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THE NOVA SCOTIA MEDICAL BULLETIN

People Medicine, the Human Approach

Few of us are perfect. Many of our maladies or maladjustments are temporary derangements which can easily be corrected. In fact, Science and Society have concentrated their resources to make this so. Results have been dramatic: tuberculosis, poliomyelitis, septicaemia, diphtheria and many other diseases have been conquered.

Chronic disorders, however, ramain with us. If you happen to have a long standing disability your lot is not so fortunate. For example, if you are a paraplegic, or arthritic, deaf or blind, if you suffer a stroke or from some incurable portracted disease, or just happen to be old, your plight is less fortunate. You may be left to your own resources and with tremendous self determination, intelligence and adequate funds, succeed in making something of your life. You may not. In most instances prolonged skilled and coordinated attention are required and this must be designed specifically for each individual. This journal is pleased to present an insight into some of the interesting developments taking place in Nova Scotia to meet these needs.

Some 10-12% of any population is handicapped, and that means some 20,000 people can benefit — probably more. The desperate need for a humane response was brought home to Ralph Byron when he was in Montreal. As a double amputee, he had gone through a prolonged period of rehabilitation and had been discharged home. He tried to contact another amputee he had previously met in hospital but he did not succeed, for she had committed suicide on Christman Day. Alone and miserable, the problems of severe disability had been too much for her. How many similar people were lingering at home? How many disabled people were hiding away afraid to meet the public or not receiving adequate medical advice?

Ralph determined to find out. During the last two years of his life, he spend nearly all his time contacting agencies and individuals who could assist the disabled. As recreation director of the Flying Wheels, he helped to establish a permanent home for the club financed by the Nova Scotia Department of Recreation. Above all he achieved wide recognition of the needs and abilities of the disabled. A "Ralph Byron" fund was established after his death, for the specific purpose of helping distressed, disabled persons.

AGENCIES GALORE

The first problem that Ralph found was the confusing number of agencies looking after the handicapped. Besides the Federal and Provincial Department of Recreation, numerous agencies exist. Dr. Shears mentions 29 that founded the Rehabilitation Council for the Disabled, which set the wheels in motion for the Rehabilitation Centre in 1956. There are many more. Fortunately the RCDNS (Recreation Council for the Disabled in Nova Scotia) has performed a great task in coordinating these activities. Under the energetic guidance of René Lyons, much has been achieved.

The Federal and Provincial governments have been made aware of the specific needs of the disabled and have become actively involved. Through a wide programme of clinics, seminars, workshops and committees, they have started many projects to provide leisure services and transport and assistance for the handicapped. In fact, there are now active groups scattered around the province, busily engaged in applying their skills and knowledge to make life more rewarding and interesting for many people with many different problems. The size of this response was evident at the first annual meeting of the RCDNS when all the groups joined in an excellent conference. The R.C.N.S. (Recreation Council of Nova Scotia) has now been able to integrate activities for the disabled and able bodied.

WHEELCHAIR ACCESSIBILITY

Much has been done to ensure that someone confined to a wheelchair can have an equal opportunity for employment, education and leisure, as the able bodied. The persistent efforts of Don Curren, through the Canadian Paraplegic Association have gradually borne results. Legislation has been established to ensure that all new public bulidings have wheelchair access. All airports are now equipped for wheelchair passengers and a comprehensive guide can be obtained.

The fact that 1700 disabled athletes, assembled at Toronto in 1976 from 47 countries, shows what can be done. Excellent guides are available now for wheelchair accessibility for all restaurants and places of entertainment in Halifax and Dartmouth.

MEDICAL ASPECTS OF REHABILITATION

The new Rehabilitation Centre, opened on June 1, 1977, is realization of a dream.[1] Some of the earlier dreamers are shown in the photographs in 1953, — Chief Justice J. L. Ilseley, Ian Campbell, National Coordinator of Rehabilitation, and Don Curren, Executive Director of Canadian Paraplegic Society. They formed the Halifax Welfare Society, and this group transformed itself into the Nova Scotia Rehabilitation Council which assumed the task of forming the Rehabilitation Centre. One of the most enthusiastic pioneers was Dr. W. D. Stephenson, who saw the project through many vicissitudes.

We must congratulate Dr. Shears and Mr. Lloyd Caldwell and their colleagues, who through many years of combined effort have at last designed and completed a superb facility for their cause. Rehabilitation has achieved a high reputation in this province. Some 30,000 patients have been treated under very cramped conditions. Now a great opportunity exists to apply all the facets of modern medicine and engineering to alleviate the difficulties of the disabled.

A MEETING PLACE

The plan to organize teams of specialists in rehabilitation medicine together with physiotherapists, occupation therapists and social workers, is an integral part of the scheme which has proved successful in other centres.[2] However, it is important to bring ideas, experience and expertise from many disciplines. A recent review[3] of the work of Dr. Freed, Professor of Rehabilitation in Boston shows how this can be done. Engineers have invented apparatus to help quadraplegics, paraplegics, the deaf and the blind. Often a handicapped person himself will invent a device.

Facilities at the new hospital are excellent. We hope that a happy interchange of knowledge will continue to flourish. Neurologists, orthopaedic surgeons, urologists, psychiatrists, prosthetists, orthotists, engineers, physicians and surgeons with special interest can contribute. With careful consideration this can be possible whilst ensuring the smooth running of this magnificent new facility.

OUR AGING POPULATION

Even if we remain healthy and without disability during our working life we cannot escape old age. Like the insane, the eolderly have often been shockingly neglected. Society is just beginning to realise that the old people do not have to be dumped or disregarded. They need understanding and excellent medical care.

We are fortunate that Dr. Martha Laurence, gerontologist has started a small revolution in geriatric care. At Camp Hill, an excellent ward atmosphere prevails, and each patient has his own room cheerfully decorated with his name. Occupational therapy and social activities keep each person busy.

Geriatric care needs patience, tolerance, care and enthusiasm, for multiple pathology is the rule. We are fortunate to have excellent contributions on pain, neurological examination and incontinence in this journal.

The story of multiple sclerosis is also well presented by a medical student, R. D. Silver. An excellent review of the problems of epilepsy is presented by Dr. LeRoy Heffernan.

DYING

The horror of death amidst a confusion of technology (an impersonal mechanical departure) is now recognized. Norma Wylie emphasizes that at all times we should treat each patient as an individual and not just as a disease process.

THE DOCTORS ART

Science continues to change the physicians role. The range and power of our remedies and means of investigation are constantly expanding.

The contributions in this journal illustrate the concern of many individuals to retain that personal element, whether it is the rehabilitation of a paraplegic, the proglems of sexual inadequacy, or the delicate attendance upon the dying, medicine remains an intensely demanding and fascinating art.

From womb to tomb
The comprehensive nature of our toil
Seeks to alleviate
the wretchedness of man,
to comfort, cure
or circumnavigate distress
making
Each trip worthwhile. □

B.J.S.G.

References

1. **Shears A H:** The Nova Scotia Rehabilitation Centre. *N.S. Med. Bul.* **36:** 64, 1957.
2. **Grogono B J S:** Help for the Handicapped. The Rehabilitation Hospital as a Community Centre. *N.S. Med. Bul.* **48:** 127, 1969.
3. Help for the Handicapped. (Ed) M.D. **21:** No. 6 (June), 1977.

The Nova Scotia Rehabilitation Centre:

The Old and The New

Arthur H. Shears,* M.D., C.M., F.R.C.P.(C),
Halifax, N.S.

For the *Bulletin* in 1957,[1] I prepared a paper outlining the origins of the Rehabilitation Centre in the old Halifax Tuberculosis Hospital on Morris Street (now called University Avenue). At this time, I feel that a brief review of the Centre will be of interest to members of the Medical Society.

In 1956, the Nova Scotia Rehabilitation Centre was founded and established as a Division of the Nova Scotia Rehabilitation Council, which in turn had been formed through the interest of the Halifax Welfare Council. This Council had brought together some twenty-nine voluntary agencies, all interested in rehabilitation, thus preventing unnecessary duplication of effort and facilities. The larger of these agencies were the March of Dimes, the Canadian Paraplegic Association, the Canadian Arthritis and Rheumatism Society, the Nova Scotia Tuberculosis Association, and the Nova Scotia Society for Crippled Children.

From the efforts of these agencies, acting through the Rehabilitation Council, space was obtained in the old tuberculosis hospital, and additional financial grants were secured from the Provincial and Federal Governments. The Centre grew rapidly, beginning with a small childrens' service and progressing to an adult out-patient service before the end of 1956. An in-patient service, with full rehabilitation nursing care, was opened in December 1957.

(From left to right) Lieut. Gov. Clarence L. Gosse, M.D., and his Aide-de-Camp Lieut. Comm. Ray Bieber; Mrs. Janice Elloway, R.N.; and Mr. Murray Sleep, Department of Public Works, during opening ceremonies at the N.S. Rehabilitation Centre, June 30, 1977.

OBJECTIVES, POLICIES AND SERVICES

From its inception, the objectives of the Centre have been:

1. to provide physical treatment and medically coordinated comprehensive rehabilitation services to all who require them, and to date 30,767 patients have benefited from the Centre.
2. to provide undergraduate teaching for medical students and postgraduate training for physicians — especially in Physical Medicine and Rehabilitation; as well as training for physiotherapists, occupational therapists, nurses, social workers, and vocational counsellors. The education of Dalhousie medical students began in 1957 and, two years later, the Centre was approved by the Royal College of Physicians and Surgeons of Canada for training in Physical Medicine and Rehabilitation. Since then, a large number of physicians have been trained successfully in this field. In 1963, the training of physiotherapy students from Dalhousie University was begun.
3. to stimulate research in Rehabilitation Medicine. A creditable number of interesting and useful clinical investigations have been carried out by the staff of the Centre, for presentation to various professional and scientific bodies, and for publication.

The Centre functions as a multidisciplinary team, consisting of a physician, who is a specialist in Physical Medicine and Rehabilitation, physiotherapists, occupational therapists, rehabilitation nurses, social workers, and a vocational counsellor. Close liaison is maintained with both government and voluntary agencies, to ensure reestablishment in the community.

The goals and objectives of the Centre remain the same for the treatment and training of all patients who require them, and even for those who may benefit to a limited degree only. Any person referred by a qualified medical practitioner will be admitted if, in the opinion of an active medical staff member, the patient can be helped to some degree of better comfort or function. Where possible, the patient is first evaluated as an out-patient, or at the referring hospital if in Halifax. However, there will be many occasions, such as for the convenience of patients arriving from some distance, that they may have to be admitted before a trial of rehabilitation is undertaken.

The types of problems dealt with include disability or handicap due to pain; limitation or mobility due to joint problems, paralysis or amputation; inability or disability to function at home, at a job, or in the community; respiratory problems; cardiac problems; certain other neurological dysfunctions; and occasionally psychological dysfunctions. These problems are also found to a large degree in children and indeed in the early years, prior to the opening of the new Izaak Walton Hospital for Children, forty percent of the services of the Centre involved children.

*Professor, Department of Medicine, Dalhousie University and Medical Director & Physician-in-Chief, Nova Scotia Rehabilitation Centre, University Avenue at Summer Street, Halifax, N.S. B3H 4K4.

THE NEW CENTRE

The new Centre, which opened on June 30, 1977, will permit us to expand all the roles so successfully carried out by the old Centre during the past 21 years. It remains to be seen whether we can continue to provide the same intensely personal care in our new facility, but we shall do our utmost to maintain the personal approach and to prevent it from becoming coldly mechanistic.

Located in the heart of the teaching hospitals and Medical School area of the city, obvious advantages are conferred on our patients. In addition, the new Centre is physically connected to the Victoria General Hospital by a tunnel, permitting access to excellent dietary, laboratory, central supplies, and pharmacy services from that institution. This will avoid undesirable duplication of services, and permit the Centre to concentrate on those functions for which it is most expert — the provision of Physical Treatment and Rehabilitation. The tunnel enters the Centre at the basement level, where there are service bays, storage facilities, and an environmental control monitor.

Level One

The Prosthetic and Orthotic Unit is siutated underground at the north end, and is an expansion of the former sister organization, the Nova Scotia Orthopedic Brace Centre. As before, braces and any adaptations required for their patients, may be prescribed by any licensed medical practitioner. This unit has been equipped so that articifical limbs may be supplied in the future, if negotiations with the Department of National Health and Welfare are successful. At the south end of this floor, there is a large occupational therapy area, connecting with an outdoor therapy section. In addition to light, medium and heavy occupational functional therapy, this section will emphasize self care and mobility retraining. Since mobility for handicapped persons if often associated with their ability to drive a motor vehicle, training in access to and egress from an automobile is provided, driver testing and training will be by simulator.

In addition to locker areas for students and staff, this level includes a cafeteria opening on to a terrace. There is access directly to this level from the parking lot.

Level Two

This is the ground level, with access through the main entrance on Summer Street, directly into Reception adjacent to Medical Records. Medical offices and examining rooms, electromyography, speech therapy, audiology, psychosocial and vocational services, are situated around the periphery of the double corridor arrangement.

Centrally, there is a large clinic room, lecture room or auditorium, where many of the special clinics, such as the amputee clinic, will be held. Also in this area, there is a double-unit X-ray Department. Patients being referred to the staff or clinics of the Centre, may be sent here in advance for their X-rays, which will then be available at the time they are later examined, thus expediting their care. Also centrally located is the Health Professions Library, and a modest Board Room for the many meetings and conferences. Finally, Administration and a lobby gift shop occupy the north end of the ground floor.

Level Three

This floor provides extensive physiotherapy facilities, both individual and group, a large treatment and training gymnasium, hydrotherapy facilities including a 22 × 40 pool, two complete immersion Hubbard style tanks, and six whirlpool baths. Both physiotherapy and occupational therapy provide small shops for the design and fabrication of special supports such as collars, splints of various materials, etc. An ultraviolet light room, space for wax baths, etc. are provided in this area. The large treatment gymnasium accommodates equipment for neuromusculoskeletal and cardiopulmonary rehabilitation programs.

