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THE NOVA SCOTIA MEDICAL BULLETIN

Halifax: Alexandria of the North

Situated as it was on the eastern corner of the ancient Grecian Empire, Alexandria was one of the most beautiful cities of the old world and a fabulous centre of learning. For nearly a thousand years, scholars of philosophy, science and literature held sway over the intellectual world. It is said that there were over 100,000 volumes in the library, although many of the manuscripts were on rolls. The schools of literature and science were thronged with orators. Lively discussion and rhetoric stirred the halls of learning and research, and criticism spiced their epic epistles. The poetry, medicine and astronomy of Alexandrians were disseminated through the world.

Modern Ptolemites are now thronging another seaboard city, similarly placed athwart the eastern shores of a great civilization. This summer, Halifax welcomes thousands of academic visitors in a series of remarkable conferences. Some 5,000 members of the Learned Societies are gathering like a vast intellectual clan, in a marathon of meetings which would do credit to those ancient scholars — societies as diverse as the History and Philosophy of Mathematics, to the Canadian Federation of Humanities.

Almost concurrent with this presentation is the meeting of the Canadian Rehabilitation Association and the Canadian Orthopaedic Association. Some six hundred members and their wives will be participating in the largest orthopaedic conference held so far in Canada. Then the annual meeting of The Canadian Medical Association will highlight the season's activities.

This *Bulletin* gives a warm welcome to all these erudite visitors and hopes that many physicians will be stimulated not only to read our journal but to contribute to future issues.

Several outstanding topical problems are presented in our Summer Issue.

The Healthy Hazards of Exercise

We would like to congratulate Dr. Murray Nixon on his literary gem about his marathon running. Competing with 1,500 other enthusiasts, is an achievement in itself, yet alone collecting the mass of statistics he flourishes so lavishly. Brian Armson's article on "Overuse Injuries in Runners" brings a timely warning to marathon runners. He gives an elegant classification of 'shin splints' — a term used for a wide variety of athletic leg complaints (stress reaction of bone, stress fractures, and compartment syndromes). He emphasizes the need for good training, sensible shoes and common sense in the management of common injuries.

Schools for Sex: Part of the Medical Text

Medical schools are more enlightened now in this field than in previous generations, when most doctors had to find their own answers to many uncensored and intimate questions posed by their patients. It is a pleasure to present Dr. Judith Gold's article describing the Dalhousie course on human sexuality, and to welcome the Guest Editorial by Dr. John O'Connor. As a practising physician, he has participated in this course which has now become part of this medical school's curriculum. Comments and discussion on both the paper and editorial will be welcomed.

Urological Incontinence in Women

Dr. M. M. Davis's article on urodynamics complements his previous paper in the *Bulletin*. Amongst other common clinical problems, eneuresis and nocturia are often encountered in practice. Incontinence and cystocele are not directly related and Dr. Davis emphasizes the need for accurate diagnosis and assessment before surgery. Since the establishment of a urodynamics clinic, there has been a change in emphasis from anterior vaginal repair for prolapse to selective suprapubic surgery to relieve this distressing condition.

Myasthenia Gravis

Finally, it is befitting that this article should be written in the true classical tradition, by a Rhodes scholar, Dr. T. J. Whelan, and it is a fine example of clear scholarly writing. Myasthenia gravis is a rare disease and may easily escape detection by the physician. The variety of symptoms, the lassitude and the protean modes of onset all make its diagnosis a matter of astute clinical acumen. This review brings us up to date with the manifestations and management of this unpleasant disease.

Offerings to Editors

Aspiring authors should study the article by Dr. Whelan for its clarity of expression and style. At a recent meeting of the Council of Biological Editors in Boston, editors expressed concern about the indifferent standards of articles submitted to journals. Some, such as *The New England Journal of Medicine,* reject 90% of all articles submitted. However, writers should not be discouraged for it was also observed that, after these articles have been rewritten, re-edited and processed, most of them will be published eventually. After all, there are some 64,000 biological papers printed each year so that there should be space for a well-written contribution.

In any case, we hope that the tremendous flurry of intellectual activity in our city of learning will stimulate you to send us some of the more classical items before the summer ends.

□

B.J.S.G.

An Appreciation

DR. RUSSELL CLARK ZINCK

Dr. Russell C. Zinck of Lunenburg was born on April 24, 1898 at Chester, Lunenburg County, Nova Scotia. He received his education as far as Grade XI at the local schools and then took a further year of high school at the Colchester Academy in Truro. For the next two years he taught school, first at Indian Harbour and later at Chester Basin.

In 1916 at the age of 19, he joined the Candian Army as a private in the 42nd Black Watch Infantry Unit, During his war service, when he was promoted to the rank of corporal, he was decorated with the Military Medal and was mentioned in dispatches.

Following his discharge from the Armed Services he returned to Truro, to Colchester Academy, for further training as a teacher. However, in the same year, 1919, he entered McGill University and graduated in medicine in 1924. After an internship at the Orange Memorial Hospital in Orange, New Jersey, he took a post as locum tenens in Stanley, New Brunswick. In 1926 Dr. Zinck started his own practice in Lunenburg, where he worked for fifty-four years until his death.

Formerly, Dr. Zinck was a member of the staff of the Dawson Memorial Hospital in Bridgewater and he held a staff appointment at the Fishermen's Memorial Hospital. He was a member of the Medical Society of Nova Scotia and has on numerous occasions served on the Executive of the Lunenburg — Queens Medical Society. Between the two World Wars he was an officer in the militia. For over thirty years he was the local health officer in Lunenburg.

Dr. Zinck enjoyed the hobbies of curling, tennis and sailing. He was often seen in summer taking groups of friends around the Mahone Bay islands in his Cape Islander. He was interested in philately, having one of the largest private collections in Canada.

His wife, formerly Vivian Smith, died in 1966, and he is survived by his two daughters and seven grandchildren.

His selfless dedication to the welfare of the people of Lunenburg was well recognized and he will be missed by both patients and confreres. □

THE NOVA SCOTIA MEDICAL BULLETIN

Published by

The Medical Society of Nova Scotia

GUIDELINES FOR AUTHORS

In 1978, a number of American, British and Canadian editors of medical journals met in Vancouver, to establish a common format for the submission of papers, and their deliberations resulted in the "Declaration of Vancouver". The Editor and the Editorial Board of the *Bulletin* have decided to adopt this new format, beginning in 1981, and the changes are chiefly in the style used for citing references.

The entire manuscript should be typed double-spaced on one side only, with generous margins on all four sides. Tables should not be included in the text but typed on separate pages, as should the references and the legends for any figures and illustrations.

Non-metric units should not be used in scientific contributions. Parts of the SI system are controversial or unfamiliar, especially concentrations of substances, gas tensions, blood pressure and radiological units, so that authors should provide conversion factors. Abbreviations should be defined when first mentioned and, if numerous, the author should provide a glossary which will be printed separately in a prominent place in the article.

In general, papers reporting on studies should adhere to the following sequence:

a) **Title page** — title of article (concise but informative); first name, middle initial and surname of each author, with academic degrees; names of department or institution to which the work should be attributed; name and address of author responsible for correspondence or reprints; source of support (if any).

b) **Summary or Abstract** — not over 150 words, summarizing the purpose, basic procedures, main findings and principal conclusions.

c) **Materials and Methods** — describe the selection of subjects, the techniques and equipment employed, the types of data collected, and the statistical tests used to analyse the data.

d) **Results** — describe in logical sequence, using tables and illustrations.

e) **Discussion** — emphasize new and important aspects, and the conclusions that follow from them. Recommendations, when appropriate, may be included.

f) **Acknowledgements** — only those persons who have made substantial contributions to the study.

g) **References** — usually limited to 10 for short papers and to a maximum of 20 for review articles. Number in sequence, in the order they are first mentioned in the text, with journal titles abbreviated as in *Index Medicus*.

Examples of the new format are:

1. Journal articles — list all authors when six or less (surnames followed by initials without periods); when seven or more, list only the first three and add *et al.*

Epstein SW, Manning CPR, Ashley MJ, Corey PN. Survey of the clinical use of pressurized aerosol inhalers. *Can Med Assoc J* 1979; **120:**813-816.

2. Book —

Fletcher C, Peto R, Tinker C, Speizer FE. *The Natural History of Chronic Bronchitis and Emphysema.* Oxford: Oxford University Press, 1976.

3. Chapter in book —

Deusche KW. Tuberculosis. In: Clark DW, MacMahon B, eds. *Preventive Medicine.* Boston: Little, Brown, 1967: pg 509-523.

h) **Tables** — type each on a separate sheet, number consecutively with *roman* numerals. Supply a brief title for each, give each column a short or abbreviated heading, and reserve explanatory material for footnotes.

i) **Figures and Illustrations** — professionally drawn and photographed, as glossy black and white prints, numbered consecutively with *arabic* numerals. List all legends on one page and state magnification of photomicrographs.

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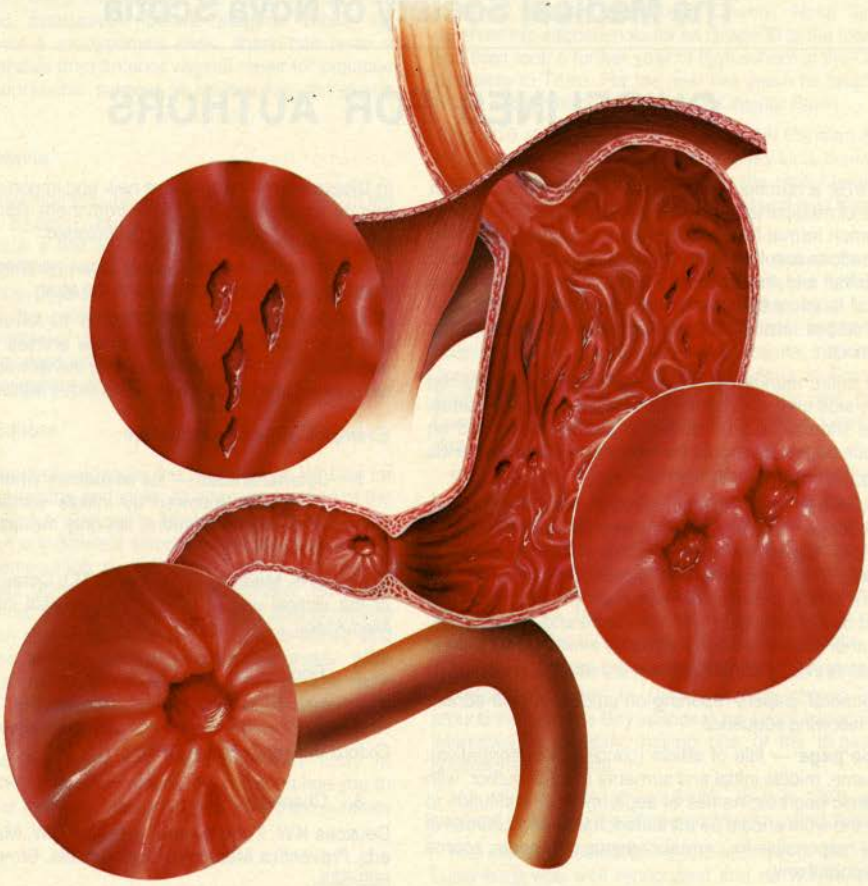
B3H 4H7

WHEN YOUR DIAGNOSIS IS

■ Duodenal ulcer ■ Gastric ulcer ■ Stress ulcer syndrome



A LOGICAL ANTACID THERAPY IS MAALOX TC

MAALOX TC Designed for greater patient compliance and low frequency of side effects in short and long-term peptic ulcer therapy.

1. Fedeli, G., et al. A Controlled Study Comparing Cimetidine Treatment to an Intensive Regimen in the Therapy of Uncomplicated Duodenal Ulcer. Dig. Dis. Sci. 24:10, 758, 1979.
2. Petersen, W.L., et al: Healing of Duodenal Ulcer with an Antacid Regimen. N. Engl. J. Med. 297:341-345, 1977.
3. Ippoliti, A.F., et al: Cimetidine versus intensive antacid therapy for Duodenal Ulcer. Gastroenterology: 74:393-395, 1978.
4. Zinner, M.J. et al: A Randomized Prospective Controlled Study of the Prevention of Upper Gastrointestinal (UGI) Bleeding in Intensive Care Unit (ICU) Patients: A Comparison of Cimetidine and Antacid Titration Maalox TC. Presented at XI International Congress of Gastroenterology, 1980.
5. Englert, E. et al: Cimetidine Antacid and Hospitalization in the Treatment of Benign Gastric Ulcer. Gastroenterology 74:416-425, 1978.

MAALOX TC
LOGICAL ANTACID THERAPY FOR PEPTIC ULCER DISEASE.

Recent clinical studies show ulcer healing occurs with a liquid antacid dose of: **80 mEq of Neutralizing Capacity[1]**

MAALOX TC:

High in Acid Neutralizing Capacity at 25.5 mEq/5 ml[2] **15.7 ml**

☐ Effective relief of pain symptoms.
☐ Makes possible smaller doses and encourages patient compliance.

Long Duration of Antacid Effectiveness **2.5 hrs.[3]**

☐ Controls the stomach pH between 3.0 – 5.0 for 49 min/5 ml.[2]
☐ Promotes prolonged pain relief and healing of peptic ulcer.

