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GUEST EDITORIAL

Rehabilitation Medicine

Jaywant J. P. Patil,* MBBS, FRCPC

The basic principles of rehabilitation were already well formed in the days of the father of medicine, Hippocrates. However, more recently the two World Wars have been a major stimulus to the future development of Rehabilitation Medicine because of hundreds of thousands of disabled people who had to be rehabilitated as a result of war injuries. With the advances in acute care medicine and surgery, more and more people with serious impairment have been able to survive, creating a greater need for the services of rehabilitation. As a result of this, the speciality of rehabilitation medicine has grown, not only in the western world, but also has started to take root in the so-called under-developed countries.

Rehabilitation services in this province were given a boost in the mid 50s by Dr. Arthur H. Shears, and Dr. Garnet C. Colwell. In this special issue on Rehabilitation Medicine, Dr. Shears provides a brief, historic perspective of the development of rehabilitation in the Province of Nova Scotia, as well as in other parts of the Western world.

In addition, in this issue, we have articles to update physicians in Nova Scotia about the new developments in the field of rehabilitation. We hope that this information will assist physicians in the care that they provide to their disabled patients.

I am indebted to my colleagues who have made contributions to this special issue. I also would like to thank the Editorial Services of Dalhousie University for its assistance.

*Assistant Professor of Medicine, Division of Physical Medicine and Rehabilitation, Department of Medicine, Dalhousie University, Halifax, N.S.

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

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

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

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

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

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

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

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

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

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

2. Book –

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

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A Brief History of Physical Medicine and Rehabilitation

Arthur H. Shears,* MD, CM, FRCPC

Halifax, N.S.

After briefly outlining the beginning of physical medicine and specific disability rehabilitation, before, during, and immediately after World War II, and reviewing the beginning of physical medicine and rehabilitation medicine in Canada, this paper describes the development of clinical services in Nova Scotia particularly with reference to the Nova Scotia Rehabilitation Centre. It also outlines the development of undergraduate and graduate educational programs in this specialty. To a more limited degree, it refers to the initiation of training programs for other rehabilitation professions. Reference is made to the long-term intensive planning and negotiations for the development of a modern complete centre, which led to completion of the first phase of the new Nova Scotia Rehabilitation Centre in 1977 and the completion of the final phase in 1991.

Prior to World War II, medical specialists whose training and experience in impaired function and pain in the neuromusculoskeletal systems, practised what was called *physical medicine.* It was called physical medicine because they used physical treatment agents consisting of various electrotherapeutic, thermotherapeutic and therapeutic exercise modalities in addition to such chemotherapeutic agents as were then available. They commonly used electrodiagnostic measures which were the antecedents of modern ones.

The interests, training and experience of these specialists, with particular emphasis on impaired function of the neuromusculoskeletal systems, made them the most likely to be sought by senior surgeons in neurosurgery, orthopedics, and plastic surgery during World War II, to direct activation or rehabilitation centres for injured soldiers, sailors, and airmen. Before this, however, there were only a few special places in the United Kingdom, United States, and Canada that offered medically based and coordinated rehabilitation to persons with major specific impairments and disabilities such as polio, spinal cord injury, arthritis and amputation. A few were specifically for children, often those with very disabling conditions such as cerebral palsy, bone growth disorders, and amputations. These were not unified. A few for the industrially injured, had been established in some countries. These might be operated by insurance companies, voluntary groups, or Workers' Compensation Boards. One such centre was operated by the Liberty Mutual Insurance Company in Boston under the direction of Dr. Donald Munroe, a neurosurgeon. He had developed a system for the treatment and rehabilitation of persons with spinal cord injury. This comprehensive unified approach to the treatment of the spinal cord injured, sometime later was often referred to as the "Munroe Doctrine".

A very large Workers' Compensation Board Rehabilitation Centre was developed in Toronto which could treat 500 patients at a time, approximately one-half of whom would be inpatients. Even before World War II, a centre for persons with amputations was developed in West Orange, New Jersey, for the rehabilitation of amputees because of the inspired direction of Dr. H. Kessler. It subsequently, became known as the Kessler Institute. After the war a comprehensive centre, called the New York Institute of Physical Medicine and Rehabilitation, affiliated with New York University, was developed by Dr. Howard Rusk. It provided a beginning and a major stimulus to the development of other centres in North America giving great impetus to the development of the specialty of physical medicine and rehabilitation as well as recognition of the need for specific rehabilitation treatment centres, which subsequently flourished on this continent. There were other special programs often attached to other hospitals in the United States, but coordinated regional centres for spinal cord injured persons were not developed in the United States until the early 1970s when they were established by federal mandate and funding.

Canada was early in the development of facilities and programs. In the mid1940s, a spinal cord rehabilitation service was developed at Christie Street Veterans Hospital in Toronto. It was continued in its successor, the Sunnybrook Veterans Hospital in conjunction with a special centre known as Lyndhurst Lodge under the direction of Dr. Albin T. Jousse. Concurrently, a similar service was begun at Queen Mary Road Veterans Hospital in Montreal by Dr. Gustav Gingras from which developed The Rehabilitation Institute of Montreal. In Vancouver, the Western Society for Rehabilitation, under the leadership of Dr. G. F. Strong, established a rehabilitation centre which became a central part of the treatment and rehabilitation of spinal cord injured persons and later, of persons with disabling consequences of arthritis. It became a major component of the training program in physical medicine later developed at the University of British Columbia.

The establishment of these early centres, in conjunction with the development of physical medicine services in major veterans hospitals in Toronto and Montreal, led to the early development of educational programs for

*Professor Division of Physical Medicine and Rehabilitation, Department of Medicine, Dalhousie University and Founding Medical Director of the Nova Scotia Rehabilitation Centre, Halifax

Correspondence: Dr. Arthur H. Shears, 5991 Spring Garden Road., Suite #265, Halifax, N.S. B3H 1Y6

physicians in this field even before the Royal College of Physicians and Surgeons made available certification in this speciality in 1944. From these training programs, graduates spread out early to Saskatoon, Halifax, Edmonton, and Fredericton. Later, other programs began in Winnipeg and Hamilton. The program at Winnipeg was developed by a graduate of the University of Manitoba, Dr. H. Dubo, who had trained in the Columbia University program in New York. British trained physiatrists had practised physical medicine in Winnipeg prior to this time and a number of university of Manitoba physicians and surgeons had participated in the establishment of the Manitoba Rehabilitation Centre. The program at McMaster and the development of services in Hamilton were initially staffed by Canadians who had trained at the University of Minnesota. Subsequent programs in Ottawa were begun by persons who had worked in the McMaster system whereas those in Quebec City, Sherbrooke, and other cities in Quebec, were initiated and staffed by graduates of the University of Montreal program.

Physiatry was practised in Halifax from 1954, when Dr. G. Colwell returned from special training in Montreal and Boston. In 1956, an organization called The Nova Scotia Rehabilitation Council for the Rehabilitation of Disabled Persons, was formed by representatives from some thirty organizations. The name was subsequently changed to the Nova Scotia Rehabilitation Council. The first objective of the Council was to establish a Rehabilitation Centre and the executive was given a mandate to seek funds and staff to develop such a centre.

While training in the specialty from 1952, I was approached by Dr. W.D. Stevenson, former head of neurosurgery at the Victoria General Hospital and Dalhousie University, to return and develop the centre and the necessary personnel and programs that would go with it. When I returned in 1956 from training in Toronto and Boston, the initial program was for children followed within a few months by an outpatient program for adults. In December of 1957, a 19-bed inpatient service for persons with very disabling impairments from many causes, but predominantly from spinal cord injuries, was established. The outpatient service was very large and served both children and adults. It, rapidly, became larger.

Because of the physical link between the Centre and the old Halifax Tuberculosis Hospital (later called the Halifax Convalescent Hospital and subsequently the Halifax Civic Hospital), and the Halifax Children's Hospital, there was ease of consultation, directive care, and interchange with relationship to the children. Children who required very major rehabilitative efforts were usually inpatients at the Halifax Children's Hospital as we did not have appropriate beds for them, but this link enabled children to move back and forth daily for those services provided by the Centre. There was an admixture of persons with many other conditions, including the sequelae of polio, strokes, severe arthritis, polyneuropathies and multiple trauma, on a space-available basis. Dr. Stevenson was constantly supportive directly and through the Rehabilitation Council from the time he encouraged me to return until the completion of the first phase of the new Centre at about which time he retired.

A close relationship was maintained with Dr. John Woodbury, who was the head of rheumatology at the Victoria General Hospital and Dalhousie, by means of interchange of consultative and directive care for persons with joint diseases in the Victoria General Hospital and at the Rehabilitation Centre. The medical staff of the old Halifax Children's Hospital, in all departments, were very supportive thus facilitating earlier consultation and habilitation for children with severe impairments.

A relative increase in the number of beds useful for severely impaired patients was made possible by the establishment of beds by the City of Halifax in the same building. This was called the Halifax Convalescent Hospital and, later, the Civic Hospital. In that hospital, patients would be under the direct care of their own physicians and we were able to provide consultative and directive care for their physical treatment and rehabilitation. This reduced some of the pressure on the 19 beds in the Centre itself.

In addition to my responsibilities for the direction and development of the Nova Scotia Rehabilitation Centre, I was physician in charge of physical medicine and rehabilitation for the Children's Hospital while Dr. Colwell was physician in charge at the Victoria General Hospital and Camp Hill Hospital. Subsequently, together we developed the service at the Halifax Infirmary at the request of the medical staff there. We also provided consultation services to the other hospitals in the area including the Canadian Forces Hospital.

Undergraduate and graduate education programs began in 1957, the first being clinical teaching to groups from the Department of Medicine. This was followed by the development of a junior internship for one live-in third year student for a full academic year in conjunction with his other studies. This was begun in the summer of 1958, and the Residency program began with the return of that same student in 1960. The first such student, and subsequent Resident, was Dr. Howard Thistle. He completed his training at the University of Toronto and New York University later, becoming a professor of physical medicine and rehabilitation at New York University and the New York Institute of Physical Medicine and Rehabilitation as well as consultant in spinal cord injuries at the large Bellevue Hospital. The continuation of the "junior internship program" with the third year student living in for the full year, proved to be extremely useful in attracting students who subsequently went on in this specialty. Two of those are currently senior members of staff of the Centre and the University Division. Dr. J.L. Sapp and Dr. R.L. Kirby completed their training at the University of Alberta and the University of Washington, respectively.

Approximately 40 physiatrists have had all or part of their training in this Dalhousie program. The core of the training is provided at the Rehabilitation Centre and the

remainder in conjunction with members of the staff of other divisions and departments in other hospitals in Halifax. These persons have had a very high success rate in the Royal College of Physicians and Surgeons' examinations. All of the current physiatry staff of the Division, the Rehabilitation Hospital and the other hospitals in Halifax had some or all of their training in this Dalhousie program.

In the beginning, there was a severe shortage of members of other rehabilitation professions to participate in physical medicine and rehabilitation programs. The most urgent need required that we set up an on-the-job nursing training program so that nurses might be trained in the rudiments of rehabilitation nursing. Fortunately, I was able to attract Ms. Yvonne Piers, a native of Nova Scotia, who was then at the Royal National Hospital for Nervous Disease at Queen's Square in London, England, to come back, take a short course at Lyndhurst Lodge in Toronto and then work with me in developing the nursing service.

There were a few physiotherapists in the Veterans Hospital and at the Victoria General. These had come mostly from the United Kingdom with a few from the McGill and Toronto schools. It was obvious that a school for physiotherapy and occupational therapy in the region was necessary and as soon as enough clinical services were available for the clinical training, it was recommended by a committee of Dalhousie University Faculty of Medicine, that action be taken to initiate a school of physiotherapy and occupational therapy. It was decided by the Atlantic Provinces Departments of Health in May 1963 that a school of physiotherapy should be initiated and developed first because of funding limitations.

I was asked by Dr. C.B. Stewart, then Dean of Medicine at Dalhousie, to establish, set up, and recruit staff for this school at Dalhousie and to be the part time Director. The school opened and began operations four months later. I continued in this capacity on a part-time basis until 1975, at which time the school was reorganized with a full-time Director to prepare for its necessary expansion. Concurrently, a good deal of effort was spent in developing the groundwork for a school of occupational therapy but, because of the financial constraints, Governments of the Atlantic Provinces felt they would not be able to fund it. A school for occupational therapy was started some years later by its present Director. In the interim, a large department of occupational therapy was developed in the Rehabilitation Centre by Ms. Ardythe Parker, a graduate of McGill, the first occupational therapist at the Centre. Subsequently she took training as an occupational therapy teacher at McGill to be prepared to initiate with me a School of Occupational Therapy if funding became available.

The first speech therapist east of Montreal, who had been brought previously to Dalhousie University by the Junior League of Halifax, was transferred to the Nova Scotia Rehabilitation Centre staff at its opening in 1956. Ms. Marie Rudd, who had trained at Oxford University school in speech therapy, provided great service in speech therapy, but also did many things over, above and beyond the call of duty of her own specialty by rendering assistance, of many types, to children and adults at the Centre. Because of the large and growing number of persons with impairments of hearing and speech and the need for professionally trained persons for audiologic testing and speech therapy, it was obviously necessary to participate in the development of a speech and hearing centre. Once again it began as a division of the Nova Scotia Rehabilitation Council and was located not far from the Centre. Subsequently, the Speech and Hearing Centre grew to be a very large organization, separately housed, giving rise to the development of the School for Human Communication Disorders at Dalhousie University.

Because of great difficulty in getting orthoses (braces) in a reasonable period of time, it was necessary to work with members of the Council in developing a "brace shop". Originally, the shop was housed in the power house of the Victoria General Hospital and staffed by only two people. The demand was heavy and long delays for orthoses continued. Some were so long that between measurement for and delivery of the prescribed brace, a child would have outgrown it. After a great deal of effort, a larger facility, also as a division of the Council, was developed. The most suitable space that could be found was at St. George's Church Hall in the north end of Halifax. It was known as the Nova Scotia Brace and Appliance Centre.

The availability of prostheses for civilians was a major problem because no civilian source existed. It was necessary therefore to arrange a liaison with the old prosthetics department in the Camp Hill Veterans Hospital where the only prosthetics department existed. Their mandate of course was to provide prostheses as prescribed for veterans, so arrangements had to be made for the provision to our civilian population. An arrangement was worked out which required that prostheses prescribed in our amputee clinic at the Centre, would be provided, if the signature of the physiatrist on the prescription was also a guarantee of payment to the Department of Veterans Affairs. It was always necessary therefore, to ensure that funding was available to pay for the prosthesis before it was prescribed. The Brace Shop and the Prosthetics Department from Camp Hill Hospital, were integrated into the Centre in 1977 as the Department of Orthotics and Prosthetics.

Psychology and Social Services as a basis for social and vocational rehabilitation was an integral part of the Rehabilitation Centre's function from the beginning and a significant psychosocial vocational group was developed as part of the team. Vocationally, many persons, for a number of reasons related to their impairment, were unable to be directly vocationally established in the community. Because of this, and because of the need, it was felt that a sheltered workshop was necessary. The Council felt unable to handle another division at this time but it was able to encourage the Junior League of Halifax to give leadership in establishing one in the city. This is called New Leaf Enterprises. The comprehen-

sively staffed original centre, with its adult and pediatric outpatient programs, its adult inpatient program, as well as other special clinics for amputees, cerebral palsy, pediatric, industrially injured, athletically injured, was able to provide clinical education for persons in all such educational programs, at least at the undergraduate and, often graduate levels.

For twenty-one years, the Centre was housed in the old Halifax Tuberculosis Hospital which later became The Halifax Convalescent Hospital and later the Halifax Civic Hospital on University Avenue. Concurrent time consuming daily efforts were required on the part of the Medical Director in conjunction with members of the board of the hospital to find a location and funding for the new, modern, Rehabilitation Centre we envisioned. Among board chairman over the years, four deserve special mention for their unflagging contribution of skills and effort to help make this possible. These are Mr. John F. Fry; the late Robert Matheson Q.C.; Mr. Lloyd Caldwell Q.C., and Mr. Howard Moffatt. In addition, Dr. W.D. Stevenson and Mr. Donald E. Curren Q.C., although never chairman of the board both personally and through their roles in the Atlantic Division of the Canadian Paraplegic Association, were towers of strength in their support of the objective. This took a period of twenty-one years, culminating in the partial achievement of the modern centre in 1977 and its completion in 1991.

Throughout the Centre history, the medical staff, in addition to operating specific rehabilitation units within the Centre, has provided ongoing care for a large number of outpatients. They continued to provide a large volume of consultative and directive services at the request of attending physicians for persons with neuromusculoskeletal disorders and injuries, including soft tissue injury, to patients in other hospitals and their offices. Regional clinics were established early, initially in Cape Breton, later in Prince Edward Island and more recently in Yarmouth, Ahmerst and Antigonish.

The heavy duty rehabilitation principles and techniques, used to assist persons with very major impairments, was based on the spinal cord injury model. This was modified for persons with stroke, polio, multiple trauma, severe sequelae of connective tissue disorders and congenital or developmental disorders. It is of considerable interest and of importance in the development of the Centre that as persons with major disorders from these conditions progressed to better functional levels, they mingled with large numbers of outpatients with lesser impairments in the outpatient department to continue their treatment as outpatients. It was a result of this "mingling" that some persons being treated only as outpatients saw and learned about what rehabilitation could do. This caused some of them to come forward and ask if they could assist in the search and development for a new Centre. Because of this, a number of community-minded persons brought some valuable skills to the Board of the Centre, thus, assisting in its further development.

There has been a gradual expansion of services as the number of trained physiatrists and other rehabilitation professionals were trained bringing special interests and skills to the provision of services, teaching, and research. This has led to the development of organized programs for persons with major impairments and disabilities, such as stroke, traumatic brain injury, amputations and various pediatric disabilities, in addition to the original spinal cord injury rehabilitation service and the general neuromusculoskeletal service. There has been a concurrent expansion and development of diagnostic services including the expansion and development of the electrodiagnostic service from the original electromyography, which began in 1956, to the present fully equipped and staffed laboratory as well as the development of a clinical locomotor function laboratory to assist in diagnosis and research. The availability of the large outpatient service had permitted smooth transition from the hospital to home and community.

These developments have permitted concurrent expansion of the educational and training programs of undergraduate medical students in physical medicine and rehabilitation. They have, also, permitted the expansion of research by physiatry staff and other rehabilitation professionals.

Physical medicine and rehabilitation services are now more readily and completely available throughout the province, and to some degree, for special patients from the rest of the Atlantic Region. □

Cognitive Remediation: Fact or Fiction

B.M. Joyce,* MD, FRCPC

Halifax, N.S.

Cognitive remediation is a challenging new technique for systematically endeavouring to improve cognitive functions that have been impaired by damage to the central nervous system. This is a controversial technique and much research remains to be done before definitive conclusions can be drawn regarding its validity and use for rehabilitation purposes.