Level Four

This floor contains a large indoor recreational area adjacent to an outdoor roof terrace, where organized recreation and recreational retraining will be provided, particularly for those patients undergoing long periods on in-patient rehabilitation. Research space, as yet unequipped until funding is secured, is provided for. These is also a large area for electromechanical controls and air conditioning, and their situation here instead of on the top floor will permit the addition of two more in-patient floors in future years, if and when the need arises and finances permit.

Levels Five and Six

These two floors containing the in-patient units, have been divided initially into four physical medicine and rehabilitation services, and these 52 beds are the only beds specified for these services in the Province. Using a double-corridor arrangement, there is an appropriate mix of four-bed, two-bed and single rooms, and the design is such that even in a four-bed, the patient has a feeling of his own territory. Each bed area is provided with appropriate lighting, the Nova Scotia Rehabilitation design overhead bar, modern adjustable beds, individual closet space, desk, wheelchair accessible lavatory, and toilet rooms.

In addition to linen and equipment storage, the centre core provides bathtub and shower rooms specially designed to permit training in self care for bathing, as early as possible. As each patient should begin to learn to feed himself and to eat with others again, for social as well as physical retraining reasons, a dining room has been included on each in-patient floor, and each accommodates 26 wheelchairs at appropriate tables. The dining room can "double" after hours for television viewing, permiting the solarium at the opposite end of each floor to be available for more quiet pursuits, such as reading or visiting.

Completely accessible by wheelchair, through the main and lower level entrances, service by three elevators (with provision for a fourth if and when indicated), this new building will permit the Centre to deliver in future a much larger quantity of rehabilitation services of the quality for which it has been recognized in the past. In this way, the founders and all Nova Scotians may be justly proud. □

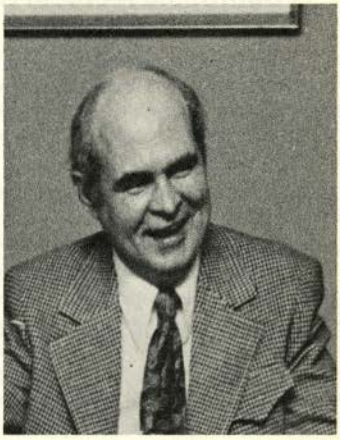
Reference

1. **Shears A H:** The Nova Scotia Rehabilitation Centre, *N.S. Med. Bull.* **36:** 64, 1957.

Pioneers in Nova Scotia Rehabilitation

Don Curren, Exec. Dir., C.P.A. (1960)

Lloyd Caldwell, Q.C. (1961)



The late Mr. Ralph Byron

Dr. W. D. Stevenson (1977)

Hon. William MacEachern, Minister of Social Services, with Archbishop James M. Hayes. June 30, 1977.

Dr. A. H. Shears, Medical Director, N.S.R.C. (1977).

A portion of the crowd attending opening ceremonies at the N.S. Rehabilitation Centre, June 30, 1977.

Letter to All Dalhousie Medical Students

From time to time, members of the Editorial Board of *The Nova Scotia Medical Bulletin* receive enquiries from medical students concerning written contributions they can make to the *Bulletin*, and I have been asked to bring a few suggestions to your attention.

Consider the following opportunities as basic sources for formal papers:

1) **Literature Search Electives** — these first-year electives often entail considerable time and effort, and a few will provide excellent reviews of a particular topic.
2) **Second, Third and Fourth-Year Electives** — always require written reports, and many can be adapted to yield interesting papers — especially those electives pursued outside our usual teaching facilities or even outside the Province.
3) **Summer Research Projects** — although these are becoming more rare, due to the current scarcity of research funds, we have published some excellent reports of student research projects — occasionally with a Faculty member as co-author.

For your manuscripts, editorial advice is available, and members of the Department of Preventive Medicine will provide assistance with the analysis and presentation of your data, when necessary. In addition, we can enlist the aid of the Audio-Visual Division to produce first-class diagrams and photographs.

Although we hope to produce one all-student issue annually, this may not always be possible and perhaps a more realistic goal is to include at least one student paper in every issue. Please feel free at any time to see me in my office in the Clinical Research Centre.

Aden C. Irwin, M.D.,
Associate Editor,
The Nova Scotia Medical Bulletin. □

The Medical Society of Nova Scotia

124th Annual Meeting

Lord Nelson Hotel, Halifax
November 17-18, 1977

Program details to follow via Presidents' Newsletter

The Neurological Examination in the Elderly

T. J. Murray,* M.D., F.R.C.P.(C),
Halifax, N.S.
W. Pryse-Phillips,** M.D., M.R.C.P., F.R.C.P.(C),
St. John's, Nfld.

Unless we are aware of the progressive changes that commonly occur with age, confusing problems can result when we apply to the aged the standards of the normal neurological examination in the younger person. Whether one can regard many of the changes of ageing as being "normal" is a moot point, but, for the purposes of this discussion, we will comment on alterations that are commonly seen during the process of ageing.

The psychological changes of the old are well known. We have all seen the elderly gentleman sitting on the front steps talking about going over the trenches in World War I, or detailing the happy adventures of his misspent youth, even though he does not know what day it is, cannot remember whether he has had breakfast and has no idea where his pipe is. The elderly often seem to "live in the past" because it is the only thing they remember clearly.

Emotional changes are also frequent, the elderly commonly becoming more withdrawn, depressed, irritable and less patient.

Anatomical changes accompany the psychological and emotional alterations. The brain decreases in weight after the sixth decade and the neurons progressively decrease in number. Pigmentation occurs in the neurons, and senile plaques and neurofibrillary tangles begin to appear. Vascular changes include endothelial proliferation, medial fibrosis and hyalinization. In the peripheral nerves there is an increase in both endo- and perineurium and a reduction in the number of nerve fibres.

Cerebral blood flow progressively decreases after age 20, cerebrovascular resistance progressively increases and the cerebral oxygen consumption decreases.

Nerve conduction velocity decreases in most nerves with age, probably due to a progressive reduction in the number of large, myelinated, rapidly-conducting nerve fibres.

NORMAL VARIATIONS ON EXAMINATION

About 80% of patients over the age of 65 will have some of the following abnormalities on the neurological examination, probably as a result of the pathological alterations in the nervous system seen with ageing. We will not discuss the neurological disorders that commonly occur in the elderly *per se*, but these changes are often found on examination in the elderly patient who is otherwise "normal" and must be interpreted properly.

Mental Status

Patients are often forgetful, with involvement of recent memory more than of past memory. Rigidity in attitudes, resistance to change, depression and withdrawal are common. They sleep less and waken early. It is common to oversedate patients to make them sleep the number of hours required by young adults, when they need less sleep.

The change in vision can be due to decreased lens elasticity, optic nerve degeneration, senile choroiditis, cataract formation, corneal opacities, retinal vessel changes of atherosclerosis or hypertension, and complications from other medical diseases. Cerebral changes may also cause difficulty in the perception or understanding of what is seen.

Hearing changes occur progressively with age, particularly for high tones. One of the complaints is the "cocktail party syndrome", when confusion of sounds occurs if many people are speaking at the same time when listening to one person, although the conversation could be clearly understood if heard individually. There is also the phenomenon of recruitment when hair cells are lost in the cochlea — as the sounds get louder, the hearing change from normal is less and when very loud may be normal. Thus the old chap will say "speak up, sonny" and when the young man yells at him he jumps back stating "you don't have to yell!" At a certain decibel level his hearing may be normal or near normal.

Cranial Nerves

The senses of smell and taste are often blunted and can contribute to decreased enjoyment or desire for food. Pupils are often irregular, sluggish in their reaction to light and smaller than in youth. Arcus senilis is common and the patient may appear to have slight ptosis. Paresis of upward gaze is common in the elderly and convergence is poor. There may be a decreased tone in the face and neck muscles. Decreased hearing, especially for high frequency sounds, and decreased vision are common. These are important as they add to the syndrome of relative sensory deprivation that can cause confusion, paranoia and disorientation in the elderly, or cause a feeling of imbalance when standing.

Motor Examination

There is a decrease in muscle bulk generally, but most particularly some atrophy of the small muscles of the hands and feet. This is particularly noticeable over the back of the hand where guttering can be noticed. Despite this, strength is often well preserved. The small muscle atrophy probably results from dropout of the peripheral nerve fibres, but may be contributed to by the cervical spondylosis that is seen in virtually all patients over the age of 70 and by other changes in muscle, in the spinal cord and brain. There may be fibrillations seen in the muscles but these are usually asymptomatic.

Minor basal ganglia changes are very common and a parkinsonian picture with rigidity and hypokinesia characterises the "typical" aged person. An actor asked to play the

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part of an octogenarian, will assume a Parkinsonian posture and move in a slow, stiff, Parkinsonian manner. Senile tremor is common and similar to essential tremor at other ages. As the decrease in muscle bulk occurs, perhaps associated with mild ischemia, muscle cramping is a common complaint.

Sensory Examination

Most elderly patients have a decrease in peripheral sensation, particularly to vibration, touch and position sense. This usually begins in the feet and slowly ascends. The sensory changes develop slowly during adulthood and old age in men but begin to develop in women progressively after the menopause. The peripheral sensory changes are consistent with degeneration of peripheral neurons. Meissner's corpuscles and other peripheral nerve structures can also be shown to atrophy with age. Although vibratory sense is the most prominent modality lost, pain, touch and temperature are also involved in some patients. These sensory changes are often associated with paresthesiae.

Reflexes

Reflexes generally decrease with age, and many elderly patients have loss of their ankle reflexes. The appearance of primitive reflexes may be noted in the elderly including an increase in the jaw jerk, the appearance of palmomental reflexes, and even occasionally a Babinski sign. If there is diffuse cerebral change then the face-hand test of Binder, and the crossed upgoing toe sign is often positive bilaterally.

Stance and Gait

With increasing age, the gait tends to be less brisk and the stride shorter and more cautious. Very elderly patients tend to move *'en bloc'* and take many small steps when turning. Their postural reflexes are poor and they tend to get off balance easily. Some will have a Parkinsonian type of gait, without the full-blown syndrome, and may have difficulty in stepping off.

An interesting occurrence in the elderly is a peculiar fear of walking even though no specific abnormalities can be found to explain their apprehension. The French refer to this as *'astasia triadante'*. Occasionally, they may be so fearful of walking that they will not take a step at all and freeze; when encouraged to step out, they may clutch on to things or even drop to their knees.

Another common phenomenon in the elderly is the *multisensory* syndrome, in which the patient has difficulty orienting himself in space. We know where we are in space because of our peripheral proprioceptive receptors, our labyrinthine system and our vision. The elderly patient often has poor peripheral proprioception, degeneration in the labyrinth and VIII nerve, and poor vision. He thus has difficulty orienting himself in space and feels as if he was swaying. Such a patient will often present complaining of "dizziness" although he really means a feeling of unsteadiness. Drachman described this as "the earthbound astronaut syndrome". □

Further Reading

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Urinary Incontinence in the Elderly

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Failure of urinary continence has devastating consequences for an individual. It leads to social limitation, rejection, feelings of low self-worth and frequently precipitates a request for institutional placement. Attitudes of families and health care professionals towards the incontinent are complicated by deep-seated aversion to excrement and the notion that failure of continence reflects personality inadequacy. Medical teaching on urinary incontinence concentrates on those disorders for which surgical procedures have been developed — stress incontinence — and situations where incontinence results from surgical treatment — post-prostatectomy incontinence. A majority of individuals with urinary incontinence are elderly and the commonest cause of urinary incontinence — the uninhibited neurogenic bladder — often unrecognized.

NORMAL URINARY FUNCTION

Urinary continence is a socially conditioned reflex which is usually developed by the age of 4 years. At birth, micturition is a spinal reflex activity but by the age of 2 years, infants become aware of the imminence of micturition and may void in a suitable receptacle if one is to hand. The capacity to inhibit the micturition reflex until an appropriate time and place develops over the next two years of life, followed by the ability to empty the incompletely filled bladder in anticipation of situations where voiding cannot be achieved, as before a journey or before entering a theatre. The ability to initiate voiding of the incompletely filled bladder is restricted to human and dogs.[1] Only in man is it under voluntary control, the apparent volitional voiding of dogs is in reality a complex reflex triggered by olfactory stimuli. The delay in the aquisition of continence reflects incomplete myelination at birth, however, by the age of 4 or 5 years a majority of children are continent by day and night, although at any age, occasional episodes of incontinence may occur if the sensory stress of a full bladder overcomes the inhibitory stimuli from higher centres.

The bladder is not a simple balloon which fills from above and drains from below and conforms to simple plumbing rules. It is a complex organ the detailed functioning of which is incompletely researched. The bladder arises from the embryonic allantoin, the Wollfian ducts and the urogenital sinus, and at the trigone these three components meet. Innervation of the bladder is primarily parasympathetic although sympathetic fibres are present at the trigone. Bladder musculature is a multi-unit syncytium arranged in outer longitudinal, circular and inner longitudinal layers. At the bladder neck the circular muscle coat disappears and the detrusor is continued into the urethra as an outer circular and inner longitudinal layer. The physiological basis of continence is the ability to maintain a urethral pressure higher than that within the bladder.

In the normal micturition cycle, urine entering the bladder is accommodated by passive distension without increase in pressure until a critical volume is reached. Sensations of distension arise from stretch receptors in the detrusor and pass to the sacral micturition centre, the lowest co-ordinating centre in the nervous system. The activity of the sacral micturition centre is modulated by several higher centres which are connected by afferent and efferent pathways in the lateral and posterior columns of the spinal cord. The desire to void is perceived in the parietal lobe and the micturition reflex is inhibited by centres in the frontal lobe and mid-brain. Waves of pressure induced by auditory or sensory stimuli or increases in volume are inhibited by the higher centres. Facilitation of the reflex is by centres in the hypothalamus and pons.[2]

THE ABNORMAL BLADDER

Urinary incontinence is defined as an involuntary escape of urine from the lower urinary tract to such a degree that it causes social or hygienic inconvenience. Table I shows a classification of urinary incontinence. Stress incontinence is very common in women of all ages. Wolin[3] surveyed 4000 healthy young females and fifty percent had some degree of stress incontinence, with 16% reporting stress incontinence on a daily basis. Stress incontinence does not seem to be increased in frequency in elderly women. Uterine prolapse is not an invariable association, and operative treatment is infrequently successful. No further consideration will be given to stress incontinence or the overflow incontinence associated with bladder neck obstruction.