Low in Sodium Content – 1.13 mg/5 ml[2] **3.55 mg**

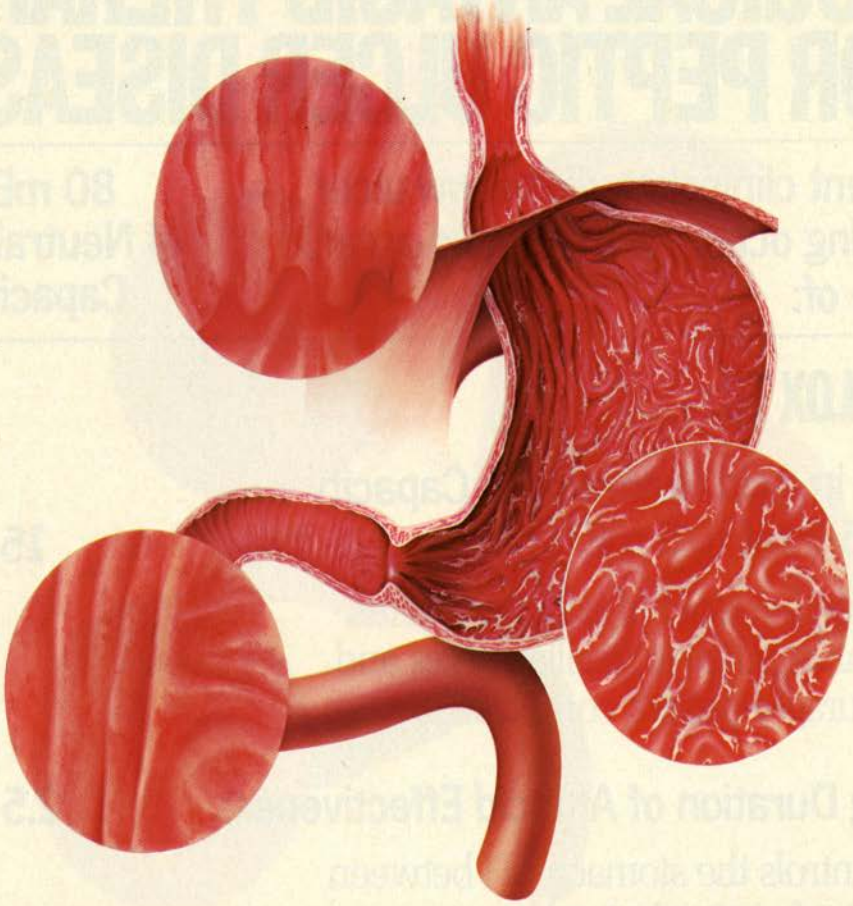
☐ Offers flexibility and freedom in diet of patients on salt restriction.

MAALOX TC. A PRACTICAL APPROACH TO PEPTIC ULCER THERAPY.

1. Since liquid antacid in a dose of 80 mEq taken one and three hours after meals and at bedtime has been proved effective in the healing of duodenal ulcer,[4] all calculations have been based on the 15.7 ml of Maalox TC needed to deliver this desired acid neutralizing capacity.
2. Independent tests conducted June, 1980. Data on file Rorer Canada Inc.
3. Duration of antacid effectiveness is related to the rate of gastric emptying. Taken on an empty stomach, the duration of antacid action is 20-30 minutes. Taken 1 hour after eating, gastric acidity can remain low for 3 hours[5] – provided the antacid is still capable of neutralizing the gastric contents. Value given is an extrapolation. Rossett-Rice time is generally proportional to the dose given.
4. Fedeli, G., Anti, M., Rapaccini, G.L. et al. A Controlled Study Comparing Cimetidine Treatment to an Intensive Regimen in the Therapy of Uncomplicated Duodenal Ulcer. Dig. Dis. Sci. 24:10. 758, 1979.
5. Fordtran, J.S. et al: In Vivo and In Vitro Evaluation of Liquid Antacids, New Eng. J. Med. 288:923, 1973.

WHEN YOUR DIAGNOSIS IS

■ Gastritis ■ Duodenitis ■ Gastroesophageal reflux.



A LOGICAL FIRST LINE MEDICAL THERAPY IS MAALOX

☐ Rapid and effective relief of pain and reflux symptoms

☐ Optimal protection of the gastric mucosa by raising the pH of gastric juice to 3.0 – 5.0.

☐ Neutralization of gastric acid provoked by such common stimulants as food, histamine, acetylcholine, pentagastrin and caffeine.

☐ Proven superior to placebo in controlling symptoms of esophagitis.[1]

MAALOX Proven effective in 31 years of clinical use.

1. Effect of Antacids on the Bernstein Test in Esophagitis, Curr. Therapy Res. 13:217, 1971.

MAALOX
LOGICAL ANTACID THERAPY FOR GASTRITIS, DUODENITIS AND GASTROESOPHAGEAL REFLUX.

A reasonable initial therapy to reduce gastric acidity and to compensate for a weakened mucosal barrier would seem to be:

40 mEq of Neutralizing Capacity

MAALOX:

Good Acid Neutralizing Capacity At 13.4 mEq/5 ml[1] **14.9 ml**

☐ Provides continuing relief of symptoms
☐ Acceptable volume per dose level which improves patient compliance.

Rapid Reaction with Gastric Acid raises the stomach pH to 3.0[2] in: **1.25 min.[1]**

☐ Gives prompt relief – especially important when pain is sudden and unexpected.

Low in Sodium Content – 1.0 mg/5 ml[1] **2.98 mg**

☐ Minimizes the risk of excessive sodium intake in patients requiring antacid therapy for an extended period.

MAALOX PERFORMS

Continue to use it with confidence in the medical management of gastritis, duodenitis and gastroesophageal reflux.

1. Independent tests conducted June, 1980. Data on file Rorer Canada Inc.
2. The in-vitro end point that correlated best with relative antacid potency in vivo as reported by J.S. Fordtran et al: New Eng. J. Med. 288:923, 1973.

Rorer Canada Inc.
Bramalea, Ontario L6T 1C3

PAAB
CCPP

Brief Prescribing information

Maalox TC

Indications: A high potency antacid for the treatment and relief of the symptoms of peptic ulcer and severe hyperacidity related disorders.

Dosage: 5-10 ml 4 times a day, taken 20 minutes to 1 hour after meals and at bedtime. Higher dose regimens may be employed under the direct supervision of a physician in the treatment of active peptic disease.

Supplied: Each 5 ml of sugar-free peppermint flavoured suspension contains 600 mg of aluminum hydroxide (equivalent to dried gel) and 300 mg of magnesium hydroxide. Sodium content: 1.13 mg/5 ml. Available in 340 ml bottles.

Maalox

Indications: For the treatment and relief of the symptoms of gastritis, duodenitis, reflux esophagitis and hyperacidity.

Dosage: 10-20 ml taken 20 minutes to 1 hour after meals and at bedtime.

Supplied: Each 5 ml of peppermint flavoured, creamy, colloidal suspension contains: 200 mg of magnesium hydroxide and aluminum hydroxide (equivalent to dried gel USP) 228 mg. Sodium content: 1.0 mg/5 ml. Available in 340 ml bottles.

Maalox TC and Maalox

Contraindications: Alkalosis, hypermagnesemia; where distension may be due to partial or complete intestinal obstruction. Not recommended for severely debilitated patients or those with impaired renal function.

Precautions: Magnesium salts, in the presence of renal insufficiency, may cause CNS depression. Aluminum hydroxide, in the presence of low phosphorous diets, may cause phosphorous deficiency. Aluminum salts tend to cause constipation. Magnesium salts tend to cause loose stools. Do not administer concomitantly with tetracycline antibiotics.

Rorer Canada Inc.
Bramalea, Ontario L6T 1C3

PAAB
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WHERE THERE'S A WILL, THERE'S A WAY.

We are the Canadian Cancer Society. We want to give your children and your children's children a chance to grow up in a world where one out of five Canadians won't die from this disease. But we need your help. Almost one-third of our needs is funded from bequests and other special gifts. When you make your will, take care of your loved ones and other responsibilities first. Then, if you leave the rest to us, we'll do everything we can to make your legacy pay off in a world without cancer–just about the best kind of inheritance our future generations could ever have. Are you willing to help?

Canadian Cancer Society
CAN CANCER BE BEATEN? YOU BET YOUR LIFE IT CAN.

So You Want to Run a Marathon?

Murray Nixon,* M.D.,
Halifax, N.S.

"So you want to run a Marathon? Contrary to popular belief you will not have to quit your job, leave your wife or husband, give up good food or alcohol.

All you have to do is three short easy runs, and three good hard workouts per week for about ten weeks. This is providing you are now running and in good physical condition. The most important thing is that you find a program which you have confidence in and that barring injury, you stick to your schedule."

These words from an article in *Canadian Runner* magazine last spring (1980) started it all, and a picture of thousands of New York City Marathoners surging across the starting line of the Verrazano-Narrows Bridge clinched it — I wanted to run in the New York City Marathon this year. I'd been enjoying jogging for relaxation and exercise for the past couple of years but had never run farther than the five miles of the annual Halifax Fun Run, but why not!

It took a little longer to talk my part-time running partner, Halifax businessman Bob Daniels, into it, but once I did, he organized a 16 week training schedule which saw us running 691 miles over 6,017 minutes. Bob's program was excellent; each week we increased our training mileage and more distant streets in Halifax became familiar. We used to stash a water jug at the old dump site; I'm sure anyone seeing us drinking there didn't suspect it was water. We concentrated on developing and maintaining a regular nine-minute-mile pace and after awhile it became a reflex, even up the hills of Point Pleasant Park.

Our other challenge was to get officially accepted for the 1980 New York run. They only accept 16,000 (yes really there are that many!) out of over twice that number of applicants. You don't have to have proven yourself as a Marathoner (as at Boston) but just have to be lucky enough to have your application drawn. We were.

So by October 26 we were all set and in New York with our wives who had gone through various phases of disbelief, surprise, resignation, acceptance, support and encouragement.

What Jim Fixx in his *Second Book of Running* says is true. "To stand after months of training at the Staten Island approach to the Verrazano-Narrows Bridge as the helicopters rattle in the October winds off New York Bay is to enjoy a sweet and poignant anxiety that echoes in memory long after your muscles have forgotten the day's torments."

I didn't surge across the starting line as in the picture, because we placed ourselves towards the back. In fact we walked slowly across the starting line and it took us six minutes after the gun went off to be able to start running. What a horde of runners — I was running with 59 airplane pilots, 546 physicians, 192 dentists, 62 priests and rabbis, 31 bartenders, 19 septuagenarians, 953 divorced people, 289 members of unmarried couples, 250 Canadians, 42 Dutchmen (including one wearing wooden shoes) and 15,000 other skimply clad bravers of the pot holes, the bridge grate and the first chill of winter.

The course passes through all five boroughs of New York, various ethnic communities, a myriad of architectural and historical landmarks and one of the largest crowds gathered in the United States to watch a sporting event. Over a million and a half spectators lined the route and did they participate! They cheered, applauded, slapped your hand and passed out everything from water to vaseline. It was an event. We ran well, finished easily and thoroughly enjoyed ourselves.

There was no Rosie Ruiz this year, but there were 60,000 bottles of mineral water, 18,000 bananas, 2,500 volunteers, 306 portable toilets, 18,000 yards of barricades and two tired but very pleased Haligonians.

I recommend it! □

*Associate Professor, Department of Family Medicine, and Director, Residency Training Programme, Dalhousie University, Halifax, N.S.

Overuse Injuries in Runners

Brian A. Armson,*B.Sc.,
Halifax, N.S.

INTRODUCTION

In most countries of the world, during the last two decades, increasing emphasis has been placed on individual and group fitness programmes. This renewed interest in physical activity appears to be the consequence of concerns regarding the sedentary lifestyle afforded by the motor vehicle as well as the troubling prevalence of coronary heart disease since the early 1960s. The majority of the exercising population have turned to jogging as their answer to a regular exercise programme. In addition, competitive runners are training harder than ever before, stressing their bodies beyond the normal limits of endurance. It is not surprising then, that overuse injury of the legs is becoming more commonly seen by general practitioner and specialist alike.

In an effort to understand, diagnose accurately and treat appropriately the many syndromes associated with overuse, the subspecialty of sports medicine was born. The medical literature is replete with descriptions of athletic ailments along with suggestions for their diagnosis and management. It is the purpose of this paper to present some of the more common overuse injuries of the lower extremity in the jogger and distance runner. Etiology, pathogenesis, differential diagnosis and management will be discussed.

The diagnosis of chronic or recurring pain in the legs with exercise, usually running, either in jogging or in competitive athletic events, is largely made clinically. The history is of pain coming on during exercise. Over a period of time, the pain begins to occur at an earlier stage and with chronicity may occur even after exercise and at rest.

Clinical examination is important, and before considering the bony, muscular and ligamentous structures of the leg which are associated with overuse injury, consideration should be given to conditions such as arterial insufficiency and claudication, and referred pain to the leg from the lower spine which may be associated with spondylosis or with disc protrusion. Also, deep venous thrombosis may be present as swelling and pain in the calf, and may be considered by the patient to be associated with some recent exercise. Thrombophlebitis can be similarly confused, and should always be excluded as a possible cause of leg symptoms.

The four compartments of the leg — 1) the anterior which is composed of the tibialis anterior, extensor hallicus longus, peroneus tertius, and extensor digitorum longus muscles; 2) the peroneal, comprising the peroneus longus and peroneus brevis muscles; 3) the deep posterior compartment, formed by the tibialis posterior, flexor hallicus longus, and flexor digitorum longus muscles; and 4) the superficial posterior compartment, which is composed of the gastrocnemius, soleus, and plantaris muscles — are all affected in overuse syndromes of the legs, although the posterior compartments are by far the most frequently affected.

Most of the leg problems associated with running can be related to either training error, anatomical factors, or shoes and surfaces. According to Brubaker[4], sixty percent of overuse injuries can be related to poor training technique. This includes excessive mileage, intense training, rapid changes in training routine and running on hills and hard surfaces. Anatomical factors which predispose the runner to injury include tibia vara, subtalar varus and/or forefoot supination, femoral neck anteversion, genu varus, tibial torsion, leg length discrepancies and excessive pronation of the foot. Brubaker also reports that fifty eight percent of the runners he has treated have had an excessively pronated foot. It is therefore extremely important that the patient's anatomical alignment and function be carefully evaluated. The overriding assumption here is that there is a position in which the foot will function most efficiently and with the least amount of stress to joints, bone, muscles and tendons of the leg. That is, prior to toe-off, the foot should be in the neutral position so that the alignment of tibia, knee and femur are optimal for stress absorption. More specifically, with weight bearing, the vertical axis of the heel should be parallel to the longitudinal axis of the distal one third of the tibia and the plane of the metatarsal heads should be perpendicular to the heel.

Having considered the general approach to the evaluation of overuse injury to the legs, specific syndromes will now be discussed.

"SHINSPLINTS": A MULTIFACTORIAL SYNDROME

"Shinsplints" is a term which has been used by athletes to describe pain originating between the tibial tubercle and the ankle associated with exercise. Some people refer to the shin as any portion of the tibia, while some dictionaries and medical literature confine the term to the anterior portion of the tibia. Severity, treatment and resulting disability may vary greatly.

