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Perspectives of Pain

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Pain is an almost universal experience, except for those rare, unfortunate individuals with congenital absence of pain nerve endings. But throughout the ages, philosophers, priests and physicians have tried to define pain concisely and completely, with a notable lack of success. The most recent definition, that of the Taxonomy Committee of the International Association for the Study of Pain, is remarkable for its apparent simplicity and its encompassing breadth:

> "Pain is an *unpleasant emotional* and *sensory experience* that is usually, though not always, associated with, or described in terms of, *tissue injury*".

I have added the italics to draw attention to the force of this definition.

An unpleasant emotional and sensory experience can only occur within the brain: what happens before that is nociceptive traffic — electrical impulses along neural pathways that have no feeling, no means of evaluating the traffic. This leads to a startling conclusion: all pain is only experienced in the head. Only when a series of impulses reach the brain can a complex pattern of signals be appreciated as pain.

This is easier for us to understand in this age of digital information. The computer spews out a sequence of 0's and 1's through a modem that sends out 1200 of these per second down a telephone wire. Provided it encounters another modem which is set to receive its configuration, these signals will pass through the central processing unit and be thrown up on a screen — perhaps as written script, as graphic images, as a TV program or as symphonic music of highest quality. The signal can be modulated into various channels so that a single cable may carry 50 different types of signals, which could in theory be directed to 50 different playback modalities.

Using the digitalisation analogy, we begin to see form and substance taking shape in the tremendous thrust of research into acute and chronic pain mechanisms over the past 13 years, since the first Inaugural World Congress on Pain in Florence in 1975.

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It is clear that injured peripheral nerves remain hypersensitive to peripheral stimuli such as circulating catecholamines for many years after injury. Hence, high levels of anxiety and circulating catecholamines not only raise blood pressure but may increase pain. Adrenergic blocking drugs not only decrease blood pressure but may reduce pain as well.

The substantia gelatinosa of the dorsal horn of the spinal cord is a multilevel grand central station of neurotransmitter outputs. We now know this level has receptors for at least three different types of opiod transmitters: mu, kappa and sigma, and maybe others that modulate pain: the serotonin pathway, the norepinephrine pathway and the GABA pathway. In this issue, the use of antidepressants that enhance the serotonin pathway is discussed, and the effect of phenothiazines on N.E. pathways is considered. GABA agonists may also have a significant role in pain modulation.

Like the conductor of a symphony orchestra, the informed pharmacologically knowledgeable physician can create a point and counterpoint of pharmacological balance that may provide elegant solutions to some patients problems, and allows us to escape the straightjacket of analgesic + codeine ± barbiturate for pain relief.

Yet working the system elegantly from the outside may not be the best solution. Our colleagues in the Hypnosis Society of Nova Scotia and in clinical psychology, activate resources within the patient to control pain through the generation of increased amounts of inhibitory hormones in response to guided imagery, relaxation and meditation.

This approach changes the orientation of the physician from being a Mr. Fix It, to a facilitator who helps the patients fix themselves. At first sight this is a less powerful role, though more difficult to play. But how much more rewarding, to help patients from dependence through to independence and responsibility for their own ongoing health!

The multifactorial model of pain generation asks for a multi-level response to complex problems: the different influences of heredity and upbringing on expression of pain behaviour ensures that pain problems will be as complex as any in medicine. As with any area of medicine, improved knowledge, careful study and cautious innovation can lead to improved patient care and comfort.

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Psychological Approaches to the Assessment and Treatment of Chronic Pain

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The Psychology Department of the Victoria General Hospital has maintained a liaison with the Pain Management Unit (PMU) now for nearly seven years. During this time, a strong working relationship has evolved between the staff of the PMU, the psychologists, and other specialties (e.g., physiotherapy, occupational therapy, psychiatry). Psychologists in liaison with the PMU conduct psychological assessments of the suitability of chronic pain patients both for psychological treatment and for invasive medical treatments (e.g., Dorsal Column Stimulator Implantation). In addition, members of the Psychology Department take part in collaborative treatment planning through team meetings and carry out their own treatment programs. Finally, psychologists play an advisory role in the areas of research and program evaluation. This article focuses on the nature of psychological assessment and treatment of patients presenting to the PMU.

ASSESSMENT

Patients referred to the PMU may see a psychologist through one of two routes. The most typical mode is a referral by a PMU physician once the latter has assessed the patient. Often, a number of other treatment modalities may have already been attempted by other specialties before the referral to Psychology. The second avenue is through joint and team assessments; i.e., simultaneous assessment by each specialty at the time when the patient first presents to the PMU. Although this mode of referral has a number of advantages (particularly for patients living a distance away from the metropolitan area), time constraints have dictated that the number of team assessments remain few.

Every psychologist has his or her own particular emphasis in assessment. Nonetheless, in general, psychologists are interested in three broad areas; that is, the patient's environment, psychological functioning, and the pain condition.

ENVIRONMENT

Environmental factors can be classed into two categories encompassing both positive and negative features of the patient's life situation.

Social Supports. The relationship of social supports to pain is a complex one. For example, although patients who are able to rely on others have been found to report lower levels of psychological distress, there has been some indication that these same patients may be more likely to exhibit pain behaviours such as grimacing or rubbing the painful areas.[1] In general, nonetheless,the possession of social supports has been perceived as a resource. Psychologists thus seek answers to the following questions: What is the nature of the patient's relationships? Can he or she rely on others for emotional support? For material assistance? For opportunities to interact socially or recreationally? How has pain influenced the nature of his or her supports? How have social supports influenced the expression of pain? Does the patient have social resources which help him or her cope with the pain in particular? Are there areas in which the psychologist could intervene to improve social supports generally or to help manage pain in particular?

Life stressors. Increasingly, health professionals have acknowledged the deleterious influence of life stressors on health. Psychologists ask themselves such questions as the following: What is the nature of the patient's life stressors? What are the sources of his or her life stressors (e.g., work, family, marriage, lack of income, litigation, pain, limitations)? Are his or her life stressors best described as mild, moderate, severe, or highly traumatic? Are life stressors interfering with the patient's ability to cope with pain? Are life stressors exacerbating the pain condition? Could the effect of life stressors be lessened through psychological interventions?

PSYCHOLOGICAL FUNCTIONING

While an assessment of environmental factors allows for the examination of external variables impinging on the patient, an assessment of psychological factors allows for an examination of the impact of these situational variables as well as the coping style brought to bear on them.

Psychological distress. The relationship between pain and affective distress (e.g., depression, anxiety) is now well-accepted. For example, a study of 73 patients referred to the PMU indicated that 39 percent admitted to mild depressive symptoms while another

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27 percent admitted to depressive symptoms in the moderate to severe range.[2] Moreover, patients indicating moderate to severe depressive symptomatology tended to report significantly higher levels of pain intensity than patients not admitting to depression. Furthermore, increased levels of depression were correlated with greater reported losses of desire and ability for activities even when controlling statistically for the overall level of pain severity.

Psychologists are thus interested in affective distress and ask such questions as the following: To what degree is the patient in distress? What are the sources of distress? What is the nature of the distress (e.g., depression, anxiety, fear, frustration, anger, loneliness, helplessness)? What thoughts, feelings, and behaviours are associated with the distress? Is distress expressed primarily through verbal or through physiological channels or through both? Is the patient able to admit to feeling distressed? Does the distress pre-date the pain condition? How is the pain condition contributing to a pattern of distress? Does the level of distress seem appropriate for the level of life problems? How is distress interfering with the management of pain on either a personal or medical level? Can psychological interventions be used to decrease the patient's distress?

Coping style. People tend to deal with life problems in a consistent manner indicative of an underlying pattern or style. Personality variables may influence both the expression of pain and the availability of resources used to cope with pain. Such questions as the following are important to psychologists in the assessment of this area: Does the patient exhibit a style considered to be psychopathological (e.g., characterized by delusions, paranoia, schizophrenia, conversion hysteria, severe anxiety disorders such as obsessive-compulsive syndromes, complex phobias, or panic disorder)? Does the patient display a style which may be extremely characterologically dysfunctional (e.g., personality disorders including extreme dependency, schizoid features, passive-agressiveness, or antisocial disorder)? Does the patient report a problem-solving style productive of low mood (depressive cognitive style), high muscular tension ("Type A" style), high cognitive arousal (obsessive worrying style), or high frustration (low frustration tolerance style)? How intrusive are these styles? Are they likely to exacerbate pain problems? Are they likely to interfere with the patient's ability to benefit from treatment approaches? Does the patient exhibit an adaptive coping style (e.g., uses "relaxation" times or rest periods effectively, takes responsibility for his or her health, develops reasonable expectations of self, others, and of the health care system)? Can the present coping style be enhanced to allow for greater management of pain? Can the patient learn new skills to increase his or her armamentarium of coping strategies?

