Parkinson described the disease that bears his name in his paper, *An Essay on the Shaking Palsy,* published in 1817. Although he wrote that the "senses and intellect were uninjured", he described the patients as "unhappy sufferers", "dejected", and feeling "melancholy" and noted their "wish for the release of death". It remained unclear for almost a century as to what extent mental and emotional disturbances were involved in this neurologic disease. French psychiatrists, writing at the end of the 19th century, first began to recognize and emphasize that the mental symptoms, including depression, of "paralysis agitans" were significant.

There has been considerable debate among authors as to whether the depression seen in P.D. is a reactive, secondary phenomenon or whether it is truly an inherent part of the disease itself. Many of the early investigations failed to use precise clinical criteria and as a result, the frequency, etiology and characteristics of depression in P.D. are still not well understood.

## CLINICAL CONSIDERATIONS

There has been wide variability in the reported frequency of depressive symptoms in parkinsonian patients. (Table I).

Warburton evaluated 140 patients referred for thalamotomy and found depression in 56% of the male parkinsonian patients and 71% of the females compared with 23% in the male controls and only 47% in the female controls.[3] In another study, Celesia and Wanamaker found the prevalence of depressive symptoms at 37% with a higher prevalence of depression in female patients. These authors found a lack of relationship between depressive symptoms and the duration and severity of the parkinsonian illness.[4]

**TABLE I**

**DEPRESSIVE SYMPTOMS IN P.D.[3]**

| Investigator | No. of Patients | % Depressed | Controls |
|---|---|---|---|
| Warburton | 140 | 60 | Medical, surgical, gynecological disorders |
| Mindham | 89 | 90 | Psychiatric in-patients |
| Celesia and Wanamaker | 153 | 37 | None |
| Brown and Wilson | 111 | 52 | None |
| Horn | 24 | ?* | "Normals" Paraplegics |
| Robins | 45 | ?* | "Normals" hemiplegics spinal cord disorders, orthopedic problems |
| Lieberman *et al.* | 352 | 30 | None |
| Mayeux *et al.* | 55 | 47 | Spouses |

*No figures reported.

Robins matched 45 patients with parkinsonism with 45 chronically disabled control patients with a significantly more severe grade of physical handicap. It was found, using the Hamilton Rating Scale, that those with P.D. were very significantly more depressed than the control group. It was concluded that patients with P.D. suffer a degree of depression which cannot be solely a reaction to the stress of the physical disability.[1] This study confirms the findings of other studies which question the long-held assumption that the depressed mood in P.D. is "reactive" to the physical limitations imposed by the disease.

Mayeux evaluated 55 Parkinsonian patients without dementia and 31 of their spouses. He found depression in 47% of the former group and 12% of the latter. He concluded that the depression in P.D. may be accompanied by mild intellectual impairment and inattention which is independent of the severity of the illness.[2] It is unclear at present whether or not neurotransmitter abnormalities play a significant role in depression in P.D. Current speculation is that degenerative changes in the brainstem and hypothalamus with resultant loss of central monoamines produces or predisposes an individual to depression in this disorder.

*Staff Psychiatrist, Nova Scotia Hospital, Dartmouth, N.S. Lecturer, Department of Psychiatry, Dalhousie Medical School, Halifax, N.S.

**Professor, Department of Psychiatry, Dalhousie Medical School.

Neuropsychiatric complications, especially depression, are important limiting factors in using most antiparkinsonian drugs in elderly and demented patients. Levodopa therapy, for instance, can cause syndromes that include depression, mania, vivid dreams, delirium, hypersexuality and, at times, frank psychosis.[28 32] Often reduction of levodopa is necessary for treatment. Bromocriptine, a dopamine agonist, has been noted to cause visual hallucinations, confusion, paranoid delusions and mania.[28 30]

It is often difficult to differentiate clearly depression from P.D., in the same patient, as the two conditions have some signs and symptoms that are similar. These include anergia, motor retardation, blank facies, social withdrawal and autonomic dysfunction. The clinical characteristics of depression in P.D. include the following: the affective change is of mild to moderate intensity, with suicide uncommon; the depression is inconsistently related to the severity of disease, age, or sex of the patient; the mood change develops just after the onset of P.D. in 15 to 25% of patients; associated intellectual changes including inattention, impairment in memory or dyscalculia may be present.[3]

## MANAGEMENT OF DEPRESSION

The antidepressants which have been in regular and successful use in psychiatry for over 25 years, are the specific therapy for the depression in P.D. However, specific therapy must be accompanied by general measures to keep the patient active for as long as possible; e.g., physiotherapy or the use of simple mechanical aids. Psychosocial aspects often need attention, especially since the degree of physical disability may be considerably worsened by the stress of concurrent depression.[26] These patients often require a great deal of encouragement and support from physicians and family members. Individual psychotherapy may be required on occasion for certain patients.

## TRICYCLIC ANTIDEPRESSANTS

Tricyclic antidepressants, still the most commonly used family of antidepressants, have been found useful in the treatment of depression in P.D. Placebo-controlled trials of imipramine, desipramine, and nortriptyline report that bradykinesia and rigidity, as well as depression, improve with imipramine and desipramine whereas only depression is relieved with nortriptyline.[5] Prior to the L-DOPA era, a number of studies of tricyclic antidepressants given to patients with P.D. reported a positive effect on the neurological signs as well as depression in both controlled and non-controlled studies. The effects were especially noted in akinesia and rigidity whereas tremor had a tendency to worsen.[5] It has been demonstrated that levodopa and tricyclic antidespressants can be used effectively together.[3]

## NEWER ANTIDEPRESSANTS

Bupropion (Wellbutrin-Burroughs Wellcome) is a "new generation" antidepressant that is pharmacologically and biochemically distinct from the tricyclic and momoamine oxidase inhibiting antidepressants. It is available in Europe and the United States but not yet available for prescription in Canada. It is a member of a new antidepressant class, the chloropropiophenones, and possesses weak dopamine reuptake blocking effects. Bupropion is distinguished from the tricyclics by its relative lack of cardiotoxic and anticholinergic effects and it has been found especially useful in geriatric patients and in patients with concomitant medical illness including P.D.[29]

Trazodone (Desyrel®), a second "new generation" antidepressant, differs from other antidepressants in its structural, biochemical, pharmacological and toxicological profile. It is a triazolopyridine derivative and has some important and unusual effects on serotonin kinetics. At low doses, it acts as a serotonin antagonist. At the higher doses used for depressions, it acts as a serotonin agonist. The latter effect is probably due to its ability to selectively inhibit serotonin reuptake.[15] In controlled trials with other antidepressants, trazodone caused little or no sedation, produced infrequent anticholinergic side effects, and evoked few adverse cardiovascular effects.[15] Since trazodone has antidepressant effects in man and documented anti-tremor effects in animals, some clinicians have given it to depressed and non-depressed parkinsonian patients and to patients with L-DOPA dyskinesia and tardive dyskinesia. The reports indicate that trazodone has a good antidepressant effect but also has a "moderate therapeutic action" (reduction in amplitude of tremor and/or dyskinesia of about 50%) within a short period of time (an average of 3 days) on parkinsonian tremor.[15] Because of its low anticholinergic activity and specific antitremor activity, trazodone has been recommended as being most useful in the treatment of depressed parkinsonian patients.[10]

## MONOAMINE OXIDASE INHIBITORS

Prior to the availability of L-DOPA, Tranylcypromine (Parnate) was used in the treatment of P.D.[31]It was found to improve rigidity and tremor as well as depression. Unfortunately, M.A.O. inhibitors are dangerous when used in conjunction with L-DOPA. However, more recently, a new selective MAO inhibitor, Deprenyl, has been developed. This drug selectively inhibits the beta type of monoamine oxidase and has little effect on the alpha type. Accordingly it may be compatible with foods containing tyramine including cheese and with drugs like Levodopa.[17]

Studies describing the successful use of Deprenyl and L-DOPA together have been reported.[16,17] In one study, the addition of L-deprenyl to L-DOPA taken as a single oral dose resulted in a statistically

significant reduction in patients' functional disability on average within 60 minutes and lasting for one to three days.[16] No doubt in the future, deprenyl will have a more significant role to play in the treatment of depression in P.D.

## OTHER BIOLOGICAL TREATMENTS

Lithium carbonate has been used in P.D. in the treatment of the "on-off" syndrome. This is a severe movement disorder characterized by alternating states of akinesia and choreoathetoid dyskinesia. The syndrome occurs in 11 to 50% of patients with P.D. who have received L-DOPA therapy for more than 2 years and it is thought to represent the progression of the underlying disease complicated by an acquired alteration in responsiveness to the drug. The pathophysiology of the "on-off" phenomenon is not known, although several mechanisms have been proposed including the fluctuation in the relative sensitivity of straital dopamine receptors.[23] Although lithium may have some value in treating certain side effects of L-DOPA, uncontrolled clinical reports show uncertain results in preventing or attenuating L-DOPA induced psychotic, manic or dyskinetic behavior.[25]

Lithium is an effective antidepressant in a small number of selected patients. It has been found useful in treating the depressive phase of some bi-polar affective disorders as well as a subgroup of acutely depressed unipolar patients.[25] Thus lithium may be useful in certain depressed P.D. patients who have movement disorders secondary to L-DOPA treatment. At the present time, however, it is not considered a drug of first choice.

E.C.T. has been used effectively for many years in the treatment of affective disorders in P.D.[20, 22] Although the number of patients treated has been small, it was found that not only did the symptoms of depression clear up after a short course of treatment but also the parkinsonian features showed improvement. It was advocated by Lebensohn that this may be related to E.C.T.-induced changes in dopamine and norepinephrine metabolism.[22] In addition, E.C.T. has been used in the treatment of the "on-off" syndrome with mixed results.[18, 19] Some patients showed an improvement which lasted for several months after treatment. E.C.T. is not contraindicated in patients with P.D. and may be used safely and effectively in those patients with marked depressive delusions or suicidal ideation who do not respond to pharmacotherapy.

Methylphenidate has been used successfully in the treatment of depressed geriatric patients who were unable to tolerate tricyclic anti-depressants.[6] In a placebo-controlled, non-blind study, methylphenidate was significantly superior to the placebo in the treatment of 54 elderly patients suffering from depression.[7] Two studies have shown methylephenidate to be effective in treating secondary depression in patients in whom the affective signs and symptoms were superimposed upon an underlying dementia.[8, 9] In P.D. depression and dementia may occur together and methylphenidate may have a limited usefulness especially for certain elderly patients with mild depression.

## SUMMARY

Depression is very common in patients with P.D. with reported frequencies varying from 30-90%. The depression is usually mild to moderate in intensity and is inconsistently related to the severity of P.D., the age or the sex of the patient. Often there are associated intellectual changes with inattention and memory impairment. The depression may precede or develop just after the onset of P.D. in 15-25% of patients.

In the management of depression in this disease, general measures such as supportive therapy and relief of psychosocial stressors are important. Additional measures to maintain the patient's mobility such as physiotherapy and mechanical aids are also necessary when relevant. Individual psychotherapy may also be indicated.

The new antidepressant trazodone as well as the tricyclic antidepressants especially imipramine, desipramine and nortiptyline are considered most useful in treating depressed patients with P.D. Methylphenidate has been used in selected patients with milder depression.

Bupropion and especially the new MAO-B inhibitor, deprenyl, are recommended for use in countries where they are available.