Many workers have attempted to define the shinsplint syndrome in terms of a specific etiological mechanism. This has led to considerable confusion in the sports medicine sphere. To clarify the term and obtain a more universal description of the etiology of leg pain, the American Medical Association's Standard Nomenclature of Athletic Injuries defines shinsplints as "pain and discomfort in the leg from repetitive running on hard surfaces or forcible extensive use of the foot flexors . . . The diagnosis should be limited to musculotendinous inflammation, excluding a fatigue fracture or ischemic disorder".[18] This definition is not widely accepted in athletic circles because only a small percentage of leg discomfort in the intensive training athlete is related to musculotendinous inflammation.

The majority of athletes with shin soreness experience a self-limited course without limitation in performance. The

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pain is usually present at the beginning of a workout period and then disappears during the warmup, and may or may not be present for several hours after the workout. In competitive athletes, once the pain interferes with vigorous training or performance, the cause has usually progressed beyond a musculotendinous inflammation. Most writers agree that the most common location of shin pain is along the distal two-thirds of the medial crest of the tibia. The clinical course of shinsplints varies considerably, but few athletes progress to severe, prolonged, and/or recurrent disabling symptoms.

Shinsplints is most often seen in the untrained athlete who has recently begun a training program that is too rigorous on hard surfaces, and in the trained distance runner who has switched to an interval track programme on a hard track.

Clancy[6] suggests that this shin soreness may be due to microscopic tears of Sharpey's fibers of the tibialis posterior or flexor digitorum longus. McNamee[26] feels that tendoperiostitis of the area of muscular and tendinous attachment to the tibia and interosseus membrane is responsible. Puranen[30] and Matsen[23] attribute pain over the posteromedial border of the shin to increased pressure within the deep posterior compartment of the leg resulting in occlusion of the microvasculature and consequent transient ischemia. This theory is disputed by D'Ambrosia[9] who found no significant intra-compartmental pressure in fourteen athletes with "typical shinsplint syndrome". Finally, Clemente[8] proposed that shinsplints were due to a stress reaction of bone which he called "the tibial stress syndrome".

As Jackson[18] and Subotnik[36] agree, shinsplints should be considered as a catch-all term which is usually associated with one or more of the following: musculotendinous inflammation, a musculotendinous tear, a stress fracture or stress reaction of bone, a muscle-bone insertion injury and/or vascular disorders (acute and chronic compartment syndromes).

Musculotendinous Inflammation

Musculotendinous tears and muscle-bone insertion injuries may involve only a few fibers of muscle or there may be complete rupture. Most often, however, just a few fibers are involved and may cause minimal symptoms. As the athlete continues to stress his legs, these tears may be associated with chronic inflammatory changes and subsequent musculotendinous inflammation.

Differentiating musculotendinous inflammation from other causes of shin-splints should be straightforward. The maximum tenderness should be determined as originating from muscle, bone or tendon. Musculotendinous pain may involve the entire length of the tibia whereas stress reactions and fractures of bone usually involve one focal area. Stressing the musculotendinous unit by passively manipulating the toes, foot or ankle should increase pain this is related to musculotendinous inflammation. Soft tissue swelling, increased temperature and x-ray evidence of periosteal thickening also occur in stress reaction and fracture of bone and therefore should not be relied upon as criteria for differential diagnosis.

Treatment depends on the severity of symptoms. The use of anti-inflammatory agents such as ASA, phenylbutazone and steroids, ice, heat and various orthotic devices have met with varying degrees of success[1]. Runners with mild symptoms may be allowed to run slowly on grass over moderate distances until symptoms disappear. Runners with persistent, severe pain should stop running until there is no longer pain on palpation of the tibial crest. This allows the more severe musculotendinous inflammations and strains to subside. Once they are free of pain they may resume training on a gradual interval program.

Stress Reaction of Bone

Jackson and Clancy explain bone pain associated with exercise as resulting from rapid bone turnover and remodeling. It is suggested that the tibia is unable to remodel itself fast enough when it is subjected to repeated strain at a vulnerable spot, so that bone resorption exceeds bone deposition in the area of stress.

Clemente[8] has also proposed an interesting etiologic theory. He postulates that with cyclic training stress the muscles of the lower leg become fatiqued and lose some of their shock absorbing function. The resulting structural stress on the tibia creates a painful periostitis reaction. The athlete compensates by decreasing the load on the affected limb, thereby introducing the potential for disuse atrophy. The affected muscles are then more predisposed to fatigue which reinforces a vicious cycle which may lead ultimately to stress fracture.

Stress reaction of bone which may have progressed to an atypical stress fracture is often not apparent on x-ray. An abnormal increase in the radio-active uptake on the technetium bone scan may be the only method by which to make a definitive diagnosis. The pain of stress reaction is often severe and localized over the medial border of the tibia just distal to the muscle insertion. It should be recalled that there are no muscle insertions in the posterior medial tibia in the distal third of the leg. Subroentgenographic fatigue fractures may be the cause of most bone pain in the distance runner (more than 30 miles per week).[18] There may also be periosteal induration at the site of maximal tenderness with increased circumference of the leg at that point. Atrophy of the anterior tibial group and gastrocnemius due to disuse is also common.[8] Rather than occurring early during exercise, the onset of pain is generally after stress overload and gradually subsides with rest. Aggravation of the pain by weight bearing is normally associated with bone reaction whereas motion related pain usually represents musculotendinous injury.

The main treatment for this condition is rest from the sport to allow the tibia to heal. Anti inflammatory drugs may be used if periostitis is suspected and a gradual rehabilitative programme for atrophied muscles should be prescribed. The importance of well cushioned running shoes in the prevention of stress reaction of the tibia cannot be overemphasized.

Stress Fractures

Stress fractures can be considered as the end stage of stress reaction of bone already described. Another theory put forward by Devas[11,12] and supported by McNamee[26], Walter[37], and Slocum[34] is that continued strenuous muscular activity causes bending of the fibula and tibia. In time, a stress fracture develops on the convex side of the bone which may progress to involve the entire cortex of the bone with subsequent fracture and displacement.

The onset of symptoms may be acute or gradual but is more often the latter. Stress fractures of the fibula are the most common and occur approximately six cm. above the

lateral malleolus in the lower third of the leg. The most common site for fatigue fractures of the tibia is at or near the junction between the middle and lowermost thirds posteromedially. Fractures in these locations are usually transverse whereas those in the upper two-thirds are usually oblique. The pain associated with stress fractures is normally located directly over the area of fracture, being most severe after exercise and gradually subsiding with rest. With continued training the pain may become so severe that the athlete can no longer participate in exercise and he may no longer find relief with rest. It is particularly aggravated by weight bearing and there may be some associated swelling and edema. However, most stress fractures are not associated with significant tissue damage.

Stress fractures are best diagnosed by x-ray. Characteristic changes include: 1) a thin crack in the cortex which spreads across the bone, 2) evidence of osteoclastic absorption in the bony cortex; 3) cortical hypertrophy; 4) periosteal and endosteal callus formation; and 5) healing and condensation about the fracture line. These changes are often not visible until two to three weeks after the onset of symptoms which may represent stress reaction or subroentgenographic fractures. Again, a technetium bone scan may be, but is generally not, necessary to establish a diagnosis.

Full blown stress fractures are usually seen in the less-conditioned athlete and those who continue to exert themselves even though they have warning pain. Hard surface running has also been identified as an etiologic factor in this syndrome.

The recommended treatment is complete rest from running for four to six weeks. Adhesive strapping has been advocated by Devas.[11,12] A gradual return to former activity levels on well cushioned shoes and soft surfaces is also advised.

Compartment Syndromes

The four osseofascial compartments of the leg are susceptible to the development of compartmental syndromes in association with strenous exercise. Although uncommon, the compartment syndromes can be extremely dramatic and may constitute a surgical emergency. The anterior compartment is affected most often followed by the deep posterior compartment. The peroneal and superficial compartments are rarely involved. The syndrome results from inadequate blood supply to the muscles of a particular compartment and this ischemic condition may cause degeneration and necrosis of the muscle and eventual replacement by fibrous tissue.

Many theories have been proposed to account for the compromise of perfusion to a fascial compartment of the leg. Horn[15] noted marked thickening of all layers of the anterior tibial artery which he attributed to the increased demands made on the artery and/or reflex spasm during exercise. Hughes[16] also defined the syndrome as a primary artery disease and proposed that the ischemia was a consequence of arterial occlusion by tension within the fascial space, interosseous membrane, embolism or thrombus or fatigue arterial spasm. None of the above hypotheses have stood the test of time, however and, as Paton[29] points out, there is no evidence to deny the possibility that arterial changes might be secondary to inflammatory changes in muscle. Most workers now feel that the common factor in these syndromes is an increase in the interstitial fluid pressure in a closed compartment with subsequent alteration in blood flow.[18,21,26,27,29,30] The suggestion is that severe exercise leads to swelling of muscle, which being confined within a walled compartment, causes increased tension. It has been shown that muscle retains water during exercise and may increase its bulk by as much as 20 percent.[29] In addition, ruptured muscle fibers may hemorrhage into the compartment and further contribute to increased compartmental pressure. Since relatively low pressures are sufficient to occlude the fine intramuscular vessels without occluding the major vessels, selective ischemia is possible.

Compartment syndromes may develop acutely or chronically. The acute form is heralded by the sudden onset of severe pain in the region of the affected compartment during or after strenuous exercise. The pain is constant, aggravated by movement and not relieved with rest. There is also rapid development of swelling over the compartment along with a woody hardness. Surface changes such as erythema and glossiness may be evident. The disappearance of distal pulses is a late sign as are the loss of motor function and sensation. This presentation should be considered a surgical emergency. Surgical decompression is achieved by fasciotomy of the involved compartment.

Experience indicates that if fasciotomy is not done within twelve hours of the onset of pain, serious disability due to ischemic necrosis, sloughing of muscle, scarring, and nerve involvement, is likely.[28,33] Complications of late fasciotomy include severe inversion of the foot and equinous deformity in the peroneal compartment syndrome, claw-foot with ischemic contracture of the anterior tibial muscle and long extensors of the toes in the anterior tibial syndrome, and permanent foot drop. Diagnosis of surgical cases of acute compartment syndromes may be made by measuring intracompartmental pressure with a wick catheter. Normal compartment pressure is 4 ± 4mm Hg. A measured pressure of 30 mm Hg or greater is considered the critical level for immediate fasciotomy.[28]

Chronic compartment syndromes may present a history identical to other overuse syndromes. They must be carefully differentiated from musculotendinous inflammation and stress reactions or fracture of the tibia. Once again, measurement of compartment pressure may be required to makejthe diagnosis. Since thisjsyndrome represents transient ischemia, conservative treatment including rest, elevation and analgesics may be sufficient. However, Puranen, has reported success in treating this entity with fasciotomy.[30]

The acute compartment syndrome is usually associated with a history of marked exertion of the lower extremities by poorly conditioned young athletes. Trained distance runners, on the other hand, are more prone to develop the chronic form.

ACHILLES TENDONITIS

Achilles tendonitis is a disabling condition of distance runners with a relatively high incidence. (12 to 15 percent of runners' ailments in one survey.[3]) In the acute stage, pain localized to a small area of the tendon is experienced during prolonged running. This usually subsides with rest but may be noted on climbing stairs. Without appropriate management the condition progresses to the chronic stage with pain over the entire tendon most of the time. The tendon is thickened and nodular on palpation.

Pathologic changes include devitalization of tendon tissue with altered collagen and granulation tissue response. There may be tendon sheath thickening, fluid accumulation, fibrosis and metaplastic calcification.

The blood supply to the achilles tendon is a critical factor in the etiology of tendonitis. The tendon is supplied in segments by mesotenons so that there is little supply at the musculotendinous junction. With prolonged extreme loads and aging there is slipping of the collagen fibers. Microscopic tears in the tendon causes tendonitis with subsequent chronic inflammation. This results in reduction of vascularity of the tendon which further predisposes it to microtraumas. These may progress to macroscopic tears.[7,10,13,31]

Usually, diagnosis of this condition can be made clinically. The middle aged marathoner, who has recently changed his training programme or who trains on hard surfaces, is particularly susceptible to this condition. Pain is usually localized to the tendon and there may also be associated swelling. The calf muscles occasionally show signs of disuse atrophy along with diminished power. In some cases, particularly in runners over the age of forty, the Achilles tendon may have ruptured. The Thompson's test is used to differentiate partial from complete ruptures. The calf is squeezed and if the tendon is still intact there will be plantar flexion of the foot.

The management of this condition is total rest from running from two to six weeks depending on the duration of symptoms. Steroid injections have been employed but have not proven particularly beneficial. Surgical repair is recommended for ruptures of the Achilles tendon and in cases where conservative management has failed to alleviate symptoms. The procedure most widely accepted encourages revascularization of the tendon and innervation after surgical excision of pathological and degenerated tissue. A longitudinal incision of the tendon sheath is followed by division of adhesions between the tendon and sheath. Clancy suggests that this procedure decreases the inflammatory reactions in the sheath and mesotenon which may promote revascularization.[7] No post operative immobilization is required and the patient may begin a graduated running programme after sutures have been removed.[35] Achilles tendonitis has also been referred to as tenosynovitis in the literature. This term is a misnomer since there is no synovial sheath to the tendon, as the sheath is composed only of a paratenon of fibrous tissue.

PREVENTION

In order to avoid the disabling overuse injuries described above, the runner must use good judgment and common sense. Excessive early season training should be avoided and changes in training programmes should be made gradually. Careful selection of well cushioned running shoes is important as well as minimizing the amount of running done on hard surfaces or up hills. Regular stretching exercises should be incorporated into any running or training programme. Careful attention should be paid to the warning signs of developing overuse syndromes rather than attempting to "run them out". Anatomical imbalances and misalignments must be carefully monitored. By paying attention to the signals of his body, the athlete can adapt to almost limitless stress.

CONCLUSION

Overuse injuries are best treated by prevention. The vast majority of injuries among runners result from improper training technique. Anatomical factors may be involved and should always be considered during evaluation.

The term "shinsplints" represents a variety of clinical entities and a specific diagnosis should be established in order to appropriately manage the patient with leg pain. Many treatment regimens have been suggested but rest is still the most effective therapy for most overuse syndromes, except where surgical repair is necessary. Achilles tendonitis represents one of the most difficult overuse injuries to treat. Much work still needs to be done in this area before the etiology pathogenesis and appropriate management of runners' ailments are fully understood.

