

# THE NOVA SCOTIA MEDICAL JOURNAL

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## Choosing a Physician

The attitude of physicians and the method of payment for services are likely to become more varied in our health care system. Graduating physicians want to work shorter hours, have less commitment to patients, and are more willing to accept the controls that salary will force upon them. Methods of treatment vary in their acceptability by physicians. The controversy over the use of transplantation and wholesale application of cholesterol monitoring are examples. The beliefs and attitudes of a physician will, in very important ways, determine the type of care received, and thus make the choice of a physician both more difficult and more significant in the future.

Patients will increasingly want to know our attitudes and where we stand with respect to medical care choices. At the same time, it is difficult for a physician to take a firm philosophical position in the face of government pressure and new information. The most important factor to be remembered, from a physician's viewpoint, is to choose what is right for each patient as an individual. As for the patient, his choices are probably going to get more difficult.

The recent review article on "HSOs: Ontario's answer to HMOs?" by Weinkauff and Scully in the *Canadian Medical Association Journal* states that there are thirty-four HSOs (Health Service Organizations) in Ontario. It is inevitable that other provinces will show an interest. The hypothesis that HSOs will benefit both consumers and providers while reducing cost has not been proven. However, if they are tried in Nova Scotia, the choice of care facing the patient, in the not too distant future, will be difficult to explain and to understand without careful thought.

Some medical care choices are described below; they are not accurate descriptions of any one system, but merely two possible scenarios.

## PHYSICIAN A

1. Paid by Health Maintenance or Service Organizations, Workers Compensation Board, Corporations, etc. and responsible to the paying agency.
2. Unless encouraged by Health Maintenance or Service Organizations, less likely to have a continuing relationship with the patient.
3. Cost concerns are prominent due to pressure from managers, administrators, etc. Income incentives for physician to provide less expensive care. High technology is rationed.
4. Follows patients when needed. Eliminates visits not proved cost effective; e.g. does little psychotherapy or counselling.
5. Personal physician less accessible to patients due to decreased hours, guaranteed vacations, sick time and study time. Not driven at all. More "balanced" life.
6. Encourages prevention when it decreases cost to agency.
7. Patient advocate for pensions, Workers Compensation Board, insurance within the limits of honesty.

## PHYSICIAN B

1. Paid by the patient through his insurance scheme. Responsible primarily to the patient.
2. Has an established life-long relationship between patient and doctor.
3. Little concern for cost as long as it is best for the patient (until recently). Often refers to high-tech care givers.
4. Follows patients when asked; e.g. does psychotherapy when asked and when possible.
5. Personal physician as accessible as is possible. Long hours with limited vacation time. Sneaks study time. Driven by need, greed or dedication.
6. Little documented preventive care but frequently takes time to talk prevention and life style and bills system despite unproven cost effectiveness.
7. Patient advocate for pensions, Workers Compensation Board and insurance within the limits of honesty.

Which doctor would you like to attend as a caregiver? If you had a heart attack, needed high tech investigation, wanted to talk new lifestyle, prevention and had concerns for your future sex life, which doctor would you prefer? While this is a simplistic and unfair question, it might help us to see the choices that will be offered our patients.

At the recent First Canadian Medical Leadership Conference, it was emphasized that physicians should have the flexibility to choose under what system they

will operate and that quality of care must be the guiding principle.

At the same conference, Dr. Maureen Law, the Federal Government's Deputy Minister of Health said, "Clinical freedom is precious to physicians, but it is not a licence to ignore considerations of cost." As we physicians make our choices of how to practice in the future, the patient also should see and understand the difficulties and choices available to him or to her.

J.F. O'C. □

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# A Program of Home Intravenous Antibiotic Therapy

Murray D. Nixon, MD, CCFP, Elizabeth MacWilliam, BN, RN, and Frederick G. Solven, MD, FRCPC,

Fredericton, N.B.

Intravenous antibiotic therapy can be safely given in the home. This article reports on a series of twenty-two patients who received intravenous antibiotics at home through the Fredericton Unit of the New Brunswick Extra-Mural Hospital. Eight patients were taught to self administer their antibiotics. No major problems were encountered; there were no therapeutic failures, and the patients appreciated the opportunity to be treated at home.

Infectious diseases such as osteomyelitis and endocarditis are usually managed with four or more weeks of intravenous antibiotic therapy. Since some of these patients begin to feel well and become mobile after a few days of treatment, both they and their physicians may become dissatisfied with continued therapy in hospital — the patients, because they are anxious to return home and to work, and the physicians because they often need the beds for sicker patients. To circumvent this long stay in hospital, home health care organizations such as the New Brunswick Extra-Mural Hospital have developed programs of home intravenous antibiotic therapy.<sup>1,2</sup>

Intravenous antibiotic therapy in the home is not suitable for all patients, all infections or all homes. The patients accepted for the New Brunswick Extra-Mural Hospital program of home intravenous antibiotic therapy meet the following criteria:

1. The patient lives in an area serviced by the Extra-Mural Hospital.
2. There is a medical need for intravenous antibiotic therapy.
3. The attending physician recommends home care.
4. The home environment is safe.
5. The patient is capable and willing to participate.
6. Venous sites are adequate.
7. A family support system is helpful but not absolutely necessary.

A sub-group of patients receiving home intravenous antibiotic therapy may be interested in learning to self administer their medication, and success in teaching patients has been well documented.<sup>1, 2, 3, 4</sup> The heparin loc system facilitates this home therapy. It is compact, allows patient mobility and is ideal for intermittent dosing schedules.<sup>5</sup>

Extra-Mural Hospital patients, receiving or scheduled to receive intravenous antibiotics, who express an

interest in self administration are given the opportunity to learn the technique. The Extra-Mural Hospital nurse conducts a teaching program based on specific learning objectives which require the patient to demonstrate satisfactory knowledge and technique for eighteen principles and procedures.

The patient is required to:

1. Exhibit knowledge about his medical condition and why intravenous antibiotics are important in its management.
2. Demonstrate good hand-washing technique.
3. Demonstrate aseptic technique when handling equipment and supplies.
4. Demonstrate the ability to assemble the syringe.
5. Demonstrate the ability to remove air bubbles from the syringe.
6. Demonstrate the ability to mix the antibiotic accurately and inject it into the partial fill.
7. Exhibit knowledge regarding (a) the labelling of the antibiotic in the vial and in the partial fill, and (b) the storage of the medication.
8. Demonstrate the ability to assemble the intravenous line while using aseptic technique.
9. Demonstrate (a) an understanding of the role of the primary and secondary lines and (b) the ability to use them properly.
10. Demonstrate the ability to flush the intravenous line.
11. Demonstrate the ability to infuse the antibiotic accurately over the prescribed time.
12. Demonstrate the ability to accurately draw up the set heparin dose.
13. Demonstrate the ability to administer the heparin.
14. Demonstrate the ability to remove the angiocath safely.
15. Exhibit knowledge of and action to be taken in the case of:
  - (a) infiltration of the I.V.
  - (b) sluggish I.V. flow
  - (c) leakage of I.V. fluid or blood
  - (d) phlebitis.
16. Demonstrate the ability to self administer the antibiotic intravenously.
17. Demonstrate the ability to wrap the intravenous line to protect the site.
18. Exhibit knowledge of possible side effects of the antibiotic.

## THE FREDERICTON EXPERIENCE

Prior to the opening of the Extra-Mural Hospital Unit, a program of home intravenous antibiotic therapy

From the New Brunswick Extra-Mural Hospital.

Correspondence: Dr. Murray Nixon, Medical Director, New Brunswick Extra-Mural Hospital, 200 Prospect Street, Fredericton, N.B. E3B 2T8



was initiated in Fredericton, beginning in 1985, with ten patients treated independently of the Extra-Mural Hospital. The diagnoses were similar to those reported subsequently in the Extra-Mural Hospital program. There were no complications. One therapeutic failure occurred in the case of a patient with a reanastomosed finger amputation. Patient teaching was done by the nursing staff of the Dr. Everett Chalmers Hospital, who viewed it as an extension of their diabetic teaching program, i.e., patient education concerning the use of needles. The contribution of these nurses and in particular Mrs. Penny Short, the Director of Surgical Nursing, is acknowledged. Funding sources included third party insurance and cooperative, small hospital pharmacies. However, the difficulty in finding adequate funding sources was the major factor limiting its more widespread application.

The Fredericton Unit of the New Brunswick Extra-Mural Hospital provided home treatment with intravenous antibiotics for twenty-two patients between January 1986 and March 1988. There were sixteen male and six female patients. Fifteen were admitted to the Extra-Mural Hospital from the local acute care hospital (where treatment had been started); one patient was admitted from the emergency department; and six were admitted directly from their homes. Thirteen physicians admitted the twenty-two patients.

AGE	NUMBER OF PATIENTS
0-14	1
15-24	3
25-34	4
35-44	5
45-54	2
55-64	0
65-74	2
75-84	5
85+	0

PRIMARY DIAGNOSIS	NUMBER OF PATIENTS
*Osteomyelitis	10
*Bacterial Endocarditis	4
Cellulitis	4
*Septic Arthritis	2
Pneumonia	1
*Septicemia	1

\* Includes self administration (8 patients).

	TOTAL	SELF-ADMINISTRATION GROUP	NON SELF-ADMINISTRATION GROUP
Number of Patients	22	8	14
Average Number of Extra-Mural Hospital Days on I.V. Therapy	24	29	21
Average Number of Nurses' Visits	49	23	66

The antibiotics used were aminoglycosides, cephalosporins and penicillins. The frequency of dosage ranged

from q12h to q6h, with the majority being q8h. The nurses noted problems with insertion of the angiocath for sixteen patients but it was rarely necessary to require the physician to insert the catheter. Although seventeen patients experienced minor local problems with the intravenous there were no problems of a serious nature.

There were no therapeutic failures in the twenty-two patients.

## DISCUSSION

Studies on home intravenous antibiotic therapy report treating a variety of infections.<sup>1,6,7,8</sup> In Gizzard's study, osteomyelitis, septic arthritis, deep wound infections and infected orthopedic appliances accounted for over two-thirds of the cases.<sup>2</sup> Self-administered home intravenous antibiotic therapy has been successful in treating bronchiectasis and adult cystic fibrosis.<sup>6</sup> The diagnoses in Fredericton were similar to those in other studies. Perhaps the base of eligible patients could be safely broadened; for example, concern about oral regimens for the treatment of pelvic inflammatory disease could be resolved by the use of home intravenous antibiotic therapy.<sup>3</sup>

There are essentially no restrictions on the types of antibiotics that can be used in home therapy but, if all other factors are equal, a drug that needs to be administered only once or twice a day is especially attractive for the home setting.<sup>1,7</sup>

Few patients in Fredericton or in the studies reviewed, experienced side effects.<sup>1,9</sup> Stiver and colleagues found the same frequency of adverse drug reactions in home patients as in a hospitalized group.<sup>1,5,7</sup> Close monitoring, including the measurement of serum concentrations when appropriate, should help minimize serious drug-related adverse effects.

The literature points out the cost effectiveness of home intravenous antibiotic care.<sup>1,10</sup> The cost savings while varying from one location to another may be considerable. Indirect savings to the patient may result from a more rapid return to work.<sup>2</sup>

Stiver states that even with daily nursing visits, home intravenous antibiotic therapy costs one-fourth to one-third that of in-hospital therapy.<sup>7</sup> The ability to keep a patient out of hospital for three to six weeks of intravenous antibiotic therapy can help relieve pressures for beds.

Careful patient selection took place in Fredericton as it did in all studies reviewed. The patients accepted into the programs were motivated, competent and anxious to be treated at home. Some were able to return to work or school during the treatment. Most patients, particularly those on self administration programs, are initially apprehensive and rely heavily on the visiting nurse for support until they become accustomed to the treatment regime.<sup>1</sup>

Home intravenous antibiotic therapy is an effective and safe form of treatment for selected patients and can



increase efficiency in health care.<sup>5</sup> It contributes to the patients' well-being by permitting them to be at home with their families. An interested, capable group of these patients can become active participants in their own care by learning to self administer their medication. Some return to productive activity while continuing to receive therapy.