Lithium and E.C.T. have both been used in P.D. but their effectiveness in treatment needs to be confirmed by further investigation.

Finally, progress in the treatment of depression in P.D. relies upon further understanding of the nature of depression particularly from the psychobiological point of view. □

### References


1. Robins AH. Depression in Patients with Parkinsonism. *Brit J Psychiatry* 1976; **128:** 141-145.
2. Mayeux R, Stern Y, Rosen J, Leventhal J. Depression, Intellectual Impairment and Parkinson Disease. *Neurology* 1981; **31:** 645-650.
3. Mayeux R, Williams JB, Stern Y, Cote L. Depression and Parkinson's Disease. *Advances in Neurology* 1984; **40:** 241-250.
4. Celesia G, Wanamaker W. Psychiatric Disturbances in Parkinson's Disease. *Diseases Nervous System* 1972; **33:** 577-582.
5. Andersen J, Aabro E, Gulmann N, Hjelmsted A, Pedersen HE. Antidepressant Treatment in Parkinson's Disease. *Acta Neurol Scandinav* 1980; **62:** 210-219.

6. Katon W, Raskind M. Treatment of Depression in the Medically Ill Elderly with Methylphenidate. *Am J Psychiatry* 1980; **137:** 8:963-965.

7. Jacobson A. The use of Ritalin in psychotherapy of depression of the elderly. *Psychiatric Quarterly* 1958; **32:** 475-483.

8. Holiday AR, Joffe JR. A controlled evaluation of protriptyline compared to placebo and to methylphenidate hydrochloride. *J New Drugs* 1965; **5:** 257.

9. Kaplitz SE. Withdrawn, Apathetic geriatric patients responsive to methylphenidate. *J Am Geriatr Soc* 1975; **23:** 271-276.

10. Ananth J. Choosing The Right Antidepressant. *Psychiatric J Univ Ottawa* 1983; **8:** 1:20-26.

Teychenne PF, Park DM, Findley LJ, Rose FC, Calne DB. Nomifensine in Parkinsonism. *J Neurol Neurosurg Psychiatry* 1976; **39:** 1219-1221.

Park DM, Findley LJ, Hanks G, Sandler M. Nomifensine: Effect in Parkinsonian Patients not Receiving Levodopa. *J Neurol Neurosurg Psychiatry* 1981; **44:** 352-354.

Bedard P, Parkes JD, Marsden CD. Nomifensine in Parkinson's Disease. *Br J Clin Pharmac* 1977; **4:** 187S-190S.

Stonier PD, Wittels PY. *Clinical Profile of Nomifensine:* Royal Society of Medicine International Congress and Symposium Series No. 25, published by Academic Press Inc (London) Ltd and the Royal Society of Medicine.

15. Ayd FJ Jr. Trazodone: A Unique New Broad Spectrum Antidepressant. *International Drug Therapy Newsletter* 1979; **14;** Nos. 9 and 10: Nov. and Dec.

16. Birkmayer W, Riederer P, Ambiozi L, Youdim MBH. Implications of Combined Treatment with "Madopar" and L-Deprenil in Parkinson's Disease. *The Lancet* 1977; **1:** 439-443.

17. Knoll J. Deprenyl — The MAO Inhibitor without the "Cheese Effect". *TINS* 1979; May: 111-113.

18. Balldin J, Eden S, Granerus AK et al. Electroconvulsive Therapy in Parkinson's Syndrome with "On-Off" Phenomenon. *J. Neural Transmission* 1980; **47:** 11-21.

19. Ward C, Stern GM, Pratt RTC, McKenna P. Electroconvulsive Therapy in Parkinsonian Patients with the "On-Off" Syndrome. *J Neural Transmission* 1980; **49:** 133-135.

20. Yudofsky SC. Parkinson's Disease, Depression, and Electroconvulsive Therapy: A Clinical and Neurobiologic Synthesis. *Comprehensive Psychiatry* 1979; **20:** 579-581.

21. Wilder J. Parkinsonism, Depression and ECT. Letter to the editor. *Am J Psychiatry* 1975; **132:** 1083-1084.

22. Lebensohn ZM, Jenkins RB. Improvement of Parkinsonism in Depressed Patients Treated with ECT. *Am J Psychiatry* 1975; **132:** 283-285.

23. Ross DR, Coffey CE, Ferren EJ, Walker JI, Olanow CW. "On-Off" Syndrome Treated with Lithium Carbonate: A Case Report. *Am J Psychiatry* 1981; **138:** 1626-1627.

24. Coffey CE, Ross DR, Ferren EC, Sullivan JL, Olanow CW. Treatment of the "On-Off" Phenomenon in Parkinsonism with Lithium Carbonate. *Ann Neurol* 1982; **12:** 375-379.

25. Jefferson JW, Greist JH. *Primer of Lithium Therapy.* Baltimore: Williams and Wilkins 1977.

26. Lishman WA. *Organic Psychiatry: The Psychological Consequences of Cerebral Disorder.* London: Blackwell Scientific Publications 1978.

27. Kaplan HI, Freedman AM, Sadock BJ. *Comprehensive Textbook of Psychiatry Vol. 2.* Baltimore: Williams and Wilkins Company 1980.

28. Chater S, Montgomery P. Parkinson's Disease: A Pharmacological Update. *Can Fam Physician* 1985; **31:** 1505-1508.

29. Ayd FJ Jr. Bupropion: A Novel Antidepressant. *International Drug Therapy Newsletter* 1984; **19** (nos. 4 and 5; April and May.)

30. Janovic J. Long Term Use of Dopamine Agonists in Parkinson's Disease. *Clin Neuropharmacol* 1985; **8** (no. 2): 131-140.

31. Barbeau A, Duchastel Y. Tranylcypromine and the Extrapyramidal Syndromes. *Can Psychiat Assoc* (Special Supplement) 1962; **7:** 591-595.

32. Sweet RD, McDowell FH, Feigenson JS, Loranger AW, Goodell H. Mental symptoms in Parkinson's Disease during chronic treatment with levodopa. *Neurology* 1976; **26:** 305-310.

---

## THERAPY IN MULTIPLE SCHLEROSIS: A BRIEF OVERVIEW.

**Continued from page 13.**

### OCCUPATIONAL THERAPY

The patient with disability will often benefit from assessment, advice and treatment by an occupational therapist and we have found this an important but often neglected aspect of the management of MS patients.

### CONCLUSION

MS is demyelinating central nervous system disease with a variable pattern and variable course. Although there is an exciting atmosphers of discovery in MS research, only limited advances have been accepted for widespread use in the treatment of patients. This brief review outlines the approaches to treatment used in the Dalhousie MS Research Unit. Some may say we are conservative, and we would agree. We use only therapies that are accepted, or that we are studying because they look promising. We cannot assess, nor do we think it is kind to administer, every new theapy that arises in MS as we hear dozens of these each year.

We do feel optimistic about the research into new treatments in MS but recognize the limitations of the current ones and look forward to more helpful advances and new ideas in the next few years. □

---

"What is above all needed is to let the meaning choose the word, and not the other way round. . . the worst thing that one can do is to surrender to them."

*Politics and the English Language*
George Orwell (Eric Blair) (1903-1950)

# Non-Invasive Vascular Laboratory Who Needs It?

J.A. Sullivan,* M.D., F.R.C.S.(C) and
Stewart Francis,** B.Sc.,

*Halifax, N.S.*

**There have been many advances over the past ten years in the field of Non-Invasive Vascular Diagnosis. Many family physicians, however, remain confused as to what investigations, if any, are necessary in the routine care of vascular disease. There are several diagnostic tests available, some of which are very expensive and elaborate, others which are very simple and easily applied. Non-invasive investigations are not a substitute for arteriography nor are they a substitute for a good clinical history and physical.**

## INTRODUCTION

Over the past 10 years great advances have been made in the "Non-Invasive" diagnosis of vascular disease. This applies to not only peripheral vascular disease but also carotid disease, arterial disease of the upper extremeties, and also venous disease in both the upper and lower extremeties. This has led to a plethora of gadgetry on the market which often has led to confusion and indeed disinterest on the part of practitioners in trying to apply these modalities in the day to day management of their patients. The non-invasive vascular laboratory should now be an integral part of the investigation and management of patients with vascular disease. It has, like any other method of investigation and treatment, its limitations and its obvious strengths. It can be invaluable to the practitioner in both arriving at a correct diagnosis and in the ongoing follow-up of vascular disease. The following is presented in an attempt to clarify the place of the modern day vascular lab in the day to day management of patients.

## EQUIPMENT

The vascular laboratory can be as simple as a normal blood pressure cuff and a small portable office doppler or as complicated as the most sophisticated micro processor for computerized interpretation of a myriad of vascular signals. The former is cheap, uncomplicated and very easily applied to a busy office practice, the latter is more suitably left to investigational labs. The average clinical non-invasive vascular laboratory should, of course, be somewhere in between the two extremes. It should provide the referring physician with the capability of performing, on a routine basis, non-invasive vascular study of at least three disorders:

a) peripheral vascular disease of both upper and lower extremeties;

b) cartoid and vertebrobasilar disease; and

c) venous thrombosis and venous disease of both the upper and lower extremeties.

## PERIPHERAL VASCULAR DISEASE

Atherosclerosis involving the lower limb is a common problem and is frequently seen and has to be dealt with in clinical practice. The elderly patient who presents with a 10-year history of claudication which has now progressed to rest pain, night pain and peripheral gangrene with no palpable pulses does not present a diagnostic dilemma. Not all cases of peripheral vascular disease, however, are so straight forward. Here in lies one of the real benefits of non-invasive diagnosis i.e. the patient who presents with leg or calf pain which is atypical and which may represent anything from osteoarthritic disease of the hip or knee to simple varicose veins or chronic venous insufficiency. This patient may or may not be felt to have normal peripheral pulses and indeed what one physician feels is a 2+ pulse another may feel is a 4+ and the diagnosis becomes very subjective. The vascular laboratory in this context can not only aid in the diagnosis or exclusion of arterial disease but it can actually assess the degree of involvement and provide the physician with an objective measurement of the patient's disability and thus allow him to plan further follow-up and management.

Another case in point is the elderly patient who presents with a history of low back pain and is known to have lumbar disc disease and coexisting peripheral vascular disease. These patients, with time, may develop increasing symptoms which one finds difficult to sort out as to their musculoskeletal verses peripheral vascular components. The non-invasive laboratory again, in this context, will provide objective data as to the degree of vascular involvement and allow one to follow this over time and thus more clearly delineate exactly which symptoms represent which disease process.

*Assistant Professor of Surgery, Dalhousie University and Director, Non-Invasive Vascular Laboratory, Victoria General Hospital. Telephone (902) 428-3792

**Chief Technican, Non-Invasive Vascular Laboratory, Victoria General Hospital, Halifax, N.S.

## METHODS

The standard tests in investigation of patients with peripheral arterial disease have recently been discussed in this *Bulletin* by Dr. Del Campo. These, in essence, are the measurement of segmental blood pressures combined with the pulse volume recordings in the standard vascular lab setting. The segmental blood pressures are simply done by applying a blood pressure cuff to the thigh above knee, below knee and ankle regions. The cuffs are inflated to occlude arterial pressure and the doppler probe is placed at the ankle while the cuff is deflated and one arrives at a pressure reading at the various levels when the cuffs are deflated (Fig. 1). Gradients greater than 30 mm of mercury are considered as indicative of arterial stenosis.