TABLE I

CLASSIFICATION OF URINARY INCONTINENCE

1. Enuresis
2. Secondary to congenital malformations
3. Overflow secondary to obstruction
4. Stress incontinence
5. Neurogenic
6. Psychogenic

The uninhibited neurogenic bladder, or unstable bladder commonly underlies urinary incontinence in the elderly. Incontinence may be "transient", occurring in 25-50% of hospital admissions of the over 65 year old patient. It is important to differentiate between "transient", and "established" incontinence, as the former frequently is remediable whereas established incontinence occurring in approximately 10% of the elderly usually reflects an uninhibited neurogenic bladder. Some common causes of transient incontinence are given in Table II. Spurious incontinence refers to a situation where a patient is apparently incontinent but environmental factors have forced him to be. An example would be of a patient admitted to hospital, placed in a bed with bed rails, given a diuretic and a hypnotic. Incontinence under such circumstances is inevitable. Similarly expecting a visually

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impaired, tremulous man to use a urinal in bed without spillage is unrealistic. Such incontinence would be spurious and may not reflect an underlying uninhibited neurogenic bladder.

TABLE II

TRANSIENT CAUSES OF INCONTINENCE

1. Spurious incontinence
2. Urinary tract infection
3. Acute brain failure (delirium)
4. Secondary to fecal impaction
5. Secondary to anticholinergic drugs
6. Secondary to sedation

Investigation of Incontinence in the Elderly

In the investigation of incontinence, the non-specialist without access to special investigation techniques will often obtain much useful information from a history, a physical examination including neurologic and mental state examination, drug history, and examination of the urine. The pattern of incontinence may be recorded on an incontinence chart — a chart on which an attendant marks every 2 hours noting whether a patient is dry or not, and if he voids when a urinal is presented. An incontinence chart may be both diagnostic and therapeutic, the latter by focusing the patient and the staff attention on regular voiding.

Cystoscopy, cystometry and urethral pressure profiles are useful studies in making exact diagnoses, however, in the absence of such tests the physician may base a therapeutic approach on the history, physical examination, examination of the urine, the pattern of incontinence and knowledge of the residual urine. Retrograde cystometry is a technique whereby saline or gas fills the bladder at a known rate and intravesical pressure is recorded. The patient records his first desire to void and the sensation of "micturition imminent". The pressure effects of cough, strain, and auditory and sensory stimuli on intravesical pressure may also be recorded. The urethral pressure profile is a procedure for measuring the functional length of the urethra and the urethral pressure which may be applied vis a vis intravesical pressure. Cystometry separates the atonic bladder from the unstable bladder. The cystometric characteristics of the unstable bladder are contrasted with the normal bladder in Table III.

TABLE III

CHARACTERISTICS OF UNSTABLE BLADDER (Uninhibited Neurogenic Bladder) VS NORMAL BLADDER

Unstable Bladder	Normal Bladder
Residual urine variable	Small residual urine
Bladder capacity around 150ml	Bladder capacity around 400ml
Late "Desire to Void"	Desire to void at 100-150ml
Uninhibited bladder contractions	No uninhibited contractions
Rapid pressure rise with filling	Slow pressure rise with filling

MANAGEMENT OF INCONTINENCE

a) Prevention

The challenge to the physician is to identify the cause of transient incontinence and prevent transient incontinence from becoming established incontinence. The urinary catheter is not a treatment for urinary incontinence despite its frequent use in hospital for this purpose. Some methods of preventing transient incontinence from becoming established incontinence are shown in Table IV.

TABLE IV

PREVENTION OF ESTABLISHED INCONTINENCE

1. Keep on incontinence chart
2. Investigate the cause of incontinence
3. Adapt ward routine to the patient's needs
4. Avoid drugs which will precipitate incontinence
5. Prevent and treat fecal impaction

b) Treatment

Many cases of transient incontinence will resolve spontaneously with improvement in the patients underlying acute illness. Prolonged catheterization for transient incontinence may lead to an infected and contracted bladder, therefore an informed approach to the problem is necessary. When incontinence has become established the management involves measures listed in Table V. Electronic devices, either implanted stimulators, vaginal or anal plugs which stimulate pelvic floor muscles to compress the urethra are of very limited use in the young and of no value in the elderly at their present state of development. Retraining of reflexes by regular voiding and intermittent bladder dilatation are of some value.

TABLE V

MANAGEMENT OF URINARY INCONTINENCE

1. Retraining reflexes
2. Bladder dilatation
3. Drugs
4. Hygienic measures
5. Electronic implants

Cholinergic drugs are of use in the atonic bladder and anti-cholinergic and muscle relaxant drugs are used in the uninhibited neurogenic bladder. A list of drugs which has been used in the management of the uninhibited neurogenic bladder is given in Table VI. Emepronium Bromide and Flavoxate Hydrochloride are not yet available in Canada and reports of the efficacy of drug treatment for this condition are not consistent. The combination of Pro-Banthine and Orphenadrine has been described as an effective combination. Estrogens applied locally to the vulva and urethra are useful in some elderly women. An atrophic urethritis often occurs in the post-menopausal woman. The characteristic appearance of the mucosa which is atrophic and keratotic reflects the estrogen deficiency. Apart from the mucosal change, the turgor of the peri-urethral tissues is reduced in the post-menopausal state.

In conjunction with drug treatment, or when drug treatment fails, hygienic measures must be employed. These include external urinary collecting appliances in the male, but

TABLE VI

DRUG TREATMENT OF THE UNINHIBITED NEUROGENIC BLADDER

1. Atropine
2. Probanthine
3. Imipramine
4. Emepronium Bromide
5. Flavoxate Hydrochloride
6. Orphenadrine
7. Estrogens

however, there is no satisfactory external collecting appliance for the female. The ancient Egyptians used a golden phallus shaped instrument for urinary incontinence and no current device is any more efficacious. Hygienic devices for incontinence in the female include incontinence pads in a bed or chair and incontinence pads worn by the patient. The latter consists of a hydrophobic woven material in the form of pants with a marsupial pouch in which absorptive material is placed. These latter, Kanga pants, are not yet available in Canada. The subject of hygienic measures in the management of established urinary incontinence is reviewed by Willington.[4] Bladder training, or more correctly, patient training, in anticipation of bladder filling may be carried out in the institution, the patient at home may use a kitchen timer or alarm to remind herself to void.

Restriction of fluid in the evening and the appropriate use of diuretics including the timing of their administration will reduce nocturnal incontinence. The prolonged use of diuretics is frequently unnecessary in the elderly. In many individuals their administration is for dependent edema which has a local cause and in any case does not respond well to diuretic therapy. Rapid acting, "loop" diuretics, cause abrupt changes in internal fluid compartments leading to postural hypotension and present the bladder with a large excretory load over a short period of time. This combined with locomotor impairment present in many elderly patients precipitates incontinence and frequently falls en route to the bathroom.

There is still controversy over the long-term indwelling catheters. In the absence of urinary tract obstruction, indications for their use within the institution are very limited. Occasional patients with decubitus ulcers or with perineal dermatitis from urine may need indwelling catheters. Most other patients can be managed without a catheter. Occasionally some patients may be returned to the community provided urinary continence is assured. In this situation long-term urinary catheterization with a collecting bag worn on the leg is acceptable.

CONCLUSIONS

The physician approaching an incontinent elderly patient should ask himself, what has precipitated the incontinence?, what place do drugs and medications play in the symptom?, what factors are reversible?, and how may I preserve the patient's dignity and independence by helping them to achieve urinary continence? A single episode of urinary soiling should not cause a patient to be labelled irremediably incontinent, "geriatric" or "senile". To understand and manage urinary incontinence we must have a broad view encompassing the individual, the urinary tract, the neural-controlling mechanisms and the external precipitating of aggravating factors. □

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Phone — 429-4299 □

Pain Management in the Elderly

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It comes as a surprise to all of us that we never feel any older: there are just more things that we cannot do as well or as often, or that we have to relinquish. This is frustrating enough, but when in addition, pain is added to the mixture, this is an unfair burden, restricting activity, reducing our capabilities, lessening our enjoyment, turning us inward upon ourselves, and finally becoming an all-corroding, ever present, unwelcome companion. Joy in the world is gone, talk becomes tedious, we don't want to inflict ourselves on others, our days stretch out endlessly, and our nights become long and lonely vigils.

We are puzzled and confused by this sudden attack on our integrity, our wholeness. We go to the doctor, and we agree with him that its just old age, and that a few pills may help. We try the pills and they don't help, but we know we have to take them because if we don't, the doctor may not help us again. We go back again, and get more pills, and become more confused, more frustrated, and the pain gets worse. How can we break out of this prison, is there no end but going on for ever and ever, day after day, like this?

Thus might the elderly patient with pain speak to us, if he or she thought we would listen. Too often, they are too puzzled and confused and we are too busy to listen. Time to listen, time to reflect and choose wisely: these are key factors in the management of pain in the elderly.

CHARACTERISTICS OF CHRONIC PAIN

The characterists of acute pain are well known and pathognomic patterns related to disease of certain organs are well recognized. The body reacts to acute pain with increased sympathetic activity: hypertension, tachycardia, sweating, immobility and anxiety. By contrast, chronic pain is characterized by decreased sympathetic activity, loss of muscle tone, constipation, introspection and social withdrawal, all concomitants of depression.

The time characteristics of chronic pain are often helpful in diagnosis, when taken with the nature and distribution of the pain. In **rheumatoid arthritis,** the pain characteristically migrates from joint to joint and is accompanied by swelling and local tenderness. Pain will often subside for long intervals, but becomes more constant as deformities appear. The pain of **osteoarthritis** increases progressively during the day with activity and, characteristically, there is morning stiffness which has to be "worked out". Sensitivity to barometric and humidity changes is more characteristic of osteoarthritis than of rheumatoid arthritis, though both may exhibit this symptom.

Nerve root compression due to disc prolapse or degeneration, or to vertebral body collapse is usually confined to a single root distribution, and commonly starts with an acute episode which settles with rest, then recurs, with subsequent episodes increasing in frequency and severity.

The **neuralgias** are also confined to a single root, though they may be bilateral. They usually start suddenly, and except for those following herpes zoster, and usually without apparent cause. They may be constant, varying in intensity with tension or emotional upset, or intermittent and severe, with multiple local triggers, as in tic doloreux.

Nerve entrapments follow the distribution of a portion of a peripheral nerve, and frequently develop with weight gain and loss of muscle tone, though they may follow minor injury. The pain is usually constant, increased by activity and quite disabling.

Sympathetic dystrophy or **causalgia** usually follows trauma to an extremity, which may be minor. The pain is characteristically burning, diffuse and poorly localized, and in a glove or stocking distribution.

Peripheral neuropathy with increasing loss of vibration and position sense, and progressive hypoaesthesia, is common in the elderly. It is often associated with diffuse burning pain which must be distinguished from sympathetic dystrophy and ischaemic night pain.

Myofascial pain syndromes show abnormal patterns of pain referral, often extending across several dermatomes, and occasionally crossing the midline. This "non-anatomical" distribution frequently gives rise to suspicion of malingering or neurosis, when in fact, the pain arises in trigger areas in muscle.

Diagnostic Clues

A thorough functional enquiry may elicit symptoms suggestive of an underlying malignancy, which require appropriate investigation. A history of heavy metal exposure, exposure to other toxic substances, or poor nutrition, in addition to other well recognized causes, may support the diagnosis of peripheral neuropathy.

A careful neurological examination may show evidence of nerve compression in decreased muscle power in certain muscle groups, with depressed reflexes and subjective or objective hypoaesthesia. Examination of the cervical and lumbar spines may indicate the likely site of compression, or indicate the presence of arthritic changes. A careful search for muscle spasm, and for trigger areas in muscle appropriate to the pain patterns, should be made. Systematic documentation of the range of motion, amount of pain, swelling, fluid, deformity and crepitus will allow later correlation with x-ray findings. It should be emphasized that gross physical deformity and positive x-ray signs may be present with little or no pain, and conversely that pain may be a major problem with minimal signs. Appropriate blood tests may help to support the diagnosis.

Management

Management of the patient's problem includes sorting out the source of the pain itself, distinguishing this from the patient's reaction to pain and providing appropriate treatment in both areas.

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Where the source of the pain is obscure, consultation with many specialists, or referral to a specialized pain clinic may be necessary.

Answers must be sought to the following questions:

1. Is the pain caused by hidden malignant disease?
2. If the pain is limited to specific nerves or joints, is it amenable to surgery?
3. Is the patient physically able to tolerate surgery?
4. Is the patient psychologically capable of rehabilitation after surgery?
5. Are there psychological aspects of the patient's relation to pain, such as anxiety, depression, or secondary gain (financial or emotional) that require treatment?
6. After all investigations, is the diagnosis still in doubt, and if so, what is the appropriate referral route?
7. Is the patient addicted or habituated to analgesics, drugs or tranquilizers? Removing all medications can frequently result in reduction of pain complaints and behaviour.
8. If the pain requires long term therapy with analgesics, antispasmodics or tranquilizers, are the detoxification systems of the liver and kidney able to handle this continuing load? And are there undesirable side effects which could be avoided by a change in medications?
9. If single nerve root involvement or substantial sympathetic nervous system involvement is present, would these respond to local therapy?

These questions are easily asked, but the answers will depend on the careful collection of all available data combined with the judgement or intuition of the physician concerning that particular patient.

In pain clinics, a common first step after history and investigation is the use of diagnostic nerve blocks. A differential spinal or epidural, carried out with concentration of local anesthetics that block first the sympathetic, then the sensory, and lastly, the motor fibres can identify sympathetic mediated pain, and point towards a diagnosis of central pain or pain of psychologic origin.