Although many of these syndromes are present in both adults and children the anatomical expression of overuse may be different in children because of the differences in the growing skeleton. □

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GUEST EDITORIAL

The Physician and Human Sexuality:

Adequacy or Inadequacy

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"The Medical establishment has become a major threat to health." Thus Ivan Illich opens his book *Medical Nemesis,* a book that challenges organized medicine's attempts to meet what we see as the needs of our society. Not only are we in medicine required to treat the hemorrhoid, heart attack and hernia, but now we must again justify our treatment. It may become more difficult to do this as we "medicalize" everything from the family argument to normal physiological functions such as running, sleep and even ageing. One can only imagine Ivan Illich's reaction to the medical profession's increasing awareness, finally, of human sexuality.

In this issue of the *Bulletin* we take note of the movement of the physicians into the swamp of our sexually oriented society. In Judy Gold's paper, giving the history and status of teaching human sexuality at Dalhousie, we can hope we are well advanced in meeting (or catching up to) the needs of our students.

To say that sexuality is causing a problem in our society seems obvious to most people. To further encourage medical personnel into taking an interest in the situation, however, still attracts argument. Ivan Illich, for one, would have us believe it is a natural function, best left to the layman to sort out. The medical profession still has those who would prefer to foster this belief. But the tide has changed.

Dr. W. D. S. Thomas, President, The Canadian Medical Association, in his address to the 1980 Annual Meeting of The Medical Society of Nova Scotia, states "We have a responsibility to help the general public to accept and come to grips with the realities of the problem". The problem he outlines is 1,000 teenage pregnancies every week in Canada, without suitable programs of sexual counselling. There are about 65,000 abortions every year in Canada, nearly 20,000 of them in teenagers.

"Too frequently counselling is done by embarrassed, ill informed and somewhat reluctant teachers, or worse, by physicians who are equally unqualified and incompetent", says Dr. Thomas. One would hope that Dalhousie's program in human sexuality might help make that statement untrue. But where does that leave the average practitioner in this province? Some, like Dr. John Savage, who is attempting to organize a comprehensive "Family Life Program" in Dartmouth City Schools, are already functioning well. Others are not so sure of their place in this relatively new field.

To quote Fred W. Whitehouse, M.D., Past-President of the American Diabetic Association, "An empathetic primary care physician who is willing to take time (and who presumably has a solid understanding of the pathogenetic mechanisms involved) is thoroughly competent to render sexual counselling. Indeed since average patients will be more comfortable in a familiar milieu, the primary care physician, provided that he or she is patient and understanding, may be the best person to undertake this task".

This having been said, one cannot help but add that there are dangers in jumping on the bandwagon if it is moving too fast. The basic M.D. degree in most medical schools does not produce sexual therapists but for one "willing to take time" for some basic reading and postgraduate learning as well as with the patient, useful sexual counselling is a possibility. Besides providing time and effort the physician must also take a close look at his or her own values. Without properly understanding ourselves, we can deliver many strong but unintentional messages to our patients and our right to judge is questioned by many. One of Alex Comfort's early books, before he became more popular with *The Joy of Sex,* was *Sex in Society* in which he deals with the fact that physicians have a great tendency to impose their values on patients, especially when dealing with sexual matters. (Bronchitis brought on by smoking brings forth a different attitude than gonorrhea brought on by promiscuity). It is estimated in every four families that a primary physician serves, there will be one person in which homosexuality is a "diagnostic factor". How accepting is the average physician of this large group of patients? Or worse, does he even recognize their existence?

Doctors have been authority figures in our society for centuries but a position of authority is our sexually oriented culture is untenable. Any physician not willing to relinquish his authority role should recognize himself and change or stay away from the area of sexuality, referring to colleagues whenever possible.

But one cannot refer all gynecological problems, obstetrics, veneral disease, abortion, "Pap" smears and anxiety rooted in sexual inadequacy. The doctor cannot ignore the sexual implications in family breakup, diabetes, physical handicaps and ageing. Sexuality is often at the core of human interaction and is a factor in almost every facet of practice.

A case could easily be made for making sexual comfort and attitude an essential prerequisite for medical school. While a most difficult thing to evaluate, it at least deserves consideration by any acceptance committee.

Physicians, in recognizing sexual problems do comprise a significant portion of their practice and overlap many nonsexual areas, have many options open that help deal with and recognize this fact.

Proper Education — support whenever possible should be offered to the student to learn treatment techniques and about his or her own values and predetermined judgments.

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We should not pass on our own inadequacies, but face them. Postgraduate courses are available and literature is abundant. Any physician closely involved with patients or families would find the journal *Medical Aspects of Human Sexuality* useful if not fascinating. Be prepared for graduates of family practice residency that have interviewing skills and knowledge of how the family functions as a unit. These physicians are better trained than many of their older colleagues in helping families meet many of the stresses of a changing society. Labelling their skills as "idealistic" or "impractical" does nothing to help our mushrooming divorce rate.

Office Arrangement — foster a relationship with patients that is open and trusting, that allows time for the patient's comfort (the worry about earning a living many interfere, of course, but ingenuity and foolhardy dedication has always abounded in the profession). Getting to know your patient's sexual problems may make his total treatment much easier and rewarding. Stay at your own level of comfort and don't hesitate to consult appropriate colleagues.

Confidentiality — Mr. Justice Horace Keever, in his recent Commission Report, gave a frightening list of infringements regarding privacy of medical records by police, insurance companies, private invesitgating firms and lawyers. Records of student employees, psychiatric patients, civil servants, etc., all need much more protection, especially so when dealing with sexual histories and therapy. Patients who may have revealed the most compromising of information, all too innocently give permission for the release of their whole medical record. Great judgment and discretion along with time consuming patient education is necessary if trust is to be maintained.

Parents must be made to understand that sometimes their adolescent children must be allowed a confidential relationship with their doctor. It is sometimes difficult to remember that parents still hold the ultimate responsibility for adolescents. If parents understand and trust your judgment prior to sensitive situations arising, the doctor-family relationship will have proved its worth.

Community — physicians must be open with their own communities to give support and basic knowledge when required. Talking to high school students, supporting boards of education in establishing family life programs, and premarital participation are possibilities.

Family — Dr. M. O. Vincent, a Dalhousie graduate, has pointed out in numerous papers and presentations that physicians and their families have their own unique sexual and relationship problems. Paying heed to some of his excellent suggestions cannot be recommend too strongly. Physicians without a strong base at home are at a disadvantage in many situations. Our pride, overwork, and sometimes basic personality types all deserve more analysis.

"A medical degree does not automatically confer mental health on the possessor", says Dr. Vincent; nor does it make him a sexual authority or therapist. That medical degree does, however, give us a professional responsibility to deal with human sexuality, whether we desire it or not.

Physicians must live up to and help society meet the responsibility that the "joy" of sexulaity inevitably brings.

In closing, remember, Mark Twain, in his wisdom said,

"To be good is noble but to teach others how to be good is nobler — and less trouble" □

A One-Week Block Course in Human Sexuality*

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The first course in sex education in an American medical school began in 1952.[1] During the 1960s other schools followed suit and, by the end of that decade, many articles were being published discussing the introduction of these courses into the medical school curriculum. By 1975 it was reported that 81% of schools replying to a questionnaire survey had some instruction in human sexuality, and 45% had organized courses.[2]

Due to the efforts of two medical students, who had chosen this as an elective topic in 1972, the Dalhousie University Medical School became interested in the introduction of a course in human sexuality. They presented their survey on sexual attitudes and information of medical students and Faculty to the Committee on Undergraduate Education, and they recommended the establishment of a course within the compulsory curriculum. A subcommittee was appointed to implement the program during the next academic year.

An earlier paper described the initial process of planning.[3] Medical schools in Canada and the United States were asked for details of their teaching of the subject, and medical education literature was reviewed extensively. Several subcommittee members attended a short course at the University of Minnesota. A tentative course was formulated, based mainly on the experiences at Minnesota. It was to be a one-week block course, early in third year, utilizing lectures, panel discussions, and sexually explicit films, followed by small group discussions. As the class would be at least two-thirds male, nursing degree students would be included, resulting in an equal number of male and female students in the groups.

Initially, it was planned to include the partner of any student but this was impossible as many worked during the class hours. Faculty and group leaders were to be both male and female. The emphasis of the course was to be on expression of values and attitudes toward human sexual behavior and on learning to discuss sexuality helpfully with patients. At that stage, a one-day "minicourse" was held for departmental and student representatives. The response was generally favourable and the course was then presented for Faculty approval of its institution in September 1973.

Since then, the course has been conducted annually with some variation in content. The teaching faculty and group leaders are furnished by Nursing, Social Work, Theology, Family Planning Clinics, Family Practice, Health Education, Obstetrics & Gynecology, Medicine, Psychiatry and resident training programmes of the latter three. In the first year some difficulty occurred in obtaining the films, because Canada Customs would not release them without written assurance from the Dean that they were to be used solely for educational purposes in the Medical School. Since then the establishment of a Canadian distributor has simplified matters.

As Rosenzweig and Pearsall point out, the . . . "curriculum design in human sexuality is not a task that can be approached in a purely academic fashion; the teaching methodology must be considered simultaneously with the course content."[4] The literature favoured the concept of an intensive course using explicit films and small group discussions in what has been described as "desensitization-resensitization."[5] Thus the students must face their attitudes through confrontation with a variety of sexual behaviours in the films. The discussion groups provide individuals with an opportunity to express feelings, experience those of others, and through this exploration improve their ability to communicate with patients. In addition lectures are given containing specific information; as well, panels such as those composed of members of the Gay Alliance and of Rape Crisis workers force the students to deal with all aspects of sexuality.

The intensiveness of the course has been found most valuable. The students are together for five days from 8:30 a.m. to 5:00 p.m. The formula of lecture or panel, film, discussion group morning and afternoon quickly results in the desired personal assessment. The groups are essential in this process. Finally, a postcourse evaluation allows the students to contribute to the planning of future courses.

Over the years that this course has been given, it has developed a set format with a slightly changing content. The choice of group leaders is an important and often difficult procedure. Gradually more medical school faculty members have agreed to participate so that the students realize that sexuality is truly dealt with in a variety of situations, not just by social workers and psychiatrists. The medical students, unlike those in nursing, are not familiar with group process and this can slow the work of the groups. Therefore, the first morning of the course is now spent learning about groups.

In earlier years, formal teaching time was spent on the development of sex roles and sociocultural determinants of sexual behaviour. In the past few years this has become unnecessary as the students are knowledgeable in this area and deal spontaneously with these in their first group meetings. Several topics, however, are essential in the formal course outline and include:

1. Myths and attitudes about human sexuality.
2. Male/female sexual response cycles.
3. Panel discussions and films of autoerotic, heterosexual and homosexual behaviors,

*Revised version of paper presented at the A.C.M.C. Annual Meeting, 1979.

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4. Panel discussions and films related to sexual offenses: incest, rape, sexual deviations. A handout on the laws pertaining to sexual activities is prepared by the Law Faculty and is given to the class.
5. Relationship of physical and mental illness, and of medications to sexuality; also of aging, and of physical disability.
6. Treatment of sexual dysfunction.

Successful small discussion groups have been found to follow a pattern during the week. The first two days consist of dealing with intellectual defences and tentative exposure of feelings. The third day, coinciding with the panel and films on homosexuality, sees a group cohesiveness and trust established. The fourth day is spent in frank discussion and attitude realignment or reassessment. The final sessions deal with group dissolution and evaluation.

The group leaders require structure and supervision during the week. This is accomplished through a meeting before the course in which the course and group objectives and process are reviewed, and through daily lunch time meetings during the course week. An experienced group therapist is in charge of this part of the week.

During the groups, the students are aided through the use of role playing to learn to take a sexual history, to deal with various sexual attitudes, and through a variety of interview situations. Actual treatment of specific sexual dysfunctions is limited to a half-day period. An extensive bibliography is provided by the library staff and the use of a textbook encouraged. As Wabrek notes: "A majority of medical educators feel that attitude modification is the most important need of the students, and there is wide pedagogic experience that students do not learn if they are uncomfortable or highly restrictive in their attitudes towards sex."[1] Attitude change, and knowledge gained, is assessed through the use of a pre- and post-course test, as well as a course evaluation. It is planned to use the same tests over the next few years to produce statistically comparable results.

The course at Dalhousie University is unusual in its intensive one-week block format. Over the past several years it has developed from a curiosity to an established part of the third-year curriculum. Student attendance during the week is constant and their evaluations positive. Our experience has taught us to reassess and revise the content yearly, and to seek new and better films regularly, in order to provide a course most suitable to the changing sociocultural milieu in which we, the students, and our patients all live. Thus in the very format of the course we illustrate its objective of learning about sexual attitudes, values and behaviours in order to best treat our patients. □

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OVERUSE INJURIES IN RUNNERS

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Canada is on a health kick, and that's good for drivers. It is just as important for a driver to be in good health as it is for the vehicle to be well maintained.

Update in Gynecological Urodynamics*

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INTRODUCTION

In 1977, one of the priorities of Dr. M. G. Tompkins, the Head of the Infirmary Division of our Department, was the establishment of a Urodynamics Clinic. When Dr. J. A. Collins was appointed as Head of the Department of Obstetrics and Gynecology in December 1977, we were very pleased to find that this was also one of his priorities.

I had attended two post-graduate courses in Gynecological Urology and, through one of the course organizers, I was put in touch with our guest speaker, Dr. Don Ostergard. Eventually, arrangements were made for me to spend approximately ten weeks with him at the Harbour General Hospital in Los Angeles. In the Fall of 1978, we started the Urodynamics Clinic at the Halifax Infirmary, and I have seen over 350 patients with a minimum of two visits per patient. Data from this experience are being computerized but, I have no firm statistics to substantiate some of the statements I will be making this morning. I hope you will understand that they are simply impressions gained over the past two and one-half years of the Urodynamics Clinic, plus some thirty-three years in gynecological practice.