PAIN

The psychological assessment of pain shares much in common with the medical assessment; that is, psychologists are also interested, for example, in the circumstances surrounding the onset of pain, the consequences of the pain, and the nature and results of various treatments. The differences between the two assessments tend to lie more in greater emphasis on the "meaning" of the pain, on psychosocial influences on the pain, and on psychosocial consequences of the pain.

Meaning. People with pain usually develop a personal understanding of their condition. Dependent on the meaning imposed on the pain, doing so may either help or hinder the individual in his or her efforts to cope with pain. Psychologists tend to seek answers to questions such as the following: To what does the patient attribute his or her pain (e.g., to an accident, an underlying disease process, to stress imposed by his or her life situation, to a "medical mystery", to problems with "nerves")? What feelings are associated with these attributions? What worst-case scenarios might the patient have about his or her pain condition (e.g., wheelchair bound, being an "invalid", dying, feeling that suicide will be the only escape)? Is the pain perceived as controllable? Is the patient's self-worth associated with his or her ability to either avoid pain or to carry on in spite of pain? Can the patient develop a more adaptive meaning system within which to view his or her pain?

Psychosocial influences. Pain conditions usually have four sorts of causal agents; that is, predisposing factors, initiating factors, maintaining factors, and exacerbating factors. An additional influence on the pain condition is those variables which serve to decrease pain. The psychologist is interested in all five classes of variables but emphasizes those of a psychological and social nature. Such questions as the following are asked: Was the onset of the pain associated with a time of significant stress? Are life stressors associated with the exacerbation of the pain? Are there people in the patient's history who may have served as models of how one copes with pain? How do mood and tension affect pain? How are self-management strategies utilized (e.g., resting, hot baths, distraction, exercises, positive self-talk)? Are these effective? What expectations does the patient have of him or herself or of health professionals? How do others respond to the patient's pain (e.g., sympathy, help with activities, disgust, criticism, indifference)? Is the patient obtaining benefits (e.g., financial, emotional, interpersonal) which could potentially serve to maintain a pain condition? Is the patient able to avoid aversive activities by virtue of having pain? Can these influences be altered to either decrease their negative impact or to increase their positive impact? Can new psychosocial influences be added?

Psychosocial consequences. The previous section suggested that some psychosocial consequences of pain (e.g., avoiding activities) may, in turn, serve as causal agents. More often than not, however, it is difficult to perceive any potential benefits which might outweigh the negative impact of pain and of associated limitations: pain, basically is a pain. At times, moreover, the consequences of pain seem to be even more aversive than the pain intensity itself. Psychologists thus ask themselves such questions as the following: What are the effects of the pain on all life areas (e.g., self-worth, mood, relationships, ability to work, take care of the home, take part in recreation, to socialize)? How intrusive is the pain? How does the patient view the interference of pain in his or her life? Can pain be made to be less intrusive?

TREATMENT

The psychological management of chronic pain patients follows from the nature of the assessment; that is, treatment addresses three broad areas including environmental concerns (social supports, and life stressors), psychological functioning (mood, and coping style), and pain (its meaning, psychological influences and consequences). Treatment is based on an understanding of the inter-relationships between those areas. Thus, treatment may focus on the development of social supports to provide the individual with greater interpersonal resources with which to deal with his or her pain. Alternatively (or additionally), it may involve psychotherapy and psycho-educational methods designed to reduce stress either potentially exacerbating pain or, at the least, hampering the individual in his or her coping. In addition, treatment may be composed of psychotherapy to help the patient conceptualize pain in a more adaptive manner in order to reduce its intrusiveness in his or her life.

Treatment approaches range from education about pain and relaxation training, to guided imagery, stress management training, assertiveness training, parent effectiveness training, coping skills training, supportive counselling, cognitive therapy for depression, individual psychotherapy, and marital therapy. In general, however, psychological treatment for pain: a) relies on psychosocial changes processes; b) is designed to foster self-management on the part of the patient; c) is patient-focused rather than technique-focused; d) is tailored to the particular needs and resources of the individual; e) is composed of diverse approaches; f) is directed towards highly interdependent systems (both within the individual and broader than the individual); and g) has a general goal of increasing life satisfaction rather than decreasing pain *per se*. With regards to the latter characteristic, for example, if a patient feels better able to cope with his or her pain as a result of treatment, the latter would be evaluated as effective even if the pain intensity itself was unaltered. The following case examples serve to illustrate the nature of the psychological management of pain.

Case Example A

This 34 year old, married woman was referred to the Psychology Department by the PMU for an assessment of her suitability for the psychological management of pain. Her assessment, by way of interviews and a standardized questionnaire battery, was indicative of continuous pain in her upper back, neck, arms, shoulders and temples, beginning two years previously after being involved in a motor vehicle accident. A number of factors increased pain including activity and tension. Numerous treatments had given little or only temporary relief. Pain was interfering in her life significantly as she was no longer working, felt much less able to take part in recreation and sexual relations, was much more irritable, and experienced considerable sleep disturbance. In addition, she was discouraged and worried about her pain and associated limitations. In general, she felt hopeless and helpless to do much about ther pain.

An assessment of her social situation revealed that she lacked social supports at home. Instead, her husband and teenage children appeared to be a strong source of stress for her. For example, she reported difficulties coping with her husband's moods and with her children's attempts to get "their own way". Psychologically, she appeared to be moderately depressed and significantly anxious. In addition, she lacked coping skills with which to deal with her problems; e.g., she was unassertive, quite vulnerable to manipulation by others (particularly by guilt), easily discouraged and self-pressured (difficulties relaxing).

In all, the individual was seen on 16 occasions. Treatment consisted of supportive psychotherapy, assertiveness training, relaxation exercises, and cognitive therapy for depression.

At termination, treatment appeared to have been most helpful for her emotional difficulties; i.e., she no longer reported depressive symptomatology in the least. In addition, she admitted to fewer difficulties with tension and anxiety. With regards to pain, she indicated that her pain was less intrusive in her life (although she was still not working), that she coped better with it, and she had reduced her pain medication intake by half. She also reported less pain (both in severity and frequency) although this decrease was not marked. Psychological treatment seemed to have been beneficial to this individual in improving her ability to cope with life stressors including pain but had been of less benefit in changing the nature of the pain itself.

Case Example B

This 48 year old, married man was seen over the course of two interviews to assess his suitability both

for psychological treatment and for an invasive medical procedure. His assessment was indicative of pain in his right foot and leg sustained through a work injury six year previous. Pain was made worse by activity and by any position held for any extended period of time. He denied any relationship between pain and either tension or emotional factors. Pain appeared to have been quite intrusive in his life as he was no longer working, and felt less able to carry out household responsibilities, socialize, take part in recreation, or to tolerate frustration. In addition, he reported much sleep disturbance.

As assessment of his social situation was indicative of adequate social supports and of no areas of psychosocial stress other than pain and associated limitations. Psychologically, he denied feelings reflective of either depression or anxiety. The assessment, on the other hand, was indicative of a coping style marked by blame of others (of health professionals in particular), some passivity, and a desire to take each day as it came without addressing psychological processes such as thoughts and feelings.

In general, this individual did not appear to be a suitable candidate for psychological approaches. His lack of either psychological distress or of interest in psychological modalities greatly reduced the probability of his gaining any benefit from such treatment. No sessions were thus scheduled.

Case Example C

This 39 year old, married woman was referred for an assessment of her suitability for the psychological management of headaches. She was seen initially in joint assessment in the PMU with an anaesthetist, and then was subsequently assessed on two occasions in the Psychology Department. Her assessment was indicative of a long history of headaches beginning around age nine without clear precipitants. Headaches appeared to be of a mixed tension-migraineous nature. Very severe headaches accompanied by vomiting and blurred vision occurred around twice a month superimposed against a background of constant dull head pain. Pain was increased by a number of factors including certain foods, driving, walking, bright lights, and tension. In the past, Fiorinal had provided some relief. However, she was anxious to reduce her use of medication. She indicated that pain had interfered with her life in the following ways: sleep disturbance (frequently awakened by severe headaches); less ability to take part in recreation and socializing (difficulties making plans because of the unpredictability of headaches); occasionally having to leave work in the middle of the day; and being more irritable.