Sympathetic mediated pain often responds to repeated nerve blocks of the appropriate sympathetic nerve supply.

Pain mediated through sensory or motor nerves frequently indicates root compression, which may respond to injections of local anesthetic drugs combined with steroid suspensions, or if this fails, indicate a need for surgical decompression.

Pain of central origins may respond to behavioural conditioning, while pain of psychologic origin may benefit from psychotherapeutic techniques.

Pain arising from arthritic joints may be temporarily relieved by steroid injection, or by arthoplasty or fusion. Developments in surgery and anesthesia have made such major surgical procedures acceptably safe in the elderly, and patients should not be excluded from correction of disabling, painful joints solely on the grounds of age or coexisting disease.

Where there is multiple joint involvement, as in the arthritides, long term drug therapy with anti-inflammatory agents and possibly steroids may be the best choice. A surprising number of these patients achieve useful long-term relief from acupuncture or the use of a trans-cutaneous electrical stimulator, to the extent that they are able to reduce drug usage and increase activity levels for periods ranging from 2-6 months following treatment.

Muscle spasm associated with joint disease frequently perpetuates pain, and may be reduced or abolished by such physical techniques as physiotherapy, massage, manipulation, or traction.

Medicine is frequently taught in terms of single disease entities, but the elderly patient with pain presents a multifaceted problem of the interaction of the effects of aging on many systems. Complete cure will seldom be possible, but the amelioration of symptoms by the treatment to the maximum extent of those problems that are treatable, combined with intelligent management of the ongoing, less amenable problems, will frequently lead to a reduction in pain, reduction of medications, an increase in activity, and an improvement in the quality of life for the elderly. □

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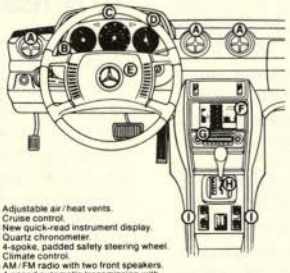

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Inner Needs, Outer Habits of the Senior Citizen

Fran Sutherland,* B.Sc., P.Dt.,
Halifax, N.S.

To quote Professor Jean Maxwell, "We need to fit our current eating pattern and needs to what we are, now, rather than from our culture."

Proper nutrition can be a means of treatment but better still, a strong arm of prevention.

The Nutrition Division of the Department of Health, in keeping with its primary role of prevention, designed a food intake record to be completed by either residents, staff, or a combination of both, in Homes for Special Care in Nova Scotia. It is hoped that physicians in the province will use this form when assessing the total health status and needs of their elderly patients.

INTRODUCTION

Interaction of Nutrition and Aging

All too often with aging there is (1) decreased income (2) disabilities (3) loneliness. Less calories are needed so the quality of food must be higher. Nutrition status evaluation should be based on general appearance, medical, social, personal, dietary, physical examination and testing. The nutritional status, proper or otherwise, of the patient cannot be discussed as an entity but must be as an evaluation of the total person.

In 1975, the Department of Health, in co-operation with the Department of Social Services, employed a full-time nutritionist to work with the Homes for Special Care. During the following year, it was determined that no record of food intake and assessment was available for the elderly. It was decided to design a simple form for use by residents or staff in these Homes.

It was recognized that no single form could accomplish all that was required, but it was felt that with appropriate introduction, education and follow-up certain improvements could be shown in the food habits of the elderly.

MATERIALS, METHODS

With this in mind, a form was designed to record daily intake relating to the four food groups. On the same page, the corresponding recommended intake for each food group was listed thus, facilitating comparison of actual intake and recommended intake. Inadequacies and over-indulgences were immediately obvious. In addition to the above, the category of "sweets" was added to portray a more complete picture of the actual intake keeping in mind "sweets" are ever popular with the elderly.

To consider other variables with relationship to food intake: **age, height, weight** and activity status were listed under "Other Information."

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At the Regional Menu Conferences Homes for Special Care, a number of homes agreed to participate in the use and evaluation of the form.

RESULTS

(A sample of 11 homes with an average of 65 residents completed the record. The comments were as follows:)

— information valuable for menu planning to meet individual nutritional needs.

— nutrition awareness for residents and staff.

— assessment of those with poor appetite.

— assessment of those with restricted intake.

— assessment of food intake with regard to energy output.

— assessment of likes and dislikes.

— a record of intake for nurses and doctors to consider in overall assessment.

It is recognized that an assessment and identification of eating habits does not necessarily imply that an improvement will result. However, deficiencies can be earmarked for action.

SUMMARY

Limited income, loneliness and social isolation will have a direct **or** indirect effect on the adequacy of the food intake. Similarly, reduced activity, poor dental health and mental disturbances will most directly affect the adequacy of the nutritional intake. Food fads (vitamin E for longevity); fallacies (honey is not sugar, or salt water cures rheumatism); and chronic alcoholism, all will take a definite toll on the nutritional status.

As age is increased, the likelihood of physical problems is greatly increased. Aging means an increase in stress and the latter increases the loss of nutrients.

Several researchers have shown that improvement or elimination of detrimental circumstances have resulted in improved appetites, food selection, and adequacy of nutrition.

With adequate nutrition, other problems may be less severe. The elderly are here because of good nutritional habits of the past. However, old habits need alteration to meet an increased demand for nutrients and decreased BMR and activity. The "Daily Intake Form — Rating Guide" will, on completion, assess the adequacy of the patient's food intake for one day and indicate the eating habit trends. It is recommended that a form be completed for each senior patient and used and maintained in the total assessment.

A copy of the form shown here will be forwarded on request.

The goal and name of the game is to keep the elderly socially and psychologically alive. Adequate nutrition is definately a positive contributing factor. Adequate nutrition must be a priority. □

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DAILY INTAKE FORM — RATING GUIDE

HOMES FOR SPECIAL CARE, NOVA SCOTIA

Name of Resident: _____ **Date of Survey:** _____

FOOD GROUP		QUANTITY — Check ( ) appropriate amount					RECOMMENDED DAILY INTAKE
		None	**4 oz.**	**4-5 oz.**	**6-7 oz.**	**8 oz. or more**	
MILK	Meal 1	____	____	____	____	____	
	Meal 2	____	____	____	____	____	12 - 16 ounces
	Meal 3	____	____	____	____	____	
		None	**<1 serv.**	**1 serv.**	**2 serv.**	**3 serv. or more**	
FRUITS	Meal 1	____	____	____	____	____	
AND	Meal 2	____	____	____	____	____	4 - 5 servings
VEGETABLES	Meal 3	____	____	____	____		
		None	**<1 serv.**	**1 serv.**	**2 serv.**	**3 serv. or more**	
BREADS/	Meal 1	____	____	____	____	____	
CEREALS	Meal 2	____	____	____	____	____	3 - 5 servings
	Meal 3	____	____	____	____	____	
		None	**1 oz.**	**2 ozs.**	**3 ozs.**	**4 ozs. or more**	
MEAT	Meal 1	____	____	____	____	____	
	Meal 2	____	____	____	____	____	4 - 6 ounces
	Meal 3	____	____	____	____	____	
		None	**1 serv.**	**2 serv.**	**3 serv.**	**4 serv. or more**	
SWEETS	Meal 1	____	____	____	____	____	
	Meal 2	____	____	____	____	____	NONE
	Meal 3	____	____	____	____	____	

OTHER INFORMATION

AGE _____

HEIGHT _____

WEIGHT _____

INACTIVE _____

MODERATELY ACTIVE _____

VERY ACTIVE _____

COMMENTS (revelent snacks, etc.)

Department of Health
July 1977

The Rights of the Dying Person

Norma Wylie,*M.Sc., R.N., and Avery Kempton,** D.Min.,
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"When a patient is categorized as terminal, he is often simultaneously deprived of his right to know the truth and his right to privacy, as well as of his right to consent to treatment and to exercise discretion in choosing a place or time to die, or to determine how his body will be disposed of after his death.
Patients would be better served both medically and psychologically if physicians and nurses see to it that these rights are preserved."

Journal of Nursing Administration, (March-April, 1974)

The technological age in which we live has helped to save lives, and this is especially true in some of the more specialized units in hospitals. As machines take over control of patients, there is a great danger of the depersonalization of their care, and this can result in the dying person being reduced to something less than a human being. Such depersonalization brings about the violation of the rights of the individual to maintain their personal identity.

"When I was born, my parents gave me the name of Mary Joe.

My doctor told me that I have cancer of the stomach, and I am going to die.

My name is still Mary Joe.

I just happen to have a terminal illness."

The Needs of the Dying

Every one admitted to hospital brings certain needs made more urgent by hospitalization. More than in any other set of circumstances, a person must have his or her personal needs met. Add to this the presence of terminal illness and the news that death is approaching and possibly very soon, it is not surprising that that individual can develop a feeling of hopelessness. What can be done to help?

First, we should treat the dying person as the living person with a right to live (until death occurs). Much reassurance and support are necessary so that he or she does not feel abandoned as just 'a dying person'. We should provide him or her someone to sit with, to talk to, to work through some of the grief and to provide companionship. We should make certain those surrounding the patient understand the changes in behavior that naturally occur to someone facing these strange circumstances, and recognize the physical changes which often occur as the condition advances. Remember, it is difficult for the patient to accept changes in his or her body image, and adverse reactions on our part often intensify the feelings of self rejection and depression.

Decisions on the place and circumstances of death

One area where we seem to be making headway is the question of people making their own decision where, when, and how death will occur. A dying person should be able to have a say in these decisions and know that they will be honored. Physical and mental changes of a terminal illness may place him or her in a dependent situation. Assurance is vital that earlier decisions will be acted upon without question and that no new rules will be introduced as circumstances change.

Dying with Dignity

This is a phrase that is on everyone's lips these days. What it means, quite simply, is the right to die with as little pain and distress as possible. The dying person must be assured that their personal dignity will be preserved. Recent development in pain control must be made readily available.

We believe the dying person does have rights. If we, who call ourselves healthcare professionals, are able to ensure that the patient's personal needs are met and allow the individual to die with dignity, we are helping to fulfill the rights of the dying person. □

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**Staff Counseller, Halifax Infirmary, Halifax, N.S.

Crossing the Bar

Sunset and evening star,
And one clear call for me!
And may there be no moaning of the bar,
When I put out to sea.

But such a tide as moving seems asleep,
Too full for sound and foam,
When that which drew from out the boundless deep
Turns again home.

Twilight and evening bell,
And after that the dark!
And may there be no sadness of farewell,
When I embark;

For tho' from out our bourne of Time and Place
The flood may bear me far,
I hope to see my Pilot face to face
When I have crossed the bar.

— Tennyson

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Epilepsy

Practical Clinical Considerations

L. P. M. Heffernan,* M.D., F.R.C.P.(C),
Halifax, N.S.

Discussion of the management of the epileptic patient generally emphasizes the use of appropriate anticonvulsant medication. This indeed is most important. Reference sources abound with proper dosage schedules for each anticonvulsant, the latter being predicated on the type of epilepsy being treated. Thus there is no need to repeat such material here. An excellent information source is contained in a recent edition of the Medical Letter entitled "Drugs for Epilepsy".[1] This should be available at all times, as it outlines succinctly the drug management of various types of seizure disorders including the therapy for status epilepticus.

NECESSITY FOR DIAGNOSTIC ACCURACY

Drug usage implies that a specific diagnosis has been made. However, the difficulty encountered in attempting to make a correct and definitive diagnosis of epilepsy, to differentiate in effect between a "fit or faint" or other causes of passing unconscious, is often much greater than the problems encountered concerning the administration of the correct medication.

Epilepsy of the grand mal variety implies, but is only one cause of, episodic loss of consciousness. The operative phrase here is "loss of consciousness". Although a patient may have collapsed and been rendered relatively non-responsive, one has to question whether unconsciousness has truly supervened. Everyone who "passes out" has not necessarily lost consciousness. Many patients use such words freely but incorrectly and thus before this situation is uncritically accepted, a few questions may well reveal significant information suggesting that consciousness has been maintained throughout the spell, i.e. the patient who admits later that while lying on the ground, though too weak to reply, was nonetheless aware of people standing and talking nearby, and who was aware of the sound of the ambulance siren during transportation to hospital, indicates that he was still conscious at this particular time. This information would render epilepsy as an unlikely causation and indicate that other possibilities must be considered.

This correct differentiation is important. There should be positive criteria for a diagnosis and not negative impressions, i.e. no other reason therefore it must be epilepsy, or even less desirable, not to even consider other possibilities with just a decision made to start the patient on anticonvulsant medication. Should medication be instituted then one is committed to a prolonged period of therapy. Such a patient will be considered an epileptic and as a consequence may well be exposed to considerable prejudice and encounter significant difficulties, i.e. when attempting to obtain employment, insurance, or permission to operate a motor vehicle, as well as the social stigmata still directed against patients with this diagnostic label.[2] Thus if the diagnosis is uncertain, anticonvulsant therapy must not be instituted.

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EPILEPSY VS. CONVULSIVE SYNCOPE

It should be emphasized that any brain sufficiently stressed may well respond with seizure activity, particularly if one's individual "seizure threshold" is low. This has important clinical implications. As an example, consider a patient who experiences an episode of vasovagal syncope as the result of a combination of hypotension, decreased perfusion, and relative cerebral hypoxia. If his seizure threshold is low, then convulsive jerking movements of the trunk and/or limbs may develop, and should his bladder be full at the time then he may indeed become incontinent. Convulsive activity plus incontinence is equated with epilepsy and such a diagnosis, although incorrect, is often made in this situation. Now it may be that the patient is a known epileptic and this syncopal episode has merely triggered off another typical spell. However, the patient may not be an epileptic and may have been perfectly well prior to this, so that the episode much be assessed on its own merits, i.e. the features indicate syncope with a complication of convulsive activity. This does not mean that the patient is an epileptic and as a consequence does not require therapy.