Patients with traumatic brain injury are becoming more familiar to us in our day-to-day practices. In the U.S.A. there are an estimated 450,000 cases each year.[1] In Canada, the annual incidence is approximately 10% of the U.S. i.e. approximately 40,000 head injuries annually. A review of head injury in Nova Scotia estimated there are approximately 520 head injuries in our province yearly.[2] Advances in neurosurgical techniques have decreased mortality in cases of severe head injury; unfortunately, however, many persons who survive these injuries are left with cognitive and resulting psychosocial difficulties. It is these difficulties, rather than physical deficits, that lead to these persons' long-term disability and dependence on family and social support systems.

Cognitive remediation or rehabilitation is a systematic endeavour to improve cognitive functions that have been impaired following damage to the central nervous system. In the U.S.A. at least, it has become one of the fastest-growing rehabilitation interventions; there are more than 600 programs of cognitive rehabilitation in the U.S. today, whereas barely 60 existed 10 years ago.[3] As with all rehabilitation, it attempts to restore persons to their highest functional level. However, controversy remains as to whether specific cognitive remediation is superior to a general rehabilitation program.

In the next few paragraphs, I will review briefly some of the background to cognitive remediation and discuss the basic approaches.

Although the roots of cognitive remediation can be traced back to the ancient Greeks, interest can be found in the medical literature after World War II, when Zangwill advocated cognitive remediation in the rehabilitation of head-injured soldiers.[4] Subsequent to this, much of the initial research in this field was directed toward stroke patients.[5]

Luria *et al.* outlined four basic principles which were felt to be important in terms of the restoration of function in persons with a diffuse brain injury.[6] These principles, which have stood the test of time and are the basis for many remediation programs are:

1) **Differential restoration of functional systems.**
Luria felt that neuropsychological investigation was the basis for planning the restoration of the cognitive ability. "If we know which links of a functional system are damaged and which are preserved, we can make a differential approach to the problem."[6]

2) **Complete extended programming of the restorative activity.**
Cognitive tasks should be broken down into underlying processes. "If only one link of the activity to be restored is omitted from this program, the successful restoration of the disturbed function is impossible."[6]

3) **Taking advantage of the intact link.**
It may be necessary to teach the patient compensatory strategies. "The injured link of the functional system may be replaced by an intact link."[6]

4) **Constant signalization of both the defect and of the effect of the action.**
Luria wrote, "Only a constant flow of feedback signals can make possible the comparison between a performed action and the original plan and enable mistakes which have been made to be corrected in time."[6]

The establishment of a cognitive remediation program by Ben-Yishay and his associates in Israel in 1974 (following Arab-Israel conflicts), marked the beginning of a "modern" approach to cognitive remediation. Ben-Yishay and Prigatano certainly embraced Luria's principles of relearning but felt they were insufficient.[7] Additionally, they emphasized the need to supplement neuropsychological tests with situational evaluation of each patient's strengths and weaknesses. They also suggested it is necessary that the patient's personality/behavioral difficulties be clarified in various settings. Additionally, the effectiveness of programming is greatest when the setting is varied. Finally, Ben-Yishay and Prigatano felt that a lack of insight or impairment of self-awareness is a significant factor in a person's lack of motivation to engage in remedial activities. Many of the cognitive remedation approaches that we see today have been built on these various principles.[8,9,10]

Currently there are three basic approaches: 1) the functional-adaptation approach; 2) the general-stimulation approach; and 3) the process-specific approach.

*Assistant Professor, Division of Physical Medicine and Rehabilitation, Department of Medicine, Dalhousie University, Halifax.

Correspondence: Dr. B.M. Joyce, Nova Scotia Rehabilitation Centre, 1341 Summer Street, Halifax, N.S. B3H 4K4

The *functional-adaptation approach* attempts to improve functional skills related to work, school or activities of daily living, by first analyzing functional tasks and then developing environmental manipulation or compensatory strategies that are presumed to enhance the underlying cognitive skills that those tasks require. The difficulty with this approach is the restricted generalizability which may lead to a person performing a task very well in a rehabilitation setting but not well at home or in the community.

The *general-stimulation approach* attempts to facilitate recovery through the use of tasks which encourage cognitive processing at many levels of complexity. Depending on the type of patient and the extent of deficits, this may or may not be appropriate, as it often overwhelms the patient and leads to failure.

The *process-specific approach* is the model favoured at many centres. Therapists use neuropsychological testing and functional evaluation in an effort to identify specific cognitive deficits towards which treatment is targeted, on the assumption that one can affect individual cognitive functions differentially. Areas that are targeted can include attention, memory, visual scanning and processing, reasoning, problem solving and executive functions. It is important to note that when a specific cognitive function is resistant to "retraining" (as memory often is), remediation strategies center around compensatory strategies.[11]

Having briefly discussed some of the background to cognitive remediation, I would like to return to the earlier statement that controversy remains as to the effectiveness or outcome of the cognitive remediation process. Much of this controversy stems from the fact that the rapid emergence of many of our programs has been as a result of societal pressures rather than sound methodological studies. In an excellent review of this subject in the February 1990 issue of the *Archives of Neurology*[12,13,14,15], and more recently in the February 1993 issue of the *Archives of Physical Medicine and Rehabilitation.*[16], the authors discuss the methodological pitfalls of studying such a heterogeneous group and note that previous studies have contained such design flaws as a lack of randomization, differences in the patients in terms of severity of injury, and a lack of blinded conditions. Furthermore, we cannot yet correlate specific patterns of neural damage and recovery of function with specific neuropsychological theory or cognitive remediation interventions.

Nevertheless, cognitive remediation, both as a theoretical concept and as a body of intervention techniques, continues to evolve. Ben-Yishay and Diller recently reviewed the subject in terms of differing conceptual and philosophical orientations and discuss emerging theories of "metacognition", or "knowing about knowing", and the pivotal effect this can have on the remediation process[16] There is promising work emerging, and studies are being published – and, I feel confident, will continue to be published – that support rehabilitation programs aimed specifically at the cognitive remediation of brain injury. I feel Hachinski summed it up very well when he stated, "Without therapeutic enthusiasm there would be no innovation, and without scepticism there would be no proof." [13] □

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"Standing on your dignity makes for poor footing."

Arnold H. Glasow

Common Medical Problems of Stroke Survivors During Rehabilitation

E.R. Harrison,* MD, FRCPC

Halifax, N.S.

Successful rehabilitation following a major stroke requires appropriately timed therapeutic interventions. In the acute phase, management is predominantly diagnostic and supportive (the Rehabilitative Phase or Phase 2). Once patients are medically and neurologically stable, rehabilitative measures to prevent potentially fatal or disabling complications, remedial measures to reduce the effect of treatable impairments, and retraining and education become paramount to ensure optimal functional recovery. The goal of this article is to update, for community physicians, the prevention and management of the common complications that follow a completed stroke.

Stroke is the third leading cause of death and most common disabling illness in Western society. The total yearly cost in the U.S.A., was estimated at between 7.5 and 11.2 billion dollars yearly in 1979.[1] Of those who survive, as many as 80% will ambulate but only 10% are capable of returning to full function, (including return to the work force), whereas 40% will be mildly disabled, 40% will be severely disabled, and 10-15% will be institutionalized.[2] Although the incidence of stroke was lower in the 1970s than in the 1960s (1.5/1000 vs. 0.8/1000 population), possibly due to better hypertension control, the prevalence of stroke survivors increased by 20% (5/1000 vs. 6/1000 population) during the same time interval, likely due to there being fewer cardiac-related deaths.[3] The majority of this increase was in persons who were most severely disabled. Early, effective, intensive rehabilitation, in appropriately selected individuals, however, has been shown to be associated with earlier discharge from hospital, greater likelihood of being discharged to the home and higher levels of functional independence which can maintained for years post-stroke.[4,5,6]

Optimal rehabilitation requires the prevention of complications and of potentially disabling impairments, retraining active function, learning means to accommodate for altered function including use of appropriate assistive devices (e.g. orthoses/bracing), modification of the home or environment to accommodate residual disability, and education of patients and their families in new social roles and resources in the community.

*Assistant Professor, Division of Physical Medicine and Rehabilitation, Department of Medicine, Dalhousie University, Halifax.

Correspondence: Dr. E.R. Harrison, Nova Scotia Rehabilitation Centre, 1341 Summer Street, Halifax, N.S. B3H 4K4

The purpose of this article is to review common medical complications that may limit patients' recovery of function in the rehabilitative phase of recovery.

DEEP VENOUS THROMBOSIS AND PULMONARY EMBOLISM

In the acute phase, the incidence of deep venous thrombosis (DVT) post-stroke is about 50% (23%-75%) by radiofibrinogen tagging.[7] Pulmonary embolism occurs in 20% of these patients and accounts for 13% of deaths (the fourth leading cause of death in acute stroke and the leading cause of death after the second week). Even in the rehabilitative phase, 11% of patients may have DVT and as many as 7% may develop pulmonary embolism, with 2.3% dying as a result. The major risk factor appears to be immobility as DVT is at least twice as frequent in the hemiparetic leg as in the unaffected leg, and the risk is five times greater in nonambulatory patients than in ambulatory patients on rehabilitation units.

Prophylaxis with subcutaneous heparin, in doses of 5,000 U s.c. bid or tid, reduces the risk of DVT by one-third and the risk of pulmonary embolism by about four-fifths. The duration of therapy is controversial. It seems reasonable to continue prophylaxis until patients are ambulatory or until the second or third month after stroke. Where low-dose heparin is felt to be contraindicated (e.g. large hemorrhagic stroke) external pneumatic calf compression is used in some centres.

All stroke patients should be considered at high risk for DVT at all stages of recovery. In some centres, impedance plethysmography is performed at two-week intervals. As this is not readily available in most secondary centres, routine low-dose heparin prophylaxis and vigilance is necessary. All suspected cases should have definitive investigation (e.g. venography) to identify DVT and be treated with full anticoagulation for 3 months for DVT and 6 months for pulmonary embolism. Mobilization can safely be begun two or three days after the start of anticoagulation, to prevent the deleterious effects of immobility, including depression. Where anticoagulation is contraindicated (e.g. hemorrhagic stroke) vena caval filters should be considered.

PNEUMONIAS

Pneumonia is the third leading cause of death in the first month following stroke.[8,9] A national stroke survey in the U.S.A. estimated that one-third of stroke patients had had respiratory infections as a complication of stroke.[10]

Unless prevented or recognized early and effectively treated, pneumonia delays or prematurely terminates rehabilitation in large numbers of stroke survivors.

Acutely ill stroke patients are particularly susceptible to developing pneumonias due to concomitant medical problems (e.g. diabetes mellitus), immobility due to hemiplegia and decreased level of consciousness, dysphagia, their age and the changes in pulmonary function that follow stroke. The incidence of dysphagia is reported as approximately 50% among acute stroke patients.[11] Among patients who are alert with normal levels of consciousness, however, only 10% have dysphagia. Choosing the appropriate time to reintroduce oral feeding is therefore an important step in diminishing the incidence of aspiration pneumonias.

With aging, changes in vital capacity, expiratory flow rate and FEV_1 occur. Dead air space increases, airways become narrowed, ventilation becomes less uniform and oxygen tension is reduced. The likelihood of nutritional deficiencies increases and humeral and cell-mediated immune responses are reduced.

In the acute phase, tachypnea, Cheyne-Stokes respiration with secondary respiratory alkalosis is common.[12] Sustained tachypnea is a poor prognostic indicator for survival. In the late phases, a variety of types of respiratory dysfunction have been noted: decreased thoracic movement on the paretic side, restrictive respiratory dysfunction, decreased lung capacity, decreased functional reserve, and reduced expiratory reserved volumes.[13] The restrictive impairments have been correlated to severity of paresis. The ability to clear airways is also likely to be diminished by reduced strength in the abdominal, accessory and inspiratory muscles. The inability to change position further predisposes to atelectasis, which increases the risk of pneumonia.

Pneumonia (and, indeed, other infections such as urinary tract infections) should be suspected when alterations in level of consciousness, strength or functional status are noted. Re-evaluating the patient with respect to the possibility of aspiration pneumonia is important. Prompt appropriate antimicrobial therapy, rehydration and chest physiotherapy can then be instituted.

SHOULDER AND ARM PAIN

Shoulder pain has been reported in 70-90% of stroke survivors.[14,15] The etiology varies based on pre-stroke musculoskeletal factors, stage of neurological recovery post-stroke and recovery of function. Differential diagnosis includes trauma to a flaccid or mobile limb caused by the patient or a caregiver, adhesive capsulitis in a spastic arm, impingement syndromes as motor recovery allows active arm elevation, peripheral neurogenic factors such as cervical radiculopathy or brachial plexopathy, central neurogenic factors such as reflex sympathetic dystrophy and central pain syndrome.

Although inferior subluxation of the flaccid shoulder is frequently observed, a close correlation between paralysis of the deltoid and rotator cuff muscles and pain has not been found. It is likely that inadvertent trauma to the flaccid unprotected shoulder is a source of pain originating from the rotator cuff. This can be prevented by ensuring that patients do careful passive range-of-motion exercises (generally within the range of 60° of abduction and 60° of flexion and 15° of external rotation) and that they support the arm with a "hemi-sling" during transfers and while walking and with an arm trough or lap board when seated in a wheelchair.

Shoulder pain in the spastic phase of motor recovery is common. Capsular contractions are noted in 77% of painful shoulders following stroke. Prevention is possible by twice-daily range-of-motion exercises, use of ice, careful positioning of the limb and electrical stimulation to decrease tone. Once capsular contractures are established, more aggressive treatment with range-of-motion exercises in a therapeutic pool, shoulder joint mobilizations, bursal corticosteroid injections or nerve blocks may be necessary. One should always look for predisposing factors for capsulitis, such as cervical spondylosis and radiculopathy, which warrant treatment. Rarely surgical intervention is necessary.

As motor recovery progresses, incoordination of the glenohumeral and scapulathoracic movements predispose patients to impingement movements. In this situation, scapula mobilization, the use of facilitative shoulder slings or electrical stimulation can be helpful. More severe symptoms warrant corticosteroid injection.

In studies of hemiplegics, rotatory cuff tears were noted in 40% of paretic shoulders but only 16% of unaffected shoulders. This should be suspected when hand and elbow strength are greater than shoulder strength, particularly when the latter movements are painful. Superior subluxation of the humeral head relative to the glenoid, humeral head osteophytes and cysts with erosion of the acromion are the radiographic findings on plain films. An arthrogram, either with plain radiographs or a CT, confirms the diagnosis. Analgesics can help to maintain a range of motion and teaching compensatory strategies, such as passive movements, may be necessary.

Neurogenic etiologies are more common causes of arm pain than most people suspect, indeed, one Japanese study found axillary and suprascapular nerve conduction abnormalities in 70% of shoulders of their hemiplegic patients.[16] Although upper trunk lesions have a good prognosis, retraining of the upper extremity may be delayed many months. Reducing traction on the brachial plexus by proper positioning and the use of upper-extremity supports and slings may reduce further trauma. Cervical radiculopathies or apparent thoracic outlet syndromes respond well to physical measures, such as appropriate postural and range-of-motion exercises, stretching of tight muscles groups around the neck and shoulder girdle, and cervical traction. Whenever motor recovery proximally is behind distal recovery or when there is a deterioration of upper-limb function, these etiologies should be considered. Electrodiagnostic studies are useful in arriving at a specific diagnosis.

Reflex sympathetic dystrophy has been reported in 12.5-25% of stroke survivors.[17] Generally this occurs be-

tween 1 and 5 months post-stroke. Although patchy osteoporosis is characteristic, at times differentiating it from disuse osteopenia on plain radiographs is difficult. Reducing any edema by briefly dipping the hands in ice water, using range-of-motion exercises (active and passive) and reducing pain with physical modalities such as TENS seem to be helpful in many instances. The use of corticosteroids, sympatholytics (e.g. phenoxybenzamine), regional guanethidine infusion and manipulation under Bier block all have their advocates. The author has found, however, that the response to stellate ganglion blocks in stroke survivors is well tolerated and effective, if followed by an appropriate program of active and passive range-of-motion exercises.

BOWEL AND BLADDER MANAGEMENT

When the patient is medically unstable and when level of consciousness is impaired, urinary incontinence is to be expected. As a patient "lightens up", any indwelling catheter should be removed. Urine for C&S should be taken before the catheter is removed and the patient should be treated for any bacterial growth identified and begun on a time-voiding regime by being offered a urinal or a bedpan every 1-2 hours. Intermittent catheterization may prevent incontinence. If incontinence persists other factors should be identified (Table I). Patients with communication difficulties frequently cannot attract the attention of the nursing staff. Hemiplegics have difficulty managing urinals and frequently spill them, but relatively "no spill" urinals are available. If a post-void or post-incontinence residual is normal and no bacterial growth has occurred, then incontinence is usually due to a loss of cortical inhibition and may respond to oxybutynin (Ditropan®) starting at 5 mg increasing slowly to 15 mg daily. If incontinence persists, a urological consultation and evaluation (including cystoscopy and urodynamic studies) is indicated.

TABLE I

CAUSES OF URINARY INCONTINENCE

Neurogenic
decreased level of consciousness
acute C.N.S. event (seizure, stroke, etc)
decreased detrusor inhibition
Urological
infection/calculi
structural changes (Benign prostatic hypertrophy)
Disability Related
communication impairments
immobility
impaired hand and arm function
Iatrogenic
medications

Initially most stroke patients are on intravenous fluids and have little oral intake. With the resumption of feeding, patients should be placed on a bowel regimen to re-establish a normal bowel pattern. When this is not done, overflow faecal incontinence is an all too common occurrence. Rectal and abdominal examinations can usually identify this. When in doubt, one should have a flat plate of the abdomen taken. Complete evacuation of the bowel with high enemas, sometimes in sequences of 2 or 3, allow resumption of a normal bowel pattern, usually with the aids of cathartics. A reasonable regimen might include Dulcolax® tablet at bedtime, followed the next morning by a Dulcolax® suppository after breakfast, with these repeated every two days. Once a normal bowel pattern is established, these interventions can be stopped.

PRESSURE SORES

A national survey in the U.S. found that 14.5% of stroke patients had pressure sores. Risk was greatest in those who had been comatose, had thrombotic or embolic etiologies, were debilitated or markedly obese, were bowel or bladder incontinent, or had severe spasticity. Inspecting high-risk sites (ear, shoulder, greater trochanter, malleoli, heels and ischial tuberosities) at least daily, turning immobile patients every two hours, preventing bowel and bladder incontinence, maintaining optimal nutrition, and attending to technique when performing transfers from the wheelchair are necessary to prevent pressure sores, which, once developed, markedly delay rehabilitation.