Social supports appeared to be quite adequate. Apart from pain, the main source of life stress appeared to be her involvement in numerous professional and community groups.However, she indicated that she derived much satisfaction from this involvement and did not perceive this to be problematic. She did feel, on the other hand, that pain was wrecking havoc with her ability to fulfill all of her responsibilities.

Psychologically, she did not appear to be depressed. A more salient feature was her level of muscle tension. The latter appeared to be self-imposed as she exhibited a coping style marked by difficulties relaxing, an orientation to activity, and a relative lack of awareness of her psychophysiological state; that is, she seemed to lack a sense of when she was over-extending herself.

Following the assessment sessions, this individual was seen on six occasions for treatment. The latter consisted of a stress management program designed to increase both her bodily awareness and her ability to decrease physiological and cognitive arousal. The modalities used included biofeedback, progressive muscle relaxation exercises, autogenic training, and coping skills training. Given that severe headaches often started while she was asleep she was instructed to awaken prior to the usual time of onset and to practise relaxation exercises at that time.

At termination, treatment appeared to have been effective. Not only had she not had a severe headache for some time, her dull headaches were no longer constant. In addition, she was taking much less pain medication, and felt less irritable and fatigued. In general, she felt that the pain was much more controllable.

SUMMARY

Throughout this paper I have pointed to the multidimensional nature of psychological pain treatment. The case examples have illustrated potential approaches as well as diverse results. Clearly, however, short-term psychological approaches are not beneficial for all patients. For example, patients with severely entrenched personality disorders or drug dependencies are unlikely to greatly benefit. Neither are patients who are strongly opposed to the involvement of psychologists in the management of their pain. On the other hand, psychological treatment can often be useful to patients with pain both as an adjunct to other modalities and as a treatment avenue in its own right.

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Post-Herpetic Neuralgia:
CURRENT CONCEPTS AND MANAGEMENT

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The pathogenesis, clinical features and common approaches to the management of post-herpetic neuralgia (PHN) are described.

Of all the acute and chronic maladies to which man is prone, this is surely one of the most debilitating and difficult to treat.

Herpes Zoster was well known to the ancients. This becomes apparent when one considers the origin of currently used terminology.

Zoster = Greek for girdle
Shingles = Derived from the Latin "cingulus" — a girdle

These terms are singularly appropriate for it is a "girdle" which encircles the patient and produces, in acute zoster, the characteristic dermatomal pattern of rash, pain and dysesthesia.

Post-herpetic neuralgia, as defined by the International Association for the Study of Pain, is: "Chronic pain with skin changes in a dermatological distribution following acute herpes zoster."[1]

PATHOGENESIS

The cause of herpes zoster is a small DNA virus known as the varicella-zoster virus (V-Z-V), and it is a member of the herpes virus family. Also included within this group are herpes simplex virus, types I and II, cytomegalovirus (CMV) and Epstein-Barr virus (EBV). Besides being morphologically identical, all these herpes viruses produce persisting infections associated with exacerbations and clinical latency.

Following a juvenile bout of varicella (chickenpox), the virus remains inactive within the dorsal root ganglia of somatic and cranial nerves. After reactivation, giving the clinical findings of acute herpes zoster, the dorsal root ganglion becomes swollen, infiltrated with round cells and generally acutely inflamed. Pathological changes in post-herpetic neuralgia seen within the nervous system include necrosis, hemorrhage, demyelination, wallerian degeneration,[2] neuronal degeneration and scar formation.[3]

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Herpes zoster is thus primarily a neurological disease — this may help to explain its resistance to therapy, particularly since most of our efforts are directed at tissues affected secondarily.

Why some acute zoster cases lead on to chronic neuralgia is unknown. Pain can persist despite neurectomy, inviting speculation about alternative pathways of pain transmission or co-existent involvement of areas higher in the CNS, e.g. the thalamus.

Other investigators have invoked the gate control theory, introduced by Melzack and Wall,[4] to explain the pain. This hypothesis proposes that pain messages carried to the CNS may be modified by the ratio of impulses in small and large unmyelinated nerve fibres. With acute zoster infection it is known that larger fibres, rather than smaller, are destroyed; regeneration of larger fibres is also slower. Hence the normal modulation of pain sensation by these larger nerve fibres is no longer present.[5]

CLINICAL FEATURES

When severe symptoms of pain and dysaesthesia persist six months after the healing of the cutaneous lesions the diagnosis of post-herpetic neuralgia can be made.

Although only 10-15% of patients who have acute herpes zoster will go on to develop post-herpetic neuralgia, the incidence in the elderly is greater and can rise to as high as 50%.[6] Symptoms may resolve within a few months or persist for much longer, even years.

The distribution of dermatomes affected by herpes zoster is well described. Thoracic dermatomes are most commonly affected (55%); cranial and cervical areas make up another 37%. However lumbo-sacral dermatomes can be affected and generalized symptoms may also occur.[7]

Lipton gives a vivid description of the various pain and dysesthetic sensations which can occur.[8] Patients may cut holes in their clothing finding even light touch unbearable. A mild draught can initiate a paroxysm of pain. As a consequence, these patients are frequently anxious and severely depressed. Suicidal ideation is not uncommon. Adding to their distress, those who are elderly may live in personal and social isolation, with little emotional or physical support available.

TREATMENT OF PAIN

We do not have a reliable, proven treatment for post-herpetic neuralgia. Any treatments used should have minimal potential side effects since the chance of providing long term relief is small.[7] It should also be readily tolerated, non-addictive and simple enough to be managed in an out-patient setting.

Initial patient contact must include a careful history and physical examination. This will help reassure the patient and may indicate the presence of other disease states. In particular, conditions which should be looked for are those that produce altered immune status. Patients having radiotherapy, or receiving immunosupressive treatment are also at increased risk for acute herpes zoster. However, most patients will be otherwise well, although elderly. Social and psychological status can also be conveniently explored at this time.

An optimistic yet realistic approach should be taken. Patients should be informed that their pain, although severe and debilitating, is not cancer or a sign of any other fatal disease. They should be told of the neurological cause of their problem, and that progressive slow remission occurs in a majority of cases.

The aims of initial management should be:

i) to eliminate or ease his or her pain;

ii) to treat underlying anxiety, depression and insomnia.

First time treatment involves the use of simple analgesics such as aspirin and acetaminophen. Narcotics in this patient group are generally contra-indicated and singularly ineffective.

Sympathetic blocks with or without steroids remain a mainstay of treatment in post-herpetic neuralgia.[6] Many regional pain clinics have traditionally performed a series of blocks over a course of several weeks. Although the long term effect and rationale of such procedures is often disputed, relief may be dramatic and some patients receive long term analgesia. A recent report has indicated no long term prophylactic benefit in preventing post-herpetic neuralgia with sympathetic blocks when performed in the acute phase of herpes zoster.[9] However, immediate improvement in pain can be achieved.

Tricyclic anti-depressants, particularly amitriptyline and doxepin, are widely used, either alone or in combination with a phenothiazine, e.g. fluphenazine. Recently, evidence has been presented to indicate that the tricyclics have a benefit independent of their serotonergic actions and unrelated to their effect on depression.[10]

Initial doses of amitriptyline (or doxepin which is often better tolerated in the elderly) are 10-25 mg at night, increasing by 10-25 mg until relief is obtained or side effects noted (to a maximum of 75 mg). These doses are less than those usually used to treat depression but are often all that is required to reduce pain and relieve insomnia. Fluphenezine can be added in doses of 1-5 mg t.i.d.

Anticonvulsants such as phenytoin and carbamazepine are sometimes useful when other remedies have failed. However, more general use is precluded because of their inconsistent effect and incidence of adverse reactions.

There are multiple other treatments used for post-herpetic neuralgia. None provide consistent relief. Transcutaneous electrical nerve stimulation (TENS) and acupuncture have helped some patients. They are, at least, safe, simple and inexpensive compared with other alternatives.

Cryotherapy has also been used to produce a local neurolysis of afferent pathways. Again results have ben inconsistent.[11]

Neurosurgical approaches are mentioned only for completeness. Recent success in some cases has been seen with dorsal column stimulation. Such techniques are only warranted in the most intractable cases.

Psychological approaches should probably be used more often. Relaxation training, lifestyle intervention, biofeedback, hypnosis and psychotherapy may be dramatically effective if patient selection is appropriate.