*"The sooner patients can be removed from the depressing influence of general hospital life, the more rapid their convalescence."*

(Charles H. Mayo) □

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**Pharmacology:** Nizatidine is a competitive, reversible inhibitor of the binding of histamine to the histamine H<sub>2</sub> receptor of the gastric acid secreting cells. Nizatidine is not an anticholinergic agent. It inhibits nocturnal gastric acid secretion and gastric acid secretion stimulated by food, caffeine, betazole and pentagastrin. Pepsin output is reduced in proportion to the reduced volume of gastric secretions. Nizatidine has little or no effect on basal serum gastrin or food induced hypergastrinemia. Nizatidine is absorbed rapidly after oral administration. Peak plasma concentrations occur from 0.5 to 3 hours after the dose. Absorption is unaffected by food or propantheline. However, antacids decrease the absorption of nizatidine by about 10%. The absolute oral bioavailability of nizatidine exceeds 90%. Approximately 35% of nizatidine is bound to plasma protein, primarily of  $\alpha_1$ -glycoprotein. This binding is not influenced by other drugs such as warfarin, diazepam, acetaminophen, propranolol, or phenobarbital. Approximately 90% of an oral dose of nizatidine is excreted in the urine within 12 hours. About 60% of an oral dose and 77% of an i.v. dose of nizatidine is excreted as unchanged drug. The elimination half-life is 1 to 2 hours and the systemic plasma clearance is about 50L/hour. The volume of distribution is 0.8 to 1.5 L/kg. Since nizatidine is primarily excreted in the urine, renal impairment significantly prolongs the half-life and decreases the clearance of nizatidine. In anephric individuals with creatinine clearance less than 10mL/min, the half-life is 3.5 to 11 hours, and the plasma clearance is 7 to 14L/hour. The dose should be adjusted in patients with moderate or severe impairment of renal function (see Dosage). The pharmacokinetic profile for nizatidine in the elderly was not significantly different from the profile in younger normal subjects. Gastric acid suppression correlates directly with nizatidine doses from 75 to 350 mg. Oral doses of 100 mg or 1.3 mg/kg suppressed gastric acid secretion in sham fed volunteers for 3 hours after the dose. The duration of acid suppression directly correlates with the nizatidine dose. 300 mg nizatidine suppressed acid secretion almost entirely early in the day, and the suppression persisted about 10 hours. Nocturnal acid was suppressed for 10 to 12 hours after 300 mg nizatidine. Treatment for up to 2 weeks with nizatidine 600 mg daily did not influence the serum concentrations of gonadotropins, prolactin, growth hormone, antidiuretic hormone, cortisol, triiodothyronine, thyroxine, testosterone, 5  $\alpha$ -dihydrotestosterone, androstenedione or estradiol.

**Drug Interactions:** No interactions have been observed between nizatidine and theophylline, chloridiazepoxide, lorazepam, lidocaine, and warfarin. Nizatidine does not inhibit the cytochrome P-450-linked drug-metabolizing enzyme system; therefore, drug interactions mediated by inhibition of hepatic metabolism are not expected to occur.

**Indications:** Nizatidine is indicated in the treatment of conditions where a controlled reduction of gastric acid secretion is required for ulcer healing and/or pain relief. Conditions include acute duodenal ulcer, acute benign gastric ulcer, and prophylactic use in duodenal ulcer.

**Contraindications:** Patients with known hypersensitivity to the drug.

**Precautions:** Gastric ulcer: Where gastric ulcer is suspected the possibility of malignancy should be excluded before therapy with nizatidine is instituted.

**Pregnancy and Lactation:** Safety of nizatidine during pregnancy has not been established. Reproduction studies performed in rats and rabbits at doses up to 300 times the human dose have revealed no evidence of impaired fertility or teratogenicity. If the administration of nizatidine is considered to be necessary, its use requires that the potential benefits be weighed against possible hazards to the patient and the fetus. Nizatidine is secreted in the milk of lactating rats; it is assumed to be secreted in human milk and caution should be exercised when nizatidine is administered to nursing mothers.

**Impaired Renal Function:** As nizatidine is excreted via the kidney, dosage should be adjusted in patients with moderately or severely impaired renal function (see Dosage).

**Hepatic Dysfunction:** Nizatidine is partially metabolized in the liver; however, in patients with uncomplicated hepatic dysfunction, disposition of nizatidine is similar to that of normal subjects.

**Geriatrics:** Ulcer healing rates in elderly patients are similar to those in younger age groups. The incidence rates of adverse events and laboratory test abnormalities are also similar to those seen in other age groups. Age alone is not an important factor in the disposition of nizatidine. Elderly patients may have reduced renal function (see Dosage).

**Children:** Safety and effectiveness of nizatidine in children has not been established.

**Adverse Effects:** In double-blind, placebo-controlled clinical trials in over 2,300 patients, the overall incidence of adverse events reported by patients treated with nizatidine was no greater than in the placebo group. Clinical pharmacology studies and controlled clinical trials showed no evidence of antiandrogenic activity or other adverse hormonal effects.

Headache, asthenia, chest pain, myalgia, abnormal dreams, somnolence, rhinitis, pharyngitis, cough and pruritus were reported with a slightly higher frequency by nizatidine-treated patients than by the placebo group. A relationship to nizatidine administration has not been established. Excessive sweating may be related to administration and has been reported by 1.1% of patients.

**Laboratory Values:** Patients treated with placebo and those receiving nizatidine therapy had mild, transient, asymptomatic elevations of transaminases; rare instances of marked elevations (>500 IU/L) occurred in nizatidine-treated patients, although causality has not been established. These abnormalities were asymptomatic and readily reversible after discontinuation of the drug. Other laboratory variables which were statistically different from placebo in the nizatidine-treated group, include serum cholesterol, serum uric acid, platelet count, serum creatinine, and white blood cell count. The clinical significance of these differences is not clear.

**Laboratory Tests:** False positive tests for urobilinogen with Multistix<sup>®</sup> may occur during therapy with nizatidine.

**Overdose:** Treatment: There is no clinical experience with deliberate overdosage of nizatidine in humans. Should overdosage occur, the usual measures to remove unabsorbed material from the gastrointestinal tract should be employed along with clinical monitoring and supportive therapy. The amount of nizatidine absorbed from the gastrointestinal tract can be reduced by activated charcoal.

**Dosage:** Duodenal or Gastric Ulcer: One 300 mg capsule or two 150 mg capsules once daily at bedtime. Treatment should be given for 4 to 8 weeks, but the duration of the treatment may be shortened if healing can be documented. Healing occurs within 4 weeks in most cases of duodenal ulcer.

**Maintenance Dosage in Duodenal Ulcer:** One 150 mg capsule once daily at bedtime for 6 to 12 months depending on the severity of the condition. Antacids may be given concomitantly if needed.

**Dosage Adjustment in Renal Impairment:**

Renal Function	Creatinine Clearance (mL/min)	Dosage	
		Acute	Maintenance
Normal	>50	300 mg/day	150 mg/day
Moderate Impairment	20-50	150 mg/day	150 mg/2nd day
Severe Impairment	<20	150 mg/2nd day	150 mg/3rd day

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**References:**

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5. Data on file, Eli Lilly Canada Inc.
6. AXID<sup>®</sup> Product Monograph.



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# Granulocytopenia Associated with Captopril Use in an Otherwise Healthy Patient

Suzanne Mack,\* BSc, Jean D. Gray,\*\* BSc, MD, FRCPC, and Robert D. Graham,† MD,

Halifax, N.S.

Among the variety of drug side effects commonly reported, few are potentially life-threatening. Agranulocytosis is one such example. Since the relatively recent introduction of the angiotensin-converting enzyme inhibitor, captopril, there have been scattered reports of granulocytopenia/agranulocytosis in association with use of this drug.<sup>1</sup> These cases have been confined almost exclusively to hypertensive patients suffering from other medical problems, most commonly renal disease, with or without collagen vascular disease, or concurrent treatment with immunosuppressive drugs.<sup>2</sup> Previously, there has been only one report of captopril-associated leukopenia in an otherwise healthy individual with severe essential hypertension.<sup>3</sup> This is the second such report of a patient with no known underlying disease who developed reversible granulocytopenia within several weeks of beginning treatment with captopril.

## CASE

A 36-year-old woman was admitted to hospital with agranulocytosis and fever. She had a nine year history of essential hypertension, poorly controlled on propranolol 20 mg t.i.d., furosemide 20 mg b.i.d. and prazosin 1 mg t.i.d. Previous investigations for a secondary cause had been negative. Nineteen days prior to admission, captopril 12.5 mg q.i.d. was started and prazosin discontinued. Blood pressure control improved. Routine blood work at that time was normal (WBC count was 4.9 with 57% neutrophils, 42% lymphocytes and 1% eosinophils).

Twelve days after starting captopril the patient complained of abdominal cramps, nausea, diarrhea, flushing and a temperature (39.4° C). Blood work revealed a hemoglobin of 14.3 g/dL, WBC count of 4,700/mm<sup>3</sup> (70% neutrophils, 26% lymphocytes and 1% bands) and an erythrocyte sedimentation rate of 15. Seventeen days following the initiation of captopril treatment the patient complained of "cold sores" on her mouth. Hemoglobin was 12.5 gms/dL and total WBC was 1,000/mm<sup>3</sup>, with a differential of 96% lymphocytes and 4% eosinophils. Two days later she was admitted to hospital. There was no history of drug or toxin ingestion other than her antihypertensive agents.

On examination, she had some tender lymphadenopathy in the anterior cervical chain and jugulodigastric nodes. There was no hepatomegaly or splenomegaly. No clinical signs of infection were identified. Vital signs were: BP 102/76 mmHg, Temp 38.5° C. Repeat CBC confirmed the absence of neutrophils. It was felt that the patient had agranulocytosis, most likely secondary to captopril therapy. Serum creatinine was 108 mm/L.

Her treatment consisted of reverse isolation after appropriate cultures. Intravenous ticarcillin and tobramycin, plus oral cotrimazole, were administered. Captopril was discontinued. Within ten days she had recovered, and her WBC was 6,700/mm<sup>3</sup> (76% neutrophils, 16% lymphocytes, 6% monocytes and 3% eosinophils).

Following this hospitalization, she was again evaluated for renovascular hypertension or collagen vascular disease. A renal digital subtraction angiogram showed a single renal artery on the left and three renal arteries on the right, with no evidence of stenosis. Rheumatoid factor, LE cells and ANA titres were negative. She refused treatment with enalapril and is now receiving furosemide, propranolol and prazosin, in the same doses as previously.

## DISCUSSION

Neutropenia secondary to captopril administration has been documented in a very small number of patients to date<sup>4</sup> and with one exception, in patients with other medical problems such as renal insufficiency, renovascular hypertension and collagen vascular disease.<sup>5, 6, 7, 8, 9, 10, 11</sup> Early studies with captopril confirmed that patients with uncomplicated hypertension had a very low risk of developing neutropenia with captopril. The risk of captopril-induced granulocytopenia was felt to be equivalent to that existing with other commonly used drugs.<sup>1</sup>

One previous case of neutropenia in a male patient with severe hypertension but otherwise excellent general health was reported by Gavras and Gavras in 1982.<sup>3</sup> This patient experience a drop in his white cell count from 5600 per cubic millimetre at the start of therapy to 3200 five weeks later. The onset of the neutropenia was slower than seen in our patient and of less severity. Recovery occurred rapidly after cessation of drug therapy, but a rechallenge with captopril demonstrated a similar reduction in white cell count which subsequently returned to normal, despite continuation of

\*Medical Student, Dalhousie University, Graduated May 1988.

\*\*Department of Medicine and Pharmacology, Dalhousie University

†Family Practitioner, Amherst, Nova Scotia

Correspondence: Dr. Jean D. Gray, Department of Medicine, Room 392, Camp Hill Hospital, 1763 Robie Street, Halifax, N.S. B3H 3G2

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# Severe Nausea and Vomiting Associated with Captopril

Frank L. Ervin,\* MD, FRCPC, and Jean D. Gray,\*\* BSc, MD, FRCPC,

Halifax, N.S.

Nausea and vomiting are not rare as side effects of medications. Surprisingly, there has been very little mention of nausea and vomiting in the post-marketing surveillance studies of captopril.<sup>1,2</sup> In the United States a total of 6,737 patients with hypertension of varying severity were enrolled in such a study.<sup>1</sup> Dysgeusia was the commonest gastrointestinal side effect noted and no patients discontinued the drug because of nausea or vomiting. Using different data gathering techniques, 4,124 patients on captopril were followed in New Zealand.<sup>2</sup> Taste disturbance was again the most frequent gastrointestinal response, but a smaller number (data not provided) did report nausea and vomiting.

This report will review a case which appears to be an unusually severe manifestation of anorexia, nausea and vomiting resulting in a 15 kg weight loss temporally related to captopril therapy.

## CASE

A 63-year-old man was seen because of nausea, vomiting and weight loss. He had suffered a cerebrovascular accident ten years before, with little residual effect. Eight years prior to admission, he underwent a left renal artery bypass graft which significantly improved his then recently diagnosed hypertension. In 1980, a right ileofemoral and right superficial femoral to popliteal artery bypass graft was performed for symptoms of claudication. There was no impairment of renal function noted at this time.

The patient was admitted to his local hospital for treatment of left ventricular failure and hypertension. Captopril was prescribed, 25 mg t.i.d. Weight at discharge was 81 kg.

Three months later he was referred to Urology for assessment of renovascular hypertension, and complained of anorexia, nausea and daily episodes of vomiting that had begun three months earlier. These symptoms were exacerbated by eating and relieved by fasting. He had mild epigastric pain, postprandial cramps and indigestion. He also had complaints of weakness, lethargy and malaise. His weight on admission was 62kg, representing a 19 kg loss. Concern about a possible gastrointestinal malignancy resulted in a consultation to Internal Medicine.

Medications on admission included captopril, hydralazine 75 mg daily, prochlorperazine maleate 15

mg daily, furosemide 40 mg o.d., potassium replacement (8 meq twice daily in a slow-release tablet) and dymenhydrinate 100 mg daily.

His blood pressure was 160/88 mmHg. Bilateral carotid, left epigastric and bilateral femoral bruits were present. The abdominal exam was unremarkable with no hepatomegaly. There was no peripheral edema.

On admission, his hemoglobin was 10.7 g/dL, hematocrit 31.5%, MCV 86, MCHC 33.9, the ESR was 74 mm/hr and the leukocyte count was 7,900/mm. Mild proteinuria was present. The creatinine was 405  $\mu\text{mol/L}$  and the BUN was 19.5 mmol/L. The creatinine clearance was 15 mls/min. Collagen vascular evaluation was negative.