FRACTURES

Fractures have been reported in 3.1% of stroke survivors, usually fractures of the hip.[18] Patients at risk include those who are impulsive or who have visuospatial perceptual impairments. High-risk activities seem to be ambulating to the washroom at night (without the usually worn ankle/foot orthosis) and transferring to and from a wheelchair. Sometimes the use of special bracing that a patient can do easily is necessary for safe ambulation at night.

DEPRESSION

Post-stroke depression can be a major obstacle to functional recovery. Studies suggest that 40% of stroke survivors are clinically depressed within the first month following stroke, and, although this number seems to be reduced to approximately 25% at one year post-stroke, some stroke survivors remain depressed for as long as two years after their stroke.[19] The role of the site of lesion on incidence of depression is controversial, but it is generally agreed that there are both reactional and organic components to depression after a stroke. Numerous depression scales have been developed, but their applicability to stroke patients is questionable. More helpful are standard psychiatric criteria for depression, which can be easily remembered with the acronym SIG-E-CAPS (sleep, interest, guilt, energy, concentration, appetite, psychomotor retardation and suicidal ideas). All patients who are failing to progress in their recovery of function after a stroke (proportional to their neurological recovery) should be evaluated specifically for depression.

Management needs to be both behavioral and pharmacological. Educating the family and encouraging its support of the patient is important, as is educating the patient in the time course of recovery from stroke and demonstrating to the patient that functional recovery is possible with effective rehabilitation. Medications such as trazodone (Desyrel®) and nortriptyline (Aventyl®) have been demonstrated to be effective in reducing post-stroke depression.[20,21] Other anti-depressant medications should be as effective, and the choice of medication should be based on minimizing potential adverse effects in a specific patient. The therapeutic window of these agents may be altered by brain injury, so one should "start low and go slow". If a response is not being noted to the above measures, then psychiatric consultation is very appropriate.

SUMMARY

In summary, this paper outlines some of the common complications — many of them preventable by appropriate medical and nursing care — that can occur among stroke patients. Optimal rehabilitation also involves evaluating the individual's pre-stroke functional status and environment, evaluation of their current functional status, optimal retraining or accommodation to their deficits to allow independent function and evaluation of the psychosocial needs and resources at home and in the community to accommodate the disabled stroke survivor. A multidisciplinary team involving physicians and other health professionals is necessary to obtain optimal functional restoration and resumption of as many social roles as possible for the stroke survivor. □

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A Calgary psychiatrist was surely guilty of ***lapsus linguae*** *when, in a newspaper interview, he questioned the validity of Statistics Canada suicide rates by cautioning, "It's dangerous to jump to conclusions".*

Books in Canada

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Traumatic Spinal Cord Injury

OVERVIEW OF INCIDENCE, CLASSIFICATION AND REHABILITATION

Robert K. Mahar,* MD, FRCPC

Halifax, N.S.

Prior to World War II, most people who sustained a traumatic spinal cord injury died, usually within 12 months, of causes such as infection, respiratory compromise, renal failure, complications of immobilization, and decubitus ulcers. With the development of antibiotics and improved acute care, physicians of the day found that more of these people had the potential for a longer life. Systems of care at that time evolved under such pioneers as Sir Ludwig Guttman, Dr. Donald Munro and Dr. Howard Rusk. Dr. Arthur Shears studied under Dr. Munro at the first specialized spinal cord injury unit in the United States (Boston) and returned to Nova Scotia in 1956, to develop and provide rehabilitation services for this population.

The goal of such programs is to provide a coordinated system of care. It has been demonstrated that provision of a full range of services, in association with maximum patient participation and education, enhances the likelihood of achieving favorable outcomes, promotes the resumption of meaningful life roles, and facilitates the opportunities for community reintegration. Through the improvements in acute care that have occurred on the medical and surgical fronts, persons with severe impairments and disabilities survive the first few months following injury and often face a marked and permanent alteration in the way they view and live their lives. Although research continues towards a cure for spinal cord injury, rehabilitation is guided by the premise that these people shouldn't be "left in bed" until this happens.

Nearly all physicians (family physicians and specialists) are called upon to provide care for this population. This is because a number of organ systems are involved as a result of the dysfunction and complications of the neurologic injury. As well, there are frequent psychological and social problems. Adoption and utilization of a standardized descriptive terminology and classification is desirable and this will be presented in this brief overview.

Rehabilitation involves a team that includes members of a number of disciplines: medicine, nursing, physiotherapy and occupational therapy, social work, psychology, nutrition, recreation therapy, vocational counselling. Liason with community agencies such as the Canadian Paraplegia Association, which provides counselling, educational and advocacy support following discharge, is important.

*Lecturer, Division of Physical Medicine and Rehabilitation, Department of Medicine, Dalhousie University, Halifax.

Correspondence: Dr. R.K. Mahar, Nova Scotia Rehabilitation Centre, 1341 Summer Street, Halifax, N.S. B3H 4K4

OVERVIEW OF SPINAL CORD INJURY

The annual incidence of traumatic spinal cord injury in which the person survives to reach hospital is about 32 cases per million population. At the Nova Scotia Rehabilitation Centre, we cared for 17 new cases in 1992. Prevalence in Canada is estimated at between 20,000 and 25,000 people who have survived a traumatic spinal cord injury.

The three leading causes of spinal cord injury in Canada are motor vehicle accidents (includes pedestrian and motorcycle), falls and sports injuries, with diving being the leading cause of sport injury.

Of all traumatic spinal injuries, 5% occur in those under age 16. The largest number occur in the 16 to 30 year age group (about 60% of total) and 20% occur to those between 31 and 45 years of age. Studies state that the age group over 60 makes up 5% of the SCI (spinal cord injured) population. It is our impression in Nova Scotia that we see a greater percentage of individuals over 60. Typically, this is a man who falls, often does not sustain a fracture or dislocation, and who presents with features of a central cord syndrome as a result of cord contusion and hemorrhage produced by posterior osteophyte impingement on the cord. The Spinal Cord Injury Program at NSRC is in the process of reviewing these data.

Males make up 80% of the spinal cord injured population. There is a seasonal variation with 1.6:1 summer to winter ratio.

With regard to levels of injury, the literature reports the ratio of quadriplegia (impaired hand function) to paraplegia has varied little over the last 30 years, with about 54% of injuries classified as producing quadriplegia. There is a trend towards a greater number of incomplete injuries, one study indicating a 38% incidence in 1973 and 54% in 1984. One hypothesis is that this is due to improved emergency and acute care. The incidence of multiple trauma is about 44%.

Patients with non-traumatic spinal cord injury are often cared for on spinal cord injury rehabilitation units utilizing the same principles of care. The exact incidence is difficult to evaluate in the literature. The most common groups of impairments seen include cervical myelopathy secondary to cervical spondylosis, vascular lesions and tumors. Less common causes include osteomyelitis or abcess, and herniated intervertebral disc.

Causes of death for patients who survive the initial injury have changed significantly with the passage of time. Death from chronic renal failure, once extremely common is now relatively rare, and is due to improved management and follow up of neurogenic bladder. Data

indicate the leading causes of death to be respiratory conditions (usually pneumonia), heart disease, subsequent trauma (includes suicide), septicemia and malignancy.

CLASSIFICATION OF SPINAL CORD INJURY

The importance of uniform standards for classification has long been recognized and a system has been adopted following interdisciplinary and interspecialty discussions. This included physical medicine and rehabilitation, neurosurgery, orthopedic surgery, surgery. In 1989, the American Spinal Injury Association (ASIA) adopted standards which have been altered and accepted in November 1992. This incorporates some functional measures. For purpose of this review, aspects of motor and sensory classification and impairment scale will be presented to encourage utilization of this terminology.

The *neurologic level of injury* is defined as the most caudal segment that tests as normal, or intact, for both motor and sensory function. Dermatomes are examined on the basis of the key sensory areas (Fig 1) and myotomes, by their key muscles (Table I). The strength of each muscle is graded on a six point scale:

0 = total paralysis

1 = palpable or visible contraction

2 = active movement, full range of motion (ROM) with gravity eliminated

3 = active movement, full ROM against gravity

4 = active movement, full ROM against moderate resistance

5 = active movement, full ROM against full resistance

NT = not testable

By convention, if a muscle tests as a grade of 3/5 (full range of motion against gravity but not against resistance), its neurological level is considered to be intact.*

There are 28 key sensory dermatomes; on examination, they are graded on a three point scale: (Fig. 1) 0 = absent, 1 = impaired, 2 = normal. There is separate grading for pin and touch sensation. If a patient cannot discriminate between sharpness and dullness, the score is zero for pin appreciation. The sensory neurologic level of injury is named by the last key dermatome to have normal (2/2) sensation.

The neurologic level of injury can be asymmetric from side to side, and the motor and sensory levels may be the same or different. Thus, a person may be deemed to have the following lesion: C8R S C7L S, C7R M C7L M (C8 right sensory, C7 left sensory, C7 right and left motor).

* It is understood to be a simplification to represent a spinal cord segment by only one muscle or one muscle group, because most muscles are innervated by two segments; however, if a muscle is innervated by only one segment, this results in a weakened muscle. If a muscle tests as grade 3/5, it is considered to have full innervation by at least one of the two segments, that is, the most rostral segment.

TABLE I

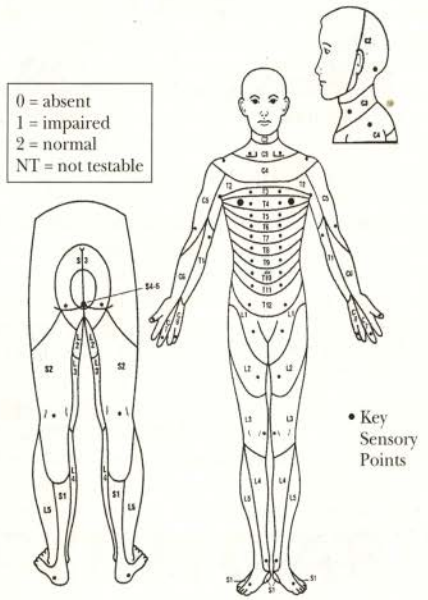
KEY MUSCLES FOR MOTOR LEVEL CLASSIFICATION

C1-4	Use sensory level and diaphragm to help localize lowest normal neurologic segment
C5	Elbow flexors (biceps, brachioradialis)
C6	Wrist extensors (extensor carpi radialis longus and brevis)
C7	Elbow extensors (triceps)
C8	Finger flexors - middle distal phalanx (flexor digitorum profundus)
T1	Small finger abductors (abductor digiti quinti)
T2-L1	Use sensory level and Beevor's sign to help localize lowest normal neurologic segment
L2	Hip flexors (iliopsoas)
L3	Knee extensors (quadriceps)
L4	Ankle dorsiflexors (tibialis anterior)
L5	Long toe extensors (extensor hallucis longus)
S1	Ankle plantarflexors (gastrocnemius, soleus)
S2-5	Use sensory level and sphincter ani to help localize lowest normal neurologic segment

From American Spinal Injury Association: *International Standards for Neurological and Functional Classification of Spinal Injury Patients.* Chicago: ASIA, 1992 (with permission).

The motor index score is a numeric scoring system used to document changes in motor function. It is a summation of the motor grades of the 10 key muscles, for both the right and left sides (Table II). The maximal score is 100. This nomenclature utilizing specific key muscles for evaluation of each level provides for more uniform evaluation of neurologic status and outcome.

KEY SENSORY POINTS



From American Spinal Injury Association: *International Standards for Neurological and Functional Classification of Spinal Injury Patients.* Chicago: ASIA, 1992; with permission.

Figure 1

TABLE II

MOTOR INDEX SCORE: THE SUMMATION OF MANUAL MUSCLE TEST FOR EACH KEY MUSCLE

Right	Key Muscle Segment	Left
5	C5	5
5	C6	5
5	C7	5
5	C8	5
5	T1	5
5	L2	5
5	L3	5
5	L4	5
5	L5	5
5	S1	5
50		50

From American Spinal Injury Association: International Standards for Neurological and Functional Classification of Spinal Injury Patients. Chicago: ASIA, 1992; with permission.

Asia Impairment Scale (Modified From Frankel)

A Complete: No sensory or motor function in the sacral segments S4-5

B Incomplete: Preservation of sensation below the level of injury extending through the sacral segments S4-S5.

C Incomplete: Preservation of motor function with the majority of the key muscles below the level of injury having a muscle grade less than 3.

D Incomplete: Preservation of motor function, with the majority of key muscles below the level of injury having a muscle grade 3 or more.

E Normal: Recovery of normal motor and sensory function.

Spinal cord injuries are further classified by the neuroanatomic area of involvement in terms of the "cross-section" or somatotopic extent of injury. Although there are other recognized cord syndromes (e.g. posterior cord syndrome), the current classification recognizes five discrete clinical entities as incomplete spinal cord injuries:

1. Central cord syndrome: a lesion, occurring almost exclusively in the cervical region, that produces sacral sensory sparing and greater weakness in the upper limbs than in the lower limbs. This is commonly produced by hyperextension of the degenerative cervical spine, with or without bony fracture.

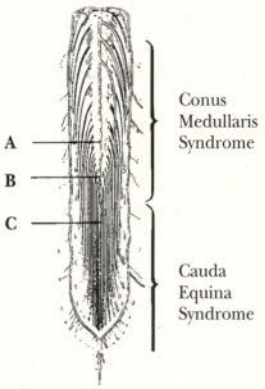
2. Brown-Sequard syndrome: a lesion (caused, for example, by stabbing or gunshot) that produces relatively greater ipsilateral proprioceptive and motor loss and contralateral loss of pain and temperature sensation.

3. Anterior cord syndrome: loss of pain appreciation and motor function with relative preservation of proprioception.

4. Conus medullaris syndrome: injury of the sacral cord (conus) and lumbar nerve roots within the neural canal, which usually results in an areflexic bladder, bowel and lower limbs, with lesions at B in Figure 2. Sacral reflexes may occasionally show preserved reflexes, e.g., bulbocavernosus and micturition reflexes, with lesions as at A in Figure 2.

5. Cauda equina syndrome: injury to the lumbosacral nerve roots within the neural canal resulting in areflexic bladder, bowel and lower limbs, with lesions at C in Figure 2.

In summary, the injury would be classified in terms of the neurologic level of injury, ASIA impairment scale, ASIA motor score and possibly as a recognized cord syndrome (e.g., C7 R M&S, C6 L M&S, motor score 65/100, ASIA scale C, central cord syndrome).



From American Spinal Injury Association: *International Standards for Neurological and Functional Classification of Spinal Injury Patients.* Chicago: ASIA, 1992; with permission.

Figure 2

REHABILITATION

Currently, prognosis is based on the results of the neurologic examination 72 hours after injury. Diagnostic studies such as MRI may become more important in the future. A great number of prospective studies review outcomes. This is important in the evaluation of outcomes from specific therapeutic interventions (e.g. early use of parenteral corticosteroids). Otherwise, our ability to predict outcome on a case-by-case basis is significantly flawed. There is, however, general agreement in the literature that individuals with Frankel A classification (which make up about 50% of all spinal cord injuries) have a poor prognosis for functional recovery. Prognosis for recovery of the ability to walk in central cord syndromes is good, with age being an important predictor (97% of those under 50 years as compared to 41% of those over 50 years of age).

The goals of rehabilitation are:

1. optimal function as predicted by neurologic level of injury and other limitations

2. optimal emptying of the bladder
3. acquisition of knowledge of sexuality and able to take part in sexual activity
4. owel continence and regular evacuation
5. health maintenance education provided
6. adjustment to disability
7. accessible home, school and workplace
8. use of appropriate equipment
9. transportation in community
10. vocational plans implemented
11. access and utilization of community resources
12. ensure regular follow-up to prevent secondary medical problems.

Inpatient rehabilitation should begin as soon as the patient is stable and able to tolerate the activity level required. The nature of this injury such that it has so many far reaching consequences including medical, psychological, functional and social issues, (Tables III and IV) that a multidisciplinary, problem oriented approach is particularly useful. Following discharge, these patients are followed in the Spinal Cord Injury Clinic.

These individuals and their families usually have permanent problems which will require preventive and maintenance care. As well, as they represent a small percentage of the population, some of their problems: autonomic dysreflexia, post-traumatic syringomyelia and heterotopic ossification may be less well understood by some physicians.

Therefore, they need to be clearly educated about the symptoms of such disorders as well as "more routine day to day issues" such as management of neurogenic bowel problems. This needs to be repeated frequently and in different settings.

Each patient's problem list to be addressed by the team and the patient (Table 4) is formulated at the admission team conference and reviewed and updated at weekly team meetings. Patients are seen off the ward in the physiotherapy or occupational therapy departments to review progress towards achieving attainable functional goals.

FUNCTIONAL OUTCOMES AFTER SPINAL CORD REHABILITATION

There is a direct correlation between the neurologic level of injury and the ultimate attained functional ability after rehabilitation. For example, individuals with C6 quadriplegia will usually be able to dress themselves, transfer from one surface to another, (e.g. wheelchair to bed), feed themselves with assistive devices and locomote with a manual wheelchair. However, they often require assistance for the performance of intermittent catheterization and bowel care because of impaired hand function, as the most distal intact muscle group is the wrist extensors. Similarly, individuals with C7 quadriplegia will be able to perform intermittent catherizations, demonstrate greater ease with transfers (including from floor to wheelchair) because of functioning triceps, but will not be able to perform rectal touch technique for bowel care as they do not have a functional first dorsal interosseous muscle (T1) that allows a sweeping movement of the digit to stimulate the rectal mucosa.

TABLE III

MEDICAL PROBLEMS ON SCI UNIT

Respiratory
- Impaired ventilation/infection secondary to altered respiratory mechanics
- Pulmonary embolus
- Ventilator-dependent quadriplegia
- Phrenic nerve pacing for high quadriplegia

Cardiovascular
- Autonomic dysreflexia - acute hypertension arising secondary to a noxious stimulus caudad to the level of injury (usually bladder etiology) - in lesions above T6
- Postural hypotension

Urinary
- Neurogenic bladder - infections/stones/incontinence/retention - detrusor/sphincter dyssynergia with "high pressure" voiding with potential for vesicoureteric reflux and upper-tract damage

Genital
- Erectile dysfunction
- Ejaculatory dysfunction - fertility issues
- Amenorrhea
- Pregnancy

Gastrointestinal
- Neurogenic bowel - incontinence/constipation predominate
- Acid-pepsin disease
- Mesenteric artery syndrome - rare

Skin
- Decubitus lesions

Musculoskeletal
- Heterotopic ossification
- Fractures
- Contractures

Neurologic
- Spasticity
- Chronic pain
- Postraumatic syringomyelia
- Peripheral entrapment neuropathy at "vulnerable" sites e.g.carpal tunnel syndrome

Psychiatric/Social
- Depression
- Marital dysfunction

This correlation is not absolute; it does, however, assist in establishing realistic functional goals for ADLs (activities of daily living), which include feeding, dressing, transfers (tub, toilet, car, etc), personal hygiene, ambulation or locomotion. Thus, in general, amongst persons with complete lesions, those patients with T1 paraplegia

or a lower lesion are independent for all aspects of self-care, including bladder and bowel management and can operate a motor vehicle with hand controls.