CONCLUSION

Post-herpetic neuralgia is a common complication of herpes zoster infection. Variations in patient selection, absence of good controls, as well as the natural trend to resolution of symptoms, makes most current treatments difficult to scientifically validate.

Initial approaches should be along general lines to provide analgesia. If this does not help, early referral to a pain unit is recommended and other treatment modalities tried, such as sympathetic blocks, epidural steroids, TENS and acupuncture.

Various combinations of drugs (tricyclics, phenothiazines, anti-epileptics) may also be useful.

Psychological and psychiatric approaches will aid selected cases. □

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Myofascial Pain Syndromes

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Myofascial pain syndromes are among the most common, poorly understood, and mismanaged disease entities in chronic pain.[1] These disorders can mimic many classical pain syndromes such as low back pain, temporo-mandibular joint dysfunction, headaches, as well as those involving other parts of the musculoskeletal system.[2]

A major difficulty in understanding these syndromes has been the many names given to them, such as muscular rheumatism, non-articular rheumatism, fibrocytis, rheumatic myocytis, myofascitis and myofascial trigger points, amongst others.[3]

PATHOPHYSIOLOGY

Myofascial pain can begin acutely with trauma, i.e. a muscle sprain, or insidiously, due to chronic muscle fatigue. The exact etiology is unknown, however, various hypotheses have been postulated. Simons and Travell believe that hyper-irritability of muscle tissue at the trigger points increases metabolic demand and impairs circulation. This results in reduction in energy production which causes heightened irritability.

Raj postulates two mechanisms. Firstly, following an acute muscle strain where overloading of the muscle fibres occur and tissue damage is caused, there is release of stored calcium from the sarcoplasmic reticulum. The presence of normal adnenosine triphosphate (ATP) and excess calcium will initiate and maintain a sustained contracture of those fibres exposed to calcium. This produces a region of uncontrolled metabolism within the muscle which leads to decreased circulation locally and shortened muscle fibres. This local vasoconstriction could be a local response or a reflex response involving the autonomic and/or sympathetic nervous systems. As a consequence a taut fibre band develops within the muscle, i.e. a trigger point. Further vasoconstriction may also be mediated by release of substances such as histamine, serotonin, kinins and prostaglandins. A second mechanism may also be involved in the formation of a trigger point whereby total depletion of ATP could lead to conditions similar to others that are known to cause muscle contracture, as in McArdle's disease and carnite deficiency.[3]

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Sola has also hypothesized that this syndrome is sympathetically mediated,[5] and Gunn has noted that trigger points often coincide with motor points (i.e. points where nerves enter muscles) and the terminal branches of the nerves that lie near the skin surface.[6]

Trigger points may be both latent and active. Latent trigger points do not cause pain during normal activity unless palpated locally. Active trigger points which are characterized by local tenderness with a twitch response, give rise to referred pain and referred tenderness, and may also initiate remote autonomic phenomenon.

SIGNS AND SYMPTOMS

Each muscle has a distinctive referred pain pattern specific to the trigger points within that muscle. Most patients present with a composite myofascial pain syndrome that is the sum of a number of individual muscle syndromes. Regardless of the mode of onset, the pain is characteristically steady, deep and aching in nature. The pain can be augmented by strenuous use of the involved muscle, at times with minimal use, passive stretching of the muscle, pressure on the trigger point, cold and damp weather, viral infections, stress and fatigue. There is usually an associated limited range of motion and weakness. This tends to be worse in the morning and recurs after periods of over-activity or immobilization. The involved extremity may feel cold due to reflex vasoconstriction and patients may also show signs of depression and sleep disturbances which in turn can lower the pain threshold.

To examine the patient with a myofascial pain syndrome the patient must be positioned so that muscles can be examined in both the relaxed and stretched condition. Comparison of the affected area with the contralateral muscle group can also be helpful. The examiner looks for tightness, ropeyness and general hyperhydrosis or coldness while palpating muscle tissues, and remains alert for a local twitch response or indication from the patient that the pressure induces a radiating pain that characterises part or all of his pain. The most effective way to locate a trigger point is by palpation with a fingertip along a taut band in the muscle. A local twitch response is pathognomonic of an active trigger point. The trigger point is a circumscribed point of exquisite tenderness. It is not unusual for the patient to jump out of proportion to the pressure applied to the trigger point.

Examination of the affected area will frequently reveal a limited range of motion, weakness, an apparent increase in muscle bulk secondary to muscle spasm, and protective positioning and use of an affected extremity. Neurologic examination, as a general rule, is completely normal. However, there are some myofascial pain syndromes whereby nerve irritation (entrapment) can occur, i.e. pyriformis syndrome where the sciatic nerve is irritated as it passes through the pyriformis muscle. Referred pain in myofascial pain syndrome at times does not follow the usual dermatomal patterns and can cause confusion, and pain is often diagnosed as psychogenic.

INVESTIGATIONS

Routine laboratory investigations show no significant abnormalities and both radiography and CT scans are not helpful. EMG may occasionally be useful showing some insertion potential and an increased number of polyphasic potentials in muscles with trigger points. Thermography has also been of use demonstrating areas of either increased or decreased skin temperature over the trigger point. A good history, physical examination and a high degree of suspicion are the most useful tools in delineating these syndromes.

CLINICAL EXAMPLES

a) Headache.

Powerful trigger points can often be located in the musculature of the upper back and neck in patients with chronic headache. Trigger points within the splenius capitis and semi-spinalis capitis muscles are commonly seen. Pain arising from these trigger points may radiate from the occiput over the top of the head and may be reported as having the focus of pain at the vertex. If the upper part of splenius cervicis also has trigger points, the patient is likely to report pain in and around the eye with blurry vision.

b) Temporomandibular Joint Pain.

Trigger points within muscles of mastication, i.e. points within lateral and medial pterygoid, masseter, etc. commonly produce pain that is centered at the TM joint and can be confused with TMJ dysfunction or arthritis.

c) Low Back Pain.

Trigger points within the longissimus thoracis, ileocostalis lumborum and multifidi produce pain with the low back and buttock. A trigger point within the pyriformis will produce pain over the lateral aspect of the thigh and the posterior iliac crest with radiation throughout the posterior buttock and thigh.

TREATMENT

Due to the chronicity of the pain in these syndromes, most patients will manifest not only the signs and symptoms of a myofascial pain syndrome but also exhibit many signs and symptoms of a chronic pain syndrome, i.e. depression, sleep disturbance, inappropriate consumption of medications and the social and financial complications seen in these patients.

The mainstay of treatment in these patients includes:

a) Injection of local anaesthetics into the trigger point after careful localization.

b) Passive stretching of the involved muscle(s).

c) Stretch and spray procedure. A vaporcoolant is sprayed on the skin while the muscle is passively stretched.

d) Dry needling or classical acupuncture into the trigger point have also been demonstrated to be effective.

e) Adjunctive physiotherapy including transcutaneous nerve stimulation, biofeedback and deep friction massage has also been used.

f) Other drugs that have been used with success include tricyclic anti-depressants in low bedtime doses, i.e. 10-75 mg qhs. Corticosteroids with a local anaesthetic are advocated for two groups of patients only, those with soft tissue inflammation and those with post-injection soreness.

These patients initially need to be seen frequently and require detailed examinations on each visit to localize trigger points. It is not unusual to uncover additional trigger points that will also need attention as treatment progresses. If the pain only responds temporarily, or not at all, it is necessary to look for other underlying disease states.

PROGNOSIS

In the absence of perpetuating factors, trigger points may resolve spontaneously, particularly if a muscle is given rest over several days or a week. If perpetuating factors such as chronic mechanical stress, structural inadequacies, i.e. a short leg or a small hemi-pelvis, etc.; poor posture or prolonged immobility are present, these trigger points may become chronic, more hyperirritable, and the trigger points may propagate themselves as satellite and secondary trigger points. The severity and distribution of pain will consequently increase. Unless the myofascial basis of the pain is recognized and treated and the perpetuating factors are resolved, the prognosis for complete return of function is grim. Reversal of this process after it is well advanced can be challenging and time-consuming. Nutritional, metabolic and endocrine inadequacies may also play a role in perpetuating myofascial pain syndromes.