Investigations to exclude malignancy included an abdominal ultrasound which revealed no abnormality of the liver, gallbladder, bile ducts, pancreas or kidneys, and an upper GI barium study which revealed a normal appearing esophagus and stomach. There were thickened duodenal mucosal folds, but no evidence of ulceration or mass lesion. Renal angiographic findings were compatible with diminished cortical function bilaterally, with no evidence of significant renal artery stenosis.

Potentially nephrotoxic drugs were reviewed. Captopril was stopped and replaced with labetalol 100 mg t.i.d. He continued on furosemide, potassium and hydralazine as previously. Within 24 hours the patient's symptoms of nausea and vomiting resolved completely, and he was able to eat a normal diet for the first time in three months. He was discharged in a much improved condition. Since discharge he has done well, with one admission to his local hospital for an episode of left ventricular failure. Four months post discharge, his weight is 65 kg and serum creatinine is 360  $\mu\text{mol/L}$ .

## DISCUSSION

An extensive clinical experience with captopril both as a therapy for severe hypertension as well as monotherapy for mild hypertension suggests a low incidence of nausea and vomiting related to this drug. For this reason, the attending physician did not consider the drug as a possible cause for this patient's dramatic gastrointestinal symptomatology. Surprisingly, the patient also did not recognize the temporal relationship to the captopril therapy.

Most publications reviewing the side effects of captopril have focussed on dysgeusia as the major gastrointestinal side effect. Early data from studies using high dose captopril therapy (> 300 mg/day) reported an

\*Resident in Internal Medicine, Dalhousie University, Halifax, N.S.

\*\*Department of Medicine and Pharmacology, Dalhousie University.

Correspondence: Dr. Jean Gray, Dept of Medicine, Room 392, Camp Hill Hospital, 1763 Robie Street, Halifax, NS, B3H 3G2



incidence of 5.4% in patients with impaired renal function and 3.4% in individuals with normal renal function.<sup>1</sup> Loss of taste was the most common abnormality but the presence of a metallic taste occurred nearly as often.<sup>2</sup> Subsequent studies with lower doses of captopril (< 150 mg/day) showed incidences of 3.2% with normal renal function.<sup>1</sup> Although 0.7% of these patients had to discontinue therapy because of dysgeusia, no patients stopped the drug for nausea and vomiting. Dysgeusia is thought to occur because of the presence of a sulfhydryl group on the captopril molecule.<sup>3</sup> Enalapril, an angiotensin-converting enzyme inhibitor that lacks the sulfhydryl group has a lower incidence of dysgeusia of 0.2%.<sup>3</sup>

Other gastrointestinal side effects reported with captopril include oral<sup>4</sup> and tongue ulcers.<sup>5</sup> Patients with this side effect had high serum creatinine levels suggesting that ulcers may result from high plasma levels of captopril or a renally-excreted metabolite.

Our patient may have been susceptible to captopril-induced emesis because of previous cerebrovascular disease. Alterations in the blood-brain barrier may have allowed penetration of captopril or its metabolites into the central nervous system. Angiotensin and angiotensin converting enzyme are known to be present in normal brain. One postmortem study has demonstrated diminished quantities of angiotensin-converting enzyme in the corpus striatum and substantia nigra of patients with Huntington's disease.<sup>6</sup> The role of angiotensin-

converting enzyme in the brain is not fully understood, but the enzyme does play an important role in the generation of kinins. It is interesting to speculate that alterations in various neurotransmitters in the chemoreceptor trigger zone or the vomiting centre of the brainstem was responsible for this man's symptoms.

The rapid improvement in his symptoms once the drug was stopped implicates captopril as the precipitating factor. The patient was reluctant to attempt a rechallenge with captopril. □

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## GRANULOCYTOPENIA ASSOCIATED WITH CAPTOPRIL USE IN AN OTHERWISE HEALTHY PATIENT.

Continued from page 46.

captopril therapy. Our patient was reluctant to attempt a rechallenge, even with a related drug, such as enalapril.

Several fatal cases have been reported in patients with underlying renal disease who developed captopril-induced neutropenia.<sup>8,9,10</sup> As captopril is excreted by the kidney, the drug may have reached toxic levels in these patients, producing a concentration-dependent marrow toxicity. Cell lines other than the myeloid line were involved in at least two cases, suggesting a generalized myelosuppressive effect.<sup>8,11</sup>

The mechanism by which captopril produced agranulocytosis in our patient remains unclear. Fortunately, marrow suppression was reversible once the drug was discontinued. The temporal relationship to the onset of captopril therapy as well as the rapid improvement with discontinuation of the drug suggests that captopril can be implicated as the cause of the agranulocytosis in this otherwise healthy hypertensive woman. □

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# Endoscopic Dilatation of Benign Esophageal Stricture

P. F. Leahy, MB, M.Ch., FRCS(I),

Windsor, N.S.

Although bouginage is widely used to treat benign oesophageal stricture, the rate of stricture recurrence and the long term effectiveness of bouginage are unknown. This study deals with the effectiveness of bouginage in a patient population hitherto treated by medical therapy alone. The results of bouginage was also studied in a smaller group who had anti-reflux procedures performed for failed medical therapy.

Eighty percent of patients required repeated dilatation during the first year of treatment. There was a high index of patient satisfaction in those patients who had long standing disease. I was unable to identify any significant factors, such as initial severity of stricture, presence of active oesophagitis, or initial calibre of dilatation, that could predict the need for subsequent dilatation. My results suggest that patients with benign strictures fall into two groups. In one group, the natural history was to improve or become asymptomatic after an initial series of dilatations, and only a small proportion eventually developed recurrent symptoms. I conclude that bouginage is effective treatment for benign oesophageal strictures, and should be utilized as primary treatment for most strictures.

The total correction of oesophageal strictures secondary to peptic ulceration, by surgical means, carries a very high morbidity and mortality rate.<sup>1, 2</sup> A more conservative method of treatment is the use of esophagoscopy and unguided dilatation of the peptic strictures under general anesthesia. However, there is a higher instance of oesophageal perforation using this technique than by using the guided dilatation procedure.<sup>3</sup> Reflux esophagitis results in stricture formation in the distal third of the oesophagus in an estimated 11% of patients who suffer from peptic oesophagitis.<sup>4</sup> Guided dilatation of the oesophagus has been performed for many years using threads or wire guides<sup>5</sup>; in recent years, the Eder-Puestow system has achieved widespread use. This system has the advantage of passing a guide wire through the stricture by using a fibre optic endoscope, thus decreasing the likelihood of oesophageal perforation.<sup>6, 7</sup> I have reviewed the early results of peptic stricture dilatation using the Eder-Puestow dilator in a patient population previously treated by medical regimens and the occasional use of bouginage.

## PATIENTS AND METHODS

Between August 1987 and July 1988, 20 patients with dysphagia secondary to peptic oesophageal stricture were treated by fibre-optic endoscopic dilatation at this General Hospital. There were 15 men and 5 women with a mean age of 51 (range 38 to 82) years. Fifteen patients had stricture formation because of reflux oesophagitis, and five patients had stricture formation secondary to surgical procedures such as Nissen's fundoplication and cardiomyotomy. All patients presented with dysphagia for solid foods and decreased intake of semi-solid foods. The diagnosis of oesophageal stricture was confirmed at endoscopy. Histology was performed in all suspicious cases to rule-out underlying neoplasia or Barrett's oesophagus.

Strictures were dilated with the Eder-Puestow dilator, guided with a steel guide wire which was inserted down the biopsy channel of the forward viewing fibre optic endoscope (Olympus G.I. & F.).<sup>8</sup> Before dilatation, a guide wire was put through the stricture under direct visual control.<sup>6</sup> All strictures were dilated under sedation and local anaesthesia, and all procedures were performed on an outpatient basis. The patients were asked to return for follow up and subsequent dilatations if necessary. The patients also had medical treatment consisting of cimetidine, gaviscon and antacids and metoclopramide.

## RESULTS

There were no complications in this series. Altogether, 70 dilatations were performed. However, the patients had different underlying pathology. In group 1, the cause of stricture was primary reflux oesophagitis, 6 patients had Schatzki rings, this group was treated by dilatations and medical treatment (antacids, cimetidine, ranitidine, metoclopramide). Altogether, 42 dilatations were performed in this group (1 to 6 dilatations per patient, mean 2.5 dilatations). All patients were able to eat solid food after one year. Ten were asymptomatic but five remained symptomatic with periods of relief after further dilatation.

There were five patients with stricture formation due to primary reflux esophagitis, but in whom an anti-reflux procedure was performed, because of failed medical therapy. In this group, 28 dilatations were performed ranging from 1 to 7 (mean 4.5). At follow up 1 year later, three patients were asymptomatic and were able to eat solid foods, while two sometimes had dyspepsia and minor reflux symptoms.

From the Surgical Department of Hants Regional Hospital, Windsor, Nova Scotia, Canada.

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



## DISCUSSION

Results presented in this paper are clearly supportive of the evidence that fibre optic endoscopic dilatation causes symptomatic improvement in the overall condition of these patients,<sup>9,10,11,7,12</sup> Medical treatment is essential for the correction of the primary cause of this peptic ulceration and in this series cimetidine, ranitidine and antacids were shown to be equally effective and were always used. The method of treatment is to primarily treat the primary disease — reflux esophagitis — and to restore the swallowing and nutritional status of the patient. These results confirm the findings of others that medical anti-reflux treatment with esophageal dilatation is sufficient in the majority of cases to maintain the patency of the oesophageal stricture in the patient population with strictures secondary to reflux esophagitis. The results are comparable to others in whom favourable results have been achieved using a conservative approach.<sup>1,9,12</sup> In addition to all the other cases of peptic-stricture formation, these patients will be pursued life long in order to ascertain the natural history of peptic-stricture formation.

## CONCLUSION

The present study confirms that endoscopic dilatation is effective and safe in the treatment of peptic-esophagitis. I was unable to identify any significant factors such as initial severity of stricture, presence of active oesophagitis that could predict the necessity for subsequent dilatation.<sup>13,14</sup>

My studies do suggest that patients with benign strictures fall into two groups. In one group, the natural history was to improve and become asymptomatic after an initial series of dilatations, and only a small proportion eventually developed permanent symptoms. The second group required further dilatations to treat dysphagia during the first year of follow-up. In this group, bile reflux was a consistent finding. This group

of patients is currently undergoing further studies at this Hospital to measure conjugate bile acids in refluxate to determine if the bile acids play a role in the pathogenesis of this esophagitis.<sup>15</sup> □

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# An Unusual Case of Obturator Hernia

P.F. Leahy, MB, M.Ch., FRCS(I),

Windsor, N.S.

Obturator hernias are relatively rare. Elderly women with chronic disease are most frequently affected. The Howship-Romberg sign is useful in diagnosing this rare condition. The often debilitated state of the patient with obturator hernia and the frequent delay of diagnosis combine to produce significant operative mortality.

Strangulated obturator hernia is a rare but a well known cause of intestinal obstruction especially in elderly, emaciated women. Because of frequent diagnostic delays the mortality rate is high, ranging from 13% to 17%.<sup>1,2</sup> The diagnosis should be suspected in patients with abdominal colic and Howship-Romberg sign, i.e. pain radiating medially in the thigh due to compression of the obturator nerve.<sup>2</sup> Because of the potentially lethal outcome, prompt surgical intervention has been advocated as soon as the diagnosis is made.<sup>3</sup>

Obturator hernias are very rare and few surgeons have seen many cases. However, the condition is curable by operation and diagnosis is easy to make.

I present a case description of a very unusual obturator hernia transmitting the left ovary which contained an ovarian cyst.

## CASE

A very frail 89 year lady presented to her own GP complaining of vague discomfort in her left groin. It was constant but not extremely painful, and she described the pain as radiating down the inner aspect of her left thigh. She had no associated symptoms but a clinically detectable swelling was palpable in the left labia majus. She was admitted to hospital and routine investigations including CBC, blood urea nitrogen, electrolytes and plain film of abdomen were normal.

A surgical opinion was sought. The surgeon described an irreducible swelling in the left labia majus which on bimanual vaginal examination could be traced easily to the obturator foramen. The swelling was firm and non translucent. A tentative diagnosis of an obturator hernia was made. It was clinically certain that the hernia did not contain

bowel as the patient was very well and had a normal soft abdomen.

The patient refused surgery but consented to an aspiration of the groin swelling and examination under sedation. At this examination 500 cc of chocolate fluid was aspirated from the groin swelling, and after aspiration the structure was easily palpated and had all the characteristics of an ovary. The suspending structures could be palpated emanating through the obturator foramen. The ovary was reduced and the patient's condition improved. This was followed by a normal post-operative course and she was discharged from hospital.

## DISCUSSION

Obturator hernia occurs through the obturator canal which is situated at the upper part of the obturator membrane covering the obturator foramen, which transmits the obturator vessels and nerves.

Obturator hernia was first described by Arnold De Ronsil in 1724. Hilton first performed a laparotomy for the condition in 1848. Sir Cecil Wakely described the anatomy of the hernia in 1939, comprehensively reviewing the literature and describing two cases of his own.