TABLE IV

PROBLEM LIST

Functional problems	Nutritional
Mobility	malnutrition/obesity
Wheelchair usage and management	
Transfers	
Negotiation of all surfaces	Psychosocial problems
Ambulation	Adjustment to disability
Self-care(ADLs)	Patient adjustment
Dressing, personal hygiene	Family adjustment/support
Feeding,	Pain
Equipment	Substance abuse
Wheelchair and seating system	Cognitive
Bathing/toileting equipment	R/O assoc'd brain injury
Bed/mattress	
Environmental control unit	Architectural barriers
Assistive devices/splints	home visit/modification
Orthotic/ambulatory aids	
	Sexuality
	feelings/actions
	potency/fertility
Medical problems	
Bladder	
Bowel	
Skin	Finances
Pain	identify sources/liason
Respiratory	
Spasticity	Discharge planning
Other	
	Recreation/avocational

Patients with C7 and C8 quadriplegia generally require assistance for neurogenic bowel management, either to perform rectal touch technique or to insert a suppository to initiate a bowel movement. They are usually independent for all other aspects of self-care.

It is not common for patients with C6 quadriplegia to be able to perform intermittent catheterizations (the preferred mode of management). They may require assistance for some aspects of personal hygiene, feed with assistive devices ("universal cuff") and transfer to level surfaces (sometimes with sliding board). They can often dress themselves completely with use of modified clothing, although it often takes significantly longer to do so. They are able to operate a motor vehicle with hand controls, often using a van because they can drive in via a lift and avoid having to transfer into a vehicle and "pull the chair" in behind them. Although they can propel a manual wheelchair, it is not uncommon for them to acquire an electric wheelchair for improved community access, i.e. longer distances. In the community, these people can often live independently if they receive assistance for bladder and bowel management. Some opt for the less desirable but more practical sphincterotomy and continuous condom drainage to facilitate this and thereby require attendant care for part of a day, rather than four times a day for intermittent catheterization.

Individuals with C5 quadriplegia locomote via an electric wheelchair, require attendant care for dressing, personal hygiene, transfers and usually for feeding. With highly modifed vans, some can operate a motor vehicle. There may be some aspects of these ADLs that they are capable of performing, but not usually enough to achieve independence.

Patients with lesions at C4 are totally dependent for all aspects of their personal care. Using environmental control systems, they can access computers, phones and household appliance controls. Electric wheelchair locomotion is achieved with "sip and puff" controls or controls built into the headrest of the chair triggered by head or neck movements.

Assistive devices and interventions of many types are utilized in our attempts to minimize the disabilities and handicaps of persons with spinal cord injuries. These include environmental-control units, orthoses to improve upper extremity function and potential for ambulation, driving-control units, interfaces to allow keyboard access in high-level quadriplegia, papaverine injections for erectile dysfunction, and velcro closures to facilitate independent dressing.

The occurrence of a traumatic spinal cord injury is a devastating physical and emotional event for the individual and his or her family. Its effects extend into nearly all aspects of life. Loss of ability to walk often greatly affects the patient's self-perception and the way we view them. It is necessary to consider the marked impact of this impairment on day-to-day activities in our attempts to assist these people. □

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Rehabilitation of People with Lower-Limb Amputations

R. Lee Kirby,* MD, FRCPC, John L. Sapp,** MD, FRCPC and Edmund R. Harrison†, MD, FRCPC

Halifax, N.S.

The rehabilitation of people who undergo lower-limb amputations is an interdisciplinary effort involving a number of medical, surgical and allied-health professionals. Ideally, it begins in the preoperative period, intensifies for the first two or three months postoperatively, but then requires only brief, periodic efforts to maintain function and to prevent complications.

In this paper we review the current practice in rehabilitating a person with a lower-limb amputation. The purpose of a program is to assist the person with an amputation regain as much functional independence as possible, and to minimize the impact of the amputation on his or her life. Before discussing management, we will briefly discuss the classification, epidemiology and etiology of lower-limb amputations.

Amputations are classified in terms of the anatomical level at which the amputation has taken place. For instance, an amputation between the knee and ankle is called a "below-knee" (or "transtibial") amputation. An amputation through (or very close to) a joint is called a "disarticulation". A "hemipelvectomy" is when part of the pelvis has been removed. We suggest that eponyms be avoided — although there are over two dozen of them, the Syme's amputation (an ankle disarticulation) is the only one that most physicians remember. Congenital skeletal-limb deficiencies are difficult to classify, but a simple description of what is missing and what is deformed will usually suffice.

Lower-limb amputation is a fairly common problem, with 383 surgical procedures having been performed in Nova Scotia during the 1991-92 fiscal year (Table I), for an incidence of 43 per 100,000 per year. These included amputations of relatively insignificant extent (e.g. loss of a single toe), second amputations on the same limb at a higher level soon after the first, and amputations on patients who died in the perioperative period. Distal amputations are generally more common than proximal ones, and significantly disabling lower-limb amputations are more common than those affecting the upper limbs. In 1991-1992, there were 103 upper-limb amputations in Nova Scotia, 90 of which were at the finger or metacarpal level. Amputation can occur at any age, depending on the cause, but those most commonly affected are the elderly. A recent study at the Nova Scotia Rehabilitation Centre (NSRC) found that the average age of 132 newly referred lower-limb amputees was 67.8 years and the male:female ratio was 3.3:1.[1]

The etiology varies with the age of the person affected. Congenital amputations (e.g. absence of the proximal femur) are those that are present at birth. Physical causes (e.g. accidents and burns), neoplasms (e.g. osteogenic sarcoma) and severe neurological lesions (e.g. flail limb) are the most common reasons for amputation among children and young adults. Infections that lead to amputations may occur at any age. By far the most common etiology is peripheral vascular disease in the elderly, associated with diabetes mellitus about half the time. These etiologies lead to amputation when the patient's health or life are seriously endangered or when pain and dysfunction are sufficient to warrant amputation.

TABLE I

NUMBER AND TYPE OF LOWER-LIMB AMPUTATIONS IN NOVA SCOTIA, 1991-1992

Level	N	%
toes, metatarsals	126	32.9
transmetatarsal	40	10.4
Symes, hindfoot	11	2.9
below-knee	75	19.6
knee disarticulation	10	2.6
above-knee	116	30.3
hip disarticulation	2	0.5
hemipelvectomy	1	0.3
other	2	0.5
Total	383	100

MANAGEMENT

Ideally, rehabilitation should begin preoperatively and continue until the patient's improvement has plateaued and the maintenance phase begins. The management can be conveniently presented as a number of issues or problems that commonly require intervention.

Surgery

As nonsurgeons, we approach this section with trepidation, but pass on to the reader what observations we can make, and comments relayed to us by the vascular, orthopedic and plastic surgeons with whom we work. The "site of election" (the level of amputation) is primarily based upon the principle of "save all available length" but there are mitigating considerations – for instance, in patients with peripheral vascular disease a longer stump may be less likely to heal well. Furthermore, amputations near a joint limit the rehabilitation team's options when replacing that joint with an prosthetic one.

*Professor, ** Associate Professor, † Assistant Professor, Division of Physical Medicine and Rehabilitation, Department of Medicine, Dalhousie University, Halifax.

Correspondence: Dr. R.L. Kirby, Nova Scotia Rehabilitation Centre, 1341 Summer Street, Halifax, N.S., B3H 4K4

The choice of the type of closure and the length of skin flaps used during surgery is based on the neurovascular supply; for example, in most below-knee amputations the posterior skin flap is longer than the anterior one. Hemostasis is important; bone wax may be used to decrease oozing from the medullary cavity of the bone. Rough edges are removed from the bone, which may also be trimmed to facilitate prosthetic fitting (e.g. in a below-knee amputation, the anterior surface of the tibia is bevelled and the fibula is amputated slightly shorter than the tibia). Muscles are drawn around the bone and sewn to periosteum or the opposing muscles (a myoplasty).

There have been some interesting advances in surgical technique, a discussion of which is beyond the scope of this paper. Some examples are reimplantation of severed limbs, transplantation from one part of the body to another (e.g. a great toe to replace a thumb) and creative reconstructive surgery (e.g. in patients affected by a congenital deficiency or tumor) in order to optimize length, shape and function.

Minimizing postoperative complications

When there is time, factors that delay healing – poor control of diabetes, malnutrition, anemia and infection – should be dealt with preoperatively (and closely monitored in the postoperative period as well).[2] There is some evidence that the prophylactic use of antibiotics decreases the likelihood of wound infection and the need for reamputation.[3] Early mobilization at the wheelchair and walker level minimizes the risk of such complications as deep-vein thrombosis, pneumonia, contractures and pressure sores.[4]

Inpatient versus outpatient rehabilitation

Patients who are otherwise in good health, who are able to care for themselves and who do not require the services of more than one or two of the allied-health professionals, may be managed as outpatients, residing at home or in a nearby hostel. Others should be managed on an inpatient rehabilitation service – the 115 NSRC admissions due to amputation between April 1991 and March 1992 constituted the second largest diagnostic group (17.4% of the total). If practical, inpatients are encouraged to go home on weekends to practice, in their own setting, what they have learned and to determine what additional equipment, architectural modifications or personal support is needed.

The rehabilitation team

The team includes the patient, the family, the physicians and surgeons involved, and members of the allied-health professions. Allied-health disciplines commonly involved in a comprehensive rehabilitation program include nursing, prosthetics, physiotherapy, occupational therapy, social work, dietetics, recreational therapy, psychology, vocational counselling and pastoral care.

Psychological adjustment

The amputation of a limb may elicit a major grief reaction or a reactive depression. The adjustment to the change in body image and diminished function should begin early; some patients appreciate the opportunity to discuss their concerns with someone who has successfully been through the process before.

Vascular risk factors

For patients whose amputations were due to vascular disease, it is wise to include secondary prevention of further vascular problems by smoking cessation, foot care, regular exercise, and control of diabetes, hypertension, hyperlipidemia and obesity.

Phantom sensation and pain

Except among congenital amputees and young children, it is normal for patients who have had an amputation to continue to feel the presence of the amputated limb, the well-known "phantom" sensation. Most patients who experience phantom pain require only stump wrapping, the use of a prosthesis, exercise and distraction in the course of a comprehensive program, but a few will require a more intensive program for the management of their chronic pain.[5] It has been suggested that eliminating pain preoperatively (e.g. by the use of an epidural anesthetic before and during surgery) may decrease the likelihood of phantom pain later.[6]

Stump pain

Stump pain can be caused by adhesions between the skin and underlying bone (that become painfully stretched with the shear forces that occur during prosthetic use), neuromas (that are irritated by direct pressure or indirect forces), ischemia, referred pain (e.g. from the lumbar spine) or infection of soft tissue or bone.

Deconditioning

Most patients who undergo an amputation have been ill for some time and require both general reconditioning and specific conditioning of muscles of particular importance to aided locomotion (e.g. the crutch muscles of the arms and the antigravity muscles of the legs).

Contractures

Unless a preventive stretching program is instituted, it is common for soft-tissue contractures to begin preoperatively, because patients usually keep the limb in the position that is most comfortable (often dangling over the side of the bed) and spend a great deal of time sitting in bed or a wheelchair. For patients with incipient knee-flexion contractures postoperatively, a retractable stump board on the wheelchair may be helpful.[7]

If a contracture develops, patients commonly experience problems due to the altered biomechanics (e.g. quadriceps fatigue due to flexion contractures at the

knee) or due to compensatory changes in their posture (e.g. lordotic low-back pain due to a flexion contracture at the hip). Splinting, stretching and periods of traction may be necessary to correct the problem. If a contracture cannot be corrected, it may be necessary to accommodate the problem by altering the alignment of the prosthesis (sometimes dramatically) or by using ambulation aids beyond the initial training period.

Wound protection

Wound protection is important during the first few months after the amputation. It happens all too often that a patient, confused by hypnotics and analgesics, gets up at night to use the toilet, tries to stride out onto the phantom limb that he or she forgets is no longer there, and falls, damaging the wound and sometimes even causing dehiscence of it. To prevent this, some surgeons apply a "rigid dressing" of plaster in the operating room whereas others use a bulky soft dressing.[8] Later, the occupational therapist can fabricate a stump protector (or "cap") of a lightweight thermoplastic material.

Stump shaping

Stump shrinkage and edema control should also begin early and continue for many months, even after prosthetic fitting is well under way. In the early postoperative period, elastic bandaging is the most commonly used technique, supplemented and later replaced by elasticised garments ("stump shrinkers").[9] Patients who are subject to edema (e.g. due to heart or renal failure) may need to use stump shrinkers on a long-term basis to limit diurnal fluctuations in stump size.

The prosthesis

Although some centres still use "immediate" postoperative prosthetic fitting, with a pylon and foot being attached to a plaster wound dressing in the operating room, generally today prosthetic fitting and training begins "early". Early means "as soon as it seems prudent" (based on wound healing and the patient's other problems) – this may vary from beginning one or two days postoperatively (using an inflatable temporary prosthesis for a young patient who has had an elective amputation) to many months later (for an elderly and ill patient with a wound that is infected and slow to heal).[10] Occasionally, when there are no other limiting factors, prosthetic rehabilitation can begin before wound healing, by the use of a "bypass prosthesis" that avoids any contact with the wound.

In the past decade there have been significant advances in prosthetic materials and in the design options available.[11] There are many combinations and permutations of suspension systems (how the prosthesis is held on), sockets (the section in which the stump fits), joints, structural sections (the "skeleton") and feet. The prescription of the prosthesis is best with input from several members of the team and is based upon the person's individual requirements and lifestyle. To provide a custom-fitted socket, the prosthetist makes a plaster-of-paris impression of the stump. The fabrication of the socket and alignment with the remainder of the limb components usually takes up to two weeks, then patients are trained in the use and care of the prosthesis.

Of 103 graduates of the NSRC amputee program who responded to a follow-up questionnaire, 65.5% reported that they wore their protheses for greater than 9 hours each day, 11.5% for about half of the day, 6.9% occasionally and 16.1% that they were not using their prostheses.[1]

Gait training

Learning to walk with crutches or a walker preoperatively helps to keep patients mobile and eases their transition to this form of ambulation later. Learning to walk with a prosthesis usually begins in the parallel bars (Figure 1), progressing to a walker or canes, and later to different surfaces (carpet, gravel) and challenges (inclines and stairs). Of the NSRC graduates, 71.4% continued after discharge to use an ambulation aid, at least outdoors.[1]

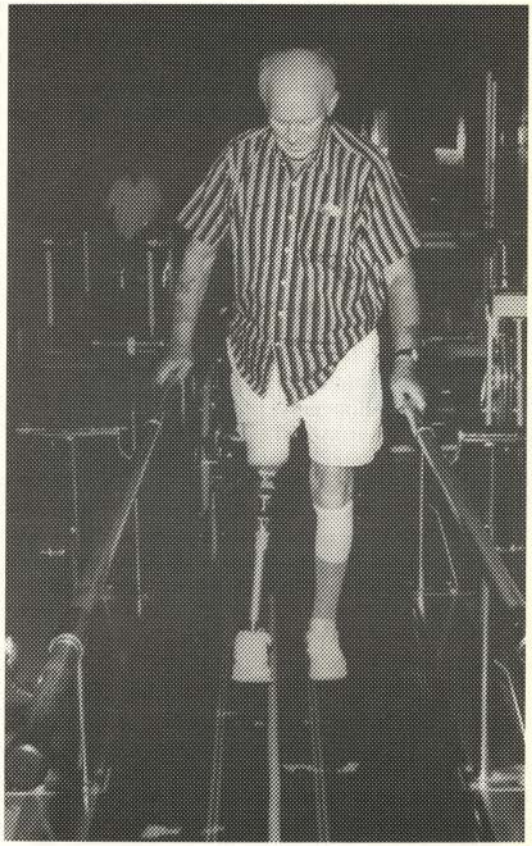

Figure 1

An above-knee amputee learning to walk with parallel bars as ambulation aids. Note that the prosthesis is in an unfinished state, to simplify changes in its alignment and components.

In another study on 43 newly referred lower-limb amputees,[12] we evaluated their locomotion function on admission (without a prosthesis), at discharge and at follow-up (about three months later), measuring their ability to walk on the level, over gravel and a carpet, and to negotiate stairs, a curb, a bus-height step and a narrow doorway.[13] On admission, the mean (SD) ability score (AS) was 63.8 (16.0)% and the speed score (SS) was 35.0 (24.7)%; at discharge the AS had increased to 76.1 (10.6)% ($p < 0.0001$) but SS was unchanged at 33.5 (20.4)%; and at follow-up, the AS was 82.6 (12.7)% ($p < 0.01$) and the SS 40.3 (23.5)% ($p < 0.0001$), demonstrating that speed improves more slowly than ability.

Footwear

For patients who have had a partial-foot amputation at the transmetatarsal level or lower, customized footwear modifications are often necessary. For patients who have had more proximal amputations, the shoe needs to be spacious enough to allow a prosthetic foot to be placed in it. (Spacious shoes, with an extra-depth toebox are also a good idea for the other foot.) Patients are encouraged to be consistent in the height of the heels on the different shoes they wear, so that the prosthetic alignment does not need to be changed (for minor changes in heel height, a spacer pad can be provided to go inside shoes with lower heels).

Wheelchair

For unilateral amputees, a wheelchair is often advisable for longer distances or when the prosthesis is not being used (e.g. when it is under repair). For people who have lost both legs, a wheelchair is usually needed to complement (and sometimes replace) walking. Of the NSRC graduates, 18.6% used a wheelchair as their only means of mobilization and 9.3% as an occasional means.[1]

In the early postoperative period, we recommend that patients rent or borrow a wheelchair rather than buy one — it is not until somewhat later during the rehabilitation program that it becomes more clearly apparent which wheelchair options are most appropriate.[14] Patients using wheelchairs may require modifications of their home to accommodate the wheelchair, such as by installing a ramp, rearranging furniture, removing carpet or widening doorways.

Driving

People who were drivers before their amputations should notify their insurance company and the Registry of Motor Vehicles that they have had an amputation, and they should not drive until they have been evaluated and provided with any adaptations necessary to ensure that they can operate the vehicle safely. A repeat driving test may be necessary.

Activities of daily living

To be able to successfully function at home (whether or not a prosthesis is prescribed), patients need to practice everyday activities such as homemaking, feeding, dressing, personal hygiene, driving and transfers. Adaptive equipment may be prescribed to make these activities easier (e.g. a raised toilet seat, bath bench, grab rails).