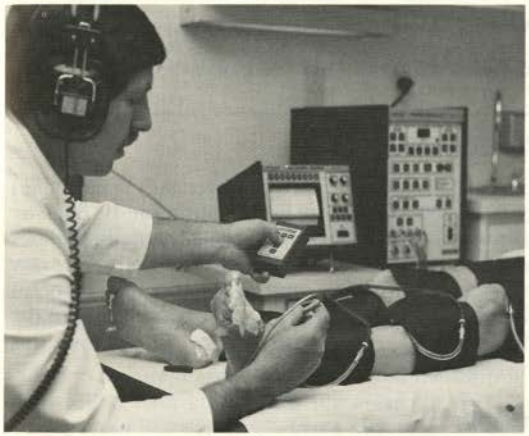


Figure 1.

In some patients such as diabetics where vessels may be heavily calcified segmental blood pressures may be falsely high and misleading. It is in these patients where pulse volume recordings may add to diagnostic accuracy. The pulse volume recording is simply done by inflating the pressure cuff to above venous occlusion pressure. The cuff is then attached to some kind of pressure transducer and the actual displacement of the cuff with each arterial cycle is transmitted into the pressure transducer and, depending on the way it is processed, can be traced on a strip chart or an oscilloscope. The tracing could be interpreted as being in keeping with mild, moderate or severe stenosis (Fig. 2). Patients with normal values at rest are exercised on the treadmill at a fixed incline and rate until symptoms occur, or to some other specified end point. The above parameters are repeated and again gradients are looked for. Ankle brachial indicies are calculated with $< 1.0$ indicating mild disease and, of course, less than .5 indicating severe peripheral arterial disease.

## CAROTID OCCLUSIVE DISEASE

Classical TIA's or frank strokes with hemispheric distribution are generally not difficult diagnostic or therapeutic problems. The practitioner, however, is frequently faced in his practice with a patient whose symptom complex is not clear cut but he finds himself reluctant to subject such patients to the morbidity of invasive angiographic investigation. Non-invasive investigation, via carotid doppler studies, can provide the physician with valuable data in either excluding significant carotid disease or in pursuing more invasive diagnostic modalities. Again, many methods of investigation have been studied and are available but essentially the most reproducable and most easily applied is carotid spectral analysis.

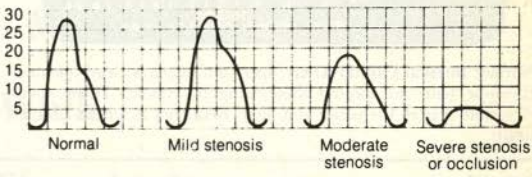
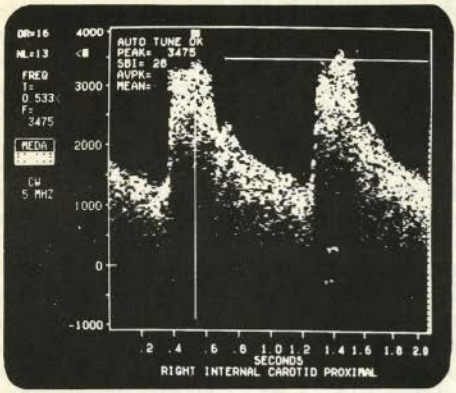


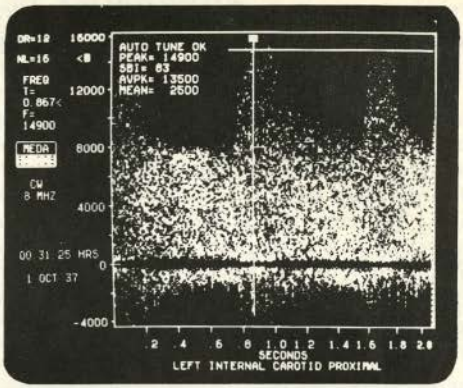

Figure 2.

Carotid spectral analysis again very simply involves a different method of interpreting a doppler signal. A small doppler probe is placed over the carotid vessels in the neck. This signal is fed into a micro-processor and the velocity pattern of flow in the vessel is displayed on an oscilloscope. Stenoses basically produces two phenomena: one being an increased velocity in the vessel and the other being turbulance which can be measured in different degrees of intensity on the oscilloscope, this phenomenon being referred to as "spectral broadening". A classical pattern of normal and severe internal carotid stenosis is shown in (Fig. 3). This method of investigation should provide the physician with greater than 95% accuracy in the diagnosis of significant carotid stenosis. That is carotid stenosis representing greater than 60% diameter reduction in the vessel lumen. It should not be expected to diagnose non-critical disease nor rule out ulcerated plaque as a source of emboli. These, however, are not accurately diagnosed even by present invasive methodology and the decision to proceed with surgical intervention in the face of less than significant stenosis is based more on the patient's clinical course.

Other investigations such as ocular pneumoplethysmography are widely used in some labs. This provides accurate data with regards to decreased pressure in the distal internal carotid artery but must be combined with some other non-invasive investigation such as spectral analysis in order to localize the disease for surgical management. Newer equipment such as duplex scanning, which combines ultrasound visualization of the vessel combined with doppler signal, is being used but to date its reproducability has not been as universal or as simple as continuous wave doppler study. None of the above modalities should




**Normal Internal Carotid Artery** Spectrum displays clearly-defined envelope with no spectral broadening in systole. There is no elevation of peak frequency and mode frequency is within normal limits.




**Abnormal Internal Carotid Artery** Spectra displays poorly defined envelope with severe spectral broadening. There is as extremely elevated peak frequency with decreased mode frequency. A bruit is clearly seen through diastole.

**Figure 3.**

be considered as substitutes for good angiographic studies. These are ancillary investigational methods which should be used in arriving at the correct diagnosis and aiding the follow-up of patients with extracranial vascular disease. Patients considered for surgical correction of the disease still require four vessel complete angiography prior to any surgical intervention.

## VENOUS DISEASE

Superficial varicose veins are, of course, seen quite commonly in clinical practice and do not represent a diagnostic problem. One is frequently faced with patients with symptoms and findings, especially in the lower extremeties, which often lead to confusion and uncertainty in the diagnosis of acute deep venous thrombosis. The gold standard for diagnosis of thrombi in the lower extremeties continues to be venography. It, however, does carry the risk of morbidity, including venous thrombosis as well as the remote possibility of fatality from anaphlactic reactions.

Non-invasive techniques have been developed in the study of venous disease. Several techniques involving outflow plethysmography and complicated techniques of filling times have been devised. The simplest and most mobile form of non-invasive venous study, however, remains the doppler study. This involves the placing of a doppler probe over several venous sites in the extremity and the audible interpretation of the venous signal. Once the technician has been trained in the interpretation of the normal and abnormal venous signals one should be able to accurately diagnose the presence of venous thrombosis in the thigh and above with an accuracy well over 90%. Accuracy using this technique for diagnosing venous thrombosis below the knee, however, falls to the region of 80 to 85%. The entire examination can be done by a trained technician in a maximum of about 10 minutes and it can be done at the bedside or in the recovery room and requires no equipment other than a pocket doppler stethescope. It is also an excellent tool in the diagnosis of venous insufficiency and valvular incompetence.

## SUMMARY

The non-invasive vascular laboratory is not a substitute for a good history and physical examination nor is it to replace conventional angiography when indicated. Patients who have indications for surgery still must undergo arteriography in order to define their anatomy. Not every patient, however, with symptoms requires arteriography. The indications for surgery especially with peripheral vascular disease still remain relative in patients with claudication. The patient with stable claudication does not require invasive arteriography unless surgical correction is being considered. Indeed, some may argue that these patients do not need non-invasive studies either.

The non-invasive studies, however, provide an objective yardstick in the assessment of these patients on a continuing basis although the patient's symptomotology may be getting worse it does not necessarily follow that his vascular disease is. The non-invasive objective parameter as described above can provide the physician with the necessary information to sort out this problem. Arteriography, on the other hand, provides only an anatomical road map. A patient with a 70% stenosis in the vessel now may have the identical 70% stenosis a year from now but hemodynamically may be much improved because of collateralization. This indeed, should be manifested in his non-invasive

studies and not necessarily on his arteriography. The physician must then think of vascular disease as an ongoing dynamic process rather than an inanimate anatomical disease.

Other areas where the laboratory is of extreme value is in following patients with vascular grafts, in diagnosing stenoses in these grafts before obvious complete occlusion occurs. It can also be used in the follow-up of carotid endarterectomies and in the follow-up response of the patient to treatment for deep venous thrombosis. The Lab should provide the physician with objective hemodynamic data which is easily reproducable with no morbidity or risk of mortality to the patient being studied. It should not be considered as a substitute for good clinical history taking and examination nor should it be considered a substitute for arteriography when indicated. □

# Your Time is My Time
## or
# Wait A While
## or
# Murderous Thoughts*

The remnants of a pimple, about 8" below my right armpit had become a small cyst.

I made an appointment with a physician for a quick "look-see" so that he could book me for the removal of the cyst. His receptionist gave me a 1:45 appointment and so to be sure, I arrived at 1:25 to find the waiting room full. I have known this physician for over 20 years, having first met him when I was on active staff at the Children's Hospital. My schedule was heavy for that afternoon, but "how long does it take for a quick peek"?

At 2:30 I became pretty restless and told the receptionist I had run out of time. She said: "He will see you now.". In I went, complained about the long wait and time wasted — and he said: "Why didn't you tell me you were here?".

I estimate that 80,000 of my neurones blasted out of existence at that moment. I said: "Thank you!" and left. Time: 3 minutes maximum.Time wasted — over one hour!

About a week later, my wife got a call. "Tell him to be at the Dixon Centre, V. G. Hospital, at 10:45 next Monday morning." It took me 15 minutes of waiting for a parking spot, so we cannot count that. Let us start counting at 10:26 when I checked in and was told to go to the waiting room with the green chairs — the place was loaded.

I had told my secretary that I would be back at 11:15 or 11:30 as I was expecting some important calls. At 12 o'clock, I went to a pay phone and called in to say that I would be delayed. I then spoke to the Physician's nurse and told her that my appointment was for 10:45. "Oh — the Doctor has been behind schedule all morning — there are only 5 people ahead of you!"

"Should I go home for a quick lunch and return?" (I live only a few minutes away from the hospital and could have been back by 12:30.) "Oh no — it will be quite soon."

At 12:30, she called me into a small surgery room. "Please sign the consent form!" She disappeared, after saying: "He will fix the spot on your face soon.".

At 12:45, in came Dr. Busy — "It's been a bad morning at Black Rock! Ha, ha, ha!" A quick shot of Xylocaine, reach for the Scalpel, I felt the sharp pain — but was quite amenable to it as I was not prepared to wait any longer — a couple of sutures and a band-aid. "All done." "See you next Monday to remove the sutures."

I am writing this to prevent a stroke or coronary — as I am still boiling. Is my time so "cheap" to him that the courtesy of a phone call before 10:00 a.m. by his recepionist that "he's way-behind schedule and please come at 12:00 or 12:15" could not be made?

I practised for 32 years and more than 95% of my patients did not have to wait more than 5 minutes. I remember hearing of a dentist who kept an executive waiting over an hour for his appointment and received a bill for $200 for the executive's time.

But that is not all. What about the total time of 10 or 15 or more people — wasted — spoiled — shot! Too many medical offices tell *all* their patients to come at 1:30 or 2:00 p.m. which is an absolute lack of concern and respect.