Obturator hernia nearly always becomes manifest only when strangulation supervenes, although occasionally a non-strangulated hernia may present with groin discomfort and with pain radiating to the knee caused by pressure on the adjacent nerves. Vaginal examination will confirm the diagnosis, a tender mass being felt in the region of the obturator foramen. This is palpable as a lump in the femoral triangle on bimanual examination through the vagina.

With the introduction of herniography, a radiologic technique using intra-peritoneal injection of an iodine contrast medium, obturator hernia can be demonstrated more frequently than hitherto.

Up to 1980 fewer than 600 cases of obturator hernia had been reported in the literature.<sup>1,5</sup> The female: male ratio is 6:1 and most of the patients were described as emaciated, very thin or frail. Most were older than 70 years. The overall incidence is greater on the right side, but in emaciated women the left side appears to be more susceptible.<sup>2,4</sup>

From the Department of Surgery, Hant's Regional Hospital, Windsor, N.S.

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



In this very unusual case, the patient presented with the classical emaciated features of the typical candidates who are susceptible to obturator herniation, as she weighed only 42kg. A definite diagnosis of which structure protruded through the obturator foramen could not be made before aspiration. The patient also refused laparoscopy and herniography which may well have presented further documentation on this unique case. As most surgeons will not see such a case, I present this brief description of an unusual groin swelling to remind us of the differential diagnosis in elderly emaciated patients. □

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#### OBITUARIES

**Dr. William A. Murray**, (75) of Halifax, N.S. died January 17, 1989. Born in P.E.I., he received his medical degree from Dalhousie Medical School in 1943 and, after the war, continued his studies in internal medicine. He practised internal medicine in Halifax for 30 years, he was consultant in medicine at the Victoria General and Camp Hill Hospitals, and was Assistant Professor of Medicine at Dalhousie University. He is survived by his former wife, and two sons, to whom the *Journal* extends sincere sympathy.

**Dr. Ian MacLeod**, (69) of Dartmouth, N.S. died on March 6, 1989. Born in Halifax he received his medical degree from Dalhousie Medical School in 1942. He was a veteran of the Second World War, returning to Canada in 1946 to specialize in pulmonary tuberculosis until 1950 when he was selected for training in radiology. In 1980 he became the first chief of radiology at the Dartmouth General Hospital and, upon retiring in 1985, he was awarded senior membership in The Medical Society of Nova Scotia. He is survived by his wife and a son. The *Journal* extends sincere sympathy to his family.

Continued on page 68.



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# Activation of the Mononuclear Phagocytic System

## IMPLICATIONS IN HUMAN DISEASE

Blaise E. Favara, MD,

Halifax, N.S.

The phenomenon of activation of the mononuclear phagocytic system and the actions of the resulting cytokines are important in a number of clinical conditions. The role of cytokines in human disease is just beginning to be appreciated.

A host of human maladies have their pathobiological bases in the phenomenon of activation of the mononuclear phagocytic system (MPS). A historical perspective on the MPS, beginning with Metchnikoff's 1882 discovery of phagocytosis in starfish larva is presented in Table I.<sup>1</sup> Cells of the MPS are active secretors of a remarkable number of biologically important substances, cytokines, or more specifically, monokines (Table II).<sup>2</sup> These cells also engage in physiochemical intercourse with lymphocytes through physical contact and cytokines (monokines from the MPS and lymphokines from lymphocytes) to form an immunologically dynamic duo. Endothelium is also a party to this intercourse.<sup>3</sup>

TABLE I

A thumbnail sketch of the history of the concept of the mononuclear phagocytic system

1882	Discovery of phagocytosis by Metchnikoff
1912	The peripheral blood monocyte discovered by Schilling
1924	Aschoff designated cells that were phagocytic as composing the reticuloendothelial system.
1939	Ebert and Florey showed that monocytes "matured" into macrophages.
1960	Mackness introduced the term "activation of macrophages"
1972	Van Furth proposed the term mononuclear phagocytic system for the continuum of monocytes, their precursors and their successors, macrophages

Although there is much to be learned about the phenomenon of activation of the MPS there is already a substantial literature on the subject. Cells of the MPS are either "down" or "up", regulated by certain "activators" causing them to secrete some monokines, to stop secreting other monokines, to proliferate, phagocytize, transform, etc. This causes interactions

\*Director, Departments of Pathology and Laboratory Medicine, I.W.K. Children's Hospital and Grace Maternity Hospital and Professor of Pathology and Pediatrics, Dalhousie University Faculty of Medicine, Halifax, N.S.

Correspondence: Blaise E. Favara, M.D., Department of Pathology and Laboratory Medicine, IWK Children's Hospital, PO Box 3070, Halifax, N.S. B3J 3G9.

with lymphocytes and endothelial cells. The resulting fever, cytopenias, constitutional symptoms, jaundice and/or shock are seen as epiphenomena in some primary conditions while MPS activation may account for the principal signs and symptoms of other disorders. There is also evidence that MPS activation may precondition the human host to pathological conditions. Examples of these three types of events will be presented briefly.

TABLE II  
SECRETORY PRODUCTS OF MACROPHAGES  
(Partial list)

Factors B,D,H,I	Prostaglandin E2
Interleukin 1	Thromboxane B2
Angiogenesis factor	6-Ketoprostaglandin F1 alpha
Erythropoietin	Leukotiene C
Colony stimulating factors	12-Hydroxyeicosatetraenoic acid
Interferon	Superoxidate anion
Fibroblast mitogen	Hydrogen peroxide
Endothelial cell mitogen	cAMP
Tumor necrosis factor	Thymidine
Tumor inhibition factor	Uracil
Listeria inhibitor	Plasminogen activator
Metal-dependent elastase	Type specific collagenases
Cytolytic proteinase	Lysozyme
Transglutaminase	Lipoprotein lipase
Arginase	Acid hydrolases
Phosphatases	Alpha-2 macroglobulin
Coagulation factors	Fibronectin
Thrombospondin	Apolipoprotein E
Complements 1-5	Properidin
Ferritin	Thromboplastin
Neopterin	Gangliosides

## GRAM NEGATIVE SEPTICEMIA AND ENDOTOXIC SHOCK

There is experimental evidence that endotoxic shock is a manifestation of limited activation of the MPS. Animals, given lipopolysaccharide (LPS) or endotoxin, develop fever, vascular collapse and other stigmata of the human counterpart of syndrome as the result of LPS activation of the MPS with consequent production of at least one monokine, tumor necrosis factor (cachectin).<sup>4,5</sup> Tumor necrosis factor (TNF), the monokine that is responsible for much of the pathophysiology of the syndrome of endotoxic shock, has also been found to be increased in the blood of patients with meningococemia<sup>6</sup> and in human subjects to whom small doses of endotoxin have been given.<sup>7</sup> Administration of antibodies to the monokine



prevents the development of the syndrome in animals.<sup>8,9,10</sup> Implications for therapeutic application in humans are not implausible.

### MPS ACTIVATION AS THE PREDOMINANT PATHOBIOLOGY

A rare familial disorder of infants, hemophagocytic lymphohistiocytosis, and the infection-associated hemophagocytic syndrome probably represent conditions, broadly referred to as hemophagocytic syndromes, in which there is more global activation of the MPS. Both disorders present with fever, pancytopenia, hepatosplenomegaly, signs of hepatic dysfunction, bleeding diathesis and neurological manifestations. Hypertriglyceridemia and spinal-fluid pleocytosis are characteristically present and there is lymphohistiocytosis and hemophagocytosis in the bone marrow (Figure 1).<sup>11</sup>



Fig. 1 A bone marrow macrophage displays a voracious appetite for leukocytes and erythrocytes in this striking example of hemophagocytosis. The open arrow points to the nucleus of the macrophage for orientation. Wright's stain. X400

In the case of the infection-associated hemophagocytic syndrome, which may mimic malignant histiocytosis, the activator of the MPS is a microbial agent, usually a virus such as Epstein-Barr virus or cytomegalovirus.<sup>12</sup> In the familial syndrome of hemophagocytic lymphohistiocytosis, a highly lethal disease with devastating brain pathology, it is possible that the affected child has a genetic defect that results in the failure of mechanisms that normally limit the effects of activators and cytokines; a deficit of containment. The suspected pathophysiology of activation of the

MPS and of the hemophagocytic syndromes is depicted in Figure 2.

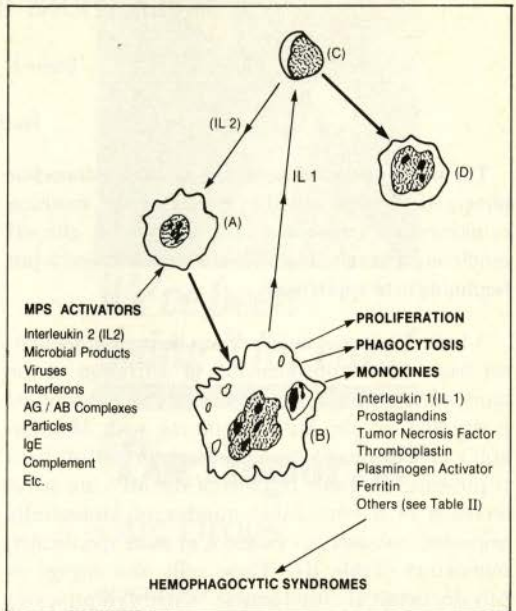


Fig. 2 Histiocytes\* (A), activated histiocytes (B), lymphocytes (C) and transformed lymphocytes (D) are linked in chemical dialogue through cytokines like IL 1 and IL 2, perhaps the most important MPS activator, with resultant proliferation and hyperactivity of some but not necessarily all cells. The role of endothelium is not depicted here.

\*The term histiocyte denotes macrophages of the MPS and dendritic cells. It is synonymous with macrophage as used here.

### MPS ACTIVATION AS A PRECONDITIONING EVENT

Chronicles concerning neopterin in human immunodeficiency virus (HIV) infection suggest that activation of the MPS and consequent transformation of lymphocytes preconditions the host to replication of HIV and progression to acquire immunodeficiency syndrome (AIDS).<sup>13</sup> Individuals with preactivated T lymphocytes and macrophages, such as those with other sexually transmitted diseases, or those who are intravenous drug users, when exposed to marginal amounts of HIV, may be more effectively infected since replication of HIV is facilitated by this preconditioning event. Strong correlations between levels of neopterin and the Walter Reed Staging Classification of HIV infections have also been documented.

For the pathologist, the morphological hallmarks of activation of the MPS, namely proliferation of macrophages and lymphocytes and hemophagocytosis, belie the paramount functional activities of the activated MPS. In the future, the identification and



quantitation of the cytokines and their actions will no doubt be an important function of the clinical laboratory and the results of such studies will form the basis of newly developed chemotherapy for disorders in which there is activation of the MPS. □

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## Career Opportunity

### Epidemiologist

The Department of Health & Fitness invites applications for the position of Epidemiologist to be located in Halifax, Nova Scotia.

#### Duties

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#### Qualifications

The successful applicant will require an M.D. plus post-graduate training in epidemiology. Fellowship of the Royal College of Physicians and Surgeons is the preferred qualifications, but equivalent training and experience may be an acceptable alternative.

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Further information may be obtained from Dr. Wayne Sullivan, Administrator, Community Health Services, Nova Scotia Department of Health & Fitness, P.O. Box 488, Joseph Howe Building, Halifax, Nova Scotia, B3J 2R8.

Applications and/or resumes should be submitted to the Nova Scotia Civil Service Commission, P.O. Box 943, Halifax, Nova Scotia, B3J 2V9.



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# Gestational Trophoblastic Disease Registry

## UPDATE 1987

R.C. Fraser,\* MD, FRCSC, I. Zayid,\*\* MD, FRCPC,  
M.L. Givner,† PhD, and M. Trott,†† RN.

Halifax, N.S.

A total of 29 new patients were entered into the Nova Scotia Gestational Trophoblastic Disease Registry and Surveillance Program in 1987. Seventeen patients were confirmed to have benign hydatidiform mole (HCG titres returned to normal with no treatment other than the original D & C); one patient developed N.M.G.T.D.; one patient presented with a placental site trophoblastic lesion; ten patients had partial (incomplete) mole. (Table I)

TABLE I

### THE EXPERIENCE OF THE REGISTRY IN 1987

Benign Mole	17
NMGTD	1
MGTD	0
Partial Mole	10
Placental Site Trophoblastic Lesion	1
TOTAL	29

### DIAGNOSIS

Ultrasound plays a major part in the diagnosis of the hydatidiform mole. Of the eighteen patients referred to the registry, sixteen had ultrasound determinations performed. Eight were positive for hydatidiform mole. Eight patients who had negative ultrasounds were subsequently found to have a molar pregnancy. This represents a false negative rate of 50% for ultrasonography, an increase from the 18% false negative rate in 1986.

Six patients were diagnosed following uterine curettage for the following: incomplete abortion (2), missed abortion (3), therapeutic abortion (1).

### Titres Normal

Of the seventeen patients confirmed to have benign hydatidiform mole, all seventeen patients had Beta HCG titres that returned to normal in 4-8 weeks.

From the Nova Scotia Gestational Trophoblastic Disease Registry, Department of Obstetrics and Gynecology, Dalhousie University, Halifax, N.S.

\*Head, Department of Gynecology, Victoria General Hospital.

\*\*Director of Anatomical Pathology, Department of Pathology, D.J. MacKenzie Diagnostic Centre.

†Head, Endocrinology Lab, Victoria General Hospital, and Associate Professor Pathology, Dalhousie University.