SUMMARY

Myofascial trigger points have been recognized as a common source of myofascial pain. The diagnosis

is suspected when the onset of pain is associated with muscular strain and when the patient's distribution of pain fits recognized myofascial pain patterns. A trigger point is located by palpation of a tender spot in a taut band of muscle, restrictive range of motion and slight weakness of the affected muscle. It is confirmed when pressure on the trigger point reproduces the patient's pattern of pain or at least part of it, or when a local twitch response occurs. Myofascial pain syndromes are treated by specific myofascial therapy using injection of trigger points, passive stretching, vapocoolant sprays, and physical therapy. It is important to treat the underlying perpetuating factors which may be mechanical, emotional or nutritional. □

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The Use of Psychotropic Medications in Treating Chronic Pain

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Because of problems with dependence, tolerance and sedation, it is important to look for alternatives to narcotics when treating chronic pain of nonmalignant origin.

This article will review the current status of psychotropic medications in treating chronic pain.

TRICYCLIC ANTIDEPRESSANTS

There is now a large body of research supporting the use of tricyclic antidepressants (TCAs) in the treatment of chronic pain. Several good reviews have concluded that the majority of controlled trials indicate TCAs have significant value in treating chronic pain.[1 2 3 4] These studies have included many different types of chronic pain and have used several different TCAs including doxepin, amitriptyline, clomipramine and imipramine.

At first, it was thought that the analgesic effect of TCAs on chronic pain was secondary to their action on an underlying depression. There is now considerable clinical evidence that TCAs possess analgesic effects that are unrelated to their antidepressant effects. Feinmann found that a significant number of controlled studies indicated that a decrease in pain was not associated with improvement in depression.[3] She suggests that pain relief may be due to an independent analgesic effect. Other arguments supporting an independent analgesic action involve dose and time of onset of effect. In general, doses of 75 mg per day or less are adequate to provide analgesic effect, i.e. while doses of 150 mg per day or greater are necessary for antidepressant effects.[4] The analgesic effect of TCAs usually occurs within a few days which is much sooner than most antidepressant effects are noted.[1]

There is also evidence from animal research that TCAs have a specific analgesic effect. Animal studies have demonstrated that TCAs not only potentiate morphine induced analgesia[1 5 6] but also have direct analgesic effects.[1 5 7 8]

What is the mechanism of action of TCAs in treating pain? There is strong evidence supporting a role for serotonin in the mediation of pain.[4 9] A drecrease in serotonin leads to increased pain and this can be reversed by administration of fluoxetine which selectively blocks the reuptake of serotonin thus increasing serotonin levels.[1] Administration of a serotonin antagonist eliminates the analgesia induced by clomipramine.[7]

There is also work to support possible involvement of opiate[5 8] and alpha-adrenergic[6] mechanisms. The research is ongoing and far from conclusive.

PHENOTHIAZINES

There are many reports in the literature supporting the use of phenothiazines, alone or in combination with other agents, in the treatment of chronic pain. Many studies lack appropriate control groups and are fraught with other methodological difficulties. A recent and extensive review[10] has concluded that there is absolutely no evidence to support a role for phenothiazines in treating pain.

The exception is methotrimeprazine (MTZ). MTZ has been found to have analgesic effects in animal[11] and human studies.[10 11] An extensive review of 10 controlled studies found MTZ had clear and significant analgesic effects in the majority of cases.[10] Two of these studies examined patients with chronic pain and both found that the analgesia provided by MTZ was not significantly different from that provided by morphine. It was estimated that the analgesic potency of MTZ, on a weight basis, was approximately half that of morphine. Long term use runs the risk of precipitating tardive dyskinesia.

GABAERGIC MECHANISMS IN PAIN. IS THERE A ROLE FOR BENZODIAZEPINES?

It has been demonstrated that both directly acting GABA agonists and indirectly acting GABAergic agents can provide analgesia in a variety of animal models.[12 13] There has been some work done in humans. THIP, (4,5,6,7 tetrahydroisoxazolo [5,4-c] pyridin-3-ol) a GABA A agonist has been found to have analgesic effects in humans.[14] Unfortunately THIP has dose-limiting side effects which have led investigators to conclude THIP is not useful for treating chronic pain of malignant origin.[14]

Baclofen is a GABA beta agonist and has been found to reduce the pain associated with spasticity caused by damage to the spinal cord.[15] It is not known yet whether this is an indirect effect secondary to decreased

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spasticity, or whether baclofen has some direct analgesic effect. Baclofen has also been found to be useful in treating trigeminal neuralgia.[16]

Although Hendler postulated that by inhibiting serotonin release benzodiazepines might theoretically increase pain perception, recent work demonstrating GABAergic mechanisms in analgesia, raise the question of whether benzodiazepines, which increase GABA actity, may have analgesic effects.

There has been very little work done in this area and it is too early to draw any conclusions.

CONCLUSIONS

There is a definite role for tricyclic antidepressants in the treatment of chronic pain. One should use much lower doses than those used in treating depression. An accepted starting dose is 10 mg po before bed. This may be gradually increased to a total dose of 50-75 mg po at bedtime. The drugs most studied to date are doxepin, amitriptyline, clomipramine and imipramine. It is necessary to start with very low doses because patients who are not clinically depressed experience an increased number of side effects such as dry mouth, sedation and blurred vision.

There is no role for phenothiazines in treating pain. The possible exception is methotrimeprazine which may be useful for short periods of time in patients experiencing nausea and vomiting or agitation along with pain.

It is too early to draw any conclusions regarding the use of benzodiazepines in treating pain. We await the results of ongoing work on GABAergic mechanisms in analgesia and controlled drug trials in humans. □

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A Proposed Mechanism of Phantom Limb Pain Based on Theories, Clinical Observations and Experimental Evidence*

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Phantom limb pain (PLP) is pain which is referred to the missing or the denervated part of a limb. Although there is much variation in the severity and quality of the pain, Jensen, *et al.* described two pain types commonly found in PLP.[1] One of the types is acute pain which is periodic, severe, sharp and knife-like, and is most prevalent just after surgery. The other is chronic pain which is cramping, pressing, crushing, or burning; this pain does not normally occur until quite some time after surgery and may mimic pre-operative pain in some cases. Phantom limb pain is seen following deafferentation injuries, eg. surgical amputation, peripheral nerve injury or spinal cord injury.

Recent surveys indicate that severe PLP in amputees is much more prevalent than the previously reported frequency of 5 to 10%. Sherman and Sherman showed in statistically valid surveys of American veteran amputees, that 78% of 2,500 respondents to their questionnaire had persistent PLP severe enough to require temporary withdrawal from work or social environments.[2,3] Assuming all non-responding veterans were free of PLP, then the lowest value of veterans with significant PLP would be about 46% which is still a substantial percentage.[5] Jensen *et al.* through the use of prospective studies, in which civilians were chosen before their amputations, obtained support for the previously stated range of values for the incidence of PLP. Of 58 patients subjected to amputation, 72% had PLP 8 days after surgery and 67% had PLP after 6 months.[1] In another 86 patients followed for 7 years, 54% had PLP.[4] From the above mentioned data, as well as from the observations of a comparative study, which demonstrated that there are no differences in the characteristics of PLP from amputations of civilian and military origins, and between PLP in men and women, an approximate value of 50% for the percentage of amputees with *severe* PLP was obtained.[5] Factors which could account for the earlier reports of low incidences are the reluctance of patients to seek medical help because they felt that they would not be taken seriously for having PLP; that they would be subjected to further ineffective treatment, and a medical lack of awareness of the problems of PLP. Surveys and studies also indicate poor results of treatment in PLP.[5,6] All these studies suggest that PLP is a significant problem to many amputees; and understanding of PLP's mechanism might prove useful in its treatment.