I have been asked by the Director of the programme today to identify my reasons for change, and to compare my management 'before' and 'after' the establishment of our Clinic. Perhaps the weakest parts of the approach in the 'before' era were the inadequacy of the functional inquiry and the objective testing of the patient before and after treatment. Many women were referred by primary care physicians with vague pelvic complaints attributed to identified pelvic relaxation and asymptomatic cystocele. Surgery was advised and performed with no more investigation than a few questions about involuntary loss of urine, frequency, nocturia, and vague pelvic pressure. Testing consisted of a stress test, a vesical neck elevation test of the Bonney type, plus the usual pelvic exam to assess pelvic floor relaxation and to rule-out gross pelvic disease. If there was an indication of lower urinary tract disease, an intravenous pyelogram with or without a consultation to a urologist was advised. If surgery was indicated, an anterior repair was elected with or without a vaginal hysterectomy and posterior repair. This is a fair assessment of the practices used in our offices.

In light of advances which have been made, the approach I have outlined must be considered grossly inadequate, since most failures in surgical therapy have been identified to result from mis-diagnosis. During this period I became unhappy with the results obtained with the treatment of urinary incontinence. Many articles were appearing in the literature concerning new instruments, new tests, new methods to improve diagnostic accuracy, and new methods of treatment — particularly the pharmacological treatment of urinary incontinence. Many of these reports were, and still are, confusing and contradictory. These facts prompted me to enroll in the post-graduate courses offered in urinary incontinence. This was not a unique decision on my part because all courses offered were oversubscribed, indicating a generalized concern with this problem. However, the picture is gradually becoming clearer and suggests that many of the concepts we have had regarding diagnosis and treatment, and the various forms of pelvic relaxation, are no longer valid.

*Presentation: Update for Specialists in Obstetrics and Gynecology. February 13, 1981. Division of Continuing Medical Education, Dalhousie University, Halifax, Nova Scotia.

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FUNCTIONAL ENQUIRY

One of the biggest changes in my functional enquiry has been the use of a history questionnaire which the patient completes. It is basically Dr. Ostergard's questionnaire, comprises some 64 questions which guarantees that no important matters are overlooked, and it serves as a basis for discussion with the patient of her problem. The questions are arranged to direct the physician's attention to the three major categories causing incontinence: namely, urinary tract pathology, bladder instability, and anatomical stress incontinence. The questionnaire will alert the physician to the fact that a mixed form of incontinence may exist (thirty-five percent of our Clinic patients present with this problem). It also alerts the physician to the psychosomatic aspects of urinary incontinence.

The ability to go through the night without voiding is one of the best cystometrograms we have and, under such circumstances, frequencing during the day may suggest a psychosomatic problem. Symptoms must be questioned in detail. For example, *nocturia*, when present, is it true nocturia? With true nocturia the patient is awakened by her bladder *at least twice* during the night. Is it secondary to early awakening for reasons unrelated to the bladder? How much fluid does she drink before retiring? How many times does she get up during the first hour after retiring to bed? Early nocturia sometimes indicates a posterior urethritis.

There is no single symptom or set of symptoms which are absolutely diagnostic of any of the three major categories resulting in female incontinence. A history of enuresis and a bladder response to running water, are very suggestive but are not diagnostic of bladder instability with urgency and urgency incontinence. However, Dr. Stuart Stanton reported urgency as one of the commonest symptoms in patients with true anatomic stress incontinence. Many normal people have a bladder that responds to running water.

DIAGNOSTIC INVESTIGATION

As with symptomatology, there is no single test or set of tests which are absolutely diagnostic of the etiology of incontinence, be it anatomic stress incontinence or any of the other forms of incontinence. The stress test with simultaneous loss of urine with cough is suggestive, but not diagnostic of anatomic stress incontinence. Vesical neck elevation test

of the Bonney type is an often quoted maneuver to aid in the selection of patients for surgery. This procedure is inaccurate. There is probably no way one can elevate the urethral vesical junction without putting pressure on the urethra. The real basis for control of urine with this maneuver is occlusion of the urethra. The Q-tip test when positive indicates descent of the urethrovesical junction and base of the bladder, but is not necessarily diagnostic of loss of the normal urethrovesical relationships. In fact, I find that most multipara have some degree of a positive Q-tip test.

In our questionnaire, patients list drugs which they may be receiving for unrelated diseases and which we know produce bladder symptoms which may result in a gynecological consultation. Dyazides may produce frequency. Phenathiazines may produce urinary stress incontinence because of their alpha-adrenergic blocking activity. Inderal is a beta-blocker and can produce retention, and so on. The number of drugs which effect the urinary tract is legion.

Many physicians feel that they can define the etiological factors in incontinence and make an accurate diagnosis on the basis of clinical examination and history alone. Many authorities in this field recognize a minimum 15% diagnostic error factor. My clinical experience at the Urodynamics Clinic would indicate a much higher error factor, more in the neighborhood of 25%. We must remember that the cure rate of 90% plus claimed by the retropubic procedure enthusiasts or the 80% cure rate claims for the vaginal approach are on patients who have had extensive urodynamic testing. Therefore, physicians who do not believe in urodynamic testing, and rely on history and clinical examination alone can expect a maximum 75% overall cure rate from a retropubic procedure, and 65% from the anterior repair. Today these figures are just not acceptable.

I would like to refer to the other aspects of our routine at the Urodynamic Clinic. I have mentioned the questionnaire, the pelvic examination, stress testing, Q-tip testing, and vesical neck elevation. We carry out a neurological examination including the testing of vaginal and anal sphincter tone. The patient is instructed to present with a full bladder. She voids, the volume is measured, and she is then catheterized for residual which is sent for C/S. A urethroscopic examination with CO_2 to rule-out urethral pathology and to observe the function of the vesical neck with hold, cough, and bearing down maneuvers, is then performed. This is followed by water cystometry and usually a chain cystogram.

One final undisputable fact with regard to patient evaluation. We know that the bladder fills with no appreciable rise in pressure and that loss of urine in anatomic stress incontinence is due to the failure of the intraurethral pressure to rise in response to stress, because of poor anatomical position. On the other hand, the unstable bladder fills with a rising pressure, creates an early sensation of filling, and has a lowered capacity. Therefore, it would seem that some form of cystometrogram is not only essential but mandatory in the original evaluation of the patient.[1]

PELVIC FLOOR RELAXATION — CYSTOCELES AND UTERINE PROLAPSE

This is probably the area which has undergone the biggest change. It is now established that there is no direct relationship between the incidence or severity of stress incontinence and the degree of pelvic floor relaxation. In fact, only 20% of women with cystoceles have incontinence and when incontinence occurs with a cystocele, it is probably coincidental. In the presence of advanced uterovaginal prolapse, if the vagina is outlined with barium paste in conjunction with a metallic bead chain cystogram, it has been shown in many instances that the urethra and bladder are well supported at a high level.

I feel that in many cases a diagnosis of cystourethrocele was made when in fact no such situation existed. A urethrocele is probably a misnomer as it may represent some loss of support of the urethra but certainly does not represent sacculation of the urethra. If this existed the woman would leak constantly. In most instances, it is usually redundant mucosa.

A true cystocele should be differentiated from an anterior vaginal wall prolapse. The former has a shiny appearance with loss, or partial loss of rugae, and is due to overstretching of the vaginal wall and underlying fascia, secondary to the advancing fetal head during labour and it is a true herniation. Anterior wall prolapse is due to tearing or attenuation of the lateral wall fascial supports of the vagina and cervix. It is usually the result of a labour where the cervix and vagina have been pushed ahead of the presenting fetal part. The vaginal rugae are still usually present. We can have a combination of both a cystocele and anterior wall relaxation.

What are the symptoms of a cystocele and uterovaginal prolapse? Very few — I think — unless conditions are advanced to the point where the protrusion either of the bladder and/or the cervix produces discomfort and irritation, purely from its size and the degree of the anatomic disarrangement. The cystocele *per se* is not a factor in recurrent cystitis or a cause of residual urine. The latter is usually due to bladder dysfunction, obstruction, or psychological inhibition.

TREATMENT

Since the late 1920s Halifax has been a centre where the vaginal approach to gynecological problems has been preferred, and in the late 1960s our ratio of vaginal hysterectomies to abdominal hysterectomies was 4:1. On many occasions, regardless of symptomatology, vaginal hysterectomy was combined with an anterior repair and a posterior repair if there was any degree of relaxation. I have done this many times in the past but during the past two and one-half years, I have performed only three anterior repairs in over 350 women seen at the Urodynamics Clinic.

Two years ago, Dr. Emil Tanagho, a urologist and recognized authority in female stress incontinence, participated in Dr. Ostergard's post-graduate course in Los Angeles. He gave the Dr. Atlee Lecture here in Halifax in 1979 and made the statement that gynecologists must stop their attack on the anterior vaginal wall — either as a method of correcting anatomic stress incontinence or treating an asymptomatic cystocele. Denervation of the urethra and scar tissue formation secondary to dissection and suturing, may result in dysfunction. He feels that a retropubic procedure is the approach to anatomic stress incontinence and it is difficult to disagree with this statement. The long-term cure rates have been proven consistently to be superior to the vaginal approach. On the other hand, if a patient has a symptomatic posterior cystocele, the anterior vaginal wall can be repaired but our efforts should be confined to the area of the cystocele and the urethra and urethrovesical junction should be avoided.

Many gynecologists feel that the repair of cystoceles is justifiable. The surgical philosophy in many areas is still, "try a vaginal wall plastic repair first, and if this fails try a retropubic procedure." Hodgkinson has often stated that this philosophy should be relegated to antiquity.[2] I feel that antiquity should also welcome the surgical treatment of the asymptomatic cystocele.

A few observations on treatment from my experience at the Urodynamics Clinic. I feel that we have been very successful with the treatment of perimenopausal women with incontinence, using vaginal estrogen cream instillations and urethral dilatation. These are women with the urethral syndrome type of incontinence due to estrogen deficiency. Our most successful anticholinergic drugs for bladder instability have been Darbid and Tofranil. As mentioned, over 35% of our patients seem to be of mixed incontinence type. When anatomic stress incontinence has been combined with bladder instability or a chronic type of urethral syndrome, we have been able to help the instability or chronic urethral syndrome in many, to the point where the patient has been able to cope with the anatomic stress factor.

Since the beginning of the Clinic, I have personally performed 52 surgical procedures from the 353 patients who have attended the Clinic. There have been another 10 to 15 procedures performed by other gynecologists who have referred their patients for urodynamic investigation. Forty-nine of these procedures have been Burch Slings and three patients have had Marlex Slings. I have had three patient failures, all occurring in the first three to four months of our Clinic operation. I feel that these failures were due to a faulty main diagnosis since I was dealing with a mixed type of incontinence in all three. The main diagnosis made was anatomic stress incontinence when in fact it was bladder instability in two cases and psychosomatic in the third. All patients were dryer than before the operation but they did not feel that the surgical procedure had really been worthwhile. If I had seen these 353 patients prior to the establishment of the Urodynamics Clinic, my operative rate would have been three to four times higher.

Last October (1980), the newly formed Gynecologic Urology Society met in New Orleans. I would like to quote from the Newsletter covering the first annual meeting of this society: "Papers originating from all over the world were presented, and the audience was privileged to hear from probably the most knowledgeable group of men every assembled to discuss gynecologic-urological problems. The majority of the university representatives felt that suprapubic procedures are the only ones that return the urethrovesical junction and the urethra to true anatomic position and physiologic function and should be the prime procedure employed by Gynecologists today."

I am a convert to the retropubic procedure as the prime surgical attack for anatomic stress incontinence and, in most instances, I believe that associated incidental anatomic defects should be ignored. Elevation of the anterior vaginal wall with the Burch procedure not only provides a satisfactory restoration of the normal anatomy of the bladder neck but, in addition, provides correction of anterior wall prolapse particularly if two or three additional sutures are placed along the base of the bladder as modified by Stanton. This procedure will also improve, although not necessarily cure, most posterior cystoceles.

Probably, uterosacral plication should be a mandatory part of a Burch procedure since there is a seven to ten percent occurrence of enterocele after such surgery due to high elevation of the anterior vaginal wall. For those physicians who still believe in the vaginal approach, it certainly should never be considered in the patient with severe stress incontinence who wears a pad at all times; the patient with minimal pelvic floor relaxation, where it is just not possible to elevate the vesical neck high enough vaginally; the patient with chronic chest disease such as asthma; the obese patient; the athletic patient; the patient engaged in a strenous type of occupation where she routinely lifts; the patient with recurrent stress incontinence; and the patient who has had a failed anterior repair for stress incontinence. All agree, these patients should always be approached retropubically. There should never be a second attack on the anterior vaginal wall.

The subject of recurrent incontinence following one or more operations is complicated and brings in other factors for consideration. Was the previous operation poorly performed? Did restoration of the vesical neck fail? Was the initial diagnosis wrong? Did the patient have an unstable bladder rather than incontinence due to poor support? Has the previous surgical scarring of the urethral and vesical area reached the point where operative repair is impossible? If surgery is indicated, should it be a sling operation because of excessive scarring or fixation of the anterior vaginal wall?

Lastly and to repeat, clinical evidence would suggest that failures in the surgical cure of urinary stress incontinence are more often due to improper patient selection, with improper pre-operative differential diagnosis rather than surgical technique. The public is increasingly aware and knowledgeable of the problems associated with urinary incontinence. Some know of studies which report cure rates varying from 50% to 100%, and reports in which patients subsequent to operation are found to have an unstable bladder rather than true anatomic stress incontinence. Perhaps more patients, or their lawyers, will be asking, "Why was a proper diagnosis not made pre-operatively?" *or* "Why was a vaginal approach selected when the majority of opinion would favour a retropubic procedure?"

The most difficult aspect of this whole problem of female urinary incontinence to me is expressed in an old Chinese proverb, "The bladder is the mirror of the soul" so never lose sight of the fact that it is the favourite target organ for female emotions and psychosomatics. □

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"A new scientific truth does not triumph by convincing its opponents and making them see the light, but rather because its opponents eventually die, and a new generation grows up that is familiar with it."

— Max Planck

Gestational Trophoblastic Disease

REPORT ON 45 CASES (1980)

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Report from the Gestational Trophoblastic Disease Registry and Surveillance Clinic 1980

Forty-five patients were registered as having gestational trophoblastic disease in 1980. All had hydatidiform molar pregnancies. All have been or are being followed by the clinic in conjuction with their referral doctor.

Of 44 patients (1 patient moved out the area) 39 (89%) were confirmed to have benign moles and 5 (11%) were subsequently found to have non metastatic gestational trophoblastic disease requiring adjunctive chemotherapy.