The social setting

Attention needs to be paid to each individual's environment to ensure that architectural barriers are identified and dealt with, and that any needed personal support is in place. Community resources may need to be mobilized to deal with problems with housing, transportation, home support, counselling, income supplementation, and funding of equipment (in Nova Scotia, prostheses are paid for by the provincial health-care system [MSI], but other equipment is not).

Follow up

The average time from surgery to the end of the rehabilitation program is about 4 months for a lower-limb amputation of significant extent (i.e. at the ankle level or above), of which the formal rehabilitation program constitutes the final 5-6 weeks.[1] After completing the initial rehabilitation program, patients usually need to be reviewed at intervals of three and six months, and yearly thereafter. They are free to contact the prosthetist directly if they cannot adjust the prosthesis fit by changing the number of socks, if they wish to change the heel height of footwear being worn, or if a part of the prosthesis is worn out or broken. However, for any more significant problems (e.g. sores, major changes in the prosthetic prescription), patients should be seen in the interdisciplinary team setting of the amputee clinic.

CONCLUSION

In closing, we would like to dispel two common myths about the rehabilitation of people with amputations. The first is that some amputees are too old to use an artificial limb. Although the feasibility of fitting older patients with prostheses has been questioned by some, we have found that age alone is not a barrier to the successful use of a prosthesis: unilateral amputees in their 90s are routinely successful, and bilateral amputees in their 70s and 80s are also commonly able to use artificial limbs for short distances.[1,15] The second common myth is that patients who do not receive a prosthesis do not need rehabilitation. Those for whom the use of a prosthesis is not appropriate for walking may benefit from the use of one for cosmesis, transfers and sitting balance.[16] Amputees who choose not to use artificial limbs at all (e.g. the bilateral short above-knee amputee) can benefit nevertheless from the nonprosthetic rehabilitation process to regain optimal independence at the wheelchair level. □

Continued on page 150.

Prevention and Principles of Treatment of Chronic Pain Syndrome in Soft Tissue Injuries

J.J.P. Patil,* MBBS, FRCPC

Halifax, N.S.

Seemingly, there has been an epidemic of cases of chronic pain syndrome related to soft tissue injury all over North America. Often, chronic pain is created by the social and health care system, including the physicians who treat these patients, as they often do not understand the difference between chronic and acute pain. Chronic pain has three major dimensions: the sensory dimension, the affective dimension and the cognitive evaluative dimension. Patients with chronic pain often dramatize their pain; suffer from depression, dysfunction and deprivation of sleep; and become overly dependant on others to control their pain. There are also certain predisposing factors to chronic pain which one must remember when dealing with any patient with pain. Physicians and therapists have to learn how to recognize the early signs of chronic pain syndrome and how to manage it.

There has been a virtual epidemic of cases of chronic pain syndrome related to soft tissue injury all over North America. As a result, some compensation agencies, such as insurance companies and workers' compensation boards spend up to four times as much of their budgets on persons with chronic pain as on all other injured persons. Along with this large increase in the number of chronic pain patients, there has been a correspondingly greater mushrooming in the number of pain clinics all over North America than elsewhere.

Since the 1965 landmark paper by Malzack and Wall in which they first proposed the gate control theory of pain, we have had a better understanding of chronic pain and its intricate, complex nature. [3]

Chronic pain syndrome is defined as a disabling pain that has existed anywhere from several to six months and beyond, and which exceeds the normal recovery period. In other words, the pain persists even after the healing process—the normal period of which has been established for various type of injuries by various textbooks of medicine and by workers' compensation boards.

We physicians often tend to treat chronic pain as we treat acute pain, which responds very well to narcotic painkillers. However, in cases of chronic pain, these do not help very much, and there is always a danger that using larger and larger amounts in patients with chronic pain due to soft tissue injury, can lead eventually to addiction and depression and often constipation.

*Assistant Professor, Division of Physical Medicine and Rehabilitation, Department of Medicine, Dalhousie University, Halifax.

Correspondence: Dr. J.J.P. Patil, Nova Scotia Rehabilitation Centre, 1341 Summer St., Halifax, N.S. B3H 4K4

There are various types of chronic pain, including causalgic pain, neuralgic pain, phantom pain, post-traumatic stress disorders, and fibrositis syndrome, but this paper will deal mainly with chronic pain related to soft tissue injury.

Over the years, Melzack and Wall have modified their pain paradigm and their concept of chronic pain.[3,4] It has become very clear that the total human pain experience generally has three major dimensions: the *sensory discriminative dimension*, which we all understand better, which generally is transmitted by the known pain pathways through the peripheral nerves, along the spinothalamic tract and then to the sensory cortex; the *affective dimension*, the mood dimension of pain, which modulates, amplifies, or even suppresses one's pain depending upon one's mood; and the *cognitive dimension*, where one experiences the mental torment and suffering as a result of pain. (Fig 1).[1]

Pain Dimensions

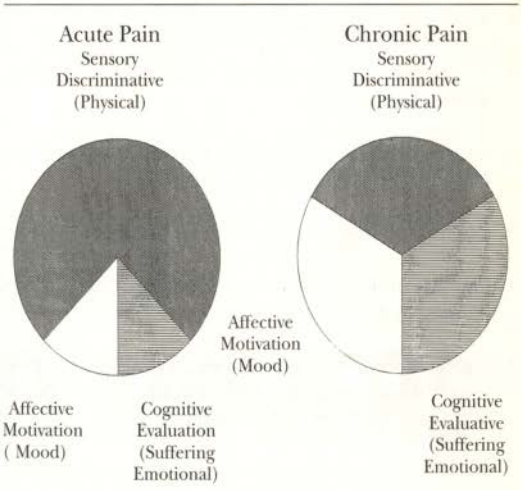

Figure 1

(Adapted from E.R. Chaplin)

Herein, perhaps, lies the most important difference between acute pain and chronic pain. On the one hand, the predominant dimension that one experiences in acute pain is the sensory discriminative dimension, which is amenable to treatment with narcotic analgesics, whereas the contributions of the affective and cognitive dimensions – perhaps in the form of anxiety, perhaps in the form of suffering – are much smaller. On the other hand,

a large component of chronic pain comes from the affective and cognitive dimensions, and much less of the pain experienced is from the sensory discriminative dimension. Hence, it would be reasonable that we physicians not concentrate all our efforts on treating the sensory discrimative dimension with narcotic painkillers and other modalities, but concentrate more on the affective and cognitive dimensions, which often can benefit from the appropriate use of tricyclic antidepressants, psychotherapy, motivational therapy and persuading the patient to be mentally and physically more active.

Many of the chronic pain programs across North America have begun to use this approach, which has been found to be far superior than the old approach of narcotic painkillers in the form of pain injections, pills and surgery. However, patients who have chronic pain tend to "doctor shop". Often, even after seeing half a dozen different specialists, they are not satisfied that their pain cannot be totally relieved. While admitting that narcotic painkillers do not help much and do not appear to be the final solution to the problem, many continue taking them, having frequently not been offered any alternate form of treatment to control the chronic pain. Some of them still continue to believe that their chronic pain can be managed with narcotic painkillers, just as acute pain is.

Clinically, it has been found that chronic pain presents itself frequently with six *D*s:

- A *duration* of months or years;
- A tendency for the patient to *dramatize* the pain, almost involuntarily;
- Overuse of *drugs* especially the narcotic drugs and this includes the so-called street drugs;
- Feelings of *depression, dysphoria,* and *despair;*
- Patients undergoing *disuse* in their daily life and becoming *dysfunctional* in society; and
- Sleep *deprivation,* as patients usually fall asleep quite easily but tend to wake through the night, so their stage-four sleep is greatly curtailed.

In our experience, if patients who have had a soft tissue injury continue to complain of symptoms, even beyond the point of healing, and exhibit at least three of the above clinical features, the likelihood of them travelling down the path of chronic pain is very high, and primary care physicians should take action immediately. Of course, taking appropriate action may require spending some time with the patient and not just writing a prescription for pills and sending the patient away. This may be a short-term solution, but in the long term, these patients will return more and more frequently.

In dealing with the duration of disability, it is important not to delay sending the patient back to work on a part-time basis, or to a lighter job, after talking to his or her employer or supervisor. Just writing a small note saying that the patient can go back to work is often not adequate, as the patient would very likely be put back to regular heavy work, which may further aggravate the pain symptoms.

Physicians should ignore pain behaviour and dramatization and should encourage increased functional capacity, such as gradually increasing the sitting, standing and walking tolerance. It will be important to give the patient some guidelines to follow. Most patients after being off work for a long period of time tend to put on excessive weight and become deconditioned physically. One also should try to reward their positive behaviour – behaviours that exhibit an attempt to do more physically – rather than their pain behaviours. It might be important to talk to the spouse and tell the spouse to do the same.

As far as narcotic painkillers are concerned, the patient should be told of the drugs' side effects (constipation, depression and addiction), and these drugs should be gradually withdrawn, with pain control being obtained by physical modalities and exercise that the patient can carry out at home. Sending patients for prolonged courses of so-called shake-and-bake physiotherapy, where they receive only modalities of heat, ice, TENS, etc., but who do not receive a regimen of therapeutic exercises and who do not receive education regarding how to modify their activities and reduce pain will, in the long run, be useless for many chronic pain patients. Indeed, continuing this type of physiotherapy for months and months at a time tends to lead chronic pain patients to develop an attitude of dependence on others to control their pain, rather than learning how to control it themselves using the information and advice that they have received from their physicians, therapists and other allied health professionals. Indeed, it has been observed that, by making patients more dependent on others for control of their pain and making them more passive in dealing with their own pain, along with excessive repeated investigation and prolonged periods of ineffective treatments – such as nerve blocks, trigger point injections and physiotherapy – can contribute to chronic pain.

Despair, depression, dysphoria and disturbed sleep may be very effectively treated with a small dose of a tricyclic antidepressant (Amitriptyline® or doxepin) which acts on the serotinin pathway. When using this type of medication, one must prescribe it in small doses at night time, which could be gradually increased. (Most patients do not have to exceed more than 75 mg of doxepin or Amitriptylline® at night). It is preferable not to use these medications during the day because of the obvious sedative effect may reduce the patient's physical activities. Some counselling or psychotherapy will also be helpful. Referral to a vocational counsellor for those patients who may not be able to return to their original work, would seem reasonable. The longer one stays off work, the less one's chances are of returning, and, after a year or two off work, the probability of returning is close to zero.

As far as the patient's disuse and dysfunction is concerned, it would be important to put the patient on a regimen of a walking or swimming program, or some other form of light physical activity, urging them to increase their activity level gradually.

The literature also suggests that patients who have suffered from chronic pain syndrome have, in the past, frequently suffered from anxiety, panic attacks, depression, child abuse, or sexual abuse, and that many times these factors can also be playing a role in their chronic pain behaviour. In such patients, psychiatric consultation, treatment and counselling is very important. Patients who have undergone some form of abuse as youngsters are thought to have learned that the only way they can escape from unpleasantness at work, at home or elsewhere, is to suffer from pain. (Although Merskey and coworkers have challenged this concept of chronic pain, their study compared non-pain psychiatric patients with chronic pain patients rather than with the normal population).[5] It is observed also that factors such as doctor shopping, not having a job to return to, desirable job modification, stress, substance abuse, multiple medical problems, English as a second language, cultural adjustment, age, female gender, lack of education, previous claims with difficult rehabilitation, inconsist examination findings, missing appointments, no response to treatment, pre-existing medical condition, and third-party litigation, all predispose one to delayed recovery from injury and often to chronic pain.[2]

The family doctor is in the best position to detect the early stages of chronic pain syndrome and to prevent it from becoming more complicated. If the measures outlined in this paper are taken by the family doctor in the very early stages, we can certainly prevent chronic pain syndrome and shorten the duration of chronic pain. If all of the above measures fail, patients who have accepted that their pain has been investigated to the maximum, that nobody can offer them a permanent cure for their pain, and that the only way to deal with the pain, is to learn to live with it, then referral to the inpatient pain program at the Nova Scotia Rehabilitation Centre or an inpatient/outpatient pain program at other centres where a behavioral/psychological approach dealing with chronic pain is used. Hard-core, difficult chronic pain patients are best treated in an inpatient program. □

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Assessing and Treating the Psychiatric Aspects of HIV Related Disorders in General Practice

A REVIEW

Bharat B. Chawla,* MB, BS and Patrick Flynn,** MD, FRCPC

Halifax, N.S.

There is probably no disease more devastating than AIDS (Acquired Immune Deficiency Syndrome). The diagnosis is seen as a "death sentence" and the course of the illness is often associated with social stigmatization, social and emotional isolation, and prolonged physical and cognitive deterioration. Psychiatric and neuropsychiatric complications are sometimes present as well.

The AIDS epidemic is a major challenge for the health and mental health professionals. All health care workers, including primary care physicians, need to be knowledgeable about the psychiatric and neuropsychiatric aspects of HIV disease in order to provide comprehensive care to persons so affected.

This article will outline the case histories of three patients with HIV infection who were recently treated as inpatients at the Victoria General Hospital and Camp Hill Medical Centre and who, in the view of the treating physicians, required psychiatric help. With these clinical vignettes as a basis, the article will describe some of the common psychiatric concomitants and sequelae of HIV infection and AIDS.

EPIDEMIOLOGY

AIDS was first reported in 1981. In 1984, the causative agent was identified as a retrovirus, Human Immunodeficiency virus (HIV-1). A second retrovirus now known as HIV-2, found primarily in Africa, is known to cause a similar illness. The HIV-1 represents the commonest form of infection in North America and United States.[1]

The evidence suggests that HIV transmission occurs only through body fluids and usually involves sexual contact, transfusion of body products, use of syringes contaminated with HIV, or transmission from an infected mother to her newborn.[2] The virus does not appear to be easily transmitted through casual contact with an infected individual.

The high risk groups in decreasing order of prevalence include homosexual/bisexual men with multiple sexual partners, intravenous drug abusers, recipients of blood or blood products, heterosexual partners of high risk individuals and persons from an endemic area. However, the majority of the AIDS cases belong to first two groups (80-90%)[3] but recent reports indicate changes in AIDS incidence trends suggesting increasing number of infected women and children.[4]

As of July 1992, over half million cases of AIDS have been reported to WHO (World Health Organization) from 168 countries but the actual number of adult cases of AIDS are estimated to be about 1.7 million. In Canada, as of January 1993, there have been 7282 reported cases of AIDS with 4685 deaths. The figures in Nova Scotia are 182 cases with AIDS with 73 deaths (personal communication).

The number of AIDS cases represent only a small proportion of HIV infection. The progression of HIV infection to AIDS is 20-30% after 5 years. The diagnosis of AIDS is not applied unless the presence of a disease indicative of immune deficiency is established.

Clinical manifestation of HIV infection varies. Acute viral infection, chronic persistent viral infection with or without constitutional symptoms and 'full blown' AIDS have been described. The detailed account of these symptoms is beyond the scope of this article and can be found elsewhere.[5]

The psychiatric complaints of HIV infection are numerous and include the adjustment disorders (often of catastrophic proportions when the patient is informed of the diagnosis), as well as the functional and organic mental disorders resulting from disturbances of functions in the central and peripheral nervous system. Table I summarizes the more commonly observed psychiatric disorders.

TABLE I

PSYCHIATRIC COMPLICATIONS OF PATIENTS WITH HIV INFECTION/AIDS

Diagnosis	Percent of Patients Affected
Adjustment Disorders	65*
Major Depression	15*
Generalized Anxiety Disorder	10*
Organic Mental Disorder	30* (Early Stage) 70* (Late Stage)
Substance Abuse	30*
Mania / Psychosis	**
Psychiatric symptoms in those without HIV infection	**

* These figures represent an approximation of the figures suggested in different epidemiological studies.

** Several case reports but exact prevalence unknown.

*Resident in Psychiatry, Dalhousie University, Halifax, N.S.

**Professor of Psychiatry, Dalhousie University, and Director, Consultation service, Victoria General Hospital, Halifax, N.S.

HIV TESTING

The screening test to detect antibodies to HIV became commercially available in 1985. ELISA (Enzyme linked immunosorbent assay) is usually the initial screening instrument employed to detect antibodies to HIV. Positive ELISA results, according to a standard protocol, are checked with a repeat test using the same specimen. Repeatedly positive ELISA results are followed by Westron blot assay to detect antibodies to the individual viral antigen.

HIV testing should always have pre and post test counselling. Before the test is performed, one should assess the risk of infection and level of social support. The physician may also emphasize 'risk education' and safer sex practice. The limitations of confidentiality of test results should also be explained. In post test counselling, disclosure of the test result should always be done in person by a physician and the clinical importance of the result should be explained in detail.

Case 1

A 42-year-old single, white, unemployed male patient who had been HIV positive for the previous 5 years. He presented to the Camp Hill Medical Centre (Infirmary Site) following an overdose of benzodiazepine and alcohol with an intent to end his life. He reported difficulties with his homosexual lover for the previous few months which made him "sad, upset and depressed". The suicidal attempt was precipitated by his lover's visit to his parents, away from the town, leaving him (patient) "alone and vulnerable". He started drinking alcohol and when he had consumed about 40 oz, impulsively took 40 tablets of oxazepam (Serax®) 15mg each in order to end his life.

He denied feelings of helplessness, hopelessness or worthlessness and any neurovegetative features suggesting a depression.

He had no stigmata of AIDS.

He had a history of two previous suicidal attempts, one after the diagnosis of HIV positivity was made and another in his childhood. He also had problems with alcohol abuse in past.

At the time of psychiatric assessment he was still voicing suicidal thoughts and was finding it difficult to go home and cope with being alone since his lover was still out of town. He was admitted to the psychiatric unit of the Victoria General Hospital with the diagnosis of Adjustment Disorder with depressed mood. There was some clinical evidence to diagnose an underlying personality disorder with dependent, immature and passive-aggressive traits.

He was given supportive psychotherapy during his brief hospitalization, allowed to ventilate his anger and cautioned against using alcohol especially in combination with benzodiazepines.

He responded well and was discharged home with an appointment to follow with a psychiatrist as an outpatient.

Case 2

A 31-year-old, white, homosexual male who had been HIV positive during the past 4 years. He was admitted to the Victoria General Hospital with a 6-week history of diarrhea, nausea and vomiting not responding to symptomatic and supportive treatment. He had two courses of appropriate antibiotics with no significant improvement. Repeated stool culture showed growth of the protozoa cryptosporidium. He was diagnosed as having AIDS because of the presence of opportunistic infection.

At the time of psychiatric assessment, he reported feeling sad, depressed, hopeless, helpless and worthless. He also admitted to some neurovegetative features including decreased appetite and insomnia. He had vague suicidal ideation but no active plans. He had no psychotic features and no evidence of any cognitive deficit on sensorial examination.