PERIPHERAL FACTORS

One explanation for phantom limb pain is abnormal afferent stimulation from various nerves in the stump. Stump pain is normally more common in amputees who experience PLP than in those who do not have PLP.[5,6] Nystrom and Hagbarth demonstrated that mechanical tapping of the neuroma of the major injured nerve resulted in a sharp stump pain in the neuroma as well as accentuation of a persistent type of phantom limb pain.[7] Microelectrode recordings of the transected nerve showed that tapping of the neuroma induced an increase in neural discharges which was correlated with the pain; furthermore, a local anesthetic injected into the neuroma eliminated these discharges, as well as the stump pain and the accentuation of the PLP. Thus, neuroma induced stump pain and PLP appear to be due to increased firing of afferent nociceptive fibres (c fibres and A-delta fibres) which comprise the neuroma of the damaged nerve.[8] Similarly, the smaller neuromata of cutaneous nerves could also play a part in PLP and account for the different characteristics and location of PLP. The immediate onset of acute PLP at and after amputation has also been correlated with an increase of firing in the damaged nerve which persists for at least two hours.[8] Lindahl believed that the increase in firing of an injured nerve was due to artificial synapses between efferent and afferent nerve fibres; but such ephaptic connections have been rarely found by other investigators.[9]

PLP can still occur without stump pain. Experimental evidence in animals[9] and in amputees[7] indicate that the tissue around the neuroma and not the neuroma itself has ongoing, spontaneous firing. In a comparison of the characteristics of firing, this type of spontaneous firing is different than the bursting and increased firing seen in fibres of a neuroma, and it also differs from the normal firing seen in people without injured nerves. This ongoing spontaneous firing was not blocked by injection of Lidocaine into just the neuromal tissue and not the other surrounding tissues. An explanation for the continuing impulses

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measured in tissues around the neuroma is the increased sprouting from axons of a damaged nerve. As a result of the sprouting, there is an increase in free terminals as well as an increase in density and complexity of innervation of the remaining part of the limb.[2] This would cause an increase in the number and complexity of the pattern of sensory impulses which are a potential source of pain. Scar tissue is often a source of pain: if the sprouts went into scar tissue from the amputation, there could be nociceptive transmission.[10] From sprouts the transmission of pain would occur along the axon from which the sprout originated and would be interpreted as being from the area which this particular sectioned nerve previously innervated.

Support of a peripheral mechanism as a source of PLP comes from the complete relief of PLP following local anesthetic block of all nerves to the stump, and its return when the anesthetic wears off. Rarely does this anaesthesia fail to give relief. Pain normally recurred with reappearance of sensitivity.[8] Wall and Gutnik[9] showed that peripheral impulse generators in neuromas and fine sprouts of damaged nerves are sensitive to blood flow, enforced activity, and sympathetic amines; i.e., stimulation of efferent sympathetic fibres caused an increase in firing rate of associated nociceptive fibres (Sherman and Tippens).[11]

Sunderland suggested that before amputation a local anaesthetic should be administered to tissue proximal to amputation site so as to decrease bombardment of central neuronal pools by abnormal impulses produced by cutting.[6] He also recommends that during amputation surgery, prevention of scarring, a good skin flap design, preservation of cutaneous innervations and treatment of nerve ends to prevent neuromas from forming should definitively be attempted. Furthermore, the stump should be treated first as the underlying cause of pain.

SPINAL CORD MECHANISMS

Many structural, physiological and neurochemical changes occur in the dorsal horn of the spinal cord after the sectioning of a peripheral nerve.

The following changes have been reported:

1. Enhanced sensitivity to remaining synapse and to neuroregulator substances due to decreased threshold of substantia gelatinosa cells.

2. Progressive depletion of neurochemical substances such as substance P (a primary afferent neurotransmitter of pain, which is depleted by 5 to 9 days in rats) and fluoride resistant acid phosphatase (FRAP) from the substantia gelatinosa.

3. Reduction of primary afferent depolarization.

4. Atrophy of some afferent terminals and spinal cord cells.

5. Decrease in dorsal root potential evoked on neighboring dorsal roots (occurs by 10-20 days in rats).[9]

6. Change in receptive fields of spinal cord neuron which have lost their normal afferent input.[12]

Deafferentation results in a decreased sensory input because the CNS is no longer receiving sensory information from the limb parts which were distal to the damaged nerve. A decrease in amplitude of input volleys in afferent fibres of a cut nerve occurs approximately between day 10 and day 20 after proximal nerve section in rats. Except for the temporary effects of local anaesthesia, there is no pure model of deafferentation in which there is no afferent information; however, the remaining afferent impulses after deafferentation are different from the normal impulse which is dispersed and patterned. After amputation there is partial deafferentation; the proximal end of a cut nerve retains the power to excite post synaptically the secondary afferents of the dorsal horn with the possible transmission of nociceptive impulses. Some of the fibres in a sectioned nerve degenerate and the primary afferent presynaptic terminals atrophy, and conduction in these fibres is blocked. Other axons of a sectioned nerve regenerate in a disorderly manner into a stump tissue neuroma or into surrounding stump connective tissue by means of multiple, wandering sprouts from each non-atrophied axon.[6] After nerve section axon sprouts begin to grow approximately two days later but neuromas do not develop until three weeks after section.

Levitt's explanation of PLP is by spinal cord mechanisms.[13] The concept is as follows: damage to the limb from amputation results in an increase in abnormal firing of peripheral nerves; this initiates abnormal firing patterns in self-exciting neurons in the spinal cord which transmit volleys of nerve impulses to the brain which result in pain. Reverberatory activity may spread to the ventral horn and produce movement in stump which in turn produces increased sensory input which results in a cycle. Once self-sustaining firing in spinal cord cells has begun, surgical removal of peripheral source of input may not stop the PLP. A decrease in nociceptive input may reinstate normal cord activity and thus produce relief. High frequency non-painful transcutaneous electrical nerve stimulation (TENS) may increase activity of large fibres, inhibiting afferent information from small fibres from reaching the brain through a gate control mechanism, or alternatively, local anaesthesia of entire stump may produce an overall decrease in sensory input, and relieve pain.

Howe proposed that some of the spontaneous activity in the dorsal horn may be due to the formation of new mono- or poly-synaptical connections between excitatory low-threshold primary afferents from adjacent and distant receptive fields and deafferentated, high-threshold nociceptic second order dorsal horn neurons.[14] Howe believes this reafferentation is the cause of PLP and that the reafferentation is secondary to neural plasticity which follows deafferentation injury. In cats, similar reorganization of the spinal cord sensory map after injury was observed. The primary afferents which synapse are either from new growth or nearby intact afferents within the spinal cored, or from previously weak primary afferents which become effective because of the loss of large fibre inhibition as a result of deafferentation. Reafferentation takes about 10 days in rats and 28 days in cats. False localization and false identification, which suggests new sensory fields, is often associated with deafferentation and PLP, and can be explained by reafferentation. Afferent information from a neuroma could be a factor because nerve endings in neuromas were sectioned prior to experiments. Surgical destruction of the dorsal horn entry zone (DREZ) would destroy most of the dorsal horn; this method is definitely effective for almost complete elimination of pain.[15]

CENTRAL FACTORS

There is evidence that other factors may contribute to PLP. Melzack in 1974 advanced the concept of a central biasing mechanism to explain PLP. The concept is that the brainstem which exerts a tonic inhibitory influence on transmissions at all synaptic levels of the somatasensory projection system.[16] This belief is supported by evidence of descending anatomical pathways from the reticular formation to the spinal cord where modulation of neurotransmission in the substantia gelatinosa of the dorsal horn occurs. In amputees, there is a decrease in the amount of normal skin sensory inputs into the reticular formation. This type of input normally maintains the tonic inhibitory influence; thus, where there is a decrease in normal sensory input, there will be a decrease in inhibition. The spino-reticular projection is important in pain transmission because there must be a certain amount of afferent impulses up this tract to permit inhibitory influences to have an effect on dorsal horn cells.

Melzack's concept of pattern generating mechanisms can explain the existence of persistent PLP even when all sensory impulses from the periphery are blocked following complete cord transection. The concept is as follows: any peripheral input would initiate abnormal firing and increased output from dorsal horn nerve cells because there is no, or decreased inhibition. This resulting abnormal firing pattern in neural pools could become self-sustaining at all levels of the CNS.[17] These hypersensitive neuronal pools all along the CNS are now susceptible to impulses from various pathways. Pain occurs when output from self-sustaining neuron pool reaches a critical lelvel. This aspect of the theory explains prolonged PLP where a critical level is maintained from within: it also explains the role of trigger zones in generating a critical level of stimulation through external stimuli which increase the firing rate of these neuron pools. Normally, dorsal horn cells responding to nociceptive afferents are modulated by both large fibre input from peripheral afferents and descending inhibitory fibres from the reticular formation. However, in amputees, modulation is mainly from the reticular formation because most of the large fibres have been observed to be destroyed.[7]

Further support of Central Biasing theory and Central Pattern Generating theory comes from clinical observations. For example, neurosurgical lesions often produce a new pain or aggravate the pain that the lesion was intended to diminish.[17] This is because further nerve sections, i.e. partial deafferentation by neurectomy, rhizotomy and cordotomy, would simply provide conditions for a decrease in centrally controlled inhibition because of the decreased afferent input, and then a new location of abnormally firing neurons in the CNS would become self-sustaining. Normal patterned inputs would be produced from movement of muscles by manipulation of stump, use of prothesis, or electrical nerve stimulation which are some of the effective treatments for PLP. These patterned inputs would be out of phase with the abnormal, spontaneous bursting of the neuron pool; thus, these treatments would disrupt the activity of the neurons resulting in relief of pain. Following treatment, the normal motor activity of a person when pain free would maintain normal pattern until some stimulus caused return of bursts of activity to neuronal pools.Induced PLP in amputees previously pain-free and even in patients with intact limbs have been reported during conduction anaesthetics.[18] This condition may occur in paraplegics, who sometimes have PLP.[17] These cases can be explained by Melzack's concepts: because of very low afferent information, Central Biasing mechanisms are activated and then release Pattern Generating mechansisms which result in pain.