SINGLE VS. RECURRENT SEIZURE ACTIVITY

It should be remembered that one seizure does not an epileptic make. Epilepsy is by definition a recurrent seizure disorder, and a patient experiencing one convulsive episode has not demonstrated any recurrent tendency. This may indeed occur in the future, but the recurrent nature of the problem has not yet become established and may never do so. An individual, for example, with a low "seizure threshold" may have a seizure brought on by various presumably innocuous events, i.e. relative sleep deprivation, excessive tiredness and fatigue, relative abstinence from food or consumption of alcohol (not necessarily an excessive amount but rather mild to moderate consumption in the face of the aforementioned factors). This patient should be considered as having suffered a convulsive episode and should not be designated as an epileptic individual.

THE USEFULNESS OF THE ELECTROENCEPHALOGRAM

This test may be of great assistance in petit mal epilepsy or in grand mal seizure disorders, where it may demonstrate significant and definitive evidence of epileptiform dysrhythmic activity. Thus it may confirm a clinical suspicion or it may provide assistance in a situation that defies correct interpretation of the clinical data, i.e. the patient who appears to have suffered a sudden unprovoked episode of true unconsciousness, who is unclear concerning the details of the spell, and for whom one has no eyewitness account. Thus a definitively abnormal EEG would allow one to make a diagnosis with some degree of conviction. Many times the EEG is only suspiciously abnormal and should the diagnosis not be felt clinically to be epilepsy, then one would be well advised not

to initiate therapy on the basis of the EEG alone, but rather to rely on one's clinical impression.

It is important to recall that a small percentage of patients who have definite epilepsy never have such activity demonstrated by means of the EEG despite repetitive tracings over many years. Under this circumstance one would not discontinue therapy just because the EEG was normal. Also a small, but definite, percentage of patients who have never had a seizure, but who have an EEG for other reasons, may demonstrate changes which, although by no means specific, are nonetheless suspicious of epileptiform dysrhythmic activity. In this situation one would not institute therapy on the basis of the EEG alone as this would be tantamount to treating the EEG rather than the patient.

SEIZURES IN THE ALCOHOLIC INDIVIDUAL

Alcohol and seizures are frequent companions. Patients with epilepsy may well have more frequent seizure activity precipitated by alcohol consumption, particularly if the patient's history includes difficult to control frequent seizure activity. Also, nonepileptic individuals who are longstanding alcoholics may, in time, develop a tendency to recurrent seizures whether or not they are drinking. Such recurrent seizure activity would then require the institution of anticonvulsant medication.

There is another type of seizure activity associated with alcohol i.e. "rum fit", which may develop one to three days following sudden alcohol withdrawal. Since it is not known which patients will develop this complication of withdrawal, not infrequently such patients upon admission to hospital are initiated on anticonvulsant therapy prophylactically. The value of this approach has been questioned, for many individuals will not develop this complication and do not require medication. Should the medication be given orally in the usual dosage (dilantin — 100 mg t.i.d.), it will take many days for a therapeutic blood level to be achieved by which time the tendency to seizure activity will have long since passed. Such patients have, at most, only one or two spells, following which the requirement for medication does not exist. Should the patient however develop recurrent seizure activity or status epilepticus, particularly if the convulsive activity is focal in nature, then the possibility of the presence of a complicating intracranial disorder must be entertained. Finally, medication specifically used for the treatment of withdrawal symptoms (chlordiazepoxide-librium) also has anticonvulsive properties and alone will likely be quite sufficient.

Should however one wish to "cover the situation" with anticonvulsive medication, then dilantin would be the preferred drug. It should be given in sufficient amounts so as to achieve a satisfactory blood level as expeditiously as possible, i.e. approximately 1 gram loading dose given either orally — 300 mg t.i.d. or preferably intravenously — 250 mg (rate of infusion must not be greater than 50 mg/min) each hour for 4 doses. Thereafter, the level can be maintained by means of the usual oral dose, i.e. 100 mg three times a day.

PETIT MAL EPILEPSY

This is a seizure disorder of childhood and although it may continue into adult life, it does not arise *de novo* in adults. What is diagnosed as petit mal epilepsy in late teens or adult life not infrequently represents clinically similar temporal lobe epilepsy. However the following differentiating points are worth remembering. Petit mal generally lasts less than 10 seconds, there is no aura, and mental clarity returns immediately. Temporal lobe attacks generally are much longer than 10 seconds, there is frequently a preceding aura and mental confusion may persist for some time.[3]

There is an association with grand mal epilepsy in approximately 50% of cases.[3] Occasionally as these patients reach adulthood, they are maintained on therapy for the grand mal component but that for the petit mal is discontinued, as this expression of the seizure predisposition may well have lessened considerably or completely with increasing age.

These individuals may at some later date experience a breakthrough of the petit mal epilepsy which may be manifested as petit mal status epilepticus.[4] This disorder is quite different from grand mal status for, generally, the patient is not rendered unconscious but is able to remain up and about although in an abnormal state. It is characterized by an attenuation of responsiveness, varying from inattentiveness and decreased spontaneity of speech and movement, to stupor resembling catatonia. Automatic pseudopurposeful movements may continue, and patients who can speak tend to respond to questions in a delayed slow and vague fashion. Disorientation is frequently in evidence. The more stuporous individuals tend to be expressionless, immobile, noncommunicative and unresponsive except to noxious stimuli, despite a superficial appearance of awareness of their surroundings. Extremities may remain for several minutes in passively placed positions. Continuous small quivering movements of the facial muscles, particularly of the eye lids and brows, may be observed frequently. Such status may last for hours to days, and an EEG at this time would show continuous 3 per second spike and slow wave activity. The clinical and EEG abnormalities can be reversed promptly to normal upon administration of diazepam intravenously. The occurence of this problem would, however, indicate that the petit mal disorder has recurred and requires the reinstitution of daily prophylactic anticonvulsive medication.

NECESSITY FOR INVESTIGATION

It is difficult to be dogmatic concerning both the necessity for and the extent of investigation for epilepsy. Even with aggressive (invasive) studies, no cause will be detected in approximately 50% of patients.[3] However if at all possible, certain investigations should be carried out even in the so-called primary "idiopathic" age group, i.e. hemotological and biochemical profiles, urinalysis, skull and chest x-rays, brain scan and an EEG. These latter two will exclude most neurological lesions[5] but their availability will be the determining factor. However should the problem arise at the age extremes, i.e. infants-children or middle-old age (groups associated with a higher incidence of secondary epilepsy), then such studies carry added importance. A lumbar puncture will be rarely helpful, unless the clinical presentation suggests the presence of an intracranial infection, and may well prove to be hazardous if performed in an individual harboring an intracranial mass lesion. Thus it should never be performed routinely.

More definitive studies i.e. CT Scan, arteriography, etc. should be reserved for those individuals who have seizures of focal onset which includes temporal lobe (psychomotor) epilepsy as this implies seizure activity originating in one or

either temporal lobe, focal abnormalities on examination or who demonstrate abnormalities (particularly if focal) on the benign studies. Such findings at any age group would make further studies mandatory.

It is important to remember not to become so obsessed with instituting therapy immediately that one forgets to look at the patient. This may provide useful information indicating whether or not further investigation is necessary, i.e. the observation of focal features to the seizure activity. Such important data may well be able to be obtained from an eyewitness to the seizure, provided one remembers to seek out such information.

MANAGEMENT OF THE SINGLE SEIZURE

Considerable controversy exists as to whether or not medication should be instituted after but one seizure which has arisen for no obvious reason, in an individual otherwise completely well. It may well be that the patient will never have another seizure or may go for years prior to the occurrence of the next spell and therefore medication administered during this time will be unnecessary although quite gratifying, i.e. one could state that the medication had prevented the patient from having another seizure although a placebo would have had just the same effect if the patient would never have had another spell regardless.

Reliance may, in such a situation, be placed on the EEG. Should this be grossly abnormal indicative of epileptiform dysrhythmic activity then even after one seizure it may well be advantageous to start the patient on medication as the abnormal tracing would suggest that the patient's seizure threshold was exceedingly low thus predisposing to further seizure activity. Should the tracing be normal then one would be quite justified in deferring therapy until if or when another spell develops. Should this occur, then the recurrent nature of the disorder has been established, indicating the need for medication.

DRUG MANAGEMENT

It is best to know a few medications well, beginning with one and increasing slowly to the point of seizure control or toxicity. Should the latter develop, then the medication should be decreased to sub-toxic levels and then a second agent added and the process repeated. It is possible by administering considerable somnolence-inducing medication to suppress completely the clinical expression of seizure activity. However, it is preferable to have the patient reasonably free of seizures, yet alert and able to function, than totally free but stunned and unable to function properly.[3]

The commonest cause of poor seizure control is poor patient compliance i.e. 30-40% take medication irregularly or not at all.[5] Many will come and complain of poor control but deny they are not taking their medication. Serum anticonvulsant levels, if available, may be of great assistance in such a situation, and they determine if indeed the medication is being taken. Also, information can be provided as to which agent of several may be the toxic one, whether or not the level is adequate despite the administration of presumed standard amounts of medication, and whether or not drug interaction is a problem.

Mysoline has specific anticonvulsant properties, but it should be stressed that one of the by-products during its metabolism is phenobarbital. Thus it is extremely doubtful whether a combination of phenobarbital and mysoline provides any advantage over the use of either drug in adequate dosage. Besides being unnecessary, a combination may enhance the likelihood of significant drowsiness as an undesirable side-effect.

Dilantin probably can be given orally in a single dose at one time daily, for patients who dislike taking medications three times a day.[5] It should be remembered that dilantin is poorly and irregularly absorbed from intramuscular sites and thus one is unable to achieve a satisfactory blood level predictably and reliably by means of this route when administering it for the first time. However, should the blood level already be therapeutic, then one is able to maintain it at this level by means of intramuscular injections. It has been suggested when switching from the oral to the intramuscular route that the latter dosage should be 50% greater than the former oral dosage for approximately one week. Should continuous intramuscular therapy be required beyond this time, then dosages equivalent to the original oral amount could be utilized. Should one switch back to the oral route then for approximately one week, 50% of the original oral dose should be administered following which resumption of original oral amounts can be undertaken.[6] The desired level can be achieved by once daily intramuscular injection which as well avoids the obvious discomfort of multiple injections. However if possible, it is perhaps best to avoid reliance on the intramuscular route for the administration of dilantin.

The question of discontinuation of medication after a seizure-free period of 2-4 years is a thorny question. It should be remembered that upwards of 40% will experience seizure recurrence and will require medication reinstitution. As a general rule the more severe the seizure disorder and the harder it has been to bring under control the more likely the problem is to recur once medication has been discontinued. Some patients however who cannot stand taking medication will accept this possibility. Others may not wish to take this chance and therefore will accept the minor inconvenience of taking medication daily.

STATUS EPILEPTICUS

This is difficult to manage and even more difficult to obtain a consensus concerning definitive treatment. All plans have much in common but no one is the same, and the reference cited initially will outline one satisfactory proposal.

Status generally develops in idiopathic epileptics who neglect their therapy. Also, rapid cessation of therapy, as for example when changing from one medication to another, may precipitate rebound seizure activity which may manifest itself as status. Should medication alteration be required then, if at all possible, withdrawal should be performed slowly (days to weeks). It may occur, however, as the first manifestation of an intracranial disorder. As a general rule, status in an idiopathic epileptic is much easier to bring under control than when it arises as a manifestation of a recently acquired organic central nervous system lesion. An adequate airway and adequate ventilation are mandatory, as the patient will continue with seizure activity if hypoxia is superadded despite the concomitant administration of anticonvulsive medication.

CONCLUSION

In the management of epilepsy, therapeutic success is enhanced if the epileptic is considered as a patient who hates

having seizures, who does not always take his medication, who fears loss of employment if his condition becomes known, and who resents yet solicits concern of those around him, and whose fondest hope is to lead a normal life.[3] □

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Positions Available

The Northern Medical Unit, University of Manitoba, has general practice positions available with the University Clinics located in Hodgson and Norway House, Manitoba, commencing October/November. The clinics are located in emerging community health centres, dealing largely with patients of native extraction. Interested applicants are asked to forward a complete curriculum vitae to the Northern Medical Unit, University of Manitoba, 750 Bannatyne Ave., Winnipeg, Manitoba R3E 0W3, Tel. (204) 786-3711.

A Physiologic Approach to Peptic Ulcer Surgery

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HISTORICAL BACKGROUND

The first surgical procedure employed successfully to treat peptic ulcer diathesis was gastrojejunostomy. This operation was used to treat the so-called "burnt out" stage in which the stomach, after years of recurrent inflammation and fibrosis, became obstructed at the pylorus. The remarkable success of gastrojejunostomy was at the time the basis of its falling into disrepute. Inappropriately applied to the active early disorder it resulted in a 20% incidence of marginal ulceration. Yet it is a curious fact, very clearly indicated by the British and Scandinavian literature, that from the overall result, gastrojejunostomy remains the safest surgical therapeutic approach, i.e. if 100 consecutive patients with peptic ulcer disease requiring surgery were routinely treated with gastrojejunostomy, and the marginal ulcerations following gastrojejunostomy were treated with gastric resection, the total overall morbidity would be less than if the 100 original patients were subjected to gastric resection initially. Add vagisection to both series and the morbidity differences increase.

FACTORS RESPONSIBLE FOR PEPTIC ULCERATION

The classical treatment of peptic ulcer diathesis has been based upon the assumption of hypersecretion of acid and pepsin as its etiology[1]. Recent elucidation of the basic physiology has destroyed this simple hypothesis. Modern concepts would present a more complex pathogenesis[2] including the following etiologic factors:

1) increased secretory capacity
2) automonic hyperactivity
3) increased hormonal drive
4) loss of mucosal barrier
5) pyloric sphincteric incompetence
6) loss of feedback mechanisms
7) pancreatic factor

In any one patient, of course, there may be an overlap or multiplicity of causative factors, so that the acid pepsin factor represents the summation of any of all of these effects (Figure 1). Above all, rational therapy must be directed wherever possible towards the pathogenesis. Thus, the modern gastroenterologic surgeon can have no single operative procedure which is the panacea. Let us consider these factors in turn.