That is all I have to say today. □

*The Author is a former Pediatric Dentist.

# Non-Invasive Evaluation of Venous Diseases:
## Guidelines for Physicians

Carlos Del Campo,* M.D., F.R.C.S.(C),

*Halifax, N.S.*

Deep vein thrombosis (DVT) may lead to pulmonary embolization and the disabling postphlebitic syndrome. The diseases plus the more common but less disabling problem of varicose veins are as frequent, if not more than arterial disease.

Clinical diagnosis of DVT is accurate in only 50 percent of the patients. In order to treat the disease and prevent complications the diagnosis must be established. Venography is the basic test for this purpose but it cannot be considered ideal because it is expensive, produces discomfort and has a minor risk, occasionally inducing venous thrombosis or allergic reactions.

Over the past two decades, several noninvasive diagnostic techniques have been developed, and have become a standard diagnostic approach to the patient with venous disorders. These techniques evaluate accurately both acute and chronic venous diseases reserving venography for more selected cases. They have also the advantage that they can be used for screening at follow-up intervals, without risk or discomfort for the patient.

### DOPPLER ULTRASOUND

A beam of ultrasound is emitted from a hand-held probe, and is reflected by the circulating blood cells; this reflection is detected by a second crystal in the probe and then transformed into an audible signal or a recordable waveform. Most superficial and deep veins of upper and lower extremities and neck are accessible for this examination.

For evaluation of the leg's deep system, the probe is placed first at the level of the femoral vein in the groin, and the side suspected to be normal should be examined first. The normal sound produced by blood flowing through a patent vein resembles a windstorm, but this sound will be absent in the presence of total venous obstruction. It must be remembered to maintain the patient in the supine position with the head of the bed elevated, with both legs slightly flexed at the hip and knees and elevated approximately 10°. The calves should be free of pressure to allow the soleal plexuses to fill properly and to avoid false positive results.

*Thoracic and Cardiovascular Surgery, Halifax Infirmary, Halifax, N.S.

**Correspondence:** C. Del Campo, M.D., F.R.C.S.(C), Room 413, Gerard Hall, 5303 Morris Street, Halifax, N.S., B3J 1B6.

The venous windstorm-like sound should be phasic with respiration and it ceases during a deep breath or valsalva maneuver. Application of sudden pressure in the thigh will produce a gunshot-like sound, and squeezing of the calf muscles will do the same but with an easily audible split second delay. When this response is missing at one level or another, the location of the venous obstruction can be determined. To reafirm the diagnosis the probe is then placed at the origin of the posterior tibial vein, posterior to the medial malleolus. When the calf muscles are compressed the flow will stop and resume rapidly upon release. Continuous flow can be heard in veins or collaterals distal to venous obstruction. These tests are essential in every patient in which DVT is suspected. In experienced hands this test has a 98 percent accuracy when compared with venography.

Competence or incompetence of venous valves can be assessed by placing the probe at the occurrence of the perforating veins in the medial aspect of the leg, and manual compression will produce audible retrograde venous flow. A tourniquet can be applied to prevent reverse flow in the superficial veins, and compression proximal to it will make the reverse flow more obvious and will differentiate from reflux from incompetent superficial veins.

These techniques can also be applied to the much less common venous disease of the upper extremity or internal jugular vein.

### STRAIN GAUGE PLETHYSMOGRAPHY (SPG)

Two separate tests are of remarkable value in the assessmentof venous incompetence of the lower extremity: 1. Venous Outflow Plethysmography; and 2. Venous Reflux Plethysmography.

#### 1. Venous Outflow Plethysmography

Plethysmography is the recording of changes in volume in a given extremity with each heart beat or during occlusion of venous return. The SPG transducers (this is the method of choice in our laboratory) are placed around the calves, and pneumatic cuffs are inflated in each thigh to occlude venous return. The increase in a calf circumference is then recorded. After two minutes the cuffs are deflated. The rate of decrease in calf volume is recorded on calibrated paper and measured. The results are reported in mm/min/100 grams of tissue. The incremental volume (capacitance) is then plotted against time in seconds. Venous

obstruction produces low values for volume increase and outflow. Extremities can be compared with one another.

2. **Venous Reflux Plethysmography**

The combination of SPG and a photoplethysmograph (PPG) provide the most accurate physiological data for chronic venous insufficiency. The patient is tested in the sitting position, while contracting the calf muscles. Patients with incompetent veins fail to relieve the venous hypertension and therefore have an increased volume.

The PPG records changes in skin blood content. The transducer is applied to the skin and the calf muscles exercised with and without a tourniquet. The recorded curve shows the speed of the volume increase. This test differentiates normal patients from those with primary or secondary varicose veins. Placing the transducer over the perforating veins (PPG) will provide additional information of reflux.

## SUMMARY

Venous diseases of the lower extremity are as common as arterial disorders. They produce a variety of symptoms ranging from benign disorders like varicose veins to the disabling postphlebitic syndrome and even death (i.e., pulmonary embolus). Clinical diagnosis has an incidence of 50 percent false positive or false negative results. Noninvasive vascular laboratory techniques are safe, inoccuous, relatively inexpensive and very accurate. These techniques not only provide objective information but also physiological data at rest and during exercise, screening and follow-up can be easily accomplished without risk to the patient. Venography can be avoided in a significant number of patients. Venography can be reserved for patients with suspected DVT but with inconclusive results, patients with suspected ileofemoral thrombosis, and for patients in which venous reconstruction for sequelae of postphlebitic syndrome is contemplated. □

**Extensive bibliography available from the author.**



# Neurodegenerative Disease and Previous Human Pituitary — Derived Growth Hormone Therapy

Growth hormone (GH) therapy was suspended in Canada in April 1985 following the report of rapidly progressive and fatal neurological disease in four young adults (three in USA and one in Great Britain aged 20-34 years) who had received human pituitary-derived GH during childhood. Creutzfeldt-Jakob disease (CJD) was confirmed at autopsy in three of these cases. Approximately 27,000 persons have received human growth hormone around the world over the past 20 years. In the general population, CJD occurs with a frequency of approximately one per million persons per year. The frequency of CJD in the GH deficient patient is unknown. It is established that CJD rarely occurs in persons less than 30 years of age. Thus the circumstantial evidence raises a question whether CJD in these four patients may have been related to previous GH therapy. Large-scale epidemiological studies are now underway in many countries to monitor this potential public health problem.

There has been no unexplained death or rapidly progressive neurodegenerative disease observed in Canada in persons treated with GH. Approximately 800 GH deficient patients have received GH in Canada since 1965 under the aegis of the Medical Research Council of Canada (MRC). A followup programme with a central registry in Winnipeg, has been established for these patients. In order that this registry be as complete as possible, all physicians in Canada who are involved in the care of these patients should ensure that the patients or families have contacted the original physician (or replacement) who distributed the GH. Any questions regarding this programme or report of a death of a person known to have received GH should be directed to:

Dr. H.G. Friesen/Dr. H. Dean
Department of Physiology
Faculty of Medicine, University of Manitoba
770 Bannatyne Ave., Winnipeg, MB, R3E 0W3
Telephone: (204) 786-3696 □

# Gestational Trophoblastic Disease Registry

## 1983 ANNUAL REPORT

R.C. Fraser,* M.D., F.R.C.S.(C), I. Zayrid,** M.D., F.R.C.P.(C),
M. L. Givner,*** M.S.c., Ph.D., B. Pierce,† R.N.,

*Halifax, N.S.*

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

This year 45 new patients were registered. Thirty-five were diagnosed initially as "benign" hydatidiform molar pregnancies, three as choriocarcinoma, and seven as hydatidiform degeneration or "questionable mole".

Seven of the thirty-five patients originally diagnosed as "benign" molar pregnancies subsequently developed either N.M.G.T.D. (5) or M.G.T.D. (2). Of the three with choriocarcinoma, two had non-metastatic disease and the third metastatic disease at the time of registration.

**TABLE I**

**THE EXPERIENCE OF THE REGISTRY IN 1983**

| Total number of patients registered | |
|---|---|
| Benign Hydatidiform Mole | 28 |
| N.M.G.T.D. | 5 |
| M.G.T.D. | 2 |
| Choriocarcinoma | 3 |
| Questionable Mole | 7 |
| TOTAL | 45 |

The seven patients in the category of "questionable mole" were found to have HCG titres which returned to normal within 10 weeks or less. A total of 18 patients have been monitored since the study regarding the "questionable mole" began in 1981. All have had uneventful follow-up. This study continues and your support is appreciated.

### DIAGNOSIS: HYDATIDIFORM MOLE

Twenty-three ultrasounds were performed on 35 patients. Thirteen were positive, or at least suggestive of hydatidiform mole (3). The diagnosis was later confirmed histologically.

Nova Scotia Gestational Trophoblastic Disease Registry, Department of Obstetrics and Gynaecology, Dalhousie University, Halifax, Nova Scotia, Director, Division Gynaecology Oncology, **Professor of Pathology, Department of Pathology, D.J. MacKenzie Diagnostic Center, ***Director, Division of Endocrinology Department of Clinical Chemistry† Co-ordinator GTD Registry.

The Nova Scotia Registry is located on the 5th floor, Ambulatory Care Center, 5820 University Avenue, Halifax, Nova Scotia, B3H 1V7.

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

Ten patients, whose ultrasounds were reported as negative for hydatidiform mole, were subsequently found to have had a molar pregnancy.

Over the past several years a false negative rate of approximately 14% as been observed. As is suggested by the above, the 1983 false negative rate is over 40%. This may or may not reflect differences is the use of this diagnostic tool and the resulting interpretations.

**TABLE II**

| The geographical breakdown of patients registered | |
|---|---|
| Nova Scotia | 20 |
| New Brunswick | 13 |
| Prince Edward Island | 3 |
| Newfoundland | 9 |
| TOTAL | 45 |

With the exception of the 13 patients diagnosed by positive ultrasound, the remaining 22 patients were diagnosed by:

| | |
|---|---|
| — tissue examination at laparotomy | 1 |
| — tissue examination of products of conception at complete spontaneous abortion | 2 |

**or**

At D&Cs performed for the following:

| | |
|---|---|
| — incomplete abortion | 11 |
| — missed abortion | 7 |
| — spontaneous abortion | 1 |

### "BENIGN" MOLE SEQUELAE

Seven patients (20%) developed non-metastatic or metastatic disease and required adjunctive chemotherapy to completely irradicate their disease. The five patients with non-metastatic disease were successfully treated with 1 to 4 courses of single agent chemother-

apy. Two patients with metastatic disease (both to lung) required 3 and 4 courses of therapy with single agent chemotherapy (methotrexate with leucovorin rescue alternately with actinomycin D). All seven patients remain in remission.

## CHORIOCARCINOMA

Three patients with gestational trophoblastic disease were confirmed, at the time of diagnosis, to have choriocarcinoma. One patient who was diagnosed and treated out of province (choriocarcinoma post normal pregnancy) is presently in her third year of follow-up. She remains in remission. The remaining two patients with choriocarcinoma are presented in the following case studies.

### CASE STUDY #1 — N.M.G.T.D.

This patient presented with PV bleeding thought to be D.U.B. and a D&C was performed. The findings were normal endometrium. Approximately two weeks later she presented again with excessive uterine bleeding for which a hysterectomy was performed. Pathological analysis of the surgical specimen revealed abnormal tissue felt to be compatible with choriocarcinoma. (Three weeks prior to the original D&C the patient had had a positive pregnancy test. Three days before the D&C it had been "negative").