††Co-ordinator GTD Registry.

### GLOSSARY:

D & C — Dilatation and Curettage

NMGTD — Non-metastatic gestational trophoblastic disease

MGTD — Metastatic gestational trophoblastic disease

HCG — Human chorionic gonadotrophin (serum-Beta sub unit assay)

### NMGTD

One patient developed non-metastatic gestational trophoblastic disease and required adjunctive chemotherapy to eradicate her disease. She had HCG titres that gradually decreased to 97 but at the five week follow-up they started to rise again. A repeat D & C was performed; showing tissue that was 'not molar' in origin. HCG titres continued to rise. She was treated with two courses of single agent chemotherapy (two different drugs) and remains in remission.

### TOTAL EXPERIENCE

A total of 416 patients (partial mole excluded) have been registered with the Nova Scotia Gestational Trophoblastic Disease Registry as of December 1987. (Table II)

TABLE II

### TOTAL EXPERIENCE (EXCLUDES PARTIAL MOLE)

	1965-70	1971-75	1976-80	1981-85	1986	1987
Nova Scotia	13(4)	27(8)	108(13)	72(14)	18(1)	12(1)
New Brunswick		8(6)	31(2)	46(8)	5(3)	3
Prince Edward Island			4(0)	9(0)	0	2
Newfoundland			15(4)	39(5)	2(2)	1
St. Pierre			1(0)			
Total number of patients					— 416	
Number requiring Rx (in parenthesis)					— 70 or 17%	

### Benign Disease

Three hundred and forty-five patients were confirmed to have benign disease requiring no treatment other than the original D & C. (Table III)

### Non-metastatic and Metastatic Disease

Fifty-three patients developed persistent non-metastatic gestational trophoblastic disease. Seventeen



patients developed metastatic disease. Nine of these patients had histologically confirmed choriocarcinoma and are dealt with separately.

TABLE III

Benign Mole	345
NMGTD post molar pregnancy	50
MGTD post molar pregnancy	11
Choriocarcinoma	9
	415
Non-gestational choriocarcinoma	1
	416
Placental site trophoblastic lesion	1
	417

### Choriocarcinoma

Nine patients had histologically confirmed choriocarcinoma. Six of the nine had metastatic disease and three non-metastatic. (Table IV)

TABLE IV

**CHORIOCARCINOMA**  
(confirmed histologically — 9 patients)

Post ectopic	(M)	1
Post molar pregnancy	(NM)	1
Post normal pregnancy	(M)	4
	(NM)	1
Etiology unknown	(M)	1 (recurrent chorio — expired 1987)
Post hysterectomy — DUB positive preg. test	(NM)	1
Five patients alive and well		

NM = nonmetastatic  
M = metastatic

Four of the six patients with metastatic choriocarcinoma died. All three were diagnosed following a normal pregnancy and died of either advanced disease or complications of chemotherapy.

Five patients (two with non-metastatic and three with metastatic disease) are alive and well following adjunctive chemotherapy and/or surgery.

### Non-gestational Choriocarcinoma

One patient presented with non-gestational metastatic choriocarcinoma. (See case study)

### Choriocarcinoma

During the 1987 year there were no new cases of choriocarcinoma registered.

### FOLLOW-UP CASE STUDY

For your information follow-up of the patient who presented with non-gestational metastatic choriocarcinoma as presented in the 1986 Trophoblastic Disease Report is chronicled.

This twenty-five year old (XY) woman presented in 1986 with metastatic non-gestational choriocarcinoma. Initially she received six courses of modified Bagshaw's regimen (EMACO), followed by abdominal hysterectomy, bilateral salpingo-oophorectomy (streak gonads) and a biopsy of the omentum. No visible tumor was found but necrotic areas of the left "gonad" and uterus were present. Following surgery five additional courses of the same chemotherapy regimen (eleven courses in all) were administered.

She continued with weekly Beta HCG titres. Our titres (at the Victoria General Hospital) remained normal at 4.0 IU/L. The Beta sub unit radioimmunoassay performed in conjunction with the New England Gestational Trophoblastic Disease Center at the Brigham and Women's Hospital became elevated (as high as 194 picograms with a normal value being <50).

In May 1987, after receiving several abnormal Beta sub unit HCG's from Boston, chemotherapy was recommended. She received vinblastin and bleomycin on day 1, bleomycin alone on day 2, 3, 4, 5 and Cisplatin® on day 6. A second course of the same therapy was given in June, 1987. The Beta sub unit HCG titres in Halifax remained <4.0 but the Boston titres continued to rise.

Chemotherapy in July was modified to VP16 (etoposide) administered over a five day period. A second course was administered in August. Chest X-ray and full lung tomograms were negative. Beta sub unit HCG titres at the Victoria General Hospital now also rose (5.8-4.5).

Further history taking revealed that the patient had discontinued her replacement hormonal medication unknown to anyone. In light of this information, she was started on hormonal medication (combination) in September. The Beta sub unit HCG titres fell to <4.0 at the Victoria General Hospital with the LH and FSH levels decreasing rapidly. Finally in December her Beta HCG titre from Boston was <50 picograms. Since then, additional Beta HCG titres over a two month interval have returned with normal results. The patient has been taken off hormone suppression using combined medication and placed on Premarin 1.25 daily for replacement.

No further chemotherapy has been necessary and to date all titres are normal and she remains in remission.

### THE INCOMPLETE OR PARTIAL MOLE

In 1981 a study to evaluate the clinical significance of the partial mole (hydatidiform degeneration) was begun.

In 1987 ten patients were diagnosed with partial (incomplete) mole. To date forty patients with partial mole have been followed by the registry. Thirty-nine patients have had their pathology reviewed by Dr. I. Zayid of the Dr. D. J. MacKenzie Diagnostic Centre, Victoria General Hospital, Halifax, N.S. Two patients were lost to follow up.



Thirty-two patients had HCG titres which returned to normal in eight weeks or less. The remaining six patients entered the study late (five at 11 weeks; one at 22 weeks) and their titres were normal at entry.

Follow-up for all thirty-eight patients has been uneventful and ranged from five to twelve months (thirty-six patients) to one to two months (two patients). Of the nine patients in 1987 being followed, eight patients had titres return to normal in 4-6 weeks and one patient had normal titres at the 10 week date.

The registry will continue to recommend 6 months of follow-up with HCG titres for the patient with partial mole. This decision is based on recent literature which suggests that 5-9% of patients with partial mole will develop persistent gestational trophoblastic disease. (Natural history of the partial molar pregnancy. *Obstet Gynaecol* 1985; **66**: 677-681).

### FOLLOW-UP RECOMMENDATIONS

Approximately 17% of the patients who have had a molar pregnancy will require adjunctive chemotherapy and/or surgery to eradicate their disease. For this reason follow-up with HCG titres is essential and cannot be too greatly emphasized.

Registration with the Trophoblastic Disease Registry is recommended for all cases and can be made by writing to the:

Nova Scotia Gestational Trophoblastic Disease Registry  
Ambulatory Care Centre, Victoria General Hospital  
Halifax, N.S. B3H 2Y9  
or by phoning (902) 428-2263.

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

(All patients should be placed on effective birth control measures for an appropriate time period.)

#### After hospital discharge:

- a) HCG weekly until three consecutive normal levels are achieved. Then. . . . .
- b) HCG monthly for one year. Pregnancy is permissible after 6 months of normal titres. If pregnancy is suspected an ultrasound is indicated for early confirmation.

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

- a) HCG weekly until three consecutive normal levels are achieved. Then. . . . .
- b) HCG monthly for one year. Then. . . . .
- c) HCG once every three months for one year. Pregnancy is permissible after 12 months of normal

titres. If pregnancy is suspected an ultrasound is indicated for early confirmation.

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

- a) HCG weekly until three consecutive normal levels are achieved. Then. . . . .
- b) HCG once a month for two years. Pregnancy is permissible after two years of normal titres. Once again if pregnancy is suspected an ultrasound is indicated.
- c) HCG once every three months for the third year.
- d) HCG once every six months for the fourth year.
- e) Yearly thereafter. □

### ACKNOWLEDGEMENTS

Best wishes and thank you to Bev Pierce who has been our co-ordinator of the Trophoblastic Disease Registry for many years. As of January 1, 1989, John Jeffrey, MD, FRCS(C) assumed the directorship of the Trophoblastic Disease Registry from Dr. R.C. Fraser. We anticipate the continued co-operation of the patients, physicians and pathologists in the future and express our appreciation for their support in the past.

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## KLINEFELTER'S SYNDROME

is a condition characterized by the presence of small testes, with fibrosis and hyalinization of seminiferous tubules, without involvement of Leydig cells, and by increase in urinary gonadotrophins, associated with an abnormality of sex chromosomes.

Klinefelter's Syndrome Association of Canada has the following goals:

- i) to educate the public, those afflicted and their families;
- ii) to promote early detection of the syndrome;
- iii) to operate a central depository of information on the syndrome;
- iv) to create and operate support groups;
- v) to encourage research into ending this affliction.

Physicians interested in the syndrome are invited to contact Mr. D.J. Rabinovitch at P.O. Box 5000, Penetanguishene, Ontario L0K 1P0.

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# Dr. Johnson and Dr. Heberden

T.J. Murray,\* MD, FRCPC, FACP, and Ernest Heberden,\*\* MA,

Halifax, N.S.

*"Had Johnson lived at a later date, science would have been able, if not to cure his oddities, at least to name them."*

## DR. HEBERDEN

The physician William Heberden (1710-1801) who attended Johnson during the last two years of his life, had, like his patient, risen from any modest beginnings to a lasting place in the annals of history (Figure 1). The relationship between the distinguished medical practitioner and the giant of 18th Century literature is perhaps of interest to historians of both.



Fig. 1 Dr. William Heberden, referred to by Johnson as, "ultimus romanorum. The last of our learned physicians".

\*Dean of Medicine, Dalhousie University, Halifax, N.S.

\*\*A descendant of William Heberden.

Correspondence: Dr. T.J. Murray, Dean of Medicine, Dalhousie University, Halifax, N.S. B3J 4H7

Heberden was the son of a Southwark innkeeper who died when he was seven. He was admitted to the free grammar school in the parish of St. Saviour's, where, under an able headmaster, he received the classical education that was essential for entry into the learned professions. Proceeding to St. John's College Cambridge as a sizar (the most penurious form of undergraduate life) with little in his pockets apart from an exhibition from the school worth £7 a year, he was soon able to balance his modest budget with the help of a "Foundress Scholarship for the Bell" — a phrase denoting his duty to ring the bell for chapel services.

At the age of 21, his academic prowess was rewarded by his election to a Fellowship and it was from that time that he began his study of medicine. Having achieved his MD, Heberden soon distinguished himself as a lecturer on *Materia Medica* and as the author of the brief but outstanding *Introduction to the Study of Physic*. He was elected FRCP in 1746 and settled in London two years later. He is remembered today for his descriptions of angina pectoris and digital osteophytes (Heberden's nodes).

His friendly disposition and love of conversation made him eminently "clubbable" and he was soon elected to the Royal Society, where he took an active interest in subjects as diverse as astronomy, electricity, horology and rainfall.

By 1761 his talents had brought him to the notice of the young George III, who offered him the prestigious Court appointment of Physician to Queen Charlotte. Though he refused the invitation on the grounds that it might interfere with his existing practice, he was tactful enough to avoid ruffling any royal feathers, and remained on good terms with the King throughout his life.

Heberden began to withdraw from medical practice in 1782 and used his new-found leisure to complete the Latin text of his major work *Commentaries on the History and Cure of Diseases* (published after his death by his son and namesake in 1802). Until his final retirement in 1788 he continued his life-long habit of making case-notes and transferring significant facts into a Latin Index. It is this Index, preserved in The Royal College of Physicians in London, that records the consultations on Johnson's stroke, painful sarcocele, asthma (heart failure) and his autopsy.

## DR. JOHNSON

Heberden was blessed with excellent health, abundant energy and a buoyant temperament — factors that helped him to pursue his career with uninterrupted



success. By contrast, Johnson's studies at Oxford were curtailed by poverty. His brief experience as a schoolmaster ended in failure and his literary output brought little financial reward until at last he received royal recognition and a pension of £ 300 a year. Ill health had dogged him from the cradle. (Table I)

TABLE I  
THE ILLNESS OF DR. SAMUEL JOHNSON

1. Birth trauma, possibly with anoxia.
2. Ophthalmia neonatorum.
3. Lymphatic tuberculosis.
4. Deafness left ear.
5. Myopia, worse in the left eye.
6. Manic depressive — depressive.
7. Obesity.
8. Gout.
9. Sarcocele.
10. Tourette's syndrome.
11. Emphysema.
12. Left middle cerebral infarction.
13. Cardiac failure and cor pulmonale.

Johnson was born to Michael and Sarah Johnson on Wednesday, September 18, 1709, in Lichfield, Staffordshire. Michael was 52 and Sarah 40 and, because there was great concern for a first pregnancy at such a late age, George Hector, a prominent Lichfield surgeon, was called rather than a midwife. In 1709 a "man midwife" was unusual. Johnson later stated that he was born almost dead and could not cry. There must have been serious concern about the infant's survival because the vicar from St. Mary's parish was called within a few hours to baptise the baby in the mother's bedroom. Dr. Swinfin, a lodger at the Johnson house, stood as the godfather.