→ **5 patients with N.M.G.T.D. 11%**

→ **39 patients with Benign Mole 89%**

The geographical source of the above 45 patients is as follows:

Nova Scotia	24
New Brunswick	9
Newfoundland	10
Prince Edward Island	1
St. Pierre	1

DIAGNOSIS

Of the 45 patients diagnosed as gestational trophoblastic disease, 19 had ultrasounds positive for molar pregnancy and later confirmed by pathology, 1 had no clinical data received from her physician, and 25 were diagnosed from the pathologists' reports of D&Cs performed for the following:

T.A.	5
M.A.	5
Inc. A.	11
Spon. A.	2 (1 dev. N.M.G.T.D.)
Complete	2
Dx by ultrasound	19 (4 dev. N.M.G.T.D.)

ULTRASOUND

Twenty-two ultrasounds were performed. One was initially negative for molar pregnancy but a repeat test was found to be positive. Two were not conclusive and the remainder were positive and later confirmed by pathology. This represents a false negative rate of 13.6%. The incidence of false positives cannot be calculated.

GLOSSARY:

G.T.D. — Gestational Trophoblastic Disease
H.C.G. — Human Chorionic Gonadotropin
N.M.G.T.D. — Non metastatic gestational trophoblastic disease
D&C — Dilation and curettage
Rx — Treatment
Dev. — Developed
T.A. — Therapeutic abortion
M.A. — Missed abortion
Inc. A. — Incomplete abortion
Spon. A. — Spontaneous abortion
Dx — Diagnosis

FOLLOW-UP

Of 44 patients adequately followed to December 31, 1980, 39 were classified as benign moles (no adjunctive therapy required) and 5 (or 11%) as having non-metastatic gestational trophoblastic disease (requiring adjunctive therapy).

Of the 39 patients with benign disease:

26% had normal HCG titres	4 weeks post evacuation
20% had normal HCG titres	8 weeks post evacuation
41% had normal HCG titres	12 weeks post evacuation
5% had normal HCG titres	15 weeks post evacuation
8% required longer than	15 weeks.

Therefore 92% were normal 15 weeks post evacuation.

The 5 patients with N.M.G.T.D. were treated as follows:

Rx immediate (ascites/hydrothorax)	— Titres normal 16 wks post Rx*
Rx 14 weeks post evacuation	— Titres normal 4 wks post Rx
Rx 13 weeks post evacuation	— Titres normal 8 wks post Rx
Rx 8 weeks post evacuation	— Titres normal 9 wks post Rx
Rx 10 weeks post evacuation	— 2nd Rx begun 22 wks post 1st Rx

*"Titres normal" indicates 3 consecutive normal HCG values. (Beta subunit HCG in serum)

INDICATIONS FOR TREATMENT

1. Rising HCG titres
2. Prolonged plateauing of HCG titres (more than 3)
3. High HCG titres associated with ascites and hydrothorax

All the above patients with N.M.G.T.D. were identified within 15 weeks of evacuation and four required one treatment only. The remaining patient is currently undergoing treatment. Twenty-two weeks following her first course of chemotherapy, a second course of treatment was deemed necessary as her HCG values had not returned to normal.

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OBSERVATIONS

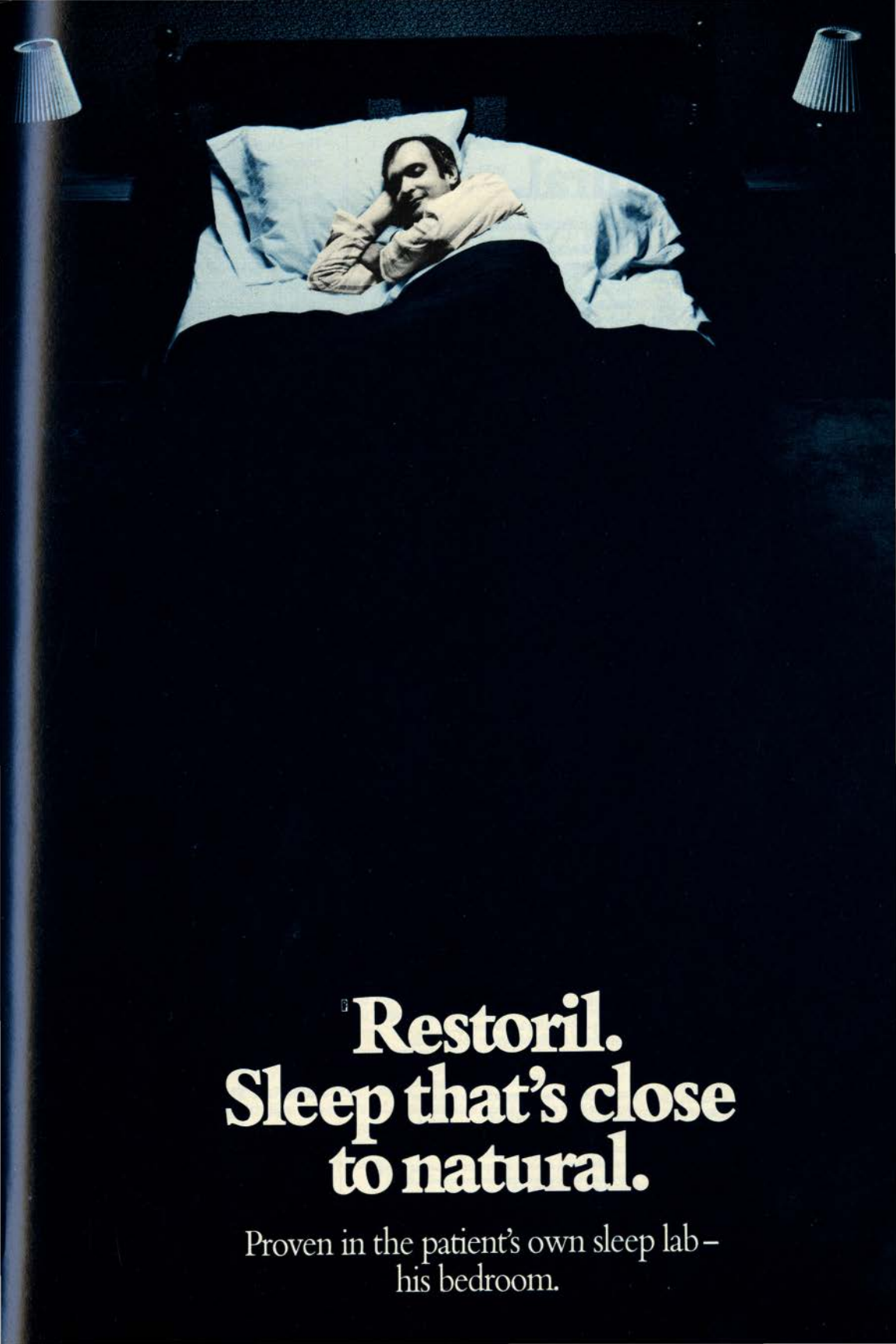
It is interesting to note that of the five patients with N.M.G.T.D., three were found to have ovarian enlargement and two presented with excessive nausea and vomiting. In the group of 40 patients with benign disease, only one was found to have ovarian enlargement and only three demonstrated excessive nausea and vomiting.

Two of the five patients with N.M.G.T.D. demonstrated uterine size greater than dates, one demonstrated uterine size less than dates, and two were not recorded. Of the 40 patients with benign molar pregnancies, uterine size was greater than dates in 13 cases, less than dates in nine cases, equal to dates in 11 cases, and not recorded in seven cases.

CONCLUSIONS

1. Increasing numbers of patients are being registered from the Atlantic Provinces each year.
2. Increasing co-operation between the clinic, the physicians, and the pathologists of the Atlantic Provinces will continue to ensure all patients with registration and proper follow up. (Direct reporting from pathologists as well as physicians is recommended.)
3. In most cases patients requiring adjunctive treatment will be identified with in 15 weeks post evacuation, providing proper follow-up programing is maintained.
4. Following subsequent pregnancies, all patients with a past history of gestational trophoblastic disease require a repeat HCG evaluation 6 weeks post abortion or delivery.

We at the clinic would like to thank all patients, physicians, and pathologists for their continued support and co-operation. □


Restoril.
Sleep that's close to natural.
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NEW MEMBERS

The Physicians listed below have joined The Medical Society of Nova Scotia between March 1, 1981 and May 31, 1981. A most cordial welcome is extended by the Society.

Dr. G. H. Bethune	Halifax
Dr. S. R. Blecher	Halifax
Dr. W. J. Butt	Halifax
Dr. A. K. Clark	Stellarton
Dr. B. K. Flemming	Halifax
Dr. Annette Foyle	Windsor Junction
Dr. B. M. Garvey	Canning
Dr. Sudarshan Goomar	Halifax
Dr. M. A. Harmsen	Greenwood
Dr. R. I. McBeath	Kentville
Dr. J. W. McNab	Windsor Junction
Dr. G. H. Nickerson	Bridgewater
Dr. J. H. L. Robbins	Lockeport
Dr. W. R. Stevenson	Westville
Dr. D. McL. Zwicker	Lunenburg

Pathogenesis and Treatment of Myasthenia Gravis

T. J. Whelan,* B.Sc., M.B.,
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Myasthenia gravis, first described in 1672 by Willis[1], is a disease characterized by weakness and easy fatiguability or striated muscle. Most frequently it affects the oculomotor, facial, laryngeal, pharyngeal and respiratory muscles. It is an uncommon disease affecting 20-40 per million population, and it occurs at all ages with a peak incidence in younger females and older males.[2] The disease is notably variable in its presentation and natural course, as it may occur insidiously or acutely, or it may involve only ocular musculature, or it may be more extensive, seriously compromising respiratory function. From its onset it may progress rapidly in a month or it may remain at the same level for a long time. Partial and complete remissions are known to occur but relapses with increased severity[3] are also common.

In recent years much progress has been made in understanding aetiology of myasthenia gravis and in providing better treatment. The discovery of antibodies to the acetylcholine receptors (AcH R) of the neuromuscular junction (Nm J) was a significant advance in elucidating the pathogenesis of the disease, and this has led to the introduction of plasma exchange and immunosuppressive drugs in the treatment of myasthenia. Advances in intensive care and respiratory support have also made a major contribution to the management and treatment, especially in conjunction with thymectomy and corticosteroids. However, the situation is constantly changing; research into defining more clearly the cause of myasthenia and comparing new and old forms of therapy is currently in progress.

Newer theories suggest that myasthenia gravis may be a group of different diseases with different pathogenic mechanisms and treatments rather than a single entity. The purpose of this essay will be to introduce current theories of aetiology of the disease and discuss the present treatment in the light of these advances.

PATHOGENESIS

Neuromuscular Junction

The typical weakness of myasthenia, induced by exercise, results from failure of neuromuscular transmission. This first became apparent when Dr. Mary Walker, noting the similarity of the disease to curare poisoning, treated a patient with anticholinesterase. Thirty years later Elmquvist, using micro electrode techniques, was able to define the defect in transmission as resulting from smaller than normal miniature end-plate potentials. An argument arose as to whether this was due to a pre- or post-synaptic lesion.[4] By a novel technique of labeling acetylcholine receptors of the post-synaptic membrane with α bungaratoxin, it was shown that the lesion was due to a reduction in available acetylcholine receptors (AcH R) of the post-synaptic membrane.

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Auto Antibodies

The suggestion that myasthenia gravis was an autoimmune disease arose from evidence showing the presence of lymphoid cell aggregates in muscle, the co-existence and association of other autoimmune diseases, and the presence of auto antibodies to muscle striations in cases of myasthenia. In 1973 Patrick and Lindström showed that rabbits immunized with AcH R developed a disease very similar to human myasthenia gravis. These experiments led to the discovery of antibodies to acetycholine receptor in human myasthenia in approximately 87% of patients.

It is now generally accepted that these antibodies are primarily responsible for myasthenia by decreasing available AcH Rs at the neuromuscular junction.[1,2,4,5] It has been shown that they are antibodies of the IgG class which may bind to various regions of the AcH R but not to the binding site itself. It has further been shown that these antibodies decrease available AcH R, by accelerating degradation of AcH R through crossbridging, by blocking AcH R by steric hindrance and by stimulating complement lysis of the post-synaptic membrane.[5] The wealth of evidence continues to support anti AcH R IgG as being instrumental in the cause of myasthenia, but other immunological factors also seem to be involved.

Thymus

The thymus is abnormal in 75% of patients with myasthenia gravis — 10% have thymoma and 65% have hyperplastic thymuses with germinal centre formation. The improvement of the disease following thymectomy had been noted by 1940. Goldstein provided evidence that the thymus might be hypersecreting a hormone responsible for neuromuscular blockade and although the theory had some initial support, the effect could not always be reproduced.[4] Recent work, showing a good correlation between the therapeutic benefit of thymectomy and decreasing thymic hormone activity, again suggests a role for thymic hormone in myasthenia gravis possibly along immunological lines, the nature of which is not understood.

It has been shown that myoepithelial cells in the thymus have an AcH R antigen and these antigens may be responsible for stimulating an abnormal immunological reaction of B lymphocytes which are increased in myasthenic thymuses.[6] However, antibody titres fall only slowly after thymectomy and it is unlikely thymectomy removes a major source of Ab. Recent work shows that thymic cells themselves induce Ab synthesis in lymphocytes outside the thymus.

The precise role of the thymus is thus unclear; it seems to have some part to play not only through hormones affecting the immunological system, but also by providing an antigenic stimulus on immune T cells.

Cell Mediated Immunity

The role of the thymus in T cell differentiation and the discovery of anti AcH R IgG in myasthenia has led to the investigation of the part played by cell mediated immunity, but the results are not clear. Peripheral lymphocytes from patients can be stimulated to undergo increased cell division, and produce T cell factors, e.g. macrophage inhibitory factor. Recent work suggests a subgroup of T Cells, suppressor T cells responsible for inhibiting Ab production are non-functional in myasthenia. This effect is mediated by IgG and mimicked by d-tubocuraraine suggesting that T cells have AcH R and that anti AcH R IgG may be inhibiting suppressor T cells. The situation thus appears to be of considerable complexity, T cells probably have a role to play, perhaps by failing to suppress an autoimmune attack.