1) Increased secretory capacity is a paraphrase of the old acid-pepsin factor. Gastric hypersecretion[1] generally derives from one or a combination of the following:

1) increased parietal cell mass
2) increased vagal activity
3) hypergastrinemia

In varying combination, these three factors describe the majority of peptic duodenal ulcers. The obvious physiologic approach would be a surgical procedure which would reduce the liberation of gastrin from the antrum and also decrease the sensitivity of the parietal cell mass. The surgical procedures available range from total gastrectomy and total vagisection, to super-selective vagal denervation of the proximal stomach. The former, most would view as inappropriate because of morbidity and mortality; the latter, some would consider inadequate and untried. The surgeon's dilemma is truly a Hobson's choice.

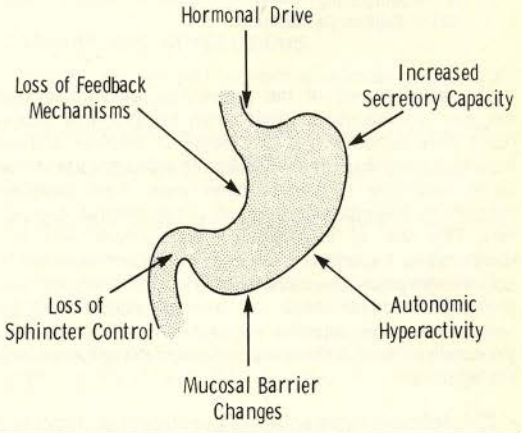

FIGURE 1

Physiologic factors in peptic ulceration.

The most rational approach would be to base the extent of surgery upon the degree of hypersecretion which, of course, should measure the augmentation of the parietal cell mass. However, data recently obtained by augmented histamine tests showed there was little correlation between rate of secretion and clinical severity. Moreover, major overlap occurred between normal and ulcer patients, and basal secretion could not be taken as an index of vagal activity. We formulated a protocol a number of years ago which attempts to evaluate both parietal cell mass, and vagal drive using 3 parameters:

1) rate of peak acid production
2) effect of vagal paralysis on secretory rate
3) 15 mEq/hr as the secretory maximum.

The protocol is as follows (Table I):

You will note that the patients are divided into relatively low secretors (15-25 mEq/hr), moderate secretors (25-50 mEq/hr) and hypersecretors (50 or more mEq/hr). If vagal paralysis, medically induced, could reduce the secretory rate below 15 mEq/hr, vagisection was sufficient, *per se,* to treat the ulcer, because ulceration does not recur in patients who secrete 15 or less mEq/hr. The net result was a reduction of gastric resection as a surgical procedure at our institution from 75% to 15% as well as the inclusion of vagisection as a *sine qua non* in treatment of ulcer.

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TABLE I

Schema relating degree of hyperscertion, response to medical vagotomy with type of ucler surgery indicated.

Rate of Hypersection	Minimal	Moderate	Marked
Augmented Histamine	15-25 mEq/hr	25-50 mEq/hr	>50 mEq/hr
Augmented Histamine & Medical Vagotomy	0-15 mEq/hr V + GE or	15-25 mEq/hr V+ A	>25 mEq/hr V+ SG
Operation	V + P		

V = Vagotomy
GE = Gastroenterostomy
P = Pyloroplasty
A = Antrectomy
SG = Subtotal Gastrectomy

The effectiveness of the protocol is attested in that marginal ulcerations occurred when inappropriate procedures were performed for one reason or another, and we have continued to use it unofficially and without regret. At the same time, the trend has been away from complete vagisection towards selective and superselective vagisection. The fear of incomplete antroneurolysis and cardioneurolysis for selective vagisection has not been borne out by experience. The more careful dissection required has prevented incompleteness, but whether selective will be replaced by super-selective vagisection is still "subjudice." Personally, I have encountered no major complication with the latter.

2) Autonomic Hyperactivity as a pathogenetic factor has been considered along with increased secretory capacity, since these factors overlap and coexist in uncomplicated duodenal ulcer. As an independent factor, it is observed in the peptic ulcer seen in brain tumor and in brain stem disease. It may possibly play a role in the evolution of stress ulceration.

3) Increased hormonal drive has an important role in peptic ulcer diathesis. It may derive from:

A) hypersecretion of hormones trophic to the stomach
   a) growth hormone-Acromegaly
   b) ACTH and corticosteroids-Cushing's Syndrome

B) hypersecretion of gastrin
   a) Werner's Syndrome
   b) Zollinger-Ellison Syndrome

The most logical solution to peptic ulceration resulting from hormonal drive is either elimination of the offending hormone by hypophysectomy or andrenalectomy, or by blockade of the parietal cell with an H_2 blocking agent. I am not aware of an extended trial with the latter therapy. The same logic would apply to extragastric sources of gastrin. Certainly the accepted procedure for "Z. E. Syndrome," total gastrectomy, is reluctantly performed by surgeons because it affords the only certain relief of catastrophic ulcer complications. I predict it will be abandoned as soon as we are wise enough to avoid it.

A special subset of hormonal drive ulceration occurs in the peptic ulcer diathesis which is seen in cirrhotics following portocaval shunting[3]. The pathogenetic mechanism includes increased stimulation of the parietal cell mass by the loss of hepatic degradation of endogenous, histamine-like secretogogues and, perhaps, gastrin, by diversion of the portal flow from the liver. Another factor is the diminution of neutralizing duodenal bicarbonate flow resulting from the portacaval diversion[3]. We have demonstrated this latter anti-physiologic effect in the experimental animal and in man. While it is customary to treat peptic ulceration following shunting in the cirrhotic in the routine fashion, it would be more logical to prevent it by the superselective shunting which maintains integrity of the portal flow through the liver and is not accompanied by pancreatic secretory depression[3].

4) The integrity of the mucosal barrier and the pyloric sphincter function are factors more specifically related to gastric than to duodenal ulcer. From the very beginning, gastrointestinal physiologists considered mucosal resistance important, and researchers were sidetracked by unfruitful study of the complicated mucins and mucoproteins. It was Davenport[1] who pointed out that as acid is secreted by the stomach, so is it reabsorbed by back-diffusion through the mucous membrane. Patients with gastric ulcer differed from those with duodenal ulcer in muscosal permeability. In gastric ulcer patients, the antral mucosa is more permeable to the back-diffusion of H^+ ion than normal; in duodenal ulcer the antral mucosa is less permeable to back-diffusion. Increased back-diffusion raises the H^+ interstitial concentration and results in injury and ulceration.

We now know that increased mucosal permeability may result from:

1) exogenous injurious substances
   a) aspirin
   b) butazolidin, cincophen
   c) alcohol
2) biliary reflux due to **incompetent** pylorus
3) hormonal action — cortisone
4) mucosal ischemia and atrophy

Moreover, gastric ulceration of an impaired mucosal barrier does not occur in the absence of acid. This gives us valuable tools in the treatment of such ulceration; i.e.

1) vagisection
2) parietal cell blockade
3) acid neutralization

These considerations afford for the first time a rationale for the treatment of peptic ulceration in the stomach. H_2 receptor blockade should provide protection against aspirin type

ulceration as well as prompt healing in non-pharmacologic gastric ulcerations. When surgery is required for gastric ulcer due to hemorrhage or intractibility, the theory would clearly indicate a place for vagisection plus gastric outlet diversion. Since gastric cancer is always a concern, antrectomy with resection of ulcer would be preferable to pyloroplasty or gastrojejunostomy.

There is evidence that the gastric erosions and hemorrhage which occur in the so-called "stress ulceration" results from a combination of autonomic neural drive and impairment of the mucosal barrier. The latter derives from hormonal factors and mucosal ischemia. Most important of all is the observation that stress ulceration does not occur in the absence of increasing gastric acid secretion following surgery. These physiologic data permit the development of a protocol of prophylaxis similar to the therapy suggested by Silen, *viz.:*

1) careful monitoring of gastric pH following surgery in patients following prolonged surgery
2) bicarbonate lavage with isotonic solution in patients with gastric hypersecretion of acid as well as those displaying an increasing rate of acid production.

6) Loss of feedback mechanisms is another pathogene to be considered. This pathogenesis comprises a disparate group of peptic ulcer patients whose diathesis appears to be related to one of the following:

1) massive small intestinal resection
2) suppression of pancreatic secretion

Following massive small intestinal resection in the experimental animal, there is physiologic evidence of loss of inhibitory factor produced by the gut capable of suppressing parietal cell secretion. In man, the evidence is less convincing. There is diversity of opinion, consequently, of the necessity or desirability of vagal denervation of the parietal cell mass as prophylaxis in these patients.

7) The pancreatic factor in peptic ulceration, originally stressed by Poth[4], can no longer be ignored. It is operative in at least three types of ulceration:

A) those exacerbated by "caffein" and "nicotine" both potent inhibitors of pancreatic bicarbonate.

B) those observed in end-stage chronic pancreatitis in which the rate of bicarbonate secretion is markedly suppressed

C) those occurring following pancreatic resection of secretory diversion.

The ultimate noxious action of excessive gastric acid secretion is the maintenance of a very low pH in the very first segment of the duodenum, as evidenced by measurements in man in typical duodenal ulcer and especially Zollinger-Ellison Syndrome. In the normal patient, submaximal pancreatic stimulation is sufficient to produce a flow of bicarbonate into the duodenum in excess of that required to neutralize the peak acid output of the parietal cell mass[5].

Even in patients with active duodenal ulcer and patients with Zollinger-Ellison Syndrome, the maximum alkaline secretory capacity of the pancreas is in excess of maximum acid output of the stomach[6]. The pancreatic factor, thus, implies loss of this ability of the pancreas to neutralize gastric acid in the duodenum whenever it is inhibited by pharmacologic agents, excluded, or its secretory capacity drastically diminished by disease.

The incidence of peptic ulceration as a complication of advanced pancreatic disease is high. Its occurrence is reported even in patients with low rates of gastric acid secretion. But above all, the incidence of marginal ulceration following surgery for such peptic ulceration is unacceptably high, about 40% in our experience. Vagisection alone as well as antrectomy alone appears to afford inadequate protection against marginal ulceration. The gastric acid protocol is inappropriate in these patients. Parietal cell denervation combined with antral resection is required.

Finally, the surgeon has learned through bitter experience that vagisecion or subtotal gastrectomy is mandatory in major pancreatic resection, i.e. Whipple procedures, in patients with normal or increased gastric acid secretory capacity. Failure to limit gastric acid secretion in these patients results in marginal ulceration.

SUMMARY AND CONCLUSIONS

A physiologic approach to peptic ulcer disease leads to the following conclusions:

1) the pathogenetic factors in peptic ulceration are diverse
2) excess acid or increased antral mucosal permeability are basic.
3) vagisection, in almost all situations, is the *sine qua non*
4) data are insufficient to assess the effect of the newer medical therapies, i.e. cimethidine, prostaglandin, etc., on these conclusions. □

This paper was originally presented at the Annual Meeting of the Surgical Society of Nova Scotia in Sydney, N.S., September 10, 1976.

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Current Concepts in Multiple Sclerosis

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Multiple sclerosis with its unknown cause, undiscovered cure, unpredictable course and peculiar geographic distribution has puzzled physicians since it was first described by Jean Martin Charcot in 1868. It is one of the most common serious neurological disorders in Canada, and its long course and disability make it a major medical problem.

Multiple sclerosis is a central nervous system disorder, usually in young adults; its exacerbations and remissions of symptoms often lead to progressive neurological impairment due to multiple plaques of demyelination and gliosis throughout the brain, brain stem and spinal cord. The patchy lesions occurring in this, the most common form of primary demyelination, result in motor, sensory, visual and cerebellar disturbances.

Theories of etiology include biochemical, viral, autoimmune, vascular and dietary factors. Evidence for genetic predisposition is not impressive but there is a higher risk in families with an MS case. The role of environmental factors may be important, since the risk of acquiring multiple sclerosis is higher in the more temperate zones of the world.

Because there have been many advances in understanding this disease, and since there is usually at least one patient with it in any family practice, a brief review of the current status of MS seems in order. The tremendous progress in research, particularly over the past five years, has been very exciting and justifies some degree of optimism about future treatment.

Etiology of Multiple Sclerosis

Most nerve fibres of the central and peripheral nervous systems are enveloped by a laminated protein-lipid myelin sheath. The pathology seen in MS is generally confined to the myelin sheath with patches of myelin breakdown, perivascular inflammation and later, gliosis or tissue scarring. Axis cylinder degeneration does not occur in primary demyelination, although it may do so secondarily as a late feature in a plaque that has remyelinated poorly and shows considerable glial scarring.

The demyelinating process is characterized by a *structural* and a *chemical* degradative phase. The structural or physical disintegration involves myelin fragmentation accompanied by the splitting of the myelin lamellae, followed by Schwann's cell proliferation and macrophage infiltration.[1] The chemical alterations found in MS patients result from accelerated enzyme activity. This intense enzymatic activity is very puzzling. It may be due to excessive enzyme liberation, suggesting that it is a primary alteration. However, upon nerve disruption, greater exposure of the proteolytic enzymes to myelin may produce the increased enzymatic activity, suggesting a secondary change. Of particular importance is the fact that the proteolytic enzymes (i.e., the neutral proteinases) are capable of disrupting lipid bound to protein, resulting in a loss of radial stability in the lamellar structure of myelin. The activity of the proteolytic enzymes is also apparent early in demyelination, though no other enzyme abnormalities are evident at this stage. This may explain why fragmentation occurs in the structural stage of demyelination. The neutral proteinases ostensibly originate within the lysosomes of the Schwann's cells. The cytolytic agent lysolecithin, normally found within the central nervous system, has been shown to release a proteinase and may be responsible for the release of proteinases from the Schwann's cell lysosome initiating the demyelinating process.

The primary change in multiple sclerosis may lie within the oligodendrocytes, the small supporting cells of the central nervous system, with cellular hypertrophy, hyperplasia and increased enzymatic activity. This theory is supported by the finding that experimental demyelination produces altered oligodendrocyte morphology before anything else.