In his childhood other medical problems developed. At ten weeks of age it was noted that his eyesight was poor and it was felt that his left eye might become blind, probably due to infection. He developed scrofula, or lymphatic tuberculosis, known as "the King's evil" from the belief that it could be cured by the touch of the Monarch. Johnson was touched by Queen Anne at one of the last of these ceremonies, and presented with a "touch piece" — a golden coin, which he kept all his life. It now resides in the British Museum.

His poor vision is suggested in many of his portraits; in one of these Reynolds showed him squinting over a book (Figure 2); Johnson disapproved and said he did not want to be known for his defects only. He added that Reynolds "could paint me deaf if he chooses, but I will not be blinking Sam.". In his dictionary he defines a blinkard as, "one who has bad eyes".

His comment about hearing is appropriate because he was deaf in his left ear. There are more than a dozen references in his own writings to his hearing problems which he felt resulted from the scrofula. It has been said that his dislike of the visual arts was due to his visual difficulty and his dislike of music and the theatre was due to his poor hearing.



Fig. 2 Dr. Samuel Johnson squinting to read. Poor vision was but one of his many medical problems.

During his life Johnson was subject to recurring depression and always frightened of impending insanity. He was immoderate in many things, including alcohol at various times, but there is little to support the suggestion that he was an alcoholic. He found it easier to abstain than to be moderate. During the years when he did not drink alcohol he drank huge amounts of tea.

Johnson also had a lifelong pattern of Gilles de la Tourette Syndrome, characterized by unusual tics and gesticulations, involuntary vocalizations and dramatic compulsive behaviour.

## HEBERDEN AND JOHNSON

We do not know when Heberden first met Johnson. Boswell mentions Heberden for the first time in connection with Topham Beauclerk, a great-grandson of Charles II and Nell Gwynn, and an original member of the Club founded by Johnson and Sir Joshua Reynolds, which met at the Turks Head coffee house in Gerrard Street:

21 March (1775): Johnson informed me that though Mr. Beauclerk was in great pain, it was hoped he was not in danger, and that he now wished to consult Dr. Heberden to try the effect of a new understanding.



Although Johnson did not himself become Heberden's patient until 1783, he had already recommended him to others, including the Rev. John Taylor, Vicar of Ashbourne in Derbyshire, who regularly visited London. In a letter advising his friend to take care of himself, Johnson writes:

Heberden's talk was rather prudential than medical: you might however perceive from it how much he thought peace of mind necessary to your re-establishment.

Presumably Johnson was present at this consultation and appreciated the importance of the "prudential" element in the advice. Heberden indeed considered that general advice to his patients on how they should take care of themselves was often of greater benefit to them than any of the accepted medical routines such as bleeding. His main concern with all patients was to assist the *vis medicatrix naturae* (in which he so firmly believed) to do its beneficent work.

When the wealthy brewer Mr. Thrale became ill, Johnson was staying in the country and Mrs. Thrale wrote to him regularly to give him news of her husband's progress. In June, 1779 he replied to one of her letters:

. . . I am glad that you have Heberden and hope his restoratives and his preservatives will both be effectual.

Four months later, the Thrales decided to visit Tunbridge Wells, and Johnson wrote again:

I earnestly wish that before you set out, even though you should lose a day, you would go together to Heberden and see what advice he will give you . . . I wish you would do yet more and propose to Heberden a consultation with some other of the doctors.

By this time Johnson's own health was failing and his friends were worried by his tendency to ignore his problems and to put off making his will. He was clearly suffering at this time from recurrent respiratory symptoms and heart failure.

At age 71, in 1780, he wrote to Mrs. Thrale saying, "Last year I perceived the remission of those convulsions in my breast which had distressed me for more than 20 years". One might wonder if these were episodes of an arrhythmia, or the symptoms of chronic respiratory disease (noted at his autopsy years later).

When he was 72, Johnson began to suffer more from chronic shortness of breath and heart failure and he began to take a lot of opium for relief, often three grains a day. He eventually found that opium was not of much help and he gave it up. Although, like Heberden, he did not place much faith in many of the medical treatments in vogue at that time, including phlebotomy, he did get some relief from blood-testings, probably because of his heart failure.

## JOHNSON'S STROKE

Among the numerous doctors Johnson consulted during his life, his favorite for some years had been Dr. Thomas Lawrence, Registrar and later President of the Royal College of Physicians. When Lawrence died early in 1783, Johnson decided to ask Heberden to take his place. A few months later — on June 16th — Miss Frances Reynolds, sister of the great artist Sir Joshua Reynolds, was painting Dr. Johnson's portrait. Johnson did not like this portrait (it has been unkindly said about Frances Reynolds that she "painted pictures that made everyone laugh — and her brother cry").

Johnson was tired after the long day and went to bed. He awoke in the middle of the night with some symptoms which indicated to him that he had had a stroke. It was not clear what these symptoms were but he became alarmed about losing his mind as a result of the stroke. He then composed a prayer to preserve his mind and intelligence, and did this in Latin to test his capabilities. Later, he stated that it was not a good verse but he thought it of significance that he was aware that it was not. He was further relieved, that he could write the verse and do it in Latin. He then took some brandy, understanding that it was good for eloquence of voice, and fell asleep again.

When he awoke the next morning his speech was still impaired but he was able to write a note to his servant asking for Mr. Allan, his next-door neighbor, to come and assist him. He then wrote the following letter to his friend the Rev. Dr. Taylor, who was on one of his periodic trips to London:

June 17, 1783

Dear Sir, It has pleased God, by a paralytic stroke in the night to deprive me of speech. I am very desirous of Dr. Heberden's assistance, as I think my case is not past remedy. Let me see you as soon as it is possible. Bring Dr. Heberden with you if you can . . .

I think that by a speedy application of stimulants much may be done. I question if a vomit, vigorous and rough, would not rouse the organs of speech to action. As it is too early to send, I will try to recollect what I can, that can be suspected to have brought on this dreadful distress.

I have been accustomed to bleed frequently for an asthmatic complaint; but have forborne for some time by Dr. Pepys's persuasion, who perceived my legs beginning to swell. I sometimes alleviate a painful, or more properly an oppressive constriction of my chest by opiates; and have lately taken opium frequently, but the last, or two last times, in smaller quantities. My largest dose is three grains, and last night I took but two. You will suggest these things (and they are all that I can call to mind) to Dr. Heberden.

Johnson's toughness and presence of mind are apparent throughout his letter to Taylor; despite his



distress, he is entirely lucid and practical; he gives the maximum amount of information to help his new physician, and remembers to include some suggestions for his own treatment.

Heberden was at Windsor, where he had bought a house the previous year for his retirement; he came as soon as possible and prescribed blisters to be applied to the patient's head and throat.

On June 19th Johnson wrote to Mrs. Thrale saying:

In penning this note I had some difficulty; my hand, I knew not how or why, made wrong letters. I then wrote to Dr. Taylor to come to me, and bring Dr. Heberden, and I sent to Dr. Brocklesby who is my neighbour. My physicians are very friendly, and give me great hopes, but you may imagine my situation, I have so far received my vocal powers, as to repeat the Lord's Prayer with no very imperfect articulation. My memory, I hope, yet remains as it was! but such an attack produces solicitude for the safety of every faculty.

On July 3rd Johnson was able to inform Boswell that Dr. Heberden and Dr. Bröcklesby were sent for and came to offer advice:

They came, and gave the directions which the disease required, and from that time I have been continually improving in articulation. I can now speak, but the nerves are weak, and I cannot continue discourse long; but strength, I hope, will return. The physicians consider me as cured. I was last Sunday at church. . . .

The consultation was recorded in Latin by Heberden, and is preserved in the Library of The Royal College of Physicians in London. Johnson is not named but the description is clear:

Sudden loss of speech in a man of 74, without any impairment of limbs or mental faculties. Recovery virtually complete within a few days. 17 June, 1783.

His speech improved slowly over the next few weeks and Johnson clearly details his recovery in daily letters and notes. However, by the 37th day of his illness he was still complaining that he had some difficulty with his voice, although it had recovered very well. Critchley examines his writings in great detail over the period of his recovery and even subjected his letters and punctuation to statistical analysis to show the difficulties that he had with his handwriting and with his ability to communicate.

## SARCOCELE

Within two months Johnson was again in trouble with a testicular swelling; an operation was carried out to puncture and drain it and Johnson was then able to accept an invitation to stay with a friend near Salisbury. But the swelling returned and after three weeks he was obliged to travel home. He wrote to Heberden begging him to visit him again, but by the time he arrived the

fluid had drained spontaneously "with relief of all distress".

## DEPRESSION

To ward off "the black dog" of melancholy that dogged him all his adult life, Johnson now decided to found a new conversation club at a small tavern in Essex Street; Heberden was among those invited to join, and the first meeting was held on 8 December. But within a few days Johnson was once again housebound by an attack of arthritis that kept him in bed and only by the use of two canes could he get from bed to chair; his old enemies asthma and dropsy added to his difficulties and he had to suffer the attentions of a dentist to extract an abscessed tooth; yet despite all these afflictions there were days when Johnson was enthusiastic and in good humour and was able to preside at small dinner parties. At other times inevitably he felt depressed, and in February he wrote to Heberden:

Dear Sir, When you favoured me with your last visit, you left me full of cheerfulness and hope. But my distemper prevails, and my hopes sink, and dejection oppresses me. I entreat you to come again to me and tell me if any hope of amendment remains and by what medicines or methods it may be promoted. Let me see you, dear Sir, as soon as you can. I am, Sir, Your most obliged and most humble servant, Sam: Johnson.

## DROPSY

A few days later Johnson wrote to Boswell that the dropsy was gaining ground and that his legs and thighs were very much swollen with water; and on the 19th of February he discharged "in about twenty hours a full twenty pints of urine" — a diuresis possibly caused by taking mercury. By now he was very short of breath and suffering from orthopnea and paroxysmal nocturnal dyspnea. His ankle swelling had worsened and he was very weak. Although he was attended by Doctors Heberden and Brockelsby who shared the dubious honour of being his unpaid physicians, Johnson asked Boswell to obtain the opinion of the Scottish physicians about his case and Sir Alexander Dick, Doctors Gillespie, Cullen and Munro were all consulted by letter. In March he was prescribed vinegar of squills to control his heart condition and by mid-April was at last able to go out of doors. Three days later he insisted on attending a dinner to celebrate an exhibition of pictures at the Royal Academy in the newly built Somerset House, an event he reported in a letter to Heberden:

Not long after the first great efflux of water, I attained as much vigour of limbs and freedom of breath, that without rest or intermission, I went with Dr. Brocklesby to the top of the painters' Academy. This was the greatest degree of health that I have obtained, and this, if it could continue, were perhaps sufficient; but my breath soon failed, and my body grew weak.



At the beginning of May, Johnson was cheered by the arrival of Boswell from Scotland and the following month the two men set out by coach to visit a friend in Oxford. In July Johnson began an even more exhausting trip first to his hometown of Lichfield and from there to Ashbourne to stay with his friend Dr. Taylor.

Considering the discomforts of long distance travel, the tours were in Lord Britain's words "a heroic defiance of death" and when Johnson wrote to Heberden in October, it was clear that his condition had worsened:

At Oxford (in June) I was much distressed by the shortness of breath, so much that I never attempted to scale the library: the water gained upon me, but by the use of squills was in a great measure driven away.

In July I went to Lichfield, and performed the journey with very little fatigue in the common vehicle, but found no help from my native air. I then removed to Ashbourne, in Derbyshire, where for some time I was oppressed very heavily by the asthma; and the dropsy had advanced so far, that I could not without great difficulty button me at my knees.

After detailing the medicines he had taken and the effects they had produced, Johnson concludes:

The summary of my state is this: I am deprived by weakness and the asthma of the power of walking beyond a very short space. I draw my breath with difficulty upon the least effort, but not with suffocation or pain. The dropsy still threatens, but gives way to medicine. The Summer has passed without giving me any strength. My appetite is, I think, less keen than it was, but not so abated as that its decline can be observed by anyone but myself. Be pleased to think on me sometimes.

## THE LAST DAYS

By now, Johnson had only a few more weeks to live. He managed to make the return journey to London and Boswell records:

Johnson being asked in his last illness what physician he had sent for, "Dr. Heberden", replied he, "ultimus romanorum, the last of our learned physicians."

His friend and biographer, Sir John Hawkins, had for many months been pressing him to make his will and had even prepared a draft, leaving blanks for Johnson to fill in. Now at last Johnson dictated his will and signed it. To Heberden and several other friends he bequeathed "each a book at their election, to keep as a token of remembrance." He began to burn many of his private papers and made arrangements for stones and suitable epitaphs to be placed over the graves of his parents at Lichfield.

His edema was marked, his shortness of breath worse and he slept fitfully sitting in a chair. He made an effort to get up and around and said "I will not capitulate". But he became so dyspneic he could barely speak.

He lay helplessly in bed, and asked Dr. Brocklesby if he could recover. He was told that he could not without a miracle. Johnson said he would refuse all medication in that case so as not to meet God in a state of idiocy with opium in his head.