There was no past psychiatric history.

The initial diagnostic impression was "Adjustment reaction with depressive symptoms". He was given brief psychotherapy with some improvement. Because his depressive symptoms continued he was started on a 10 mg dose of desipramine (tricyclic antidepressant). He was also started on an investigational drug for his opportunistic infection.

He showed a marked improvement of emotional and physical symptoms within a few days. His emotional improvement was likely due to the combination of psychotherapy, the desipramine and an improvement in physical symptoms.

He was discharged home to continue the antidepressant and follow up with a psychiatrist as an outpatient.

Case 3

A 33-year-old, white, homosexual male who had been HIV positive for 3 years. He presented to the Emergency room of the Victoria General Hospital with a 2-month history of acting "very strange" and drinking "much more" than usual. On psychiatric assessment he was floridly psychotic with persecutory delusions that his phone was being tapped and people were going to kill him and his family. He denied any feelings of depression and there were no neurovegetative features of depression. He denied abusing any drugs other than alcohol which was also confirmed by his friend who accompanied him.

He was admitted to the psychiatric unit of Camp Hill Medical Centre.

Physical examination revealed no significant abnormalities other than generalized lymphadenopathy. Liver function test results were elevated consistent with acute alcohol abuse. Other laboratory investigations were normal including x-ray chest, EEG and urine for toxic screen.

He had no stigmata of AIDS.

He had no past psychiatric history.

The psychiatric diagnosis was Acute Psychotic episode NYD, probably related to HIV infection.

He was started on flupenthixol 1 mg TiD (Fluanxol, a thioxanthene). lorazepam 0.5 mg BiD and 2 mg qHS and

thiamine 100 mg OD. He developed severe extrapyramidal side effects and was started on benztropine (Cogentin®) 2 mg bid. He was also given prn Haldol® for episodes of extreme agitation.

His symptoms improved and he was discharged home with plan to follow up at the Infectious Disease (I.D.) clinic as well as at the psychiatric out-patient clinic.

ADJUSTMENT DISORDERS

Adjustment disorders are the most common and often the immediate psychiatric reactions provoked by HIV infection and its consequences. They are also likely to reoccur during the course of the illness.

They are seen to some extent in almost all the cases and disorders requiring psychiatric attention are seen in about two-thirds of patients with HIV infection.[6,7] The manifestations and severity are dependent on the premorbid personality, maturity, range of experience and skills, integrity of their social network and the prior mental health of the infected individual. The reactions of patients with the diagnosis of HIV positivity or AIDS are similar to those reported in patients with terminal illness for example, cancer. There is, however, the additional burden of stigmatization and social isolation because of society's mis-perceived fear of contagion with HIV infection.

The acute reaction following the diagnosis includes some of the following: shock, emotional numbing, disbelief, feelings of guilt and self-blame, denial, acute anxiety, anger, social avoidance and so on. Some patients develop transitory depression or show serious withdrawal behavior while others have despair and suicidal preoccupations. Pathological levels of denial are also found as part of the adjustment process. Suicide risk is considered to be high shortly after the diagnosis is confirmed.

Results of a Canadian Needs Assessment National Survey (CNANS) of people with HIV found that their most distressing problems included uncertainty about the future, not being able to realize life goals, getting sick, feeling helpless, not being able to care for themselves, feeling angry, financial difficulties, feeling lonely, problems in sex life, feeling anger and frustration at the health system and the side effects of medications.

Management

Early diagnosis and intervention is very important for the following reasons:

i) Intensity of distress associated with the diagnosis may lead to considerable acute and chronic psychosocial morbidity (including self-destructive activities) and interference with the patient's interpersonal relationships.

ii) Psychosocial morbidity will substantially affect the subsequent "coping response" of the patient faced with a future life with HIV disease and its consequences.

iii) It may be possible to ameliorate the condition that would otherwise lead to eventual serious psychiatric/psychological disturbances that might require psychiatric hospitalization.

The adjustment process is helped by having available:

i) Competent and compassionate clinicians who understand and are comfortable with the issues involved.

ii) Peer support services to provide models for coping and to give information and emotional support.

iii) Various types of individual and group counselling to include ventilation, emotional support for the patient and, where appropriate, for the family and friends.

An adjustment disorder with depressed and anxious mood is the most common psychiatric diagnosis.[6,7] In some patients depressive symptoms predominate whereas in others, anxiety symptoms are prominent. When anxiety and depressive symptoms increase in spite of appropriate psychotherapy then anxiolytics and/or antidepressants can be helpful. Two important points to remember when using drugs in these cases are that modest doses are used (because of poor tolerance to side effects) and the prescribed quantities are small because of the risk of overdose.

Lorazepam (Ativan®) 0.25 mg - 0.5 mg bid/tid for patients with anxiety and alprazolam (Xanax)® 0.25 mg - 0.5 mg tid for patients with mixed anxiety and depression adjustment reaction, are reported to be safe and beneficial.[8]

An adjustment disorder with depressive symptoms usually responds well to supportive psychotherapy, as illustrated in Case 1, and pharmacological intervention is seldom indicated or necessary. Sometimes a low dose of a benzodiazepine hypnotic is necessary for concurrent insomnia with anxiety and, in some cases, the use of antidepressants may be helpful, as illustrated in Case2.

MOOD (AFFECTIVE) DISORDER: DEPRESSION, MANIA

Depression

It is obvious that much sadness typically accompanies the knowledge of being HIV positive. Individuals who are diagnosed with HIV infection undergo a process analogous to grief as they adjust to live with a terminal illness. Major depression with the standard characteristic features develops in about 20% of patients.[6] The patients who react in this way may be known to have a history of unipolar or bipolar illness or the HIV infection may have precipitated an attack in someone with a genetic tendency for affective disorder. Further, a depression may be "reactive" to having the illness and its consequences or it may be secondary to the drug treatment of the disease.

Those with the symptoms of classical depression may complain of anhedonia, sadness, pessimism, low self-esteem, diminished interest, a sense of helplessness,

hopelessness, worthlessness and uselessness. They often express intense feelings of guilt over real or imagined shortcomings and may feel suspicious of others (including ideas of reference or delusional beliefs).

As with other medically ill patients, differentiating symptoms attributable to progression of HIV illness from symptoms of depression may be difficult. Weight loss and low energy, included in the DSM-III-R criteria for major depression, are included as features of the 'AIDS wasting syndrome' and are also symptoms of general physical debilitation.

Symptoms of early 'AIDS dementia complex' (see later) especially apathy and motor/cognitive slowing should not be misdiagnosed as depression. Patients are usually able to distinguish between a depressed mood and apathy when careful inquiry is made.

Management

The most important step in the management of these patients is establishing the diagnosis of a major depression before prescribing any treatment. Further, patients who are actively suicidal may need immediate psychiatric hospitalization.

Once the diagnosis is established and a decision to start an antidepressant is made, the choice of drug may depend upon a previous positive response, a positive family response to a specific antidepressant or upon a patient's known sensitivity to psychotropic drugs. As these patients are very sensitive to side effects and are at high risk of developing 'anticholinergic induced delirium', an antidepressant with a low anticholinergic profile eg. desipramine or nortriptyline might be used. As with cancer patients, many in this group respond to antidepressants more rapidly and at lower doses than ordinary patients.[9] Therefore, one-third or one-half of the average therapeutic dose may only be required. One might initiate treatment for example with desipramine 10 mg daily and increase the dose gradually to 75 mg/day. Plasma level monitoring is very useful and is often necessary in establishing the therapeutic dose in those patients because of erratic absorption secondary to HIV related gastrointestinal disturbances.

In 'mild to moderately' depressed patients with HIV infection, Ritalin® (methylphenidate) is often rapidly effective and is generally well tolerated. It is best to start with 5 mg or 10 mg in the morning and at noon. If necessary, dosage increments up to a maximum of 20 mg bid might be tried.[10,11] In some patients a triazolobenzodiazepine like alprazolam (Xanax®) may be useful as an antidepressant. There is some evidence that newer SRI antidepressant drugs which specifically influence serotonin eg. fluvoxamine, and which have less anticholinergic action might be of benefit in refractory cases. Other treatment approaches including MAOIs, ECT etc are used if the above treatments are not helpful.

In addition, tricyclic antidepressants may also be helpful in the treatment of HIV related pain conditions and may be used in combination with opiate/non-opiate analgesics. So far there is no reported evidence of any toxicity when antidepressants and Azidothymidine (AZT) are combined.

If a patient presents with confusion or other sensorial changes associated with a depression, neuropsychological testing may be helpful since "AIDS dementia complex" is often misdiagnosed and mistreated as depression during the early stages.

Suicide

The shock and turmoil resulting from the diagnosis of HIV infection and AIDS is understandable and a high incidence of suicide in this group of the patients is not surprising.[12] Suicide appears to occur shortly after the diagnosis is confirmed, most likely because the patient is psychologically devastated and has powerful feelings of panic, guilt, depression and hopelessness. The risk is high again in the later stages of the illness, when it is related more to biological factors due to the development of confusion and disorientation etc. associated with delirium and dementia.

The relative risk of suicide in men with AIDS in the 20 to 59 year age group was found to be 36 times that of a group of the same age without AIDS and 66 times that of the general population.[13] Risk factors as reported in different studies include being unmarried, white, homosexual male, in one's thirties with a diagnosis of less than 6 month duration.

A physician, faced with a patient who has suicidal ideation, should complete the standard history and mental state examination usually performed for these patients.

Mania

Several authors reported that hypomania or mania can be a presenting feature of patients with HIV infection/AIDS.[14] Most manic swings in these patients are not often associated with a bipolar psychopathology but are secondary to other factors such as drugs, infections, delirium etc. Patients who show this behavioral change present with typical manic symptoms including decreased sleep, pressured speech, poor judgement, disorganized thinking, irritability, euphoria, etc.

Management

Managing manic patients includes tranquilosedation while trying to establish the etiology. The more serious and uncontrolled manic behaviours are hazardous for the patient and those around them, making psychiatric hospitalization necessary where rapid control of their behaviour can be achieved. This can be best done by using parenteral neuroleptics eg. haloperidol, loxapine etc. The haloperidol given intravenously appears to decrease the incidence of severe and persistent extrapyramidal side effects, to which these patients are susceptible, especially when haloperidol is given in combination with a benzodiazepine eg. lorazepam. It is best to initiate treatment with 5 mg iv haloperidol. If needed, additional iv doses of 1-2 mg haloperidol every hour will

usually control the manic behaviour. Haloperidol should be tapered over 3-4 days once the patient's behaviour is settled.

Loxapine has also been used because of its decreased incidence of extrapyramidal side effects. It is best to initiate treatment with loxapine 12.5 mg IM bid and gradually increase the dose to 25 mg IM or more bid depending upon patient's response.

A switch to the oral neuroleptics should be made as soon as possible.

Should neuroleptics evoke an extrapyramidal reaction, it may be treated with amantadine 100 mg orally once or twice daily rather than with the usual anticholinergic antiparkinson agents.

When the acute manic behaviour is improved a decision has to be made regarding long term treatment with lithium. HIV infected individuals with preexisting bipolar mood disorder will require and benefit from the use of lithium for the prevention and treatment of affective symptomatology regardless of the stage of HIV infection. Lithium has been used in 'AIDS related complex' and to treat AIDS patients diagnosed as bipolar manic or with a manic organic mood disorder.[12] However, although the efficacy of lithium for the treatment of mania secondary to the organic effects of HIV has not been studied, it could be of great clinical benefit for these patients.

Once a patient is started on lithium, close monitoring of the lithium level is very important. Once every 2 weeks is usually recommended because of erratic GI absorption.

ORGANIC MENTAL DISORDER: DELIRIUM AND DEMENTIA

Organic mental disorders are typically characterized by disturbances of affect, behaviour and cognition. Estimates are that as many as 70% of patients with HIV disease will develop an organic mental disorder during the course of their illness.[16]

Delirium

Delirium is most frequently encountered in patients with HIV disease who are hospitalized. The increased vulnerability in these patients is related to viral invasion and damage to brain tissue, as well as to the adverse cerebral effects of opportunistic infections, fever, hypoxia, narcotics and steroids.

At an early stage the delirium may present with prodromal features including disorganized thinking, irritability and insomnia. Later on there may be in addition decreased attention span, perseveration, reduced level of consciousness and perceptual disturbances (illusions or hallucinations). The clinical features may develop over a few hours to a few days and may fluctuate over the course of a single day.

Management

It is important to recognize the early signs of delirium, since in patients with HIV disease, many of the causes are correctable. The management of these patients include the following:

i) Determine and correct the underlying cause(s) if possible.

ii) Provide appropriate medical management and stabilize the patient.

iii) Psychosocial and emotional support, as appropriate.

iv) Pharmacological interventions including:
   a) discontinuation or minimal use of all medications that may further disturb cerebral metabolism and function;
   b) use of high potency neuroleptic such as haloperidol or loxapine in patients who are extremely agitated or violent. (Using neuroleptics is outlined in some detail in the section on the management of manic patients).

Dementia

Mental (sensorial) changes in HIV infected individuals may be due to systemic diseases, opportunistic infections, neoplasms within CNS, metabolic dearrangements or due to direct neuropathogenic effects of HIV infection.[17] Psychosocial factors and medications used in these patients can also contribute to mental changes.[7]

AIDS Dementia Complex (ADC)

This was first described by Snider and associates in 1983[18] and several reports have appeared since then.[17] Other terms used by different authors include: HIV encephalitis, subacute encephalopathy, subacute encephalitis and 'AIDS related dementia'.

The clinical picture in ADC is one of "subcortical dementia" and is similar to multi-infarct dementia. The patients have more problems with alertness and attention but are usually aware of their deficit. It has an insidious onset as a rule but, in a minority, there is a rapid onset and progression. This dementia is chronic and progressive with a fluctuating course and is often exacerbated by a concomitant illness eg. infection.

The early clinical symptoms may resemble a depression and are often indistinguishable from it without neuropsychological testing. These early symptoms consist of forgetfulness, poor concentration, loss of interest, loss of libido, apathy, blunting of affect, psychomotor retardation and withdrawal. They may be associated with neurological symptoms such as hand tremors, leg weakness and balance problems.

The progression from "early" to "late" symptoms may occur within a few weeks in some patients but more often, it may take several months. "Late" symptoms often consist of confusion, disorientation, agitation and psychotic symptoms such as visual hallucinations and delusions.

Management

i) It is important to differentiate between the primary dementia caused by the direct affects of HIV on the

CNS, from the secondary dementia caused by systemic disease. In the primary type, the cognitive decline is gradual over months and amnesia, aphasia etc are unusual until very late in the course. There is no effective treatment available. In the secondary type, cognitive decline may be abrupt and amnesia etc are often prominent early on. There seems to be significant improvement after treatment of the underlying condition.

ii) The marked apathy seen in some patients may lead to a misdiagnosis of major depression.

iii) In patients with significant cognitive and affective problems associated with ADC, the use of psychostimulants (methylphenidate) can provide an improved quality of life.[11] Patients with ADC show marked improvement in cognitive and affective components without significant side effects.

AZT (Azidothymidine) also appears to alleviate, and in some studies, may partially reverse cognitive impairment.

iv) Psychotic demented patients may require a neuroleptic, eg haloperidol, for specific target symptoms such as paranoia, agitation, restlessness, confusion and insomnia.

v) Psychological and environmental support as appropriate.

vi) Neuropsychological testing is important, since the characteristic abnormalities (difficulty with complex sequencing, reduced verbal abilities, visual spatial difficulties, impaired fine motor control) are often not detected by a sensorial examination such as the Mini-Mental State Exam (MMSE).

vii) Baseline and regular monitoring of cognitive and sensorial function should be on integral part of the overall assessment of patients with ADC.

Psychosis

There have been a number of case reports of patients at various stages of HIV illness who present with psychotic symptoms.[19,20] The case reports provide clinical descriptions of the full range of psychosis from the appearance of one circumscribed delusion, to a fulminant psychotic break with delusions, auditory hallucinations, rambling or repetitive speech and an overall poor reality testing. The psychotic symptoms described may appear with "organic" signs of delirium or they may appear or without clouding of sensorium, as illustrated in Case 3. Although psychotic symptoms can appear at anytime during the course of this disease, they occur most commonly at the time the diagnosis of antibody positivity is made or at a subsequent change in status to AIDS.

Management

The most important steps in the management of these psychotic disorders are to establish the diagnosis and to identify and treat any organic factors that may be causing or contributing to the disorder. The differential diagnosis of psychotic symptoms in the context of the spectrum of HIV illness is quite complex. It includes a 'catastrophic reaction' to the diagnosis; a manifestation of demonstrable CNS dysfunction as a result of direct invasion of the CNS by HIV or by opportunistic infections or lymphoma; effect on the brain from hypoxia due to pneumonia or from electrolyte changes associated with severe diarrhoea. Side effects of medications also must be considered. Many of these are at least partially correctable.

For the treatment of psychotic symptoms in HIV illness, neuroleptics are quite effective whether the symptoms are of "organic" or "functional" origin and differential diagnosis for the purpose of treatment is less critical. Many different neuroleptics have been used to treat psychotic symptoms and syndromes in HIV infected patient. High potency neuroleptic eg. haloperidol, have relatively low anticholinergic effects and low doses can be effective. However, there may be an increased incidence of extrapyramidal side effects as well as the possibility of a neuroleptic malignant syndrome. Low potency neuroleptics eg. chlorpromazine have high anticholinergic side effect profiles and are not very useful. For chronic treatment, a medication intermediate in both anticholinergic and parkinsonian profile, eg. loxapine, may be better tolerated. If a patient with psychosis is also depressed or anxious, it may be necessary to prescribe a combination of appropriate psychotropic medications.

PSYCHIATRIC SYMPTOMS IN THOSE WITHOUT HIV INFECTION

Psychiatric manifestations are increasingly seen in people who have been involved in high risk activities in the past but remain seronegative. They often have misunderstood the established routes of transmission and many of them have enhanced psychological vulnerability. They realize that their life style has put them at risk for developing AIDS. They are rarely reassured for more than a brief period, as shown by their repeated request for antibody screening. The terminology used to define this group includes "worried well"[7] and "Pseudo-AIDS".[21]

The clinical presentation of these patients includes severe anxiety, panic attacks, obsessive compulsive behaviour, hypochondriasis (excessive somatic preoccupation and fear of the disease). Occasionally, in the more psychologically vulnerable, psychotic episodes will occur.

Management

i) Establish the serostatus regarding HIV infection;

ii) Careful assessment of the patient's risk for HIV;

iii) Make available support systems;

iv) Discuss HIV transmission, especially if misunderstanding or ignorance is a factor;

v) Repeated reassurance;

vi) If patient does not respond to above measures then the patient should be referred for psychiatric assessment and treatment.