Slow virus infection is a leading etiological theory at present. This infection is characterized by initial exposure to an agent, probably a virus, then a long incubation period culminating in a progressive neurological deficit several years later. A specific MS virus has not been proven, but there are reports that support the possibility as a cause of MS.[2] Indeed, there may be several different viruses which could initiate a similar demyelinating process.

The measles virus, particularly if contracted in early adolescence, is a highly suspicious agent. A number of studies have confirmed the finding that about 40-60% of MS patients have a higher measles antibody titre than the general population.[3] However, many patients have normal measles antibody levels and, of course, most people who have had measles never develop multiple sclerosis.

The measles virus etiology becomes more plausible when one notes the similarities between multiple sclerosis and another neurological disorder with a demyelinating component called subacute sclerosing panencephalities. First, in patients with this relatively rare disease, CSF and blood contain markedly elevated measles antibody titres. The disease is known to be caused by the measles virus which has been isolated from patients. Secondly, the CSF contains a larger than normal percentage of gammaglobulins (i.e. greater than 13% of total protein), a finding in 60-70% of MS patients. Further evidence supporting a viral origin for multiple sclerosis comes from the epidemiological, geographical, social and familial patterns of the disease and these are very similar to poliomyelitis.[3]

There is some evidence that the important factor may not be the measles infection *per se*, but the *age* at which it was contracted.[4] We have found evidence that MS patients in Nova Scotia had measles infection at an average age of thirteen years.[5] The critical age of susceptibility to whatever agent or factor causes MS seems to be around puberty.

Autoimmunity or delayed hypersensitivity in which the patient becomes allergic to and eventually destroys the myelin component of his central nervous system, is another popular etiological theory. The autoimmune theory is

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supported by finding antibodies to various brain and myelin components in the CSF and the elevated gammaglobulin in the CSF.[6,7] Among MS patients, 60-70% have above 13% of the total protein as gammaglobulin which, with a few exceptions, is diagnostic of MS. It has also been shown that the gammaglobulin is IgG and that it is an active antibody formed in the nervous system.

There has been controversy over the years about the role of *genetics* in multiple sclerosis.[3] As yet, no definitive evidence exists to support a hereditary factor although there is a 10% familial incidence. Whether this is due to some sort of genetic predisposition, or is simply a result of the common exposure of two members of the same family to the same causative agent, remains to be determined.

Studies on HL-A antigens in different diseases show an association with the HL-A antigens detectable in the serum.[8] A consistent association between multiple sclerosis and the HL-A antigens has been illustrated, with an increased frequency of HL-A3 and HL-A7 and a decreased frequency of HL-A2 and HL-A12 antigens. A multiple sclerosis susceptibility gene believed to be present on the No. 6 chromosome adjacent to the area coding for the HL-A antigen complex has been postulated.

There does appear to be at least a *familial tendency* in multiple sclerosis with an incidence in families 20 times and an incidence in siblings 50 times that of the general population.[3] However, studies comparing the incidence of MS between siblings and identical twins show that there is *little* difference between the two groups, a finding that tends to preclude a purely hereditary basis.

A **vascular** thrombotic or embolic origin has been another etiological consideration in the past. Increased platelet adhesiveness, perhaps a consequence of depressed linoleic acid levels, could lead to the formation of microemboli resulting in local areas of hypoxia. This could promote lysolecithin synthesis and hence demyelination. These are theoretical considerations and their role in the cause of MS is uncertain. Although an unlikely explanation by itself, the fact that most plaques are perivascular has to be explained by whatever theory we accept.

Recently there has been heightened interest in the possible role of **nutrition** in the onset of multiple sclerosis. Individuals who consume a higher than normal level of lipid (particularly cholesterol) may be at a higher risk of acquiring multiple sclerosis.[9] For example, the consumption of dairy products, like the world distribution of multiple sclerosis, seems to increase with geographical latitude in both a northerly and southerly direction from the equator. A high fat intake may result in an alteration of the blood-brain barrier leading to a diminished immunological defence system within the brain and spinal cord, the production of defective myelin and increased susceptibility to some agent, such as a virus. Myelin synthesis commences in utero and continues well into the 3rd and 4th decade and thus the diet consumed early in an individual's life may influence this process.

Epidemiology

Epidemiology, the study of the distribution of a disease in a population and of the factors influencing that distribution, has contributed greatly to our understanding of MS. Population studies have confirmed that multiple sclerosis is more common in temperate climates and relatively rare in tropical regions.[3] It is doubtful, however, whether this particular distribution is temperature related, since the north-south distribution is not consistent throughout the globe. In Japan, multiple sclerosis is less common than expected, whereas, in other countries at a similar latitude in North America, the incidence is much higher.

There are other epidemiological factors worth consideration. MS is slightly more prevalent in women. It is rare in childhood, and most patients experience the onset of symptoms between the ages of 20 and 40 years, the average being 27 years. Thus, the risk of developing MS seems to be age-related, with the so-called critical age being before 15 years. Based on immigration studies, individuals migrating before 15 years of age appear to take on the risk for developing MS that exists in their new place of residence, whereas those moving at a later age retain the risk of their native country. Multiple sclerosis is more prevalent in areas of higher socio-economic status and sanitation level; the significance of these findings which were also seen with polio is not understood.

The questions of trace metal abnormalities in multiple sclerosis patients has been looked at for 50 years, but no definitive relationship has been established. There are several isolated areas of increased prevalance in the world including New England, the Orkney and Shetland Islands, Washington State, and closer to home, Nova Scotia.[5]

DIAGNOSIS

The scattered plaques of demyelination found in multiple sclerosis affect many of the nerve fibre pathways throughout the brain, brain stem and spinal cord with motor, sensory, ocular, brain stem nuclei and occasionally cerebral involvement. To review, pyramidal tract involvement, a frequent occurrence in MS patients, results in spasticity, hyperreflexia, loss of abdominal reflexes, ankle clonus, positive Babinski signs and general weakening of the limbs. Cerebellar involvement is characterized by an intention tremor, ataxia and dysarthria. Sensory disturbances may include paresthesiae, trigeminal neuralgia, and posterior column involvement with depressed two-point discrimination, diminished vibratory and postural sensation, and a positive Lhermitte's sign which is most useful in diagnosing multiple sclerosis.

The optic nerves are very commonly involved, with visual blurring and frequently the development of a retrobulbar neuritis marked by pain on eye movement, diminished visual fields and large visual scotomata. Funduscopic examination may reveal pallor in one or both optic discs. A propensity for brain stem demyelination in MS can affect any of the brain stem nuclei. Demyelination of the oculomotor or 3rd cranial nerve produces diplopia, though there is generally no observable strabismus. Intranuclear ophthalmoplegia, resulting from demyelination along the medial longitudinal fasciculi produces an ataxicnystamgmus.

Frequently, one observes in the MS patient a mild facial weakness and a facial myokymia, or fine undulating rippling of the facial muscles. Cerebral involvement, though not characteristic of multiple sclerosis except in advanced cases, may produce emotional changes such as depression, euphoria and hysterical reactions, and impairment of intellectual functions, apathy, paranoid or schizoid reactions and mental confusion. Other common symptoms include genitourinary disturbances, impotence in males, and constipation. Typically, symptoms are intermittent and are provoked by such factors as heat, humidity, cold, trauma, fatigue and emotional stress.

A combined presentation of motor, sensory, cerebellar and visual abnormalities, scattered in time and space, usually determines the diagnosis of multiple sclerosis. However, the differential diagnosis may be obscured. Frequently the patient does not present to the physician until he is significantly disabled and initial symptoms may go unreported. At the time of examination certain diagnostic clues may not be observed simply because the patient is experiencing only very early manifestations of the disease. This may lead to considerable delay in diagnosis.

The analogies between MS and other neurological disorders are numerous. Cervical spondylosis is characterized by a paraplegia resembling MS. A typical clinical presentation of subacute combined degeneration of the spinal cord is tingling followed by numbness radiating to the most distal portion of both the upper and lower limbs. This, of course, would also be the initial manifestation of multiple sclerosis. Spinal cord compression and MS are similar with respect to the paraplegia that can occur. Myasthenia gravis in its early stages may simulate MS by a presentation of intermittent double vision and limb weakness. Finally, pyramidal signs and a rise in the gammaglobulin fraction of the cerebrospinal fluid in subacute sclerosing panencephalitis make differentiation between this leukodystrophy and multiple sclerosis difficult.

Though there are as yet no pathognomonic tests for multiple sclerosis, a number have been found useful in confirming diagnosis. Some of the typical laboratory findings are the presence of only 5-10 mononuclear cells in the CSF, an elevation of total CSF protein in 50% of patients, a first or mid zone rise in the mastic curve in 50% of patients and a CSF gammaglobulin of greater than 13% in 80% of patients.[3]

Computerized tomography (CT scans; EMI scans) has demonstrated abnormalities in many patients with MS but these are not usually diagnostic.

TREATMENT

Due to the absence of any specific cure for multiple sclerosis, the aim of therapy is to provide maximum relief from symptoms and to prevent further complications. The treatment and management of the MS patient depend on the particular clinical course he is following. A course of chronic progression occurs in about 30% of MS patients, whereas, one of exacerbations and remissions superimposed on chronic progression is evident, at least early in the disease, in about 70%. The malignant form of multiple sclerosis is unusual; it is characterized by acute episodes of great severity, ultimately leading to a very rapid progression to disability within several years. Fortunately, this is unusual and a benign form of the disease seen in about one third of MS patients is more common.

Steroids are often used to manage the MS patient during an acute attack. For example, ACTH may be helpful in relieving the signs and symptoms of an **acute** attack but do **not** appear to be much use in managing chronically progressive multiple sclerosis.[3] Orally administered prednisone is another mode of steroid therapy, but it is not comparable with ACTH in its ability to deal with acute exacerbations and, like ACTH, has no beneficial effect on a long-term basis. A long-term course of any steoid therapy can be detrimental, with such side effects as Cushingoid features, hirsutism, ulcers, recurrent infections, psychic changes and, in the more extreme case, osteoporosis.

Various antispasticity regimens have been tried. The intrathecal administration of phenol is usually restricted to patients who are completely immobile. Diazepam is frequently used for muscular relaxation but is successful only in dosages that induce drowsiness. Dantrolene sodium and baclofen are most recent advances in antispasticity therapy and have proven more useful. The latter drug is still experimental. Antispasticity drugs in general have one serious drawback — they can be used only in patients who have significant strength remaining in their spastic limbs. Our experience with these drugs has generally been disappointing. The removal of the spasticity tends to make the weak limbs even weaker and, therefore, less functional. Probanthiline, an anticholinergic medication, is frequently used to control bladder spasticity. It is important, however, for the patients to learn to regulate their fluid intake in relation to their bladder function. A more radical procedure against spasticity is a myelotomy, a surgical procedure designed to sever tracts in the spinal cord. Experiments with dorsal column stimulation are promising but the place of this type of therapy is as yet unclear.

For the relief of trigeminal neuralgia in MS, carbamazepine is very effective. It has also improved dysarthria and ataxia in some cases. Immunosuppressive therapy such as azathioprine and cyclophosphamide has been studied but has *not* shown any appreciable benefit to MS patients. Particularly for those who experience acute attacks, complete rest is necessary. Physical and mental stress have been shown to hasten the rate of progression of the disease. Physiotherapy or an exercise program, or both, are important for maintaining muscular strength and mobility. Several diets have been used for multiple sclerosis patients with varying degrees of success. The low cholesterol diet with a linoleic acid supplement in the form of sunflower seed oil is currently being studied in Canada and England.

Experiments using transfer factor in the treatment of MS have been disappointing. A trial of A-1 protein to treat acute attacks is now underway but the results will not be available for a few years.

Because MS is characterized by acute attacks and remissions, the patient usually presents when the symptoms are at their worst, and appears to respond to any form of therapy. Unfortunately the "response" is usually the expected course of the acute attack and not a result of therapy.[11] The appearance of a dramatic improvement in this manner explains why hundreds of forms of therapy used over the years are usually no better than placebos, and why new "breakthroughs" and treatments appear in newspapers and magazines each year.

As a final note, after nearly 100 years of research, medical scientists may at last be "zeroing in" on the cause of multiple sclerosis. Hope for a major breakthrough is ever increasing despite the mysterious nature of this disease. This optimism reflects, of course, the growing interest of both the public and medical communities. New technology has made possible the study of various chemical and physical processes within the central nervous systems that only several years ago were beyond the scope of our knowledge. As we look to the future, we hope that the combined efforts of clinicians, neurologists, pathologists and epidemiologists will reach the ultimate goal of prevention and cure for this devestating disease. □

References on page 138.

Recognition and Management of Sexual Dysfunction

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With the publication of *Human Sexual Inadequacy*[1] in 1970, a promising new therapeutic format for the treatment of sexual dysfunction became available.

It is not unusual now for patients to ask their physicians for help with sexual problems. It was the increasing frequency of this phenomenon that prompted us to obtain training in the rapid treatment of sexual dysfunction at the Reproductive Biology Research Foundation, in St. Louis. However, the majority of patients are still reluctant to mention sexual dysfunction as a chief complaint. They are more likely to describe their fatigue, headache, backache, dysmenorrhea, etc. and the physician may miss those instances in which the couple's sexual dysfunction is a major contributing factor.

Sexual dysfunctions include erection failure (impotence), premature ejaculation and ejaculatory incompetence in the male; orgasmic dysfunction and vaginismus in the female; dyspareunia and sexual aversion. With the exception of dyspareunia, 95 per cent of sexual dysfunctions are of psychogenic origin.

RECOGNITION

If the physician is going to succeed in identifying a sexual dysfunction, his or her own attitude and manner are very important. If he shows discomfort or embarrassment in asking sex-related questions, the patient's answers are likely to be prejudiced. Straightforward questioning is essential.