The surgeon, William Cruickshank, was asked to make cuts in his legs to drain the edema. Afraid of gangrene he made very superficial cuts. Johnson cried out "deeper, deeper. I want length of life, and you are afraid of giving the pain which I do not value." It was probably on this occasion that Johnson reproached Heberden with being "timidorum timidissimus" for supporting Cruickshank in refusing to make deeper cuts. When Cruickshank left, Johnson took scissors and stabbed deeply into the calves of his legs. He bled a great deal and Cruickshank had to be called back to dress the wounds. Although there is debate about Johnson's last words, Bate states that on Monday, December 23, Johnson in a delirium murmured, "Jam moriturus" (now am I about to die), echoing perhaps the ancient salutation of the dying gladiators to Caesar. He died that evening. (Figure 3)



Fig. 3 The death mask of Samuel Johnson. The scars of scrofula can be seen on his neck.

As Bates says in his biography: "With all the odds against him, he had proved it was possible to get



through this strange adventure of life, and to do it in a way that is a tribute to human nature."

## POSTMORTEM

An autopsy on Johnson was performed by James Wilson and his handwritten account can be found in the library of the Royal College of Physicians in London. His death mask shows the scars of scrofula and a slight right facial weakness from his stroke. His emphysematous lung was used as an example of this condition in Baillie's textbook for a century, with no indication of the origin of the specimen (Figure 4).

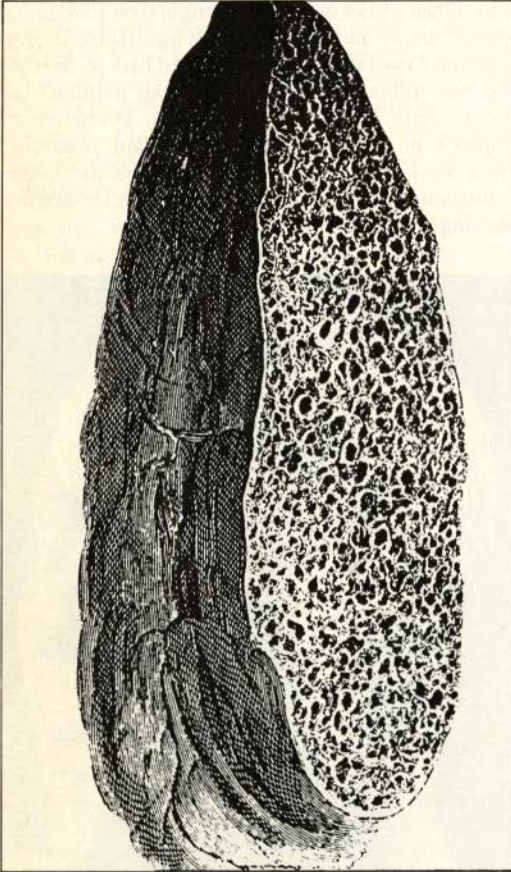


Fig. 4 The drawing of Johnson's emphysematous lung printed without comment on its origin in Baillie's textbook.

At his autopsy it was noted that:

"On opening the cavity of the chest, the lungs did not collapse as they usually do when air is admitted, but remained distended, as if they had lost the power of contraction; the air cells on the surface of the lungs were also very much enlarged".

A few days after the death of Johnson, Dr. Brocklesby, writing to inform Boswell of the sad news, included this illuminating passage:

The last time all the doctors consulted together when we entered his room he began thus from Swift. "The Doctors tender of their Fame, wisely on me lay all the blame. We own indeed his case was nice, but He would never take advice, He had been ruld for what appeared He might had livd these twenty years, for when we opend him we found his vital parts were sound."

"Now", says he, "Brocklesby will lay my death to disobedience and my taking lately 4 times as much squills as he advised. Dr. Heberden will say, I disturbed Natures operation in the outlet she made spontaneously in one leg, when I maugre all advice punctured myself the other leg which never ouzed any, but stopped by not ouzing the current of tother..." The good Man had his wishes answered for at last he dyed possessed of his mind, in a full vigour as ever and reconciled to the final close."

Johnson's good-humored prediction was very much to the point; he knew he had been a difficult patient and had frequently disregarded his physician's advice; moreover by "disturbing Nature's operation" he had acted against the *vis medicatrix naturae* instead of cooperating with it as Heberden would have wished.

Dirckx put forward the unusual suggestion that Heberden may have been at least partly responsible for Johnson's death by administering a lethal dose of digitalis in the treatment of Johnson's heart failure. Withering had reported the new remedy for dropsy, made from foxglove plants, in 1785, but his treatment learned from an old woman in Shropshire was becoming known to other physicians before this and Heberden knew of it. Dirckx feels Heberden gave Johnson an overdose of digitalis on December 31, 1784. Heberden's case notes suggest Johnson was given ten hourly doses of one spoonful of a solution made from 2 ounces (16 drams) of leaves of digitalis to 8 ounces of water. This is five times the strength of Withering's solution and could produce toxic effects and may have hastened Johnson's death.

## CONCLUSION

Although Heberden and Johnson had known each other for a comparatively short time, their relationship had become much closer than a merely professional one. If it was beyond the powers of any physician to cure the catalogue of Johnson's physical ailments, (Table I) his letters leave us in no doubt of the value he set on Heberden's visits; the latter's ability to evoke "cheerfulness and hope" was especially important to Johnson at a time when any of his earlier friendships, notably with the Thrales, had been terminated by death or other reasons. Heberden, close to him in age, sympathetic in temperament and sharing his love of literature and conversation, was able to alleviate some of his suffering and provide comfort during the closing months of his life. □



# Current Topics in Community Health

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

## THE HEALTH CARE SYSTEM UNDER SCRUTINY

Recent months have seen the national media carrying reports of ambulances with critically ill patients being re-directed by some Toronto hospitals, the shortage of nursing staff in critical care units, and the long lists of patients waiting for cardiac and hip operations. These are manifestations of a health care system struggling to cope with limited resources. While each sector may make a case out for increased resources, what is sometimes not appreciated is the interdependency of various components of the health care system — Peter is often robbed to pay Paul. Further, our Society's expectations of health care has been likened to the "onion skin effect" — as each layer of demand is peeled off, another one surfaces.

The appropriate use of different levels of care within the health care system is an important issue to be addressed if resources are to be used wisely. Criticisms have been directed at the inappropriate use by the public of emergency departments. This could be due to the lack of public education on appropriate use of such facilities as well as a reflection of inadequate primary care facilities in the area, among other factors.

The allocation of beds and operating time within hospitals is often based on historical precedent rather than on a rationale examination of need. The efficient use of beds may also be compromised by a lack of attention to pre-admission and discharge planning, and the paucity of support services in the community. Several hospitals have converted procedures traditionally done as in-patients to out-patient procedures, but the necessary changes in remunerative practices have been somewhat slow to develop. Trends towards day surgery for conditions managed previously with longer periods of hospitalization have also meant a more efficient utilization of existing resources. However, as "lengths of stay" shorten, it does not necessarily follow that hospital costs reduce, as the greatest costs are incurred in the initial days of hospital care. While mean lengths of stay have been used as standards to compare deviations from, the validity of these measures in the context of clinical outcomes is questionable.

Much has been written in recent months about intersectoral cooperation. This concept is best illustrated with an example. In planning for services for runaway children, it would be desirable to have on the planning committee representation from the housing authorities, police, hospital sector, family physicians, public health sector, social services, parental groups as well as representatives of those directly affected — the runaway

children. The potential contribution that each sector of society can make to the eventual resolution of what are complex problems has to be recognized, and mechanisms to enable this to occur have to be established.

Health promotion and disease prevention are only now being recognized as areas worthy of financial support. The lack of glamour and the long latency before benefits are realized often result in the displacement of funds in favor of the acute services.

The strengthening of community-based services such as home care programs can be expected to pay great dividends in the long term. In regions where additional funds cannot be injected into the development of such programs, the deinstitutionalization of patients can only be cost-saving when significant reductions in institutional beds accompany the process. The economic concept of marginal analysis suggests that significant cost-savings would only be made if whole wards or services shut down, with resultant lay-offs in manpower. Such measures are obviously unpalatable and difficult to realize.

When the health care budget pie is finite and cannot expand, the whole area of resource allocation begs serious study. Some countries have devised complex formulae taking into account such factors as standardized mortality ratios to allocate resources between regions, whereas others are struggling with the concepts of quality of life indicators as a means of deciding between different treatments. Decisions taken in these areas can have wide ranging impact on physicians' patterns of practice, and it is prudent for our profession to better understand the issues at hand and be a major player in the events that unfold.

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## THE GARBAGE BUSINESS

Recycling waste is attracting a lot of attention in the business world, as Patricia Lush of *The Globe and Mail* reports.

Businesses are finding ways to reduce the amount of waste they produce, to reuse or recycle some of what used to be garbage, to cut the amount of disposal and to diminish the hazardous properties of some wastes. In the process, they are saving money, using resources more efficiently and cutting pollution.

Scrap nylon from a plant in Kingston is being transformed into hockey helmets because of a philosophy at DuPont Canada Inc. that it should try to turn waste materials into saleable products. Winnipeg Photo



Ltd. recycles all of its developing and bleaching chemicals. The recycling equipment paid for itself in about a year. Developer now costs the company about 90 cents a gallon rather than \$3.40 off the shelf.

Recyclers are turning plastic soft-drink bottles into roofing, insulation and fibre-fill for pillows and parkas. Old tires and broken glass, mixed with asphalt, are making roads last longer. Further, businesses are finding new ways to make money from scrap materials, using advanced technology to make fence posts from used foam coffee cups and reviving classic ideas, including composting food waste into fertilizer.

Increasingly, industry is accepting, adopting and even promoting the philosophy inspired by the 1987 report of the United Nations Brundtland Commission: the world has to find sustainable forms of development or face environmental collapse. Businesses are pushed by their increasingly environmentally aware customers, shareholders, employees and communities. They are threatened by new, tougher laws, more aggressive prosecutors and stiffer fines. The cost of handling waste is also skyrocketing. Commercial tipping fees at dump sites in the Toronto area, for example, are jumping from \$18 a ton a few months ago to a forecast \$100 by next fall. The increases reflect both shrinking landfill capacity and more realistic accounting that includes, in addition to operating costs, the price of restoring the site when it is full and establishing a new dump to replace it.

Envionics Research Group Ltd. has found in its quarterly environmental surveys that 89 percent of

Canadians believe their health has already been affected by pollution. The public's fears are translating into enthusiastic participation — typically 80 to 90 percent — in household recycling programs.

The Federal Government has passed its long-promised Environmental Protection Act, promised \$306 million to clean up the St. Lawrence River and Halifax Harbor, set up a centre for Sustainable Economic Development in Winnipeg and started a program to identify environmentally preferred products. This year, Ontario is considering doubling its \$2.5 million fund to help companies install waste-reduction processes. It also plans to implement MISA — the Municipal Industrial Strategy for Abatement — a piece of legislation lauded by environmentalists as the most progressive in North America. MISA will require industry to use the best available technology to contain pollutants before they reach Ontario's waterways. Complying is going to cost millions of dollars to push even the most progressive companies to the limits of their technology and expertise.

Nevertheless, the business community tends to agree with MISA's philosophy while — predictably — complaining about its cost, extensive reach, and fast-track timetable. □

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Source: Patricia Lush. Good news from the garbage pile. *The Globe and Mail*. Report on Business. Saturday, February 4, 1989.

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## Notice Board

Notice Board will be a regular feature of the Journal consisting of topical developments pertaining to the more technological side of medicine. For further information, a contact person will be identified in each notice.

Possible contributors or suggestions to:

Dr. J. Phillip Welch, Professor of Paediatrics, Clinical Research Centre, 5849 University Ave., Halifax, N.S. B3H 4H7

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### CHORIONIC VILLUS SAMPLING

Chorionic villus sampling (CVS) is a means of obtaining fetal tissue late in the first trimester for purposes of chromosomal or biochemical study. Ideally, the procedure is carried out between nine and eleven weeks gestation. Most centres will report a karyotype within two weeks of sampling. The initial hope of an immediate report has not been borne out.

Results from a Canadian multicentered trial comparing chorionic villus sampling with amniocentesis has recently been reported. The findings indicate a miscarriage rate of 3-4% following CVS, carried out between 9-11 weeks gestation. This represents a loss rate of 1-2% in excess of the natural abortion rate at this gestation. This means, CVS carries a risk three times that of amniocentesis. Problems of chromosomal interpretation, particularly mosaicism, are more frequent in villus culture than in cell culture after amniocentesis.

It may be that chorionic villus sampling will turn out to be the prenatal diagnostic tool of choice in a certain select group of circumstances.

At the present time, the procedure is subject to much publicity, and many women request such testing. Work is underway to implement a program in Halifax but currently, women undertaking chorionic villus sampling are required to travel out of the Maritimes. It is important that women considering CVS understand the practicalities and implications of the procedure. Physicians wishing further information are advised to call Carole Smith, R.N., Prenatal Diagnosis Coordinator at the Atlantic Research Centre for Mental Retardation, telephone number 424-6491. □



# Appreciations

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## DR. CLYDE MARSHALL

Dr. Clyde Marshall died on July 11, 1988, after a distinguished career in medicine. He was the first Administrator of Mental Health Services for Nova Scotia, and he made this Province a leader in the field in Canada and the United States, through his innovative plan for the development of community-based mental health services.