Investigations done in hospital included a CAT scan of the abdomen and pelvis which showed no evidence of metastases to liver or spleen; a chest x-ray which was normal; and routine blood work which was also within normal limits.

The patient subsequently received three courses of single agent chemotherapy. As the patient had a severe reaction to methotrexate, actinomycin D was used for her second and third treatments. She is in her second year of follow-up and remains in remission.

### CASE STUDY #2 — M.G.T.D.

This 37 year old female was referred to the Gestational Trophoblastic Disease Registry following diagnosis of a mediastinal mass compatible with a mediastinal teratoma. Thorocotomy performed revealed choriocarcinoma without other germ layers being present. Most recent pregnancy (seven years previously) had resulted in a missed abortion. The patient has one 12 year old child.

Immediate past history included investigation of possible ectopic pregnancy with EUA, laparoscopy, and D&C. At this time there was no evidence of any pregnancy or of pelvic pathology. However, her Beta Subunit HCG titre was elevated and a chest x-ray revealed the mediastinal mass.

Following surgery this patient subsequently received four courses of combined chemotherapy (methotrexate, actinomycin D, and cytoxin) to which she responded well. She is in her second year of follow-up and remains in remission.

**TABLE III**

**TOTAL EXPERIENCE**

| | 1965-1970 | 1971-1975 | 1976-1980 | 1981-83 |
|---|---|---|---|---|
| Nova Scotia | 13(4) | 27(8) | 108(13) | 47(11) |
| New Brunswick | | 8(6) | 31( 2) | 27( 6) |
| Prince Edward Island | | | 4( 0) | 8( 0) |
| Newfoundland | | | 15( 4) | 30( 5) |
| St Pierre | | | 1( 0) | |
| Benign | | | | 260 |
| NMGTD | | | | 42 |
| MGTD | | | | 17 |
| TOTAL | | | | 319 |

As noted above a total of 59 patients required adjunctive chemotherapy for non-metastatic or metastatic gestational trophoblastic disease. Fifty-six patients were treated successfully. Three patients with choriocarcinoma died of advanced disease unresponsive to treatment, or of complications related to the disease. All three were diagnosed following a normal pregnancy.

This year an attempt was made to contact these surviving 56 patients regarding their fertility status following chemotherapy. Forty-four patients were successfully contacted. Twelve were lost to follow-up. The findings appear in Table IV.

**TABLE IV**

| | |
|---|---|
| Number of patients with subsequent normal pregnancies | 12 |
| Currently pregnant | 5 |
| Currently in follow-up (not yet pregnant) | 9 |
| Hysts on T.L. unrelated to GTD | 15 |
| Hyst dueto drug resistant chorio | 1 |
| Hyst — Dx of Chorio made at time of surgery | 2 |
| TOTAL | 44 |

These findings suggest the continued fertility of women who have had non-metastatic or metastatic gestational trophoblastic disease (requiring adjunctive chemotherapy) and are in keeping with the findings of other centers.

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

**After hospital discharge:**

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

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

**If chemotheraphy was required then follow-up is as follows:**

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

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

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

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

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

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

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

d) Yearly thereafter.

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

**ACKNOWLEDGEMENTS**

We would like to take this opportunity to thank Dr. M. Givner, Director, Division of Endocrinology, Department of Clinical Chemistry, and his staff for their continued assistance and support. Likewise, our sincere thanks to the patients physicians, and pathologists for their interest and cooperation.

---

"Nothing is more repsonsible for the good old days than a bad memory."

Anonymous

# The Medical Society of Nova Scotia
## Policy Statement Re:
# Electronic Fetal Heart Rate Monitoring

The use of electronic fetal heart rate monitoring continues to be a controversial issue, and the value and potential problems this procedure may lead to is still a matter for debate. In 1981, at the request of the Medical Society, the Reproductive Care Program formed an Ad Hoc Committee, consisting of physicians and nurses from the province. Their task was to review the literature and formulate a policy statement for the use of electronic fetal heart rate monitoring in obstetrical units in Nova Scotia. A statement was prepared and approved by the Action Group and the Planning and Directing Group of the program. Subsequently the statement was sent to The Medical Society of Nova Scotia for approval and dissemination to physicians. Since 1982, the statement has been reviewed once by the Action Group of the Reproductive Care Program.

In 1985 the Ad Hoc Committee was re-activated at the request of the Medical Society, and the attached statement prepared. The Medical Society requested that particular emphasis be placed on antepartum monitoring. This item has been addressed in this review.

Electronic fetal monitoring (EFM) continues to be widely used in North American hospitals. However, much of the controversy surrounding the use of EFM continues with many centres continuing to conduct studies. Although the recommendations that follow are appropriate at this review time, an annual review by the Action Group of the Reproductive Care Program is recommended.

1. EFM is accurate in predicting the normal well oxygenated fetus during labour. However, abnormal Fetal Heart Rate (FHR) patterns are difficult to interpret and are frequently misleading. In most cases of abnormal heart rate patterns additional diagnostic measures, particularly fetal scalp blood sampling and pH estimations, are necessary. This is in addition to the overall clinical assessment of the patient, presence or absence of meconium, etc. Fetal scalp sampling requires extra training and facilities, and the results should be interpreted with caution.

2. There is still no proof but a good deal of circumstantial evidence to indicate that EFM in *low risk* pregnancies does not reduce perinatal mortality or morbidity, compared with good clinical and auscultatory assessment of fetal condition during labour. Good assessment is demonstrated by a 1:1 nurse/patient ratio throughout labour and auscultation of fetal heart every fifteen minutes in the first stage of labour, and with every other contraction in the second stage of labour. It is important that this auscultation is carried out continuously for approximately 30 seconds before, during and for 30 seconds after, the uterine contraction.

3. Some studies have shown that EFM plus fetal scalp sampling have contributed to a decrease in perinatal mortality in *high risk pregnancies.*

4. Clinical antenatal screening should detect 60 to 70 percent of high risk pregnancies. Therefore, 30 to 40 percent of high risk pregnancies first manifest themselves in labour. However, many of these, e.g. premature rupture of membranes, meconium staining, abnormal FHR on auscultation, hypertension, antepartum haemorrhage, etc. arise early enough in labour to allow transfer of the patient to a hospital where facilities for more exact monitoring and treatment in labour are available. Thus, although any hospital with an obstetrical service will have to deal with unpredictable high risk cases that arise in labour, the majority of these cases can still be safely transferred to an appropriate hospital. This will obviously depend on local geographical conditions.

5. Even in large tertiary care hospitals, the overall incidence of caesarean section for fetal distress is only one to two percent. Thus, in a hospital with 200-300 deliveries per year, fetal distress as an indication for caesarean section would only arise three to five times each year. Assuming that such hospitals manage fewer high risk cases, then this figure would be even lower, probably one to three cases each year.

6. Potential drawbacks to EFM at present include:

a) Reduced mobility of the patient in early labour. This can easily be overcome by common sense, i.e. allowing the patient to remain ambulant without EFM in early labour if all is well clinically.

b) Patient resistance, due to misunderstanding of the purpose of the monitor can be overcome by an explanation of the purposes of the monitor.

c) A possible rise in caesarean section rate. This is controversial. In units that have extensive experience with EFM allied to fetal scalp sampling, this has not been shown to occur. However, in units with a limited experience of EFM and that do not have the additional diagnostic aid of fetal scalp sampling, this may occur.

d) Infection. Again, this is controversial but the consensus seems to be that this is not a major factor. Fetal scalp absesses occur about once in 200-300 cases. These are usually of little significance and respond to local treatment. It is also theoretically possible to traumatize the fetus by inaccurate placement of the scalp electrode (e.g. in the eye). This is unlikely but more prone to happen with the inexperienced.

7. EFM also plays an increasing role in antepartum fetal assessment. This method, using the non-stress test (NST), is now a widely used antenatal test of fetal well being. The principles of this test are simple and safe. The application of EFM in antepartum fetal assessment is much easier and has far fewer pitfalls than the application of EFM for intrapartum fetal monitoring. However EFM is only an adjunct to clinical judgement and should not lull physicians into patterns of non referral of high risk cases.

## RECOMMENDATIONS FOR NOVA SCOTIA

1. Tertiary care hospitals should have available full facilities for EFM and fetal scalp blood sampling.

2. Regional hospitals should have facilities for EFM both antenatally and intrapartum. In addition, efforts should be made for all regional hospitals to have fetal scalp blood sampling facilities.

3. Community hospitals. There is a wide variation in the type of community hospital in the province, and in particular the number of deliveries they perform. Other factors such as the availability of other facilities and personnel and distance from regional centres, will dictate a certain amount of flexibility.In general, based on the lack of demonstrable beneficial effect in normal pregnancy and looking at possible drawbacks, we do not recommend EFM in labour in these hospitals. However in hospitals that have greater than 50 deliveries per annum and are more than 30 minutes away from a regional hospital, an antepartum fetal heart rate monitor for the purpose of doing NST may be appropriate for the patient convenience. In general, when an NST is carried out because a problem in the pregnancy is suspected, a telephone consultation with an obstetrician is desirable.

4. In the interest of economy if a monitor is purchased for a community hospital, it is recommended that antepartum fetal heart rate monitors be chosen due to the significantly lower cost.

5. Regarding fetal heart rate tracings, it is recommended that the guidelines outlined by the Society of Obstetricians and Gynaecologists of Canada in 1980 be followed.

a) All fetal heart rate records obtained from patient under hospital supervision be retained in total as a portion of the patient's medical record.
b) A statement summarizing the interpretation of the fetal heart rate record against a background of other clinical and/or laboratory information should be placed on the chart by a physician.

## EDUCATION

Providing adequate initial and ongoing training in order that physicians and nurses too can develop and maintain skill in intrapartum monitoring, presents a problem. One-day workshops in fetal monitoring are sometimes provided but this provides only the most basic skill and does not provide the more essential component that of ongoing education in the clinical setting.

Training for intrapartum monitoring which includes fetal scalp sampling, is a complex matter. It requires an initial intensive educational programme which should include practical as well as theoretical training.

However training for carrying out and interpreting a Non-Stress Test is simpler. An acceptable approach would be to train a cadre of physicians and nurses from each hospital who in turn could share their knowledge and expertise with their colleagues.

Any educational programme is expensive in both time and money to individuals and institutions. Nonetheless, if institutions wish to offer this service to their patients, the commitment must be made to buy the appropriate equipment, to have both physicians and nurses trained, and to ensure that expertise is maintained.

The question of who should provide the initial education needs to be addressed. In the past this has been done by Continuing Medical Education, the Grace Maternity Hospital, the Reproductive Care Program and one of the Fetal Monitoring companies. It would be beneficial to develop an organized approach. The way to ensure reaching a maximum number of people would be to offer educational programmes regionally. One possible option would be as part of the workshop programmes offered by the Royal College of Physicians and Surgeons.

## PAPER SPEED

Paper speed should be standardized throughout Nova Scotia 3 cm per minute. □

**January 18, 1986**

### References


Douvas SG, Meeks GR, Graves G, Walsh DA, Morrison, JC: Intrapartum fetal heart rate as a predictor of fetal distress and immediate neonatal condition in low-birthweight (less than or equal to 1,800 grams) infants. *Am J Obstet Gynecol* 1984; **148:** 300-302.