CONCLUSION

Increasing evidence suggest that the clinical psychiatric manifestations of HIV infection encompass the full spectrum of psychiatric diagnoses including organic, affective, psychotic and anxiety disorders. HIV patients who present with major psychiatric disturbances should initially have treatment directed at correctable organic abnormalities. Once this is done, the remaining psychiatric symptoms should be treated in the usual way using a combination of biological, psychological and social measures as appropriate.

The traditional psychotropic medications including antidepressants, antianxiety agents, neuroleptics and psychostimulants can be useful for symptomatic treatment. Because these patients have an extreme sensitivity to psychotropic drugs and manifest adverse affects more often, drugs with a low anticholinergic side effect profile should be used whenever possible. Doses should be kept modest and side effects should be treated appropriately.

Physicians can play an essential role in minimizing the psychological and social impact of HIV infection through counselling. The psychotherapeutic aim should be to improve the patient's sense of self-worth and control and to try and decrease the feelings of victimization created by the relentless progress of the disease.

In the absence of an effective treatment, a decrease in HIV infection is not expected. Since most individuals infected are asymptomatic and are unaware that they are infected, the potential for further spread of the disease is frightening. □

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REHABILITATION OF PEOPLE WITH LOWER-LIMB AMPUTATIONS

Continued from page 140.

ACKNOWLEDGEMENTS

We thank the Editorial Service of Dalhousie University, Mr. V. Hicks of the Department of Health and Fitness and Mrs. B.L. Yorke for their help.

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Medical Humanities

T.J. Murray,* OC, MD, FRCP

Halifax, N.S.

"You may be sure that some men, even among those who have chosen the task of pruning their fellow creatures (surgeons) grow more and more thoughtful and compassionate in the midst of their cruel experience. They become less nervous, but more sympathetic. They have a truer sensibility for other's pain, the more they study pain and disease in the light of science."

Oliver Wendell Holmes
The Professor at the Breakfast-Table, 1860

READING WEEKEND, OCTOBER 1993

Would you and your companion like to spend a Reading Weekend in a lovely inn during the autumn colors in the Annapolis Valley? We have reserved the Tattinghouse Inn for a discussion of books that relate to "The Patient's Story", so we can view and understand illness from the patient's perspective.

There will be a list of books that you can read over the summer. Twelve people will attend and all are expected to do the reading and join in the discussion. All these books are available from Mary Jo Anderson at Frog Hollow Bookstore.

If interested in further information, call 902-494-2514.

READING BINGES

A friend, Nick Davies, once wrote a tantalizing article in the Christmas edition of the *British Medical Journal* (1989; **299**:1209-10) (The Christmas Edition always has wonderful historical, philosophical and whimsical articles) on his secret and embarrassing personal problem. He explains that his problem is the inordinate amount of time he spent thinking about what he would read on his summer vacation. He blushingly admitted he planned all year for his summer reading. I note that when he wrote this article his vacation plan was interrupted by his responsibility for one of his patients. When he was about to leave, a patient who just had a colostomy went into ventricular fibrillation, so he stayed behind, indicating that when he caught up with his family he would get to his delicious reading list.

He was one of those open, warm, enthusiastic people who touched everyone he met, and we all felt we had a very personal relationship with Nick. I remember one meeting of the American College of Physicians when he arrived at a session I was chairing on medical humanities, proudly clutching under his arm the nameplate from the World War II ship *USS William Osler*, and enthralled everyone with the story of the ship and how he acquired the plate.

He felt, and I agree, that bibliomania is passed via some quasi-genetic mechanism from the parents. My mother felt that just having lots of books around would affect you by osmosis. I was interested to note that Nick, like me, was essentially a slow reader, which didn't stop us, especially if you read continuously. I once wrote him, outlining the books in my bathrooms, as I have bookcases in each loo (a gastroenterologist colleague visiting my cottage told me he did not find this amusing). Nick responded with his long list for his next vacation.

Tragically, Nick didn't get his next vacation, as he was on the small commuter plane that crashed on a short flight near his home in Atlanta, Georgia. Many remember the crash because a well known Senator was on board, but American medicine was profoundly affected because a few days later Nick was to become President of the American College of Physicians, at a time that the ACP was evolving a concept of American health care reform.

Nick Davies was an avid Oslerian, and like Osler, felt reading was the lifeblood of the physician who continued to learn throughout life.

ILLNESS IN LEADERS

The recent US Presidential race again raised the question about the health of national leaders. What if Tsongas didn't drop out of the race, and won? He was quite open about his malignancy, but when he dropped out it was apparently because of inadequate funds for his campaign. Only a few months after Clinton was comfortably in the Oval Office, we hear that Tsongas is suffering with a recurrence and undergoing chemotherapy and feeling very unwell.

I rather liked Tsongas. He seemed a thoughtful, responsible, warm and charming candidate, and with a social conscience that would be refreshing in the White House. But I have to ask if it was responsible for him to seek the Presidency when his health might restrict his ability to do what his country asks and expects. And I have to ask if there shouldn't be a process that assesses candidates for high office and which then monitors the health of the leader. Some years ago I wrote about this, but struggled with the process that would allow a Canadian leader to have a personal physician, while the leader and the physician were also responsive to a confidential panel that reported to the Governor General if a serious situation arose.

I wasn't satisfied with my solution, and some would suggest it isn't a medical issue anyway. They would argue

* Professor of Medical Humanities, Dalhousie Medical School, Halifax, N.S.

it is a political issue, and the political process should struggle with it, like they do with any other conflict situation.

The only country that has seriously, but unsuccessfully, struggled with the question is the USA, spurred on by President Eisenhower after he became concerned that his three serious illnesses would have precluded him managing any national crisis while he was ill. The resulting solution was two articles to the 25th Amendment, which were unworkable, and really no solution. It should not be a surprise that the USA made the effort, because they have had a lot of problems. Grover Cleveland was operated on (secretly) during a national financial crisis; Roosevelt was weak and in failing health when he was deciding the fate of post-war Europe at Yalta; Nixon was out of control on alcohol and sedatives during the Watergate crisis. The following Presidents had periods of incapacitation while in office: Harrison, Taylor, Garfield, Cleveland, MacKinley, Roosevelt, Eisenhower, Johnson, Nixon, and Regan.

The problem is not a new one. George III was ill during much of the conflict with the American Colonies. Ramsay MacDonald was demented during his last years in office. Begin of Israel was seriously depressed and suffering from recurrent myocardial infarcts and angina, ignoring his cabinet, but refusing to step down. Hatoyama, Nehru, Kozlov, Zigney, Salisar, Brezhnev and Begin all had strokes while in office, and stayed in office!

History tells us we have a serious problem about illness in leaders. My review of history also suggests physicians are not a help in these cases. Hugh L'Etang has written and spoken extensively on this subject, (*The Pathology of Leadership*, London: William Heinemann, Medical Books Ltd) and he comments that by covering up, shielding everyone from the truth, lying to the media, and by even misleading the leader, physicians are part of the problem.

Should physicians be concerned about this, or should we ignore the issue and regard it as just another vexing problem for the politicians to solve? Is it conceivable that we could develop a process that would protect citizens from incapacitated leaders. Would such a process recognize the rise of another Hitler, for instance?

I spoke to L'Etang recently in London, and he is busy on a new edition of his book. Why? Because so many cases of ill leaders in office have occurred since his book was published in 1969.

THE NATIONAL LIBRARY OF MEDICINE

When you visit the largest medical and research library in the world you expect to see long rows of tall bookstacks running off into the distance. When I was in Washington in April I went to nearby Bethesda, Maryland, to the National Library of Medicine, and looked forward to browsing through all the wonderful collections of books and documents, fondling the leather covers and the ageing crisp pages. Anyone is welcome to visit the Library.

I walked from room to room and hardly saw a book. What I saw were rows of computer terminals. There is no problem accessing material, but you do it with computers and the helpful staff will appear with the requested books shortly after. Oh, there are books, lots of books, some 4,926,000 of them, and 21,863 journals, but they are in floors below. They will get you the books you want, but you can't browse, or even go on those floors.

Even though they are a major resource because of the huge collection, their real activity has moved from a repository of printed material, to a vast communication centre with computerized data bases. When you go to the Kellogg Medical Sciences Library at Dalhousie Medical School, and log on to MedLine or many other databases, you are talking to the National Library through the computer networks.

The library began in 1836 as the library of the Army Surgeon General's Office. By the time it moved into the Ford Theatre in 1866 (it was thought to be in bad taste to use it for plays after Lincoln was shot there), it was still a small collection of shelved books, with a handwritten catalogue. At that time John Shaw Billings took over, he had the idea of producing an index of published articles, and the Index Medicus has been a major effort of the National Library ever since.

Remember the story of why the huge railway companies failed, even though they were so powerful and rich early in this century. Because they tried to get more and bigger trains, and more tracks and new routes, when the airplane and better highways started to compete with them. They failed because they forgot what business they were in. They thought they were in the railway business.

They weren't – they were in the transportation business. They built bigger trains when they should have moved to the newer forms of transportation.

The National Library knows what business they are in. They aren't in the book and journal business, they are in the communication business, and so they have not only moved but led the way in the methods to better communicate medical and scientific information.

But for those of us who like to hug a book, they still maintain the world's largest medical collection under ground. There is a story about the underground construction that the staff like to tell. The current building was built at the height of the Cold War, so bomb shelters were all the vogue. The library collection below ground is designed as a large bomb shelter, covered with a concrete dome. The dome has a windowed ring at ground level that will collapse down to cover the shelter in the event of an explosion. They had to put the least important facility in the windowed area, as it would be crushed in an atomic attack. That's where they put the administration.

When told the story I wasn't surprised. I've worked in buildings where people would die for a window.

DEATH OF A ROCK SINGER

A second year Dalhousie medical student has recently written an excellent review of the mysterious death of Jim Morrison, lead singer of The Doors, in life a charismatic and shocking rock and roll singer, and in death a cult figure. (*Medical Post*, Dec 1, 1992)

The mystery began when Morrison apparently died of an overdose of heroin in Paris. His girlfriend called in a "fly-by-night" physician to sign the death certificate and he was buried in a sealed casket without notifying the family or the American Embassy.

Green reviewed his early career as a brilliant and well read student, his rise to fame, and his alcoholism.

By the time he died at age 27, he was failing in music and in life and unable to control his drinking. Green gives a differential diagnosis of alcoholic cardiomyopathy, myocarditis, suicide, and heroin overdose, among other possibilities, but leaves the verdict open.

The mystery continues, and fans still turn up daily at his Paris grave. As might be expected, rumours circulate that he is still alive somewhere. Perhaps he's sharing a flat with Elvis.

DR. ROBERT J.T. JOY TO VISIT DALHOUSIE

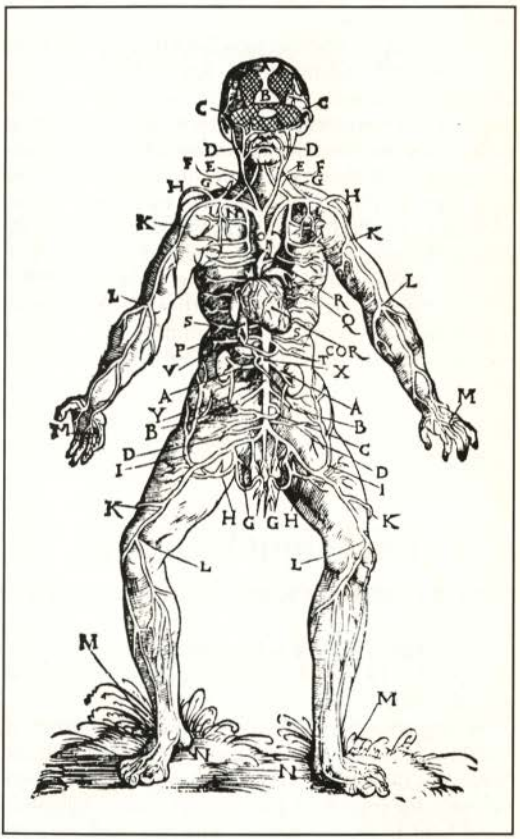
Dr. Robert J.T. Joy will give the first Friday-at-Four lecture on September 17. Dr. Joy will deliver the first Dr. T. J. Murray Visiting Scholar address at that time, and will be a visitor at the medical school, giving various presentations, rounds and discussions with students, faculty and community physicians.

He directs the largest program in medical history in the United States, as Professor and Chairman of the section of Medical History at the Uniformed Services University of the Health Sciences, Bethesda, Maryland. He is an expert on the history of military medicine, infection and antibiotics, and surgery.

Dr. Joy has a long list of visiting lectureships in Canada and the United States, and received outstanding teacher awards from his medical students. He has a reputation as an enthusiastic and outstanding clinician, teacher and historian, and we look forward to his visit to Dalhousie and the medical community.

ART IN MEDICAL TEXTS

Dr. Kenneth Roberts, Emeritus Professor at Memorial University, has recently co-authored the outstanding book *The Fabric of the Body: European Traditions of Anatomical Illustration*, which displays the manner that physicians and medical artists have seen and displayed the human body through the centuries.



Dr. Roberts gave an address at the Dalhousie Art Gallery on "The Social and Artistic Aspects of Anatomical Drawings" and his examples of anatomy in art will be displayed in an exhibit sponsored by the National Gallery of Canada entitled "The Dead and the Naked: The Interrelations Between Anatomy and Art".

Dr. Roberts was the Clinch Professor of Medical History at Memorial for many years and very influential in medical history in Canada. □

Appreciations

DR. OTTO HENRY HORELTT

Dr. Otto Horeltt, was born in 1933 at Dalhousie, New Brunswick. Otto began his education in the Dalhousie public school system, but eventually transferred to Mount Allison Academy and thence to Mount Allison University. He came to Dalhousie University in 1953. He played football under the tutelage of coach Dr. Merv Shaw where he played on a line with Drs. Brian Chandler and Don Nicholson. He was a permanent member of Phi Rho Sigma fraternity. Following graduation from medical school in 1960, he did general practice at Petit Rivier where he was known as "that big German doctor". He was revered by most of Lunenburg County for his availability and his wise counsel.

I lived with Otto when we were both first year residents in the 1966/67 academic year. It was a delightful experience. Otto's patients followed him wherever he went, and that year was no exception. It was not uncommon to come home and find the apartment filled with friends and patients. Following attainment of his anaesthesia certificate in 1969, Otto practised anaesthesia at the Victoria General Hospital in Halifax for 15 years. He eventually returned to a limited general practice which he maintained until his death on December 21, 1992.

At his memorial service, the overflowing chapel was a testament to the respect and confidence his patients placed in him. He will be greatly missed by all those whom revered him.

Dr. C. Lamont (Monty) MacMillan
Halifax, N.S.

DR. GEORGE ARCHIBALD WATSON ANGUS

Dr. George A. W. Angus, 77, of Yarmouth, passed away on April 11, 1993 in the Yarmouth Regional Hospital, after a short illness.

Dr. Angus was born in Paisley, Scotland. A graduate of Edinburgh, Scotland, with a Bachelor of Medicine and Surgery in 1945. He achieved his diploma of psychological medicine from the University of London, England.

He held posts in Scotland, England, and Australia before accepting a position as Executive Director of Western Nova Scotia Mental Health Clinic, which he held for 30 years. He also served as Regimental Medical Officer in the 24th Field Regiment, Royal Artillery. He was a psychiatrist in private practice, with the Yarmouth Regional Hospital, until he retired in 1990.

He was also a member of the Rotary Club of Yarmouth, which he served two or three years. In 1983, he was the recipient of the Citizens of the Year award given by the Lions Club.

Dr. Angus was a lover of books, good food, and helping anyone he could. He loved animals and always had a dog that he walked everyday; a drive in the country to look at scenery was one of his favorite pastimes, if he was not reading. He had a large collection of books which filled most corners of his home. Antiques were a special interest and, as a hobby, he collected many fine pieces.

One of his main goals in life was to help children to understand and "set them on the right track". Although many "did not seem to get to the end of the road" he listened and never judged.

He was a familiar sight sitting on the sofa that faces the street, or standing in his front hall waiting for his morning paper to do the crossword puzzle which he did faithfully everyday.

May he find peace and contentment in his new resting place.

A Special Friend of All.

Dr. and Mrs. Audrey Macdonald
Yarmouth, N.S.

Personal Interest Notes

Dalhousie medical graduates of the late '60s and early '70s were pleased to learn that **Miss Margaret Cragg** was awarded an honorary LLD on May 26, during the Dalhousie Health Professions Convocation. For this period, Miss Cragg was in charge of the Comprehensive Health Care Program, where medical students were assigned to families and then provided assessment and primary health care.

OBITUARIES

Dr. George A. W. Angus, (77) of Yarmouth, Nova Scotia died on April 11, 1993. Born in Scotland, he received his medical degree from Edinburgh in 1945. He accepted a position as executive director of the Western Nova Scotia Mental Health Clinic – a position he held for 30 years. He was psychiatrist at the Yarmouth Regional Hospital until his retirement in 1990. He was a senior member of The Medical Society of Nova Scotia and the Canadian Medical Association. He is survived by his brother, to whom the *Journal* extends sincere sympathy.

Dr. Allen D. Cohen (50) of Halifax, Nova Scotia, died on June 25, 1993. Born in Yarmouth, Dr. Cohen received his medical degree from Dalhousie Medical School in 1968. He studied nephrology at Yale University and at Charing Cross Medical School in London. Dr. Cohen was a member of Dalhousie University's Senate, a Professor in the Department of Medicine, and Chair of the Financial Management Committee. He was Head of the Division of Nephrology as well as Director of the Renal Transplant Unit at the Victoria General Hospital. Among his many outside activities, Dr. Cohen was President of The Nova Scotia Society of Internal Medicine. He was also the Chief Negotiator of the Negotiations Team and Co-Chairman of the Joint Management Committee of The Medical Society of Nova Scotia. Dr. Cohen is survived by his wife, and four sons. *The Nova Scotia Medical Journal* extends sincere sympathy to his wife and family.

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Conference Notice

Restoring the Integrity: An education conference on the dimensions of sexual exploitation by professions involved in relationships of trust.

An Interdisciplinary Conference will be held in Fredericton, October 15th and 16th, on Professional Sexual Misconduct. The conference will bring together professionals from various disciplines to examine this complex issue within an educational context. Registration for this conference will be limited.

For more information, please contact:
The New Brunswick Medical Society, 176 York Street, Fredericton, NB, E3B 3N8 1-506-458-8860.

once daily Nasacort*

(Triamcinolone Acetonide Nasal Inhaler)

THERAPEUTIC CLASSIFICATION

Corticosteroid for nasal use

ACTIONS AND CLINICAL PHARMACOLOGY: Triamcinolone acetonide is a potent anti-inflammatory steroid with strong topical and weak systemic activity. When administered intranasally in therapeutic doses, it has a direct anti-inflammatory action on the nasal mucosa, the mechanism of which is not yet completely defined. The minute amount absorbed in therapeutic doses has not been shown to exert any apparent clinical systemic effects.