Human Leucocyte Antigen (HLA)

In recent years there have been a number of reports of an association between myasthenia and certain HLA groups. Reports vary but there has been a consistent association between HLA-B8 and myasthenia occurring in young females. The relationship of HLA system to disease is poorly understood. HLA seems to be related to immunological responsiveness and general susceptibility to disease.

In summary, the direct role of anti AcH R IgG in causing the neuromuscular transmission defect in myasthenia is more or less accepted. The role of the thymus and cell mediated immunity is less obvious but may be more important in the origin and maintenance of the disease state. Recently, a new light has been given to the involvement of so many factors in myasthenia. It has been suggested by Drachman[2] and others[7] that myasthenia gravis may represent more than one disease. Types of myasthenia gravis may be differentiated on the basis of age, sex and association with different HLA types. Unpublished work by Newsom-Davis indicates that they can be further differentiated by levels of anti Ach R IgG and other parameters such as the presence or absence of thymoma and response to different treatments. This is further supported by the observation that the response of cell mediated immunity to AcH R correlates very well with myasthenia in the older male population. It is felt that the different types of myasthenia suggest different mechanisms of disease triggered off by different factors. Further study into considering anti AcH R IgG, T cells and the thymus, in terms of the different types of myasthenia distinguished, may clarify the picture more. It has already been shown that drugs, e.g. penicillamine, can induce myasthenia with measurable levels of anti AcH R IgG. Study into how this occurs may also be very rewarding.

TREATMENT

Treatment of myasthenia largely developed empirically. As new theories of myasthenia gravis were advanced, different treatments were tried and many have been retained. Some guidelines can be given and the increasing tendency of relating therapy to different types of myasthenia gravis discussed, but it is important to realize that definite statements cannot be made often owing to controversy and lack of clinical trials.

Anticholinesterases

Until recently, the anticholinesterases were the basis of medical treatment of myasthenia gravis, and virtually all patients were started on these drugs. Neostigmine and the longer acting pyridostigmine are most commonly used.

Recently, it has been shown that prolonged anticholinesterase treatment damages the post-synaptic membrane independently of the disease process and effectively reduces the number of AcH R. This may explain why patients fail to respond to anticholinesterase therapy after years of treatment. Because of this complication and the inability of anticholinesterases to restore all muscle strength to normal, plus the problems of overdosage, the current trend is to relegate anticholinesterases to a minor role in the treatment of myasthenia gravis.

Thymectomy

Most studies show a beneficial effect of thymectomy with remission rates ranging from 21-36% and improvement in symptoms in 57-86%.[2] One of the most recent and best organized studies is a computer analysed retrospective comparison of 160 cases from the Mayo Clinic; half surgically and half medically treated and matched for age, sex, severity and duration of the disease. The study proved thymectomy to be significantly better than anticholinesterase treatment with 70% significant improvement compared to 25% in the control.

Determining the indications for thymectomy has always been a problem, because the response to thymectomy may vary in different types of myasthenia. Thymomas are an obvious case, where thymectomy increases survival significantly but has relatively little effect on amelioration of symptoms. In a similar fashion it has been observed that younger female patients do better than older male patients.

Other areas of controversy exist over whether or not early thymectomy and thymectomy involving a very hyperactive thymus results in a better prognosis. The analysed study did not support early thymectomy but it did show a significant increased benefit from removal of a hyperplastic thymus. Currently accepted indications for thymectomy include thymoma, and patients less than age 55 not responsive to anticholinesterases. Good prognostic factors include younger age, under 40, female, HLA type A_1B_8 DRw3. Results are probably best if the thymectomy is done early in the course of the disease.

In recent years a new method of surgery — the transcervical approach to the thymus — has been offered as an alternative to the classic sternal splitting procedure. The latter is still widely used but the newer procedure may prove useful in isolated cases, e.g. debilitated patients. Because it is possible to miss thymus tissue or ectopic thymus, the transcervical method is not widely used.

Steroids

The use of steroids in the treatment of myasthenia gravis is a recent innovation. Theories that myasthenia gravis was an autoimmune disease first led physicians to use ACTH and cortisone. Initial reports were unfavourable: adrenocorticotrophin hormone often made the patient worse; many patients had to be placed on respirators temporarily, and any improvement did not last. With the introduction of more

potent synthetic steroids and the possibility of long-term treatment, the therapeutic effects became evident. It was also noted that the early adverse results were in long-standing severe myasthenics and these complications were much less and the results better in early and less involved cases. Many small studies and preliminary reports of larger studies support their use.[8] The largest recent study claims 69% complete remission and 89% improvement of patients maintained on long-term maintenance dose.

The main disadvantage of steroids is that once started, it is often impossible to wean patients off them and they are then subject to the complications and side effects of prolonged steroids. About 60% become Cushinoid and a third of cases develop serious complications, although these effects can be minimized to some extent by alternate day therapy for maintenance treatment. Another disadvantage is that patients may suffer initial increase in weakness, starting at six days and often maximal by ten days. This can be life threatening and hospital admission is mandatory before starting this therapy. This effect can be minimized if steroids are began gradually. Increasing the dose at any time will cause similar effects and patients should be warned. Steroids are usually started at 25 mg, working up gradually to 100 mg prednisone alternate days. Once normal function has been achieved, the dose should be decreased to the lowest possible amount.

The indications for steroid treatment are even less clearly defined than for thymectomy. It is interesting that whereas younger female myasthenics seem to respond better to thymectomy, older male myasthenics seem to respond better to steroids. Many physicians are now using a course of steroids in all myasthenics in an attempt to achieve a remission.

The therapeutic mechanism of steroid therapy remains uncertain. Steroids undoubtedly exert suppressive effects at many levels of the immune system. In myasthenia, they have been shown to decrease levels of anti AcH R IgG and suppress the stimulating effect of AcH R on peripheral cells. Recently, experimental evidence has revealed a direct effect of prednisone on the neurovascular junction either facilitory or inhibitory depending on concentration. The early weakness seen in myasthenic patients on steroid treatment may be due to the inhibitory effect of a relatively high concentration of prednisone.

INDICATIONS FOR STEROID THERAPY IN MYASTHENIA GRAVIS

Accepted Indications

1. Thymectomy not appropriate,
   i.e. patient more than 55 years old.[1]
2. Failed thymectomy.

Good Prognostic Factor

1. Males.
2. Older age (more than 40 years old).
3. HLA type A_3B_7 DRW2.

Thymectomy versus Steroids

It is generally held that thymectomy and steroids are comparable forms of therapy in different types of myasthenia. It is still uncertain whether the combination will give better results. This seems unlikely: in the few early cases reviewed, patients responding to long-term steroid therapy did not seem to improve after thymectomy significantly and had to be continued on steroid treatment, and younger patients responding well to thymectomy did not respond further to steroids.

It is really too early to draw any definite conclusions, especially with regard to matching treatment to specific types of myasthenia. But, considering the risks of prolonged steroid treatment and the well-established therapeutic role of thymectomy, it seems reasonable at present to suggest the following: early thymectomy in all cases of generalized myasthenia in the appropriate age group, not responding well to anticholinesterases, with the possibility of steroid treatment post-operatively, if necessary.

Immunosuppression

Because of evidence of an immune process in myasthenia gravis, other therapeutic mechanisms have been explored recently. Immunosuppressive drugs azathioprine and 6-mercaptopurine produced improvement in twenty out of twenty-six cases.[9] The beneficial effect takes as long as one year to become established and risks of this treatment such as bone marrow suppression and opportunistic infection should be realized. It has been suggested that they might be useful in cases resistant to thymectomy and steroids. Thoracic duct drainage has also been shown to play a beneficial role, but improvement is maintained only as long as drainage is continued,[9] and this is not very practical clinically.

Recently, treatment by plasma exchange has been introduced, based on the theory that it reduces the levels of serum antibodies and possibly other unidentified circulating factors, and it may be combined with immunosuppressive drugs to suppress cells stimulated by the reduction of antibodies they produce. Initial reports were encouraging, showing plasma exchange leading to short-term remission in a number of cases. Other studies combined immunosuppressive drugs with steroids — an increasing trend which may prove of real benefit in its own right.[10] It may still be possible that different treatment regimens may prove plasma exchange a worthwhile long-term treatment but, in view of the risks associated — notably arterial embolism, anaphylactoid reaction and septicaemia, and the high cost of materials and time — it seems responsible to reserve plasma exchange for controlling severe diseases not responsive to conventional therapy. Such would be the case in an acute fulminating crisis or where one was waiting for immunosuppressive drug therapy to take effect.

Recently, other forms of treatment have been instituted. Anti T cell antiserum shows some therapeutic effect, but results are not encouraging. A new drug 4-aminopyridine, acting on the pre-synaptic membrane to release acetylcholine, has been tried but again does not seem any more useful than current forms of treatment.

It is important to realize that the treatment of myasthenia gravis involves not only the use of separate therapies such as anticholinesterases, thymectomy and steroids, but also requires careful clinical management in the broadest sense.

SUMMARY AND CONCLUSION

In summary, the treatment of myasthenia gravis is still in a developing stage. New trends involve less emphasis on anticholinesterases and increasing use of immunosuppressive measures, notably drugs combined with steroids in resistant cases. A constant feature of both the study of the pathogenesis and treatment of the disease is the growing awareness of different types of myasthenia responding differently to treatments available. It is to be hoped that increasing experience will enable one to recognize the different patterns of myasthenia more easily and to tailor treatment accordingly.

Finally, two possible future lines of research may involve either specifically manipulating the autoimmune response, e.g. with antibodies against idiotypes of the anti AcH R IgG or, as Simpson recently suggested, if damage to the neuromuscular junction is permanent, induction of repair of regeneration of the neuromuscular junction may provide a better therapy. □

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Tension Headache as Part of a Headache Spectrum

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INTRODUCTION

Headache causes many patients to consult their physicians and to lose time from work. There are many types, and the two most common are muscle contraction (tension) and vascular (migraine) headaches. Combinations of the two are also quite common although it is difficult to determine prevalence because most reports have ignored the combined headache category.[1]

Headaches are a protective response to potentially harmful or toxic stimuli. However, they cannot be regarded as a simple one-to-one relationship between stimuli and sensation. Although tension is felt to be the primary stimulus for muscle contraction headaches, tension plays some role in all headaches, regardless of etiology.

This paper questions restricting the term "tension headache" to muscle contraction headache. It also introduces the concept of a spectrum of headache symptoms for evaluating tension headache sufferers. The spectrum is based on clinical observations of the combined headache and is supported by review of literature on the mechanism of tension headache. (Table I)

TABLE I

THE SPECTRUM OF HEADACHE

MUSCLE CONTRACTION	COMBINED	VASCULAR
(Tension) Pain is dull, non-throbbing and bilateral. Can last for days. Has trigger point pain.	Has one or more symptoms from each group.	**(Migraine)** Pain in sharp, throbbing and unilateral. Lasts for hours. Has prodrome and somatic symptoms.

TRADITIONAL DEFINITIONS

Headache classification was formalized in 1962, based primarily on the theories of Ray and Wolff.[2] According to this classification tension headaches are considered to result from muscle contraction, specifically in the scalp and cervical muscles. These headaches vary widely in intensity, duration and frequency, and intensity varies throughout the day.[3] In the chronic form, the patient awakens with a headache which remains throughout the day without any relation to stress experienced. They are the only type of headache that commonly lasts for days without relief.

Migraine headaches are traditionally thought to result from transient changes in the intracranial vessels: constriction followed by dilatation. Migraines are usually intense but last for hours, not days, and are often relieved by sleep.

Other characteristics of both headaches are listed in Table II.

MECHANISM OF TENSION HEADACHE

The original definition of tension headache has been criticized because it does not consider the cause-effect relationship of muscle contraction, head pain, and other factors such as vascular symptoms. To develop a better understanding of the mechanism of tension headaches, we can examine it at three levels: painful stimulus, physiological contributions and psychological aspects.

Painful Stimulus

Although the primary stimulus is often not known, identical symptomatology is seen in headaches resulting from neck pathology, local trauma or muscle stretching in the neck-scalp area by a mechanism of referred pain.

Comparative analysis of soft tissue trigger points and acupuncture points (or "tsubo") shows that location and associated pain syndromes of each corresponds closely.[14] It has been suggested that although these points were discovered independently and labelled differently, they represent the same phenomenon.[15,16]

Although most tension headaches are thought to be caused or aggravated by chronic tension, anything that produces muscle spasms in the scalp or cervical muscles can produce such headaches. Local pathological conditions in the head, neck and shoulders can produce identical headaches.[7] Often, these headaches are related to a previous, sometimes trivial, injury that the patient has forgotten.[7-11] Whiplash is a common example of injury related to tension headache.