Placek PJ, Keppel KG, Taffel SM, Liss TL: Electronic fetal monitoring in relation to cesarian section delivery for live births and stillbirths in the U.S., 1980. *Public Health Rep* 1984; **99:** 173-183.

Wood C, Renou P, Oats J, Farrell E, Beischer N, Anderson I: A controlled trial of fetal heart rate monitoring in a low-risk obstetric population. *Am J Obstet Gynecol* 1981; **141:** 527-534.

Banta HD, Thacker SB: Policies toward medical technology: the case of electronic fetal monitoring. *Am J Public Health* 1979; **69:** 931-934.

Hobbins JC, Freeman R, Queenan JT: The fetal monitoring debate. *Obstet Gynecol* 1979; **54:** 103-109.

Parer J: Benefits and detriments of fetal heart rate monitoring. *Sem Perinatol* 1978; **2:** 113.

Haverkamp A, Orleans M, Langendoerfer S, McFee J, Murphy J, Thompson H: A controlled trial of the deferential effect of intrapartum fetal monitoring. *Am J Obstet Gynecol* 1979; **134:** 399.

Zuspan EP, Quilligan EJ, Iams JD, vanGeijn HP: Editorial. Predictors of intrapartum fetal distress: The Role of Electronic Foetal Monitoring. Report of the National Institute of Child Health and Human Development Consensus Development Task Force. *Am J Obstet Gynecol* 1979; **135:** 827.

# "No Resuscitation"

## Guidelines for Patients with Irreversible Illness*

### 1. IRREVERSIBILITY

A "no resuscitation" order should be considered only in the case of a patient whose condition (or damage done by that condition) is considered irreversible.

If there is any doubt concerning the irreversibility of the patient's condition a qualified second opinion should be obtained.

### 2. INITIATION

Discussion of "no resuscitation" may be initiated by the attending physician, a member of the house staff or nursing staff, the patient or his family, or others legitimately involved in the situation.

The persons involved in the discussion or treatment options and outcomes shall include the attending physician, house staff, head nurse or delegate, appropriate nursing staff, and other legitimately involved in the situation.

### 3. PROCESS

It must be recognized that although the ultimate responsibility for the "no resuscitation" decision rests with the attending physician, the ideal outcome is a consensus amongst those involved in the decision-making process.

If a patient is considered *competent,* i.e., capable of understanding the nature and effects of the proposed treatment, it is required that the attending physician (or his designate) will discuss with the patient:

a) treatment options (see notes)
b) the patient's wishes
c) the physician's recommendations

**NOTES:**

The decision for "no resuscitation" does not imply that all care will be withdrawn. General levels of care can be designated as follows:

1. All components of sophiscated medical care including emergency resuscitation.
2. Intensive care and advanced life support but excluding emergency resuscitation.
3. General medical care including antibiotics and other drugs, surgery, cancer chemotherapy, and artificial hydration and nutrition.
4. General nursing care and assurance of the patient's comfort, including pain relief and such hydration and nutrition as is dictated by the patient's thirst and comfort.

Unless in the opinion of the attending physician, the patient is emotionally incapable of participating in such discussions.

If the patient is incompetent, i.e., incapable of understanding the nature and effects of the proposed treatment, or is documented to be *emotionally* incapable of participating in such discussions, the above procedure is followed with the patient's legal guardian or next-of-kin.

It is advantageous for this discussion to take place in the presence of a member of the nursing staff whose primary role is to:

a) support the patient and his family
b) reinforce the information given by the physician

The family is informed of the decision by the attending physician or his designate unless the patient requests otherwise.

### 4. DOCUMENTATION

The "no resuscitation" order must be written on the order sheet by the staff physician or promptly co-signed by the attending staff physician if written by the resident. The reason for this clinical decision must be clearly documented with the order at the time the decision is made.

The relevant facts leading to this management decision must be documented on the progress notes by the attending staff physician or his designate.

Similarly, relevant facts, from a nursing perspective, must be documented on the nursing notes.

The head nurse or delegate must tag the progress note and transcribe the order from the physician's order sheet to the nursing Kardex.

### 5. REVIEW

The "no resuscitation" order must be re-assessed and re-written at intervals determined by the physician having regard to the patient's condition.

The order must be rescinded *immediately*.
a) upon request of the patient
b) in the case of the incompetent patient — upon the request of the next of kin.

The order must be re-assessed immediately if the patient's status changes in a way which would favourably affect the prognosis.

### 6. TRANSFER

When a patient with a "no resuscitation" status is transferred to another medical service, the transferring and receiving attending physician should discuss the existing decision. The new staff physician must assess the patient to review the order, preferably prior to transfer or, if this is impossible, atleast within 24 hours to transfer.

If the transfer is outside of regular working hours, the transferring service will maintain responsibiality for the resuscitation status of the patient until the new attending physician can assess the patient. □

*From the Department of Medicine, Victoria General Hospital, Halifax, N.S.

# Current Topics in Community Health

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

## LIFELONG LEARNING AND THE PROFESSIONAL

After a distressing episode with her local veterinarian, a farm neighbor observed:

> "He has a 1985 clinic — but he's running it with a 1946 diploma. I simply can't count on him to be up-to-date".

She went on to explain that while the doctor has a proclivity to collect the latest instruments and gadgets for his practice, he had not refurbished his intellectual kit of tools since graduation nearly four decades ago.

The professions are increasingly significant in the everyday life of each of us. Directly and indirectly, the various professional fields influence the quality, the character, and the security of daily living. They do this in major and pervasive ways. The very term *professional* connotes competence and evokes a sense of confidence. But society can be the victim of intellectual obsolescence in its professional cadres.

Preservice preparation in the various professions is prolonged and generally sophisticated. But because of burgeoning knowledge and technology, and because the areas to which professional efforts are directed are increasingly complex and interrelated, it is apparent that learning must continue throughout the lifetime of practice.

**Source:** Chairmans Report, W.K. Kellogg Foundation, Annual Report 1985.

## PRINCIPLES OF HEALTH PROMOTION

Health promotion is the process of enabling people to increase control over, and to improve, their health. This perspective is derived from a conception of 'health' as the extent to which an individual or group is able, on the one hand, to change or cope with the environment. Health is, therefore, seen as a resource for everyday life, not the objective of living; it is positive concept emphasising social and personal resources, as well as physical capacities.

1. *Health promotion involves the population as a whole in the context of their everyday life, rather than focusing on people at risk for specific diseases.* It enables people to take control over, and responsibility for, their health as an important component of everyday life — both as spontaneous and organized action for health. This requires full and continuing access to information about health and how it might be sought for by *all* the population, using, therefore, all dissemination methods available.
2. *Health promotion is directed towards action on the determinants or causes of health.* Health promotion, therefore, requires a close cooperation of sectors beyond health services, reflecting the diversity of conditions which influence health. Government, at both local and national levels, has a unique responsibility to act appropriately and in a timely way to ensure that the 'total' environment, which is beyond the control of individuals and groups, is conducive to health.
3. *Health promotion combines diverse, but complementary, methods or approaches,* including communication, education, legislation, fiscal measures, organisation change, community development and spontaneous local activities against health hazards.
4. *Health promotion aims particularly at effective and concrete public participation.* This focus requires the further development of problem-defining and decision-making life-skills both individually and collectively.
5. While health promotion is basically an activity in the health and social fields, and not a medical service, *health professions — particularly in primary health care — have an important role in nurturing and enabling health promotion.* Health professionals should work towards developing their special contributions in education and health advocacy.

**Source:** World Health Organization, *A Discussion Document on the Concept and Principles,* WHO Regional Office for Europe, 1984.

## DEATH RATES IN CANADIAN PROVINCES

### — the importance of age structure —

Crude death rates cannot be compared across provinces due to differences in age structure. Age specific death rates must first be calculated, then applied to a standard age distribution, such as the Canadian population. Crude and age standardized rates for Canada and provinces are presented in the accompanying table for the census year 1981. It is relevant to note that *Nova Scotia exhibits the highest age standardized overall mortality for both males and females.* Why?

**DEATH RATES (per 1000 population): CANADA AND PROVINCES 1981**

| | Crude | | | Standardized | | |
|---|---|---|---|---|---|---|
| | M | F | T | M | F | T |
| Canada | 8.3 | 6.0 | 7.0 | 7.2 | 4.3 | 5.6 |
| Newfoundland | 6.8 | 4.6 | 5.7 | 7.1 | 4.2 | 5.6 |
| Prince Edward Is. | 9.4 | 6.8 | 8.1 | 6.9 | 3.7 | 5.2 |
| Nova Scotia | 9.5 | 7.0 | 8.2 | 7.9 | 4.5 | 6.0 |
| New Brunswick | 8.7 | 6.1 | 7.4 | 7.6 | 4.2 | 5.8 |
| Quebec | 7.8 | 5.5 | 6.6 | 7.7 | 4.3 | 5.8 |
| Ontario | 8.1 | 6.5 | 7.3 | 7.2 | 4.3 | 5.5 |
| Manitoba | 9.7 | 7.2 | 8.4 | 7.3 | 4.4 | 5.7 |
| Saskatchewan | 9.1 | 6.4 | 7.8 | 6.6 | 3.9 | 5.2 |
| Alberta | 6.7 | 4.8 | 5.7 | 7.0 | 4.3 | 5.6 |
| British Columbia | 8.2 | 6.3 | 7.2 | 6.6 | 4.1 | 5.3 |

**Reference**

Vital Statistics, Volume I, Births & Deaths, 1981 (Cat. #84-204), Statistics Canada.

**Comment**

For a partial answer to this question, read "The Health of Nova Scotians" in the August 1985 issue of *The Nova Scotia Medical Bulletin.*

## PREVENTION AND TREATMENT OF TRAVELERS' DIARRHEA

More people than ever before are travelling to developing countries where they will be at risk for acquiring travelers' diarrhea (TD). TD is usually a self-limited illness of several days duration which is characterized by frequent unformed bowel movements, abdominal cramps, nausea, fever, and malaise. High fever, vomiting, and bloody stools are uncommon. Where surveys have been conducted, enterotoxigenic *Escherichia coli* is the most frequently identified causative agent. Other bacterial agents (*Shigella, Salmonella,* and *Campylobacter*), viruses (rotavirus and Norwalk-like viruses), and parasites (*Giardia, E. histolytica* and *Cryptosporidium*) have also been identified as causes of TD. The following is a summary of recommendations on the prevention and treatment of TD that were developed at a consensus conference on TD held at the National Institutes of Health in the summer of 1985.[1]

TD is acquired by consumption of fecally contaminated food and water. Careful attention to food and beverage selection can decrease the likelihood of developing TD, but does not eliminate the risk entirely. All uncooked foods (especially salads and unpasteurized milk products) pose greater risks of contamination. A prudent traveler consumes food which has been thoroughly cooked and is maintained hot. In areas where chlorinated tap water is not available, travelers are advised to drink canned or bottled carbonated beverages, or beverages made with boiled water.

Antimotility medications, such as diphenoxylate (Lomotil®) and loperamide (Imodium®), are *not* effective in preventing TD. Studies have, in fact, shown that diphenoxylate actually *increases* the incidence of TD in persons taking it prophylactically. Also, bismuth subsalicylate (Pepto-Bismol®) is not recommended for TD prophylaxis, even though a single study has demonstrated efficacy when used in large doses (2 oz. four times daily), because potential side effects (especially in persons already taking salicylates for arthritis, and those with peptic ulcer disease) outweigh the purported benefit. Likewise, two antibiotics, doxycycline and trimethoprim/sulfamethoxazole have been shown to prevent TD when taken prophylactically, but they are also *not* routinely recommended because side effects (skin rash, photosensitivity, blood dyscrasias, Stevens-Johnson syndrome, and/or staining of the teeth in children) once again offset the possible benefits. Antimicrobial therapy can also induce colitis and vaginitis. Hence, the only prophylactic measure routinely recommended is education of the traveler as to appropriate dietary practices.