INDICATIONS AND CLINICAL USE: Nasacort™ (triamcinolone acetonide) nasal inhaler is indicated for the topical treatment of the symptoms of perennial and seasonal allergic rhinitis unresponsive to conventional treatment.

CONTRAINDICATIONS: Active or quiescent tuberculosis or untreated fungal, bacterial and viral infection. Hypersensitivity to any of the ingredients of Nasacort™ (triamcinolone acetonide).

WARNINGS: In patients previously on prolonged periods or high doses of systemic steroids, the replacement with a topical corticosteroid can be accompanied by symptoms of withdrawal, e.g. joint and/or muscular pain, lassitude, and depression; in severe cases, adrenal insufficiency may occur, necessitating the temporary resumption of systemic steroid therapy. Careful attention must be given to patients with asthma or other clinical conditions in whom a rapid decrease in systemic steroids may cause a severe exacerbation of their symptoms.

Pregnancy: See Precautions.

PRECAUTIONS:

1) The replacement of a systemic steroid with Nasacort™ (triamcinolone acetonide) has to be gradual and carefully supervised by the physician. The guidelines under "Administration" should be followed in all such cases.
2) During long-term therapy pituitary-adrenal function and hematological status should be assessed.
3) Patients should be informed that the full effect of Nasacort™ therapy is not achieved until 2 to 3 days of treatment have been completed. Treatment of seasonal rhinitis should, if possible, start before the exposure to allergens.
4) Treatment with Nasacort™ should not be stopped abruptly but tapered off gradually.
5) Corticosteroids may mask some signs of infection and new infections may appear. A decreased resistance to localized infections has been observed during corticosteroid therapy; this may require treatment with appropriate therapy or stopping the administration of Nasacort™.
6) The long term effects of Nasacort™ are still unknown, in particular, its local effects; the possibility of atrophic rhinitis and/or pharyngeal candidiasis should be kept in mind.
7) There is an enhanced effect of corticosteroids on patients with hypothyroidism and in those with cirrhosis. Acetylsalicylic acid should be used cautiously in conjunction with corticosteroids in hypothrombinemia.
8) Because of the inhibitory effect of corticosteroids on wound healing, in patients who have had recent nasal surgery or trauma, a nasal corticosteroid should be used with caution until healing has occurred.
9) Patients should be advised to inform subsequent physicians of prior use of corticosteroids.
10) Until greater clinical experience has been gained, the continuous, long-term treatment of children under age 12 is not recommended.
11) **Pregnancy:** The safety of Nasacort™ in pregnancy has not been established. If used, the expected benefits should be weighed against the potential hazard to the fetus, particularly during the first trimester of pregnancy.
Like other glucocorticosteroids, triamcinolone acetonide is teratogenic to rodents and nonhuman primates (see under TOXICOLOGY). The relevance of these findings to humans has not yet been established. Infants born of mothers who have received substantial doses of glucocorticosteroids during pregnancy should be carefully observed for hypoadrenalism.
12) **Lactation:** Glucocorticosteroids are secreted in human milk. It is not known whether triamcinolone acetonide would be secreted in human milk, but it is suspected to be likely. The use of Nasacort™ in nursing mothers, requires that the possible benefits of the drug be weighed against the potential hazards to the infant.
13) **Children:** Nasacort™ is not presently recommended for children younger than 12 years of age due to limited clinical data in this age group.
14) Fluorocarbon propellants may be hazardous if they are deliberately abused. Inhalation of high concentrations of aerosol sprays has brought about cardiovascular toxic effects and even death, especially under conditions of hypoxia. Aerosols are safe when used properly and with adequate ventilation, but excessive use should be avoided.
15) To ensure the proper dosage and administration of the drug, the patient should be instructed by a physician or other health professional in the use of Nasacort™ (see Patient Instructions).

ADVERSE REACTIONS: Adverse reactions reported in both controlled and uncontrolled studies involving 1148 patients who received Nasacort™ (triamcinolone acetonide) are provided in the following table:

Adverse Experience	Nasacort % (n = 1077)	Placebo % (n = 545)
Headache	20.4	19.4
Upper Respiratory Infection	5.3	8.1
Nasal Irritation	5.1	4.2
Throat Discomfort	4.6	3.3
Dry Mucous Membranes	3.5	2.2
Epistaxis	4.6	6.6
Sneezing	3.1	5.5
Sinusitis	2.1	3.7

When patients are transferred to Nasacort™ from a systemic steroid, allergic conditions such as asthma or exzema may be unmasked (see Warnings).

SYMPTOMS AND TREATMENT OF OVERDOSAGE: Like any other nasally administered corticosteroid, acute overdosing is unlikely in view of the total amount of active ingredient present. However when used chronically in excessive doses or in conjunction with other corticosteroid formulations, systemic corticosteroid effects such as hypercorticism and adrenal suppression may appear. If such changes recur, the dosage of Nasacort™ (triamcinolone acetonide) should be discontinued slowly consistent with accepted procedures for discontinuation of chronic steroid therapy. (see Administration). The restoration of hypothalamic-pituitary axis may be slow; during periods of pronounced physical stress (i.e. severe infections, trauma, surgery) a supplement with systemic steroids may be advisable.

DOSAGE AND ADMINISTRATION: See Warnings.

Nasacort™ (triamcinolone acetonide) is not recommended for children under 12 years of age.

Careful attention must be given to patients previously treated for prolonged periods with systemic corticosteroids when transferred to Nasacort™. Initially, Nasacort™ and the systemic corticosteroid must be given concomitantly, while the dose of the latter is gradually decreased. The usual rate of withdrawal of the systemic steroid is the equivalent of 2.5 mg of prednisone every four days if the patient is under close supervision. If continuous supervision is not feasible, the withdrawal of the systemic steroid should be slower, approximately 2.5 mg of prednisone (or equivalent) every ten days. If withdrawal symptoms appear, the previous dose of the systemic steroid should be resumed for a week before further decrease is attempted.

The therapeutic effects of corticosteroids, unlike those of decongestants, are not immediate. Since the effect of Nasacort™ depends on its regular use, patients must be instructed to take the nasal inhalations at regular intervals and not as with other nasal sprays, as they feel necessary.

In the presence of excessive nasal mucus secretion or edema of the nasal mucosa, the drug may fail to reach the site of action. In such cases it is advisable to use a nasal vasoconstrictor for two to three days prior to Nasacort™ therapy. Patients should be instructed on the correct method of use, which is to blow the nose, then insert the nozzle firmly into the nostril, compress the opposite nostril and actuate the spray while inspiring through the nose, with the mouth closed.

An improvement of symptoms usually becomes apparent within a few days after the start of therapy. However, symptomatic relief may not occur in some patients for as long as two weeks. Nasacort™ should not be continued beyond three weeks in the absence of significant symptomatic improvement.

Adults and Children 12 years of age and older: The recommended starting dose of Nasacort™ is 400 μg per day given as two sprays (100 μg/spray) in each nostril once a day. If needed, the dose may be increased to 800 μg per day (100 μg/spray) either as once a day dosage or divided up to four times a day, i.e., twice a day (two sprays/nostril), or four times a day (one spray/nostril).

After the desired effect is obtained, patients may be maintained on a dose of one spray (100 μg) in each nostril once a day (total daily dose: 200 μg per day).

AVAILABILITY: Nasacort™ (triamcinolone acetonide) is a metered-dose aerosol unit containing a microcrystalline suspension of triamcinolone acetonide in the propellant dichlorodifluoromethane and dehydrated alcohol USP 0.7% w/w. Each canister contains 15 mg triamcinolone acetonide. Each actuation releases approximately 100 μg triamcinolone acetonide of which approximately 55 μg are delivered from the nasal actuator to the patient (estimated from in-vitro testing). There are at least 100 actuations in one Nasacort™ canister. The device should not be used after 100 inhalations, since the amount delivered thereafter per actuation may not be consistent. It is supplied with a nasal adapter and patient instructions: Box of one.

REFERENCES:

1. Product Monograph: NASACORT (triamcinolone acetonide); Rhone-Poulenc Rorer, 1992. 2. Ziemniak J.A. J. Resp. Diseases, 1991; 12(3A): S41-S45. 3. Storms W.W. et al., Annals of Allergy, 1990; 66 : 329-334. 4. Tinkelman D. et al., Annals of Allergy, 1990; 64 (February Part II): 234-240. 5. Spector S. et al., Annals of Allergy, 1990; 64: 300-305. 6. Data on File: Rhone-Poulenc Rorer; 5029-02. 7. Data on File: Rhone-Poulenc Rorer; 5029-01.

†unresponsive to conventional treatment

Atrovent®

(ipratropium bromide)

INHALATION SOLUTION

THERAPEUTIC CLASSIFICATION

Bronchodilator

INDICATIONS AND CLINICAL USES

Atrovent (ipratropium bromide) solution is indicated for the therapy of acute exacerbations of chronic bronchitis. Atrovent solution, when used in conjunction with a β_2-adrenergic stimulant solution such as fenoterol or salbutamol, is indicated for acute asthmatic attacks. It is to be administered by compressed air or oxygen driven nebulizers.

CONTRAINDICATIONS

Known hypersensitivity to Atrovent (ipratropium bromide), to any of the product ingredients, or to atropinics.

WARNINGS

Atrovent (ipratropium bromide) solution in the 20 mL multidose bottle contains preservatives (benzalkonium chloride and disodium ethylene diamine tetraacetic acid-EDTA-disodium). It has been reported that these preservatives may cause bronchoconstriction in some patients with hyperreactive airways.

The 2 mL unit dose vial (250 mcg/mL, 125 mcg/mL) does not contain preservatives.

Atrovent should not be used alone for the abatement of an acute asthmatic attack since the drug has a slower onset of effect than that of an adrenergic β_2 agonist.

Care should be taken to ensure that the nebulizer mask fits the patient's face properly and that nebulized solution does not escape into the eyes. There have been isolated reports of ocular complications (i.e., mydriasis, increased intraocular pressure, angle closure glaucoma) when nebulized ipratropium bromide either alone or in combination with an adrenergic β_2 agonist solution has escaped into the eyes. In the event that glaucoma is precipitated or worsened, treatment should include standard measures for this condition.

PRECAUTIONS

General:

- Patients should be instructed in the proper use of the nebulizer.
- Caution is advised against accidental release of the solution into the eyes.
- In patients with glaucoma, prostatic hypertrophy or urinary retention, Atrovent (ipratropium bromide) should be used with caution.
- If a reduced response to Atrovent becomes apparent, the patient should seek medical advice.
- Atrovent solution, when administered to patients with acute severe asthma, should be used with concomitant β_2 -adrenergic stimulant therapy.

Use in Pregnancy:

The safety of Atrovent in pregnancy has not been established. The benefits of using Atrovent when pregnancy is confirmed or suspected must be weighed against possible hazards to the fetus. Studies in rats, mice and rabbits showed no embryotoxic nor teratogenic effects.

Use During Lactation:

No specific studies have been conducted on excretion of this drug in breast milk.

Benefits of Atrovent use during lactation should therefore be weighed against the possible effects on the infant.

Use in Children:

The efficacy and safety of Atrovent in children younger than 5 years has not been established.

Use with Other Drugs:

In patients receiving other anticholinergic drugs, Atrovent should be used with caution because of possible additive effects.

In patients with glaucoma or narrow anterior chambers, the administration by nebulizer of a combined Atrovent-β_2 agonist solution should be avoided unless measures (e.g., use of swimming goggles) are taken to ensure that nebulized solution does not reach the eye. Exposure of the eyes of such patients to a nebulized combination of Atrovent and a β_2 agonist solution has been reported to result in increased intraocular pressure and/or acute angle closure.

Atrovent solution with preservatives (i.e. from the 20 mL multidose bottle) should not be mixed with sodium cromoglycate, as this produces a cloudy solution caused by complexation between the preservatives and sodium cromoglycate. If the patient's condition requires the administration of sodium cromoglycate, it should be given in combination with Atrovent solution without preservatives (i.e., from the unit dose vial).

ADVERSE REACTIONS

The frequency of adverse reactions recorded in 214 patients receiving Atrovent (ipratropium bromide) solution was as follows, given by percentage of patients reporting: Dry mouth or throat, 9.3; Bad taste, 5.1; Tremor, 4.2; Exacerbation of symptoms, 4.2; Burning eyes, 0.9; Nausea, 0.9; Sweating, 0.9; Cough, 0.9; Headache, 0.5; Palpitations, 0.5.

The adverse effect judged to be most severe was exacerbation of symptoms. This occurred in 8 patients treated with Atrovent solution alone, 6 of whom withdrew from the clinical studies. Bronchospasm occurred in 3 patients with acute severe asthma who received Atrovent solution alone. In two patients, this was reversed after therapy with a β_2 sympathomimetic solution. The third patient received no other therapy.

The following table compares the incidence of adverse effects of the combination of Atrovent and a β_2 agonist (either fenoterol or salbutamol) solution with that of the β_2 agonist alone.

ADVERSE EFFECT	ATROVENT + β_2 AGONIST (% of 94 patients)	β_2 AGONIST (% of 96 patients)
Tremor	31.9	26.0
Dry mouth	16.0	28.1
Bad taste	16.0	13.5
Vomiting	2.1	2.1
Palpitations	2.1	1.0
Headache	1.1	2.1
Cough	1.1	0.0
Flushing	1.1	0.0
Dizziness	0.0	1.0
Numbness in leg	0.0	1.0

There have been isolated reports of ocular effects such as mydriasis, increased intraocular pressure, and acute glaucoma associated with the escape of nebulized ipratropium bromide–alone or in combination with a β_2 agonist solution into the eyes.

DOSAGE AND ADMINISTRATION

In adults, the average single dose of Atrovent (ipratropium bromide) solution is 250-500 µg of ipratropium. In children, aged 5-12 years, the recommended dose is 125-250 µg of ipratropium bromide solution. This should be diluted to 3-5 mL with preservative free sterile Normal Saline [Sodium Chloride Inhalation Solution, USP 0.9%] or with a bacteriostatic sodium chloride solution, 0.9% preserved with benzalkonium chloride (see PHARMACEUTICAL INFORMATION). Nebulization should take place using a gas flow (oxygen or compressed air) of 6-10 L/minutes and the solution nebulized over a 10-15 minute period. The Hudson Updraft™, Bennett Twin Jet® and Inspiron Mini-Neb® nebulizers, with facemask or mouthpiece have been used. The manufacturers' instructions concerning cleaning and maintenance of the nebulizer should be strictly followed. Treatment with Atrovent solution may be repeated every 4-6 hours as necessary.

PHARMACEUTICAL INFORMATION

Stability and Storage Recommendations:

20 mL Bottle: Unopened bottles of Atrovent (ipratropium bromide) solution should be stored at controlled room temperature (below 30ºC). Solutions diluted with presevative free sterile Sodium Chloride Inhalation Solution, USP 0.9% should be used within 24 hours from time of dilution when stored at room temperature and within 48 hours when stored in the refrigerator. Dilutions may also be made with a bacteriostatic sodium chloride solution 0.9% which contains benzalkonium chloride as the bacteriostatic agent (see WARNINGS). This diluted solution may be stored at room temperature and used within 7 days.

Controlled laboratory experiments using mixtures of Atrovent solution with Alupent® (orciprenaline sulfate), Berotec® (fenoterol hydrobromide) or salbutamol sulfate (6mg/mL preserved with benzalkonium chloride) solutions and diluted with a sterile bacteriostatic sodium chloride solution 0.9% (i.e. normal saline), preserved with benzalkonium chloride, indicated that such mixtures were stable for 7 days at room temperature. For the preparation of such mixtures, it is recommended that only sterile solutions of bacteriostatic sodium chloride 0.9% preserved with 0.01% benzalkonium chloride be used to maintain the level of preservative in the mixture.The safety of preservatives other than benzalkonium chloride has not been established.

Incompatibilities: Atrovent solution with preservatives (i.e. from the 20 mL multidose bottle) should not be mixed with sodium cromoglygate solution, as this produces a cloudy solution caused by complexation between the preservatives and sodium cromoglycate. If the patient's condition requires the administration of sodium cromoglycate, it should be given in combination with Atrovent solution without preservatives (i.e., from the unit dose vial).

2 mL Unit Dose Vials (250 mcg/mL and 125 mcg/mL):

Unopened unit dose vials of Atrovent solution should be stored at controlled room temperature (below 30ºC) and protected from light. If required, the solution should be diluted with a preservative free sterile sodium chloride solution 0.9% and used immediately. Any solution remaining in the vial must be discarded.

The solution is physically compatible with Alupent® (orciprenaline sulfate), Berotec® (fenoterol hydrobromide) or salbutamol sulfate (6 mg/mL) solutions. If such mixtures are prepared, they should be diluted with preservative free sterile sodium chloride solution 0.9% and used immediately. Any unused portion of such combined solutions must be discarded.

AVAILABILITY

20 mL Bottle: Atrovent (ipratropium bromide) solution is provided as 20 mL clear, colourless or almost colourless solution containing 250 µg/mL (0.025%) Atrovent in isotonic solution. This solution is preserved with benzalkonium chloride 250 µg/mL and EDTA-disodium 500 µg/mL at pH 3.4 in an amber glass bottle with screwcap.

2 mL Unit Dose Vial: **250 µg/mL** Atrovent solution is also provided as 2 mL of clear, colourless solution containing 250 µg/mL (0.025%) ipratropium bromide in isotonic solution, presented in a plastic single use vial. One vial contains a total of 500 µg of ipratropium bromide.**125 µg/mL** Atrovent solution is also provided as 2 mL of clear colourless solution containing 125 µg/mL (0.0125%) ipratropium bromide in isotonic solution, presented in a plastic single use vial. Each vial contains a total of 250 µg of ipratropium bromide.

The complete Product Monograph for Atrovent (ipratropium bromide) Inhalation Solution is available to health professionals on request. Patient Information/Instructions are provided with the solution.

REFERENCES:

1. Reisman J, Galdes-Sebaldt M, Kazim F, Canny G, Levison H. Frequent administration by inhalation of salbutamol and ipratropium bromide in the initial management of severe acute asthma in children. *J Clin Immunol* 1988;81:16-20. 2. Product Monograph – Atrovent (ipratropium bromide) Inhalation Solution (rev. Mar. 24, 1993). 3. Beck R, Robertson C, Galdes-Sebaldt M, Levison H. Combined salbutamol and ipratropium bromide by inhalation in the treatment of severe acute asthma. *J Ped* 1985;107(4): 605-608. 4. Watson WTA, Becker AB, Simons FER. Comparison of ipratropium solution, fenoterol solution, and their combination administered by nebulizer and face mask to children with acute asthma. *J Allergy Clin Immunol* 1988;82:1012-1018.

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