Clyde Marshall was born in Halifax, Nova Scotia in 1900. He graduated from Dalhousie University Medical School in 1924. During his first year of practice in Senora, Guysborough County, he was granted a Rockefeller Fellowship to study psychiatry at the Boston Psychopathic Hospital and neurology at the Massachusetts General Hospital. He was to return to head up a new Department of Psychiatry at the Victoria General Hospital, one of only two or three such departments in Canada. However, while he was in Boston, the Nova Scotia Government changed, and the position at the Victoria General was no longer a priority; instead, the focus was to be on treatment of mental retardation.

Reasoning that he no longer had a commitment to return to Nova Scotia, he almost accepted another position. However, as he put it, "his conscience got the better of him" and he returned to the Province as a provincial psychiatrist, and became heavily involved in the establishment of the Nova Scotia Training School in Truro between 1927 and 1930. He returned to the United States in 1930 to do research in neurophysiology with Dr. John Fulton at Yale University and to teach in Yale Medical School's Department of Anatomy. In 1941, he came home for good and accepted a position as Neurologist and Professor at Dalhousie Medical School. He was also Head of Neurology, Neurosurgery and Psychiatry at the Victoria General Hospital and the Children's Hospital.

In 1945, Dr. Marshall became Administrator of the Nova Scotia Hospital and, when asked by the Government to evaluate the Hospital "in white heat," he wrote a stinging criticism of the Hospital and the whole mental health system in Nova Scotia, with recommendations to improve it. To his surprise, the report was read, and to his dismay, the Department of Health agreed to implement his entire program, provided that he would run it. In 1947, he became the first Administrator of Mental Health Services.

Shortly after his appointment, he instituted the Nova Scotia Mental Health Plan, known to many as the "Marshall Plan." This divided the Province into ten areas, all of which were to be served by psychiatrists in community Mental Health Centres, with referral services to the Nova Scotia Hospital and the Nova Scotia Training School. For chronic cases, there were community-based municipal or county hospitals. The second phase of the program involved the formation of Mental Health Centre Boards and placing Mental Hospitals within the framework of the Hospital Insurance Commission. *The Municipal Mental Hospital Act* of 1966 reflected these changes. Throughout his career, Clyde Marshall was known as a forceful, skilled negotiator, determined to provide comprehensive, community-based care for mentally-ill patients throughout the Province and to make treatment facilities for them efficient and humane and as similar as possible to those for physically-ill patients.

After his retirement in 1967, Dr. Marshall maintained his interests in photography, music and travel. Despite a long bout with cancer, he remained outgoing and deeply involved with people.

His wife, Frances, a psychologist, whom he met while in Boston, died in 1972. He is survived by two children, a son and a daughter.

Dr. Douglas Archibald  
Dr. William Draper

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## DR. ALLISON HOUSTON BARSS

Dr. Allison Barss passed away at home on December 1, 1988 at the age of 68. He was born in Rose Bay in 1920 and was the son of the late Dr. Geoffrey and Clara Barss.

He graduated from Acadia University with a B.Sc. and then attended Dalhousie University where he received his MD in 1944. He served in the Royal Canadian Air Force from 1944 to 1946 and commenced medical practice in Rose Bay, serving the community for more than 40 years. He was a member and past president of the Riverport Board of Trade, the IOOF Lodge, Riverport, and was a member of the Royal Canadian Legion Branch 23, Lunenburg. He was a member of the Medical Society of Nova Scotia, President of the Fishermen's Memorial Hospital



Medical Staff and belonged to both the Fishermen's Memorial Hospital and the Dawson Memorial Hospital Medical Staff.

He was involved in all community affairs in the area. He enjoyed gardening, painting, writing and travelled extensively around Nova Scotia and abroad. He particularly had a love for sailing and of the sea. He was born and practised out of the same house which his father before him had practised.

He is survived by his wife, Genowefa; two daughters, Anna, Toronto; Nancy, at home; five sons, Dr. David Barss, Bridgewater; Dr. Peter, Baltimore, MD; Dr. Kenneth, Halifax; Robert, Rose Bay; James, Martin's River; nine grandchildren.

He will be sadly missed by those who had the privilege of knowing and working with him over the years.

Sincere sympathy is extended to his family.

M.S. McQuigge, M.D.

## DR. HENRY A. MYERS

With the passing of Dr. Henry A. Myers of Amherst, the medical profession lost a very loyal supporter. He was 80 years old at the time of his death on December 18, 1988 at the Highland View Regional Hospital in Amherst.

Born in Moncton, N.B., he received his medical education in Edinburgh, Scotland and served in the Medical Unit treating prisoners of war. He returned to Canada in 1945 and started his practice of medicine in Amherst until his retirement in 1984 due to illness. For many years he worked as a Medical Examiner for Cumberland County. He was made a Grand Knight of the Venerable Order of the Hospital St. John of Jerusalem in 1980. He was a member of the St. John Ambulance Society for forty years, serving in the capacity as its director.

He was a parishoner of St. Charles Roman Catholic Church in Amherst.

He was a member of the Nova Scotia Medical Society and the Canadian Medical Society.

He is survived by his wife, Dora; a daughter, Lindsay (Mrs. John Murphy) of Antigonish; a son, Richard of Kingston, Ontario; a brother, Ralph of Moncton, N.B.; a sister, Harriett of London, Ontario; five grandchildren, Flora, Michael and Lindsay Murphy, and Kenneth and Anne Myers.

To his family we extend our sympathy.

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Continued from page 52.

**Dr. Clennel E. van Rooyan**, (81) of Halifax, N.S. died on March 16, 1989. Born in Crown Colony of Ceylon he received his medical degree in Scotland in 1934. In 1946, he emigrated to Canada and was Professor and Head of the Department of Virus Infections, University of Toronto until 1956, when he came to Halifax as Professor and Head of the Department of Microbiology, Dalhousie University and Director of Public Health Laboratories. He was made senior member of The Medical Society of Nova Scotia in 1986 and he was a member of the Canadian Medical Association. He is survived by his wife, a son and a daughter, to whom the *Journal* extends sincere sympathy.

**Dr. John K.G. Grieves**, (68) of New Glasgow, N.S. died on March 22, 1989. Born in London, England he received his medical degree in London in 1947 and then stayed to continue his studies in ophthalmology. He practised in Africa before going to New Glasgow and was one of the first physicians in Eastern Canada to perform corneal transplants. He was a member of The Medical Society of Nova Scotia and the Canadian Medical Association. He is survived by his wife and two sons. The *Journal* extends sincere sympathy to his family.

**Dr. T. Clare C. Sodero**, (79) of Truro, N.S. died on March 14, 1989. Born in Sydney he graduated from Dalhousie Medical School in 1935; he then continued his studies in surgery at the Toronto General Hospital. He was a member of The Medical Society of Nova Scotia and was nominated for senior membership in the Canadian Medical Association in 1983. He retired from his practise in Truro in 1984. He is survived by his wife and son, to whom the *Journal* extends sincere sympathy.

**Dr. Joseph P. McGrath** (95) of Kentville, N.S. died on April 6, 1989. Born in Stewiacke he graduated from Dalhousie Medical School in 1917. He served on the executive of the Medical Society, the council of the Canadian Medical Association, and was a member of the Provincial Medical Board. He was founder and first president of the Nova Scotia Society of Ophthalmology and Otolaryngology and received an honorary doctorate from Acadia University in 1972. He was made a senior member of The Medical Society of Nova Scotia in 1964 and was made a senior member of The Canadian Medical Association in 1966. He is survived by his son, to whom the *Journal* extends sincere sympathy.

□



# Correspondence

## To the Editor:

Dr. McCormick and Ms. June Penny have to be complimented for drawing attention to the hitherto most neglected aspect of the Hippocratic Oath ("Confidentiality, Lets Try Harder", by William McCormick and June Penny, in the *Nova Scotia Medical Journal*, Feb., 2 1989). I wholeheartedly agree with their contention that confidentiality is paramount in a doctor/patient relationship and it is a matter of grave concern that not enough weight has been placed on this important aspect of medical ethics.

My own feeling is that this neglect is partially due to the fact that the medical licensing authorities throughout the world have not been consistent in their rulings on the subject. Whilst I am aware that the General Medical Council in London takes a very serious view on breaches of confidentiality, I believe that it is tantamount to serious professional misconduct though some other countries, notably Australia, have been relaxed in the application of this standard. Indeed, looking through the annual reports of different licensing authorities, it is remarkable how few cases are brought up before the disciplinary committee.

The other possible reason for this ambiguity may be the legal requirement clause which compels a medical practitioner to divulge information normally regarded sacrosanct. As medical personnel, we all have to take a very serious view of the recent ruling in California which stated that public interest overruled every possible dissent on grounds of confidentiality. In effect, it amounted to saying that the medical profession is primarily required to keep public interest above the patient's interest and, I personally, find it disturbing.

My own experience through different appearances in the courtrooms has been that, by and large, judges have been sympathetic to the ethical dilemmas faced by the medical profession and never place the expert medical witness onto the brink. For instance, in one particular case where I was required to appear as an expert witness, the interrogating counsel insisted that I divulge certain pieces of information which I regarded confidential. My refusal prompted an intervention by the presiding judge but a simple response, that is, "your honour, if you were my patient probably you would expect the same consideration", persuaded him to ask the counsel to withdraw the question.

In the medical profession, we enjoy a great deal of public prestige and trust. A recent opinion poll in the U.K. demonstrated that people turn to their doctors before they turn to their ministers in times of need. I feel quite strongly that if we were to stay on this pedestal we owe it to the society and to our patients to respect their wishes and even if the presiding judge was not as sympathetic as I encountered, they should be prepared to face up to the consequences of non-disclosure.

Another legal ambivalence that I discovered since my arrival in Nova Scotia is the Child Protection Act which

compels all professionals to breach confidentiality. Whilst it is perfectly desirable for the medical profession to state its position in advance, in such cases I am still uncertain that the ethical requirements can be so arbitrary over-ridden. After all, lawyers do tend to enjoy this privilege and I feel that the medical profession should be accorded the same consideration.

The obligations of confidentiality, as McCormick *et al.* point out, extend beyond the patient's death. In my view, they also extend beyond a clinical situation. Any information obtained through administrative experience and confidential enquiries, not necessarily clinical, plus the confidence accorded to us by a medical and paramedical colleagues, has to be respected with this same scrupulosity as the situation that McCormick *et al.* so astutely elaborated.

Another, perhaps, the most prevalent reason for non-observance of this important principle, which the authors briefly touch upon, is the cavalier attitude that has been shown by the senior members of the medical profession. I, personally, know of a senior professor of psychiatry who insisted on naming one of his patients who was a ballet dancer of international repute, just immigrated to Australia. This was to a group of fifty individuals and then he went about to distribute her autobiography which she had presented to him with her signature. When, pointed out to him, he dismissed the objection as invalid, claiming that many people already knew that she had a problem that he was trying to illustrate and, therefore, there was no duty or obligation of confidentiality on his part. I know of another senior professor who had come to know about a colleague's medical condition through a strictly confidential inquiry, initiated by the Health Department, which he insisted on divulging to his colleagues without seeking permission from the source and without informing the institution which had commissioned the inquiry. The disease in question was not AIDS or any other infectious illness. Both these cases were presented to the licensing authority which refused to rule on them, regarding the former as just a restatement of facts, despite the ballet dancer's objections and the latter as the inevitable consequence of the inquiry.

Our primary duty is to our patients and not to preserve public interest. Whilst there are cases where exception to this rule may be justified, for instance, G.M.C. has relaxed its guidelines if the physician comes across an AIDS patient but by and large, I feel, the principle should be irrevocable. My hope is that Dr. McCormick and Ms. Penny's article will provoke some important discussions in this important area which has hitherto been neglected.

Yours sincerely,

Ashoke Prasad, M.B., M.R.C. Psych  
Anna. Co. Mental Health Clinic  
P.O. Box 728  
Middleton, N.S.  
B0S 1P0

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# MSNS POLICY SUMMARY

## HIV POSITIVE PHYSICIANS

### THE PUBLIC RISK

The Society's research and communication with local infectious disease experts have concluded:

1. that there is NO risk of transmission of HIV to a patient during professional procedures which do not involve the possibility of blood transfer.
2. for procedures (mostly surgical) where there is the possibility that the HIV Positive Physician's blood may contaminate the patient, there is a risk but it is very low.

There have been no cases reported to date where an HIV positive physician has transmitted the disease to his patient in the course of professional activity. We assume that the risk to a patient from an HIV positive physician is the same as that which occurs in the reverse situation (is) when a health care worker sustains a needle stick injury or other type of contamination of blood fluid from an HIV positive patient - here the risk is less than 1%. There is good data to support this figure with several thousand health care workers followed for long periods of time after such exposure.

### SCREENING FOR HIV IN THE PHYSICIAN POPULATION

Informed opinion would indicate that this is NOT routinely indicated. Testing of a physician may be indicated however for epidemiological reasons. An example would be the investigation of a cluster of HIV infections where epidemiological studies point to the physician as being the source.

Physicians in high risk groups are encouraged as is the general public to participate in voluntary screening programs.

### THE RESPONSIBILITY OF THE HIV POSITIVE PHYSICIAN

The physician testing positive for HIV is urged to:

- (a) confine medical practice to non-surgical procedures. It is not felt in this context that informing the public of his/her HIV status would serve any useful purpose.
  - (b) if the physician is going to continue to perform a procedure/s where he/she could possibly infect a patient then the physician has the responsibility to inform the patient that he is a carrier of blood born disease and that there is a risk, albeit small, of transmitting this disease to the patient.
-