When symptoms of TD occur, several therapeutic measures are recommended. In mild cases, fluid and electrolyte balance can be maintained by drinking caffeine-free soft drinks and eating salted crackers. Symptomatic relief of abdominal cramping and diarrhea in the absence of high fever, may be provided by diphenoxylate or loperamide. Alternatively, a regimen of 1 oz. of bismuth subsalicylate suspension every 30 minutes for a total of 8 doses may alleviate symptoms. If the symptoms are moderately severe (more than 3 stools in an 8 hour period with cramping and especially fever), self-directed treatment with antibiotics may help ameliorate the illness. For adults, either trimethoprim/sulfamethoxazole (160mgTMP/800mgSMX), trimethoprim alone (200mg), or doxycycline along (100mg) taken twice daily for 3 days is recommended. Doxycycline should not be given to children under the age of 9 years. Also, pregnant women and nursing mothers are advised to avoid these medications whenever possible. If diarrhea persists or if blood or mucous is present in the stools, medical attention should be sought. These symptoms may reflect a more serious illness.

**Reference**

1. *NIH Consensus Conference,* Travelers' Diarrhea. *JAMA* 1985; 253: 2700-4.

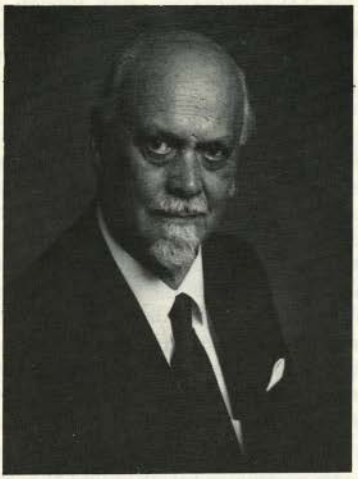
**Source:** California Morbidity, #28, July 19, 1985.

## BRYONY HOUSE

Bryony House is a place where a woman and her children can go if she has been beaten or is under threat of violence by her husband or common-law partner. **24-hour telephone 422-7650 (Halifax)** □

# Appreciations

## DR. NEVILLE MASON-BROWNE



On December 20, 1985, the medical communities of Cape Breton were grieved at the passing of Dr. Neville Mason-Browne of Louisbourg.

To say the least, Neville was a champion of a man. Born in 1919, he was the son and grandson of surgeons. His academic qualifications were numerous and included, an M.S. (Philosophy and Latin), M.B., D.P.M., C.R.C.P., and F.R.C.P. (Can.). He served in the British Armed forces from 1939 to 1945, achieving a rank of Lieutenant-Colonel. He was a glider pilot in the 1st and 6th British Airborn Division, and was awarded the Military Cross.

Having received his medical training in Edinburgh University and London University, his practised psychiatry in the British Honduras in Central America, Saskatchewan, Ontario, British Columbia, the Yukon Territory, and lastly Nova Scotia. He left behind a trail of enviable accomplishments. He was Director of Mental Health Services in the Yukon and Director of Medical services in the British Honduras, where he was directly responsible to the Minister of Health. He holds to his credit nine professional publications on psychiatry, had a weekly column on lay topics under a pseudonym in a Canadian newspaper, and has written a book entitled *The Crazy House*.

Dr. Mason-Browne has been fervently interested in medical politics all his life. He has been an executive and founding member of the Canadian Psychiatric Association (1970-3), President of the British Honduras Medical Association,) and President of the Yukon Medical Association. He was a student of anthropoligy at UBC. His interest in Nova Scotia medical politics was no secret to our Society. His addresses to the Medical Society of Nova Scotia were always met with respect, as his command of the English language was exceptional as the quality of his voice.

He leaves to mourn his wife, Rosemary, who is a trained, qualified medical illustrator, two daughters and a son. His presence in Sydney has left an indelible impression, and we share with his family a deep remorse.

P.F. Murphy, M.D.

## DR. FRANCIS B. MACDONALD

Dr. F.B. MacDonald, 69 of Sydney, a senior member of both the Nova Scotia Medical Society and the Canadian Medical Association died Friday, December 28, 1985.

Born in Glace Bay, Dr. MacDonald was a son of the late John D. and Alexandra (MacDonald) MacDonald. He was a graduate of St. Francis Xavier University in arts and science following which he studied medicine at Dalhousie University, graduating with honors.

After practising in Sydney for several years, he began graduate studies in obstetrics and gynecology at the University of Pennsylvania where he received his masters degree in medical science. He then resumed his practice in Sydney.

In 1983, he was made a senior member of the Nova Scotia Medical Society and the following year was similarly honored by the Canadian Medical Association. He was chief of staff at St. Rita Hospital for many years. He was President of Cape Breton Medical Society.

A life-long scholar, his particular interests were poetry, history and all things Scottish. He was author of a book, *To The Old and New Scotland,* a collection of addresses given at St. Andrew's Day dinners and his own essays. He wrote several Medical articles in reported medical magazines.

He was also co-author of the *25th Battalion, C.E.F.,* honoring this celebrated First World War combat unit. After the War, Dr. MacDonald continued his association with the armed forces, remaining several years with the militia and retiring with the rank of lieutenant-colonel.

He will be remembered as much for his kindness and integrity as for his professional skill. His concern for life led to his involvement with the pro-life movement, for which cause he made himself available in addressing meetings on various occasions.

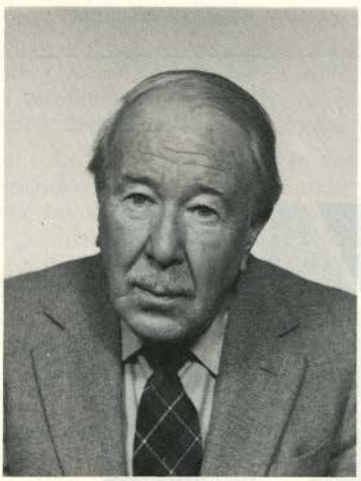
He was a member of the Knights of Columbus, Clan Donald Association, Canadian Physicians for Life, Royal Canadian Legion, Royal United Services Institute and a life member of St. F.X. University Presidents' Club.

He is survived by his wife, the former Betty Ronan; three sons, Frank W., Dartmouth, Andrew and Ira, both of Halifax; two daughters, Audrey, Winnipeg and Clare, Halifax; a sister, Mrs. R.F. MacKinnon, Glace Bay; and two grandchildren. To his wife and children we offer our sincere sympathy.

M.A. Naqvi, M.D.

---

## DR. ARTHUR LISTER MURPHY



The sudden death of Doctor Arthur Lister Murphy on Saturday, November 9, 1985, came as a shock and a surprise to those of us who knew him.

Although 79 years of age, a senior member of the profession and long ago retired from his surgical practice, he had developed parallel careers as an higher education advisor, drama teacher at Dalhousie University, playwright and author in which he continued to be active until he died.

Arthur graduated MDCM from Dalhousie in 1930, did residency training in surgery at the Montreal General Hospital and returned to practise in Halifax with his father at the Halifax Infirmary. He was appointed Assistant Surgeon at the Victoria General Hospital in 1934 and Assistant Professor of Surgery, Dalhousie University. He was promoted to Associate Professor in 1944 and continued practice on the Surgical staff of the V.G.H. until his retirement in 1970. During the later years of his surgical practice, he became chief of the Head and Neck Surgical Service at the V.G.H.

He first became interested in theatre during his student days at Dal, and tried his hand at playwriting while a resident at the Montreal General Hospital. He wrote scripts for local Halifax radio shows and for the CBC radio, but got his first major breakthrough writing episodes for the USA TV programs featuring Ben Casey and Doctor Kildare. "I wrote more episodes for those T.V. series than I would care to recall" he once said. Soon after he was asked by the Mayor of Halifax to chair a Theatre Feasibility Committee. Out of this the Neptune Theatre was born and Arthur became it's founding father and first President. During the 1960s several of his plays were produced by Neptune Theatre including an adaptation of *Diary of a Scoundrel* and *The Sleeping Bag.* His most successful play, *Tiger, Tiger,* about Doctor John Hunter was produced at Neptune in 1970.

Arthur received several drama awards and an honorary Doctor of Laws degree from St. Francis Xavier University. He was Chairman of the Nova Scotia University Grants Committee 1968-1974, and subsequently was appointed an adviser on higher education to the government. He was invited to join the Faculty of the Theatre and Drama Department of Dalhousie University as a part-time teacher in 1977, and continued to give classes until he died.

His most recent publication was *Three Bluenose Plays* in 1984 and at the time of his death he was starting to work on a play about Sir John Thompson based on Professor Waite's recent book, *The Man from Halifax.*

Arthur was a keen yachtsman, loved schooner sailing, and was one of a group instrumental in getting the St. Margaret's Bay Sailing Club started near his home at Tantallon.

We extend our sympathy to his wife, Barbara, his sons, Arthur and Paul, and his daughter, Sylvia Joanne, all of Halifax.

He was truly a Renaissance Man.

H.C. (Curly) Still, M.D.

## DR. RONALD MORRISON RITCHIE

On December 9, 1985 this community lost one of its most respected and admired paediatricians. Ronald Ritchie passed away after fighting against a prolonged and delibitating illness. He continued his practice until early summer, when his poor health forced the discontinuance of it.

Ron was loved by his patients and he had many "second generation" patients which he especially enjoyed. He approached his paediatric practice firstly by knowing the conditions of the home environment, the family circumstances and the interaction of the children and parents and applied this to his care of the patient. He particularly enjoyed getting to know his young patients, looking after them and watching them progress through their various stages and ages. I know he will be sorely missed by his patients, their parents and his academic colleagues.

He played a major role in teaching paediatrics at Dalhousie from 1951 to 1982. Following his official retirement from the university and hospital staffs, he worked in the out-patients at the IWK and taught for 3 years there where his clinical acumen was much appreciated. He was an Associate Professor of Paediatrics at Dalhousie.

Ron was born in Sydney, N.S., attended school there and then St. Francis Xavier University, followed by graduation from Dalhousie in medicine. Following this he spent three years in the active service in the Royal Canadian Army Medical Corps overseas. On completion of his wartime service he spent three years in Inverness, Cape Breton in general practice with Dr.Frank MacLeod. Following this, he went to the Montreal Children's Hospital for two years postgraduate training in paediatrics. In 1951 he came to Halifax and joined Dr. Henry Ross in a very busy paediatric practice and started teaching at Dalhousie. Many generations of medical students will long remember his practical tips and pointers that are not found in medical textbooks.

Ron was a valued member of St. Andrew's United Church, Saraguay Club, Phi Rho Sigma Fraternity, Canadian Paediatric Society and the American Association and the Medical Society of Nova Scotia.