Evidence for parts of the brain other than the reticular formation being involved in PLP exist. The cortex with its "map of the body" appears to play a role in PLP because of surveys which show that the location of the pain is mostly in the foot and hands.[1 3] These areas of the extremities have the largest representation in the central image. Phantom limb pain often has the same characteristics as that experienced before amputation, and PLP is more

common in patients with preoperative pain.[1,4] These two observations concerning preoperative pain can be explained by the establishment of a nociceptive memory engram of a particular pain in some cortical structures so that the pain continues even when isolated from its peripheral input.[19] Transneural changes from deafferentation have been observed as far as the thalamic nucleii.[6]

NEUROCHEMICAL FACTORS

A specific biochemical change has been proposed as a cause of PLP based on various experiments and pharmacological explanations. Rat models of PLP produced by peripheral nerve section were used in both experimental and control groups. When a monoamine oxidase (MAO) inhibitor was administered to the experimental but not the control rats, autotomy, which is the self-mutilation of a painful or damaged limb in response to the pain of PLP, was markedly facilitated in the experimental rats compared to control rats in whom autotomy was also present.[10]

The MAO inhibitor would cause an increase in dopamine, serotonin and norepinephrine. The increase in PLP cannot be attributed to an increase in dopamine and serotonin because an increase in these neurotransmitters would cause a decrease in pain.[21] However, the increase in PLP could be due to an increase in norepinephrine (NE) in the synapse. MAO inhibitors would prevent degradation of NE by MAO and thus an increase of NE in the cell would coccur and more NE would be released into the synapse. NE is capable of increasing nociceptive activity in injured peripheral nerves. NE is also released from neurons in the brain; NE is released from the lateral reticular formation of the medulla. The effects of the induced increase in NE being related to PLP are supported by NE's ability to reduce morphine's analgesic effect when injected intracerebroventicularly and the unresponsiveness of deafferentation pains, such as PLP in humans, to morphine for pain relief.[13] Conversely, inhibition of NE synthesis; i.e., decrease amount of NE in synapse, results in a significant increase in analgesia produced by electrical stimulation in the mid brain. There is also a peripheral location where increase in NE seems to have an effect on PLP; this is based on the observation that when NE was injected around the neuroma there was an increase firing rate of the fibres of the injured nerve.[8]

Increase of NE as a probable source of PLP is supported by dramatic response to chlorpromazine in a patient suffering for 30 years from persistent PLP of two types; i.e. lancinating and gnawing. This PLP had not responded to most common treatments. Although chlorpromazine is used mostly as an antipsychotic agent, this patient was evaluated and found to be mentally stable.[22] Chlorpromazine is a phenothiazine which can block post synaptic NE receptors; therefore, there would be a decrease in NE entering post synaptic neurons which would cause a decrease in pain transmission. Major support of chlorpromazine as working in the brain and acting as anti-NE agent comes from the increase in reticular brain stem activity seen when taking chlorpromazine. Chlorpromazine has also been reported to be effective in the treatment of thalamic pain syndromes.[23] Chlorpromazine also has peripheral anti-NE activity where it blocks sympathetic adrenergic receptors. Carbamazepine has a similar effect to chlorpromazine in the brain; carbamazepine causes a decrease in NE because it inhibits uptake and release of NE from brain synaptosomes. Elliott, Little and Milbrandt found carbamazepine best for lancinating type of PLP.

SUMMARY

In seeking to elucidate possible mechanisms for the generation of PLP, evidence has been produced that peripheral factors, changes in the architecture, connections and neurochemistry of the dorsal horn region, changes in descending inhibitory modulation, the postulated existence of central biasing mechanisms and neural engrams (CNS tape recordings) may have a role to play in the generation of PLP from time to time. The plasticity of CNS responses over time is becoming clearer, and suggests that at any given moment, one or two of these mechansism may be dominant, but that with the passage of time there is a tendency for mechansisms to gradually move centrally and become more fixed.

The multiplicity of treatments which may be at times successful in treating PLP and the overall low success rate of any one treatment attest to the complexity and multifactorial origins of this condition. Through a knowledge of the possible background mechanisms at work it may be easier to evolve a logical plan of progression of treatments that may offer a higher overall likelihood of success. □

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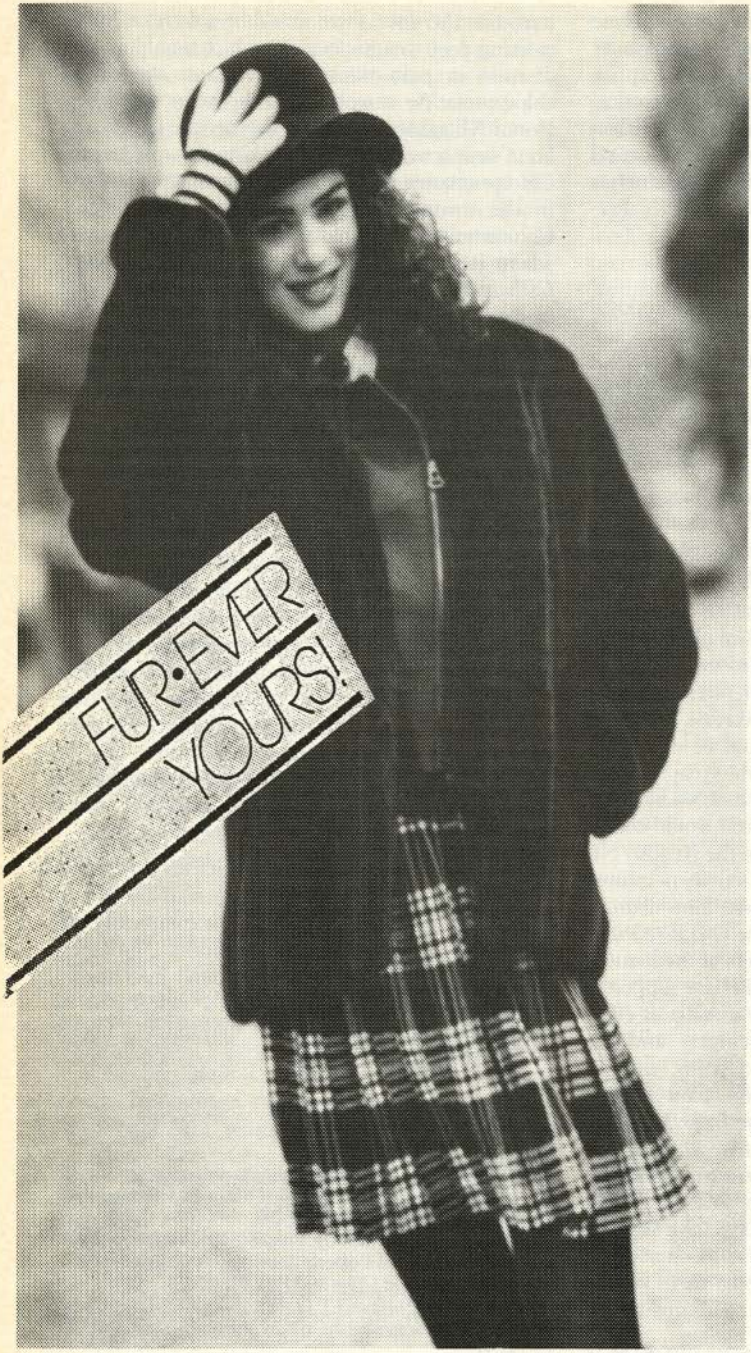

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Cancer Pain Management

AN OVERVIEW

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People with cancer need not suffer pain, but they do. Why is this so? The World Health Organization declared the year 2000 as a target date to achieve universal cancer pain control. Unfortunately this is unlikely to be achieved even in highly developed countries, let alone poorer nations.