Masters, Johnson and their staff at the Foundation, suggest that the following 5 questions will help to reveal a sexual problem in a very few minutes, much the same as in a simple system review:

1. Any difficulty in sexual relationship: e.g. Is the sexual part of your life living up to your expectations?

2. Frequency of sexual intercourse: e.g. Is your sexual activity as often as you would like?

3. Masturbation: e.g. When did you begin to masturbate? (for male)
Did you ever stimulate yourself? (for female)

4. Homosexuality: e.g. Have you any sexual experience with members of your own sex?

5. Invite patient to ask you questions: e.g. Do you have any questions about sexual function?

The chief reason for asking about masturbation and homosexual experience is to evaluate the amount of anxiety and/or guilt which these activities have engendered.

MANAGEMENT

a) Counselling by Family Physician

Meyer deals with sexual problems in office practice in his recent book.[2] The family doctor can deal effectively with straightforward sexual problems or lack of knowledge about sexual response, if he is adequately informed himself, is comfortable in discussing the topic and is willing to set aside sufficient time for counselling. If these requirements are not met, it is usually more helpful to refer the patient to a colleague who is devoting some time to this type of problem. Complicated or major sexual dysfunctions are best referred to those with training in the field.

There is no such thing as an uninvolved partner in a marriage troubled by sexual dysfunction[1]. Treatment is usually much more effective if the partners are counselled together. For couples just starting sexual activity and looking for reliable information about the anatomy, physiology and psychology of human sexual response, one interview may be sufficient. Some physicians carry out physical examinations on each partner while the other partner watches (the conjoint physical examination), commenting on the functions of the genital organs as they are examined and described. A comfortable, matter-of-fact approach by the doctor helps to demythologize sex for the couple.

Sexual dysfunction in relationships of somewhat longer duration usually requires counselling beyond the simple provision of information. The physician may suggest that the couple return for several visits if the sexual problem is not ingrained and there is no evidence of significant psychopathology. If this option is chosen a more detailed history of their sexual interaction is required. Frequently one or both partners harbour unrealistic romantic or sexual expectations. All too often they feel pressure to measure up to some supposed standard of sexual performance. This type of performance concern can sometimes be removed by having them set aside intercourse for awhile in favor of non-coital types of stimulation — to orgasm if desired. Or if one partner experiences less arousal than the other, the doctor may suggest that the less aroused partner decide when they move from non-coital love play to intercourse. Afterplay may be suggested if one partner has not been satisfied during coitus.

b) Referral

Couples with longstanding or more complete disability are best referred to specialists trained in the field. Psychiatric assessment is indicated when major psychopathology is suspected. The gynecologist, urologist, neurologist or endocrinologist may aid in the recognition or exclusion of physical disorders in selected cases.

We have been trained in the assessment and counselling of couples with psychogenic sexual dysfunction — with or without difficulties in the non-sexual aspects of their relationship (and with or without associated physical disorders). We almost always focus attention on more effective communication between the partners. In our private practice, the Masters and Johnson standard therapy format is followed, because the dual-sex counselling team, isolation of couples from work and family, daily counselling sessions for 12 to 14 days, etc. provide greater effectiveness. It is also essential that both partners be genuinely motivated. Only 20 per cent of couples fail to experience reversal of the

dysfunction when the standard format is used on a properly selected population[1]. But, in a relationship with major dysfunction, all the cards must be stacked in their favour in order to achieve this remarkable reversal rate. □

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CURRENT CONCEPTS IN MULTIPLE SCLEROSIS

Continued from page 136.

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Guidelines for Anesthesia

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A document entitled "Guidelines for Anesthetic Services in the Province of Nova Scotia" has been endorsed by the Executive Committee of the Medical Society, the Health Services and Insurance Commission, and by the Department of Health. It will be available for use by the administration and medical staff of provincial hospitals.

These anesthetic guidelines came from the cooperative efforts, over three years, of an Advisory Committee of ten practising anesthetists who represented well their regions of Nova Scotia. Full-time and part-time anesthetists worked together to produce a reasonable approach toward providing safe anesthetic services and capable anesthetists for Nova Scotian hospitals.

At a recent branch society meeting, several questions were asked that probably come to most minds, of those presented with this subject. Hence, it seems worthwhile to go over them in the *Bulletin*.

Why guidelines for Anesthetic Services? Until now, there have been no guidelines for organization of anesthetic services in most hospitals: how to set up a department, or criteria for equipment or safety. The only clear criterion of eligibility to be an anesthetist has been a medical degree. Present-day anesthesia, with a wide spectrum of potent drugs, has a low morbidity and mortality, provided the anesthetist knows well what he does and why. A minimum period of formal training is considered essential by the Advisory Committee — two weeks or a month is unlikely to produce a capable, safe anesthetist for present-day needs. A basic practical course of formal training provides a solid background of experience for the practice of anesthesia in the smaller hospitals of this province. Hospitals in which the volume of surgery and obstetrics is sufficient to occupy full-time anesthetists, should have fully trained and qualified people.

There is the need to define more clearly the practice of anesthesia and put some reasonable boundaries on it, to assure that anesthetic practice is of good quality. Anyone doubting that should read the Report of the Board of Inquiry on Anaesthetic Practices in Saskatchewan, of December 1976. This was an investigation of anesthesia that looked into anesthetic deaths and such practices as one anesthetist having several patients anesthetized simultaneously, with varied help to monitor them. Comparatively, anesthesia in Nova Scotia is quite good, but proper care and support is essential, as recommended in the guidelines, to assure that we grow stronger from the present base.

Will this force out or threaten the present practising anesthetists? No: the recommendation for formal training applies for the granting of new anesthetic privileges. Present anesthetists are urged to look closely to their quality of service and continue their anesthetic education by refresher courses.

Will not such restrictions on who can practice anesthesia result in a severe shortage of anesthetists when the present ones retire? How will we keep our operating rooms going?

The answer is: that is highly unlikely to happen. There is probably a 10-15 year period over which most of the present anesthetists will retire. During that time, sufficient capable new people must be trained, which depends upon anesthesia being chosen as a career by more graduates. There is beginning evidence that this is happening across Canada. The federal manpower study identified anesthesia as one of the best-opportunity specialties. Certain other specialties and general practice are not in short supply of practitioners.

In the last five years, the Dalhousie training programme has been completely reorganized and Dalhousie graduates are entering it: high-quality doctors making a wise career choice. The Department at present has 19 residency posts in Halifax and 10 in Saint John. As the only Maritime training centre we are totally committed to peopling these provinces with capable anesthetists, both full and part-time. When they are from here and train here, they will stay here.

The question seems more likely not to be, where can we find anesthetists, but, when fully trained anesthetists are looking for practices, will the medical staff of our hospitals welcome them for their specific capabilities? Providing of course that a need exists, one hopes that the answer is yes; if not, then the pessimism is fully warranted. The trained anesthetists will go west or go south: the opportunities are there.

The recommendations embodied in the guidelines are already being used in hospitals to improve their practices and patient safety. When the guidelines say that "All anesthetic areas should have immediately available . . . cardiac monitors . . . for use . . . in prolonged major operations", that is useful in persuading the "powers" that you need a monitor. Anesthetists are now involved in a major thrust to upgrade equipment in operating rooms and recovery rooms — all needed and proper, and resulting from the stimulus of the guidelines. In this respect the Dalhousie Department is ever available for advice, visits or phone calls, and we exist to help the practising anesthetists of the Maritimes. We will do everything possible to aid in providing competent anesthetists for every hospital.

So the Guidelines for Anesthetic Services now come to you, not as a threat but as a needed, reasonable move to try to provide a safe anesthetic experience for everyone requiring operation. As the present anesthetists retire, in every hospital in Nova Scotia, capable, safe anesthetists must replace them. The guidelines are the best hope of accomplishing this. Surely all associated with surgical and obstetrical patients are in agreement. □

*Professor and Head, Department of Anaesthesia, Dalhousie University, Halifax, N.S.

Personal Interest Notes

Late in June, the community of Chester held a special festival evening, honoring **Dr. Edward K. Woodroofe** for his 45 years of dedicated service to general medicine in Nova Scotia. A native of Charlottetown, Dr. Woodroofe graduated from the University of Toronto in 1931, practising first at Canning, and then continuously at Chester since 1936.

Dr. Willem O. Kwant, a 1931 graduate of the University of Toronto, has been awarded a $12,000 grant from the Nova Scotia Division of the Canadian Cancer Society. This "local level grant" was made after an appeal for funds had been approved by the Provincial Medical Advisory Committee of the Canadian Cancer Society. Dr. Kwant, who is a haematologist, is investigating malignant tumours of lymph glands.

OBITUARIES

Dr. R. W. Murray MacKay (75) died at his home in Dartmouth on May 9, 1977. A 1928 graduate of Dalhousie, he received postgraduate training in psychiatry in Brandon and Detroit, returning to Nova Scotia in 1931. He joined the staff of the Nova Scotia Hospital, finally retiring from there in 1967 after 30 years as Administrator-Superintendent. Our sincere sympathy is extended to his family.

The death occurred on June 5, 1977 of **Dr. William Rawson Barlow** (88) of Lake Echo. Born in England, he graduated from London in 1911 and came first to Labrador, later practising in Port-aux-Basques and Kentville. The *Bulletin* extends sincere sympathy to his family.

Dr. James Charles Vibert (53) died August 26, 1977 following a car accident. Born in Stewiacke, he was an honors graduate of the former Colchester Academy where he was awarded the Governor General's Medal in 1938. Following service with the Canadian Army overseas he attended Dalhousie University Medical School where he was the gold medalist in the graduating class of 1951. He graduated in Surgery from the Mayo Clinic in 1958 returning to Truro where he practised until his death. Our deepest sympathy is offered to his wife and family. □

The Medical Society of Nova Scotia

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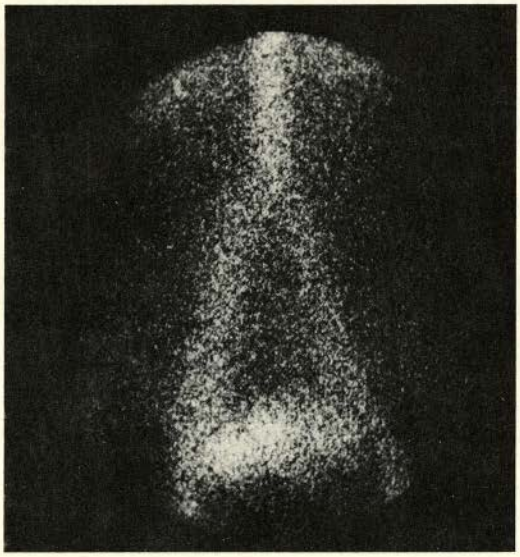
Correspondence

To the Editor:

The Nova Scotia trophoblastic disease registry announced in Doctor Fraser's paper (N.S. Med. Bul. **55:**178, 76) is timely. The following case report may be of interest:

Mrs. A. B. was delivered of a "benign" molar pregnancy in March 1974. She was placed on careful surveillance and urine assay for hormones; physical examination was done monthly and was initially normal. In June 1974 she brought her urine specimen but did not remain for examination. LH level (4,000 units) was grossly elevated. The patient did not return until September 1974 when she began PV bleeding. This time, she had a right pelvic mass; LH level was more than 20,000 and radioactive angiogram (see illustration) showed the mass to be bilateral.



A registry could have enabled us to trace and treat this patient in June with much improved prognosis.

This patient was fortunate that with Actinomycin-D and Methotrexate therapy her tumour regressed. Patient has remained well, although the angiogram is still abnormal.

Yours very truly,

J. F. Filbee, M.B., B.S.,
Radiotherapist,
Radiotherapy Department,
Halifax Infirmary,
Halifax, N.S. □

NO SMOKING

Once again there is a tip-in which may easily be removed for use as a poster in your waiting room or office.

NEW MEMBERS

The Physicians listed below have joined The Medical Society of Nova Scotia between February 1, 1977 and July 31, 1977. A most cordial welcome is extended by the Society.

Dr. Peter G. Bartlett	Liverpool, N.S.
Dr. Richard J. Bedard	Dartmouth, N.S.
Dr. Terrance E. Brennan	Antigonish, N.S.
Dr. Dougal R. Chisholm*	New Westminster, B.C.
Dr. Carolyn R. Covert*	London, Ont.
Dr. Nancy J. Dalrymple,	Lower Sackville, N.S.
Dr. Albert D. Doucet	Liverpool, N.S.
Dr. E. Stephen Farrell	New Waterford, N.S.
Dr. Wilfrid J. Gardiner	Bridgewater, N.S.
Dr. Daniel F. Glasgow	Canso, N.S.
Dr. Bhagwandas D. Gokul	Dartmouth, N.S.
Dr. Judith H. Gold	Halifax, N.S.
Dr. Alan L. Goldbloom	Halifax, N.S.
Dr. Richard O. Healy	Halifax, N.S.
Dr. V. Ann Heath	Dartmouth, N.S.
Dr. David B. Hogan*	Edmonton, Alta.
Dr. D. Nicholas P. Holmes	Oxford, N.S.
Dr. Stephen J. Katz	Digby, N.S.
Dr. Robert R. Kimball	Windsor, N.S.
Dr. William B. Kingston	L'Ardoise, N.S.
Dr. Lawrence H. Leech*	Regina, Sask.
Dr. Gerard Mallon	Antigonish, N.S.
Dr. Nigel Merchant	Dartmouth, N.S.
Dr. John D. Miller*	Windsor Jct., N.S.
Dr. Colin M. Muir*	Ottawa, Ont.
Dr. Iain F. McCaw	Halifax, N.S.
Dr. H. Paul MacDonald	Stellarton, N.S.
Dr. Ewan C. McPherson	New Glasgow, N.S.
Dr. T. A. (Sandy) Peacocke*	Ottawa, Ont.
Dr. Patricia A. Pearce	Stellarton, N.S.
Dr. David Rider	Liverpool, N.S.
Dr. Mary M. Roddis	Saulnierville, N.S.
Dr. Christine H. Rolton	New Waterford, N.S.
Dr. Ingrid A. Romney	Bridgewater, N.S.
Dr. Ronald L. Rondeau	Truro, N.S.
Dr. Harold G. Walker	Glace Bay, N.S.

*Internes/Residents outside Dalhousie Program (Dal. Grads.)

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