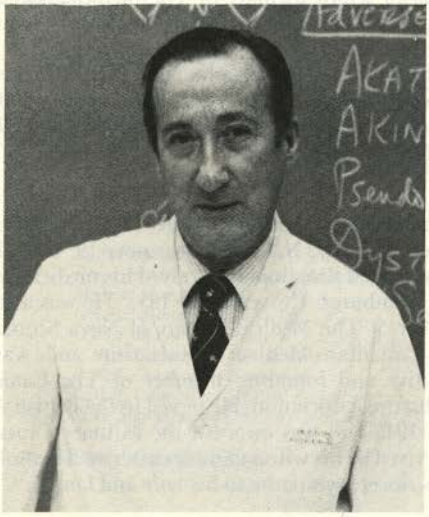
He is survived by his wife Doris (Craig), 2 daughters Judi (Kent), Whitehorse, Susan at home and three grandchildren in Whitehorse.

J.M. Crosby, M.D.

□

# Personal Interest Notes

**Dr. Patrick Flynn** was recently named best teacher at Dalhousie University's Medical School by the 340-member Intern and Residents Association of Nova Scotia.




Dr. Flynn, a Halifax psychiatrist, has been teaching undergraduate and graduate students, teachers, social workers and practising physicians for more than 20 years. He attributes his success to his ability to "sympathize with them and show lots of good humor."

This isn't the first time students recognized Dr. Flynn's superior teaching skills. In 1976 undergraduate medical students named him teacher of the year. Three years ago psychiatry residents presented him with a "special certificate of spectacular status" naming Flynn a lifelong resident in psychiatry. As a director of psychiatric residency training for 11 years, he was noted by the students for his "unquenchable lust for life and learning, and his consistent ability to treat trainees as fellow humans."

Dean Murray calls Dr. Flynn "one of our most committed, excellent teachers".

---

The appointment of **Dr. John P. Finley** as Head of the Division of Cardiology in the Department of Pediatrics and as Head of the Diagnostic Cardiology Department of The Izaak Walton Killam Hospital for Children was recently announced. Dr. Finley replaces **Dr. D.L. Roy,** who was recently awarded an Honorary Life Membership by the Board of Governors of the IWK. Dr. Finley is currently an Associate Professor of Pediatrics at Dalhousie University.

---

## DALHOUSIE MEDICAL ALUMNI

The Annual Meeting of the Dalhousie Medical Alumni Association was held on November 18, 1985 at the Nova Scotian Hotel. This year the format was a reception, dinner and dance with Joe Skowronski's Band, and it was a delight. The food was good, the music superb and the attendance good. Two outstanding medical alumni were honoured, namely **Dr. Donald I. Rice,** M.D. '51, who was appointed Honorary President, and **Dr. Harold L. Scammell,** M.D. '27, who was appointed Alumnus of the Year.

In making the presentation to Dr. Rice, Dr. Carlyle Phillips outlined the many contributions he had made to the organization of Family Medicine, both nationally and internationally. Dr. Rice has recently retired as Executive Director of the College of Family Physicians of Canada.


Dr. Donald I. Rice.

In Dr. Byron Reid's citation for Dr. H.L. Scammell, he outlined his many contributions to organized medicine and education, noting that he had served Dalhousie very well as Registrar, Special Assistant to the President, and that he was very influential in the establishment ofthe Dalhousie School of Nursing and Faculty of Graduate Studies. He served as Registrar of the Provincial Medical Board of Nova Scotia for 26 years, he was Medical Superintendent of the Victoria General Hospital, 1931 to 1937, when he transferred to the Workman's Compensation Board, of which he is still serving as a Consultant. His list of contributions goes on and on, noting many papers and articles written on the History of Medicine, History and Ethics. Dr. Scammell is Life President of his Class and he graduated with honours. An outstanding Career.

Dr. Merv. Shaw, Barbara Blauvelt, and Mrs. Shaw.

Medical Reunions were very evident with alumni returning and attending from as far away as Ghana, the United States and all over Canada. All in all, a fine time was had by all and we look forward to increased attendance in the future when our meeting will be held the first October. □





## OBITUARIES

**Dr. John W. Sutherland,** (89) of Burlington, Ontario died on January 11, 1986. Born in Dalhousie Mountains, Pictou County he was a veteran of the First World War and, upon receiving his medical degree from McGill University, he became a research scientist at the paper mill in Hawkesbury, Ontario. He is survived by his two sons, to whom we extend sincere sympathy.

---

**Dr. Francis B. MacDonald,** (69) of Sydney, N.S. died on December 27, 1985. Born in Glace Bay he received his medical degree from Dalhousie University in 1944. He was a senior member of The Medical Society of Nova Scotia and The Canadian Medical Association. He did his graduate studies in obstetrics and gynecology in Pennsylvania and then returned to Sydney to resume his practice. He is survived by his wife, three sons and two daughters. Our sympathy is extended to his family.

---

**Dr. Neville S. Mason-Browne,** (66) of Louisburg, formerly Sydney, N.S. died December 19, 1985. Born in the United Kingdom he received his medical degree from Edinburgh University in 1951. He was a senior member of The Medical Society of Nova Scotia and The Canadian Medical Association and was an executive and founding member of The Canadian Psychiatric Association. He served in the British Army until 1945 and was awarded the Military Cross. He is survived by his wife and three children. The Bulletin offers sincere sympathy to his wife and family. □

**HEALTH CARE PROFESSIONS GROUP OF A.A. AND DRUG DEPENDENCY**

Meetings held Sunday evenings — 8:00 p.m.
Bethany United Church

First three Sundays of month closed meetings
Last Sunday of the month open meeting

(Meeting place and parking rear of building)

For further information please call 422-5875

**ADVERTISERS' INDEX**

# THE MEDICAL SOCIETY OF NOVA SCOTIA

NOVA SCOTIA DIVISION OF THE CANADIAN MEDICAL ASSOCIATION

## MEMBERS OF EXECUTIVE COMMITTEE

### OFFICERS

*President* ..... J.C. Kazimirski
*President-Elect* ..... W.C. Acker
*Past President (Immediate)* ..... M. G. Shaw
*Chairman, Executive Committee* ..... R. Stokes
*Vice-Chairman, Executive Committee* ..... M.E. Lynk
*Treasurer* ..... V.P. Audain
*Honorary Secretary* ..... J.H. Gold
*Officer-at-large* ..... W.H. Lenco
*Executive Director* ..... D.D. Peacocke

### OBSERVERS AND STAFF

*Economics Committee Chairman* ..... W.C. Acker
*Manager, Economics Department* ..... A.A. Schellinck
*Director of Communications* ..... Bill Martin

### BRANCH SOCIETY REPRESENTATIVES

*Antigonish-Guysborough* ..... D.P. Cudmore
*Bedford-Sackville* ..... G.L. Myatt
*Cape Breton* ..... P.K. Cadegan & D.P. Hickey
*Colchester-East Hants* ..... F.E. Slipp
*Cumberland* ..... W.G. Gill
*Dartmouth* ..... R.J. Gibson & G.L. Roy
*Eastern Shore* ..... D.P. Sinha
*Halifax* ..... I.A. Cameron, D.A. Gass & J.H. Gold
*Inverness Victoria* ..... G.W. Thomas
*Lunenburg-Queens* ..... G.H. Nickerson
*Pictou* ..... E.R. Sperker
*Shelburne* ..... W.H. Blair
*Sydney* ..... P.F. Murphy & G.E. Boyd
*Valley* ..... J.R. MacEachern & R.R. Kimball
*Western* ..... P.L. Loveridge
*Student Members* ..... D. Campbell, G. Raymond & M. Conrod
*I.R.A. Representatives* ..... Allan Patrick & Dianne Mosher

### OBSERVERS

*Editor-in-Chief — The Nova Scotia Medical Bulletin* ..... J.F. O'Connor
*Representative to Provincial Medical Board* ..... G. McK. Saunders
*Medical Director M.M.C. Inc* ..... H.J. Bland
*General Manager M.M.C. Inc.* ..... D.L. McAvoy
*C.M.A. Board of Directors* ..... R.D. Saxon
*C.M.A. Council on Health Care* ..... D.S. Reid
*C.M.A. Council on Economics* ..... P.D. Muirhead
*C.M.A. Council on Medical Education* ..... J.D.A. Henshaw
*M.D. Management Limited* ..... G.A. Sapp

### STANDING COMMITTEES

| | Chairman |
|---|---|
| *Allied Health Disciplines* | J.H. Quigley |
| *Annual Meetings* | President |
| *Archives* | I.A. Cameron |
| *Awards Committee* | P.E. Kinsman |
| *By-Laws* | P.W. Littlejohn |
| *Cancer* | A.J. Bodurtha |
| *Child Health* | G.H. Nickerson |
| *Community Health* | M. Kazimirski |
| *Drug & Alcohol Abuse* | E.L. Reid |
| *Editorial* | J.F. O'Connor |
| *Ethics* | K.R. Murray |
| *Finance (Treasurer)* | V.P. Audain |
| *Hospitals & Emergency Services* | D.F. Craswell |
| *Maternal & Perinatal Health* | E.R. Luther |
| *Medical Education* | G.J. Butler |
| *Membership Services* | K.R. Langille |
| *Nutrition* | M.P. Quigley |
| *Occupational Medicine* | J.D. Prentice |
| *Pharmacy* | J.P. Anderson |
| *Physical Fitness* | M.R. Banks |
| *Presidents' Committee* | President |
| *W.C.B. Liaison* | J.C. Kazimirski |

### BRANCH SOCIETIES

| | President | Secretary |
|---|---|---|
| *Antigonish-Guysborough* | P.J. Mckenna | T.D. Smith |
| *Bedford-Sackville* | J.W. McNab | G.B. Mader |
| *Cape Breton* | B.J.M. O'Brien | J.A. Stevens |
| *Colchester-East Hants* | C.K. Bridge | R.L. Rondeau |
| *Cumberland* | M.F. Husain | K. Thakkar |
| *Dartmouth* | W.R. Lee | L.B. Marr |
| *Eastern Shore* | W.C. Brown | D.R. Barnard |
| *Halifax* | I.A. Cameron | S.B. Gibbon |
| *Inverness Victoria* | J.O. Belen | K.R. Murray |
| *Lunenburg-Queen* | F.G. Bell | |
| *Pictou* | G. E. Farrell | C.S. Giffin |
| *Shelburne* | D.H. Wilson | M.E. Riley |
| *Sydney* | S.M.A. Naqvi | |
| *Valley* | R.R. Kimball | W.N. Enright |
| *Western* | D.S. Armstrong | W.M. Gorman |

### SECTIONS

| | | |
|---|---|---|
| *Anaesthesia* | A.C. Walker | K.R. Hamilton |
| *General Practice* | M. J. Fleming | R.A. Oliver |
| *Internal Medicine* | L.P.M. Hefferman | B.J. Cookey |
| *Internes & Residents* | B. Wright | S. Robinson |
| *Obstetrics and Gynaecology* | C.A. Maley | S.C. MacLeod |
| *Ophthalmology* | J.H. Quigley | D.M. Keating |
| *Orthopaedic Surgery* | J.C. Hyndman | G.H. Reardon |
| *Otolaryngology* | G. Shimo | F.S.H. Wong |
| *Paediatrics* | A.F. Pysemany | A.E. Hawkins |
| *Laboratory Medicine* | D.A. Taylor | A.J. Wort |
| *Psychiatry* | C.W. McCormick | R. Morehouse |
| *Radiology* | J.A. Aquino | W. F. Barton |
| *Surgery* | D.M. Nicholson | P.D. Roy |
| *Urology* | S.G. Lannon | R. W. Norman |
| *Vascular Surgery* | J.A.P. Sullivan | R.S. Dunn |