Diseases in both intracranial and superficial tissues may be felt as pain in the same target area. Even somatic visceral structures can give rise to pain and muscle contraction in these areas.[2,12] The mechanism behind some patterns of referred pain is often difficult to understand and does not always follow a clear segmental distribution. One example is the distribution of trigger points found in muscle and fascia[7,12] and ligamentous structures.[8](Figure 1)

A trigger point is a localized area of hypersensitivity that produces a characteristic pain pattern when palpated. These patterns are relatively constant from person to person and may be accompanied by vasoconstriction and other autonomic effects.[13] Trigger points often serve as foci for muscle spasms and pain in patients with headache.[8]

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TABLE II

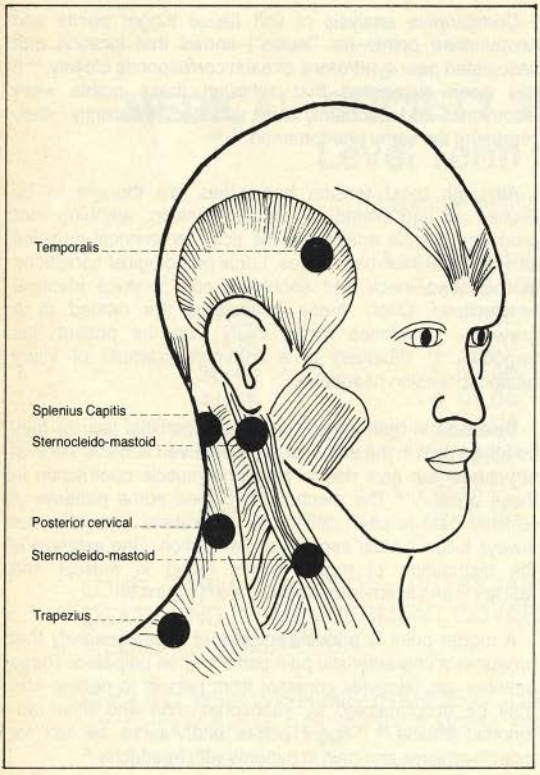
MAJOR DIFFERENCES BETWEEN MUSCULAR CONTRACTION AND VASCULAR HEADACHES

Factor	Muscular Contraction Headache	Vascular Headache
Cause	Contraction in scalp and cervical muscles caused by stress	Constriction followed by dilation of intra-cranial vessels
Premonitory symptoms	None	Visual, sensory or motor disturbances
Type of pain	Dull, persistent, and non-pulsatile	Sharp, throbbing, associated with photophobia and irritability
Intensity	Varies throughout the day	Very intense
Duration	Few hours to weeks	Few hours, often relieved by sleep
Distribution of pain	Bilateral (90%)	Unilateral
Sex of patient*	Male	Female
Severity*	Mild	Severe
State of vaso-constriction**	Most of the time, with an increase during headache	Only prior to headache
Effect of vaso-constrictors***	No effect	Relieves headache

*Usual distribution found by Phillips.[4]
**As found by Cohen.[5]
***As found by Dalessio.[6]

FIGURE 1

TRIGGER POINTS



Physiological Contributions

In the past, techniques such as electromyography (EMG) and arteriography have been used in an attempt to differentiate tension from migraine headaches.[2] The traditional classification did not thoroughly relate the two groups (See Table I), or compare either to non-headache controls with respect to common signs and symptoms or underlying physiological factors.[14] Recent studies demonstrate many similarities between muscle contraction and vascular headaches.[4,5,17]

Muscle contraction has been found to be a non-specific characteristic of all headaches — a result rather than a cause of head pain.[17] It is considered non-specific because contraction of neck and temporalis muscles has been found in all headache sufferers, regardless of etiology. Muscle contraction in tension and migraine headaches has frequently been compared. There is usually no difference in pain location. Somatic symptoms (for example, visual disturbances and unilateral throbbing pain) always found in migraine patients are now being noted in tension headache patients as well.

One main difference between tension and migraine headache patients has remained, however. Migraine patients vasoconstrict just prior to headache while tension headache patients are vasoconstricted most of the time with an increase during headache.[5] Only migraine patients respond to vasoconstriction agents.[6]

Psychological Factors

Psychological factors play a significant role in determining the frequency of headache. Vaughn *et al.* found that a main difference between low and high frequency headache groups was reflected in electromyographic (EMG) responses during

stress.[18] The high frequency group showed no significant change, remaining at a sustained level during stress and relaxation. EMG levels in the low group, however, were correlated to stress, confirming that acute headache is a reactive anxiety response. The chronic headache group was not as responsive to day-to-day changes.[18] These headaches may be related instead to underlying depression; antidepressants are very effective in treating many chronic headaches.[19,20]

Chronic pain leads to many complex behavioral changes. Fordyce points out that in order to treat a chronic pain patient, not only must the presence of pain be considered but also the absence of "well behaviour."[21] Pain behavior is difficult to treat but many "behavioral intervention" methods have been used successfully to assess and to treat chronic pain sufferers.

COMPARISON OF TENSION AND MIGRAINE HEADACHES

Recent clinical observations have shown frequently that patients with muscle contraction headaches have some vascular symptoms and vice versa.[1] The relative contribution of each type of headache may even vary from headache to headache in one patient.[22-24]

Many of the distinguishing features of each type are no longer found to be unique for that headache. Comparisons of patients suffering from mild-severe headache pain and from low-high frequency headache have shown that all patients are familiar with the same wide range of symptoms.[22] The main difference is probably quantitative rather than qualitative,[5] with the type of headache possibly related to severity and the gender of the patient.[4]

Muscle contraction and vascular headaches therefore appear to be part of a headache spectrum (Fig. 1). Perhaps most headache sufferers belong in the combined category, having symptoms from both types, although one type may predominate.

CONCLUSION

The headache due to tension can be of the muscular contraction, vascular or combined type. It is important to consider these categories as part of a spectrum of symptomatology when evaluating the tension headache sufferer.

Separating headaches into categories by muscular, vascular, physical and psychiatric factors is artificial in many cases and can detract from effective patient management. A better concept of the tension headache population is that of a heterogenous group with individual variations in etiology, but unified by the complaint as a reaction to environmental stress. □

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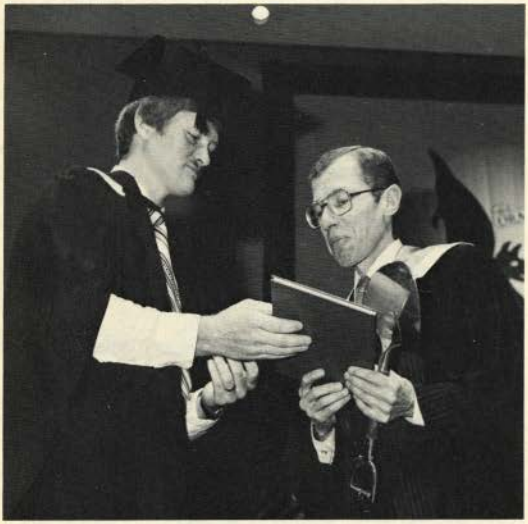
Personal Interest Notes

1981 DALHOUSIE UNIVERSITY MEDICAL SCHOOL CONVOCATION

The 1981 Faculty of Medicine Convocation was held at the Rebecca Cohn Auditorium on May 27, 1981. Eighty-nine students graduated with the degree of M.D., of these students, 49 were from Nova Scotia, 27 from New Brunswick, 4 from Prince Edward Island, 1 from British Columbia, 2 from Ontario, 1 from Quebec, 1 from Newfoundland, 1 from Nigeria and 2 from the United States.



Dr. James G. Holland being presented with the Silver Shovel and Class Yearbook by Dr. Patrick Gill. Dr. Holland was elected Professor of the Year by the Class of '81

HONORARY DEGREES

Honorary Degrees were presented to **Dr. Léon Richard,** Chancellor of l'Université de Moncton and President-elect of The Canadian Medical Association, **Dr. Lloyd B. Macpherson,** biochemist and former Dean of the Faculty of Medicine of Dalhousie University and **Dr. Albin T. Jousse,** distinguished physician, teacher and leader in rehabilitation medicine in Canada.

Dr. Leon Richard receiving his honorary degree from President W. A. MacKay

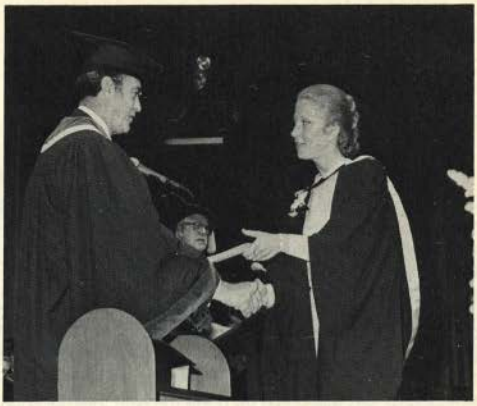
Dr. Lloyd Macpherson receiving his honorary degree from President MacKay

Dr. Albin T. Jousse receiving his degree from President MacKay

Prize Winners At The Convocation Were:

Dr. C. B. Stewart Gold Medal	**Preston Albert Smith**
Dr. John F. Black Prize for highest standing in Surgery	**Patrick Joseph Whelan**
Dr. Clara Olding Prize for highest aggregate in Fourth Year	**Christopher Joseph Gallant** **Peter Hugh Poulos**
Andrew James Cowie, M.D., Memorial Medal for highest standing in Obstetrics and Gynaecology	**Barbara McNichol Stanford**
Poulenc Prize in Psychiatry for highest standing in Psychiatry in Fourth Year	**Rachel Louise Morehouse**
Prize in Medicine for highest standing in Medicine in all four years	**Christopher Joseph Gallant**
Dr. J. W. Merritt prize for highest standing in Surgery in all four years	**Preston Albert Smith** **Patrick Joseph Whelan**
Dr. W. H. Hattie Prize for highest standing in Medicine in Fourth Year	**Joanne Elizabeth Embree** **Katalin Kovacs**
Dr. A. F. Miller Prize for best written review in the field of Respirology in Fourth Year	**Aven Jean Poynter**
Department of Surgery Prize for highest standing in Surgery in Fourth Year	**Patrick Joseph Whelan**
Dr. Albert A. Schwartz Prize for best essay on Orthopaedics in Fourth Year	**Brian Anthony Armson**
Dr. Gordon B. Wiswell Prize for greatest distinction in Paediatric Studies in Fourth Year	**Lyn Susan MacBeath** **Joanne Elizabeth Embree**
Dr. S. G. B. Fullerton Award for Family Medicine in Fourth Year	**Donald Byrne McDonah**
Dr. Lawrence Max Green memorial Award	**Lyn Susan MacBeath**

Dr. Preston Smith being presented with the Dr. C.B. Stewart Gold Medal in Medicine from The Honourable Dr. Gerald Sheehy, Minister of Health, Province of Nova Scotia



Dr. Katalin Kovacs receiving her M.D. degree from President MacKay

FACULTY OF MEDICINE'S CLASS OF '81 STARTS A TRADITION.

The 89 new young doctors who graduated from Dalhousie University Medical School this year started a new tradition when they collected $800 amongst themselves and gave it for medical research in The Maritimes. At a reception held for them by Dalhousie Medical Alumni Association, May 21, the President Dr. Douglas Brown, a Halifax paediatric orthopedic surgeon, received a cheque from newly qualified Dr. Tony Kelly, Enniskillen, N.B., life member of the executive of the Class of '81, with a class pledge to continue giving equal or larger amounts for the next 30 years. The money is to be given initially to The Dalhousie Medical Research Foundation, and after the Foundation has a well established capital fund, the annual gift will be used by the Association for other worthy projects.

Dr. Kelly, former President of Dalhousie Medical Students' Society, and an originator of the idea, said: "In our own hearts we have always considered our Class and its spirit as something very unique and many of us feel we should mark our graduation with some gesture that will benefit not only the medical school, but also future generations of students." He said junior colleagues support the idea of an annual donation from each graduating class and though the first contribution may seem meagre, it will quickly snowball into substantial income for the recipients. "This final gesture as students will create a tradition that will continue to benefit our medical school and its students for many years to come."

Dalhousie Medical Research Foundation was established less than three years ago with the aim of raising $10 million to further research at Dalhousie, The Medical School of The Maritimes, and to encourage medical students to do research for at least part of their career. The Class of '81 has already helped the Foundation financially with money raising projects and has helped to increase students' awareness of the importance of research. Grants by the Foundation to first year medical students of merit have already provided career focus and summer employment to seven successful contendents who now do research under the guidance of senior investigators at Dalhousie University and its affiliated hospitals. □

Correspondence

To the Editor:

RE: NURSING AND M.S.

The Dalhousie M.S. Research Unit is interested in information related to occupations and the incidence of multiple sclerosis. We are interested in learning about any nurse with this diagnosis.

We wish to maintain confidentiality of such information but would need to know the name to ascertain that we are not counting the same patient sources twice. If there is a need for further data we would wish the nurses to fill out a questionnaire and this could be done through their individual physician's office, so the contact would be through the doctor.

Thank you for your assistance.

Yours truly,

T. J. Murray, M.D., FRCP(C), FACP
Professor of Medicine,
Dalhousie University.
Director,
Dalhousie M.S. Research Unit.

To the Editor:

I have just received and read with great interest the February issue of *The Nova Scotia Medical Bulletin.*

All the members of our chapter have not had the opportunity of reading your Bulletin with its many excellent articles about the diseases and conditions familiar to so many of our ostomates. However for those who have read it and for others who will, may I offer our deep gratitude to you and the other members of your profession, who gave of their time to prepare this issue.

We of the Metro Halifax Chapter U.O.A., realize full well how very fortunate we are to have the support and concern of so many eminent surgeons, physicians and others in the medical profession and are deeply grateful to all of you.

Again our most sincere gratitude and thanks to all of you.

Most sincerely,

Rose Chisholm per.
A. Bertha Power, President,
United Ostomy Association,
Metro Halifax Chapter Inc.

To the Editor:

The United Ostomy Association, Inc., would like to commend *The Nova Scotia Medical Bulletin* for its February, 1981 issue which addressed ostomy and inflammatory bowel disease. Using almost an entire issue for these two subjects will certainly help inform the readers.

We are recommending our chapters to make similar approaches to the Medical Associations in their areas. This type of coverage would benefit many people, including both professionals and patients.

Would it be possible for the UOA to get at least 20 copies of the February issue? We would like to distribute them to our Board of Directors. If larger quantities are available we would appreciate your advising how we might obtain them.

Thank you for any consideration you can give our request, and for your concern for ostomy rehabilitation.

Sincerely,

Donald P Binder, Executive Director,
United Ostomy Association,
Los Angeles, California.

To the Editor:

Have just finished reading Dr. Ernst's article on the History of Urology in Nova Scotia. This was exceedingly interesting and well done. I congratulate Dr. Ernst and the *Bulletin.*

I hope that others with such knowledge and ability will prepare more material.

Kindest regards,

John C. Wickwire, M.D.,
Liverpool, N.S.

OBITUARIES

Dr. Roy A. Moreash (74) died in Halifax April 25, 1981. Born in Halifax, he received his Medical Degree from Dalhousie University in 1931. He practised in Berwick, N.S. for 43 years and returned to Halifax upon retirement. Sincere sympathy is offered to his wife and sons.

Dr. Jeptha S. Munroe (90) of North Sydney died May 22, 1981 in the Victoria General Hospital. He graduated from Dalhousie Medical School in 1917 and practised in Cape Breton. He was involved in Church and Community affairs and was a former mayor of North Sydney. He was a Senior Member of The Medical Society of Nova Scotia. Our sympathy is extended to his family.

Dr. James T. Balmano (61) of Yarmouth died May 29, 1981. Born in Scotland, he came to Joggins at an early age and graduated from Mount Allison University before serving overseas with the R.C.A.F. during World War II. After the war he graduated from Dalhousie Medical School in 1949. He is survived by his wife and two daughters to whom we extend our sympathy.

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