The capability of achieving this state is technically not far from us today, but it is not being achieved for several reasons. These include: lack of knowledge, interest and organization. Fortunately, however, this is changing.

In order to control cancer pain, one must have an understanding of the causes and nature of the pain. More than 60 percent of cancer sufferers have significant pain if untreated. This leaves, however, more than 30 percent who do not. When pain is present, in many instances the cause is multifactorial and also more than one pain may be present. For example, pain can result from direct tumour invasion, be associated with treatment, bodily dysfunction or from recumbancy and prolonged inactivity.

Twycross reviewed the number of pains suffered by cancer patients; many had 4 or 5 different pains simulataneously and some up to 8.[1]

TABLE I

SOME CAUSES OF PAIN

Related to the Tumour	Related to Treatment
Bone Pain	Post-operative Pain
Nerve Compression	Colostomy
Soft Tissue Infiltration	Nerve Blocks
Visceral Involvement	Adhesions
Muscle Spasm	Radiation Fibrosis
Lymphedema	Oesophageal Pain
Raised Intracranial Pressure	
Associated Pains	**Incidental**
Constipation	Myofascial Pain
Bowel Cramps	Low Back Pain
Bedsore	Osteoporosis
Catheter Spasms	Joint Stiffness
Pathological Fractures	Traumatic

CAUSES OF CANCER PAIN

In discussing the causes of pain, there are some that warrant a closer look.

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Bone Pain can cause extreme problems and yet is often potentially manageable. It is caused by compression, expansion or lysis by the tumour, sometimes with resulting pathological fractures. Prostoglandins are released as a result of osteolytic tumour activity and play a significant part in pain medication.

Nerve Pain may result from compression of collapsed vertebrae of pathological fractures. Direct tumour compression, stretching, invasion and ischaemia, are all frequent causes. The pain may result from single nerve involvement or from groups of nerves such as the various nerves plexi. Characteristic pain patterns may result with associated loss of function such as that of an arm or leg.

Referred Visceral Pain, from internal organs such as the stomach, pancreas and ureter may be felt at somatic, cutaneous sites with devastating effect. These structures arise from embryologically corresponding areas to the somatic presentation of the pain. A classic example, of course, is shoulder tip pain, resulting from diaphragmatic irratation.

Myofascial Pain, originating from muscles and their associated structures, often occurs as a result of muscular imbalance due to postural problems, tension or awkward positions adopted by patients by necessity to avoid other pain stimuli or by paralysis and contracture.

Thalamic Pain, a notoriously difficult problem to manage, may result from metastatic tumour invasion. This presents a diffuse pain on the contralateral side of the body.

Dysaesthetic and Deaffrentation Pain present as severe burning pain, unresponsive to normal analgesia. Dysaesthesia frequently results from sympathetic nerve over activity while deafferentation pain results from the destruction of a peripheral nerve, nerve plexus or spinal cord. The nerve damage may be complete or partial resulting in pain being felt in areas of altered or absent sensation.

ASSOCIATED CAUSES

Briefly outlined above is the organic aspect of the mechanism of cancer pain, but as indicated in the table, other factors play a significant part in contributing to the pain state. These factors include general bodily dysfunction and, perhaps more significantly, the psychological and spiritual aspects as they affect the patient.

Physical Aggravators of the Pain. Gastrointestinal problems of excessive or absent saliva, nausea and persistent vomiting, bowel obstruction with distension or constipation, all may add greatly to the discomfort level or potentiate the overall pain experience. Bladder and catheter discomfort, incontinence and soiling all add to the misery, but can be effectively managed. Contractures and bed sores serve only to aggravate the situation.

Psychological Factors. These have been even greater disturbing effects on the cancer sufferer, greatly aggravating any existing organic pain. Upon learning about the diagnosis of cancer, various intense emotions may be experienced. These frequently include initial denial, followed by anger, isolation, loneliness, depression and then sometimes acceptance. Each of these is a major psychological event and may cause marked lack of sleep and exhaustion on top of the effects of the disease process itself, thus disabling the patient further and reducing their coping ability. Sleep lack is particularly potent in this effect.

MANAGEMENT OF CANCER PAIN

In order to help the sufferer cope with his/her pain, the whole person must be considered. This in turn requires that their environment, both physically and emotionally, be attended to. The physical environment desired by most people is home; failing this, a quiet, pleasant hospital or hospice environment may be the next best option. Emotional support should be available from family and friends as well as experienced hospice staff. The hospice staff will often have to care for the family members as well.

Fears, Anxieties and Myths. These should, if possible, be tackled openly and clarified. There are many myths with respect to pain and cancer and these include such ideas that all cancers cause pain; that morphine is addictive and must be reserved for use at the last minute only; and that the dose will inevitably escalate with excessive side effects.

Many of today's problems would disappear with greater knowledge, understanding and, regretably to say, interest by some sections of the health profession. Fortunately things are changing with appropriate effect.

Pain, being multifactorial in aetiology, must therefore be tackled from several aspects. Some of these have just been discussed.

PHYSICAL PAIN CONTROL

The mainstay of pain control is by oral medication. Initially simple, non-narcotic analgesics should be used, increasing the potency of the drugs only as required.

The drug regimen is of great importance. It is much easier, and requires less total analgesic dose to keep pain under control than to constantly attempt to regain control. *Therefore regular dosing is essential.*

If simple analgesics, such as Tylenol® or aspirin are insufficient, minor narcotics such as codeine can be substituted, As a general rule, combination drugs should be avoided since change in the dose of one component cannot be done without the other and this may not be desirable. It is better to administer different drugs separately.

Strong Narcotics. Drugs such as morphine should be introduced as soon as required. Regular dose scheduling is again the rule with the exception of occasional use of additional medication for breakthrough pain. However, if such additional medication is required frequently, then the regular dose is inadequate and should be increased.

The oral route of administation should be used where possible, but other routes have a definite place and will be discussed later.

Most cancer pain is best managed by morphine. Demerol® (meperidine) is too short-acting and offers no advantage. Diamorphine® (heroin) is converted in the body to morphine before it is biologically active.[2] Its only advantage over morphine is in the injectable form where it is in a more concentrated and, possibly, less irritant but the oral or rectal routes have no advantage over morphine. Heroin can be administered only by specially licensed practitioners and has the disadvantage of being very popular as a street drug. Hydromorphine (Dilaudid®) is a synthetic opioid with similar advantage to diamorphine in the injectable form and is readily available in Canada. It is also available as an oral preparation while Diamorphine® is not.

M.O.S. Morphine sulphate syrup, M.O.S., is the preferred drug to initiate potent narcotics, and frequently remains the mainstay of treatment. Tablets are available but are less flexible when dose titration is required. The dose should be increased until control of pain is achieved and the starting dose depends on the age and general condition of the patient. For patients previously not on strong narcotics, this should generally be 10 mg every four hours, except for the frail patient, where the dose should be 5 mg. If the patient was already taking a strong narcotic, the dose may be adjusted according to the equivalent tabled below.

The dose should be increased by 50% to 100% after 24 hours if pain control is little better than before, then tailored more gradually (10%-30%) when control is approaching.

The following is an example (in mg): 5 -10 -15 -20 -30 -40 -60 -80 -100 -120 -160 -200, etc. every 4

hours. Constant review is required while titrating the dose, with changes made daily or even more frequently.

Respiratory depression is not a problem unless the dose is greater than that required to control the pain, or the pain is relieved by other means, in which case the dose must be reduced.

At this point, long-acting SUSTAINED RELEASE MORPHINE (M.S. Contin) can be substituted with the same total 24 hour dose but administered every 12 hours. Apart from simple administration, a more even serum level is achieved with fewer side effects.

Twelve hourly dosing must be observed although shorter acting morphine may be used for breakthrough pain but such pain often indicates a need to raise the overall dose. These long-acting tablets must not be crushed or bitten, since this destroys the sustained release effect.

The side effect of sedation associated with morphine is often only an initial effect and will diminish within a few days of use.

TABLE II

ORAL DOSE EQUIVALENTS

Analgesic	Potency	Duration (hours)
MEPERIDINE (Demerol®)	0.125 (1/8)	2-3
PAPAVERETUM (Pantopon®)	0.66 (2/3)	3-5
MORPHINE	1	3-5
OXYCODINE (Percodan®)	1	3-5
DIAMORPHINE® (Heroin)	2	3-5
METHADONE	3-4	6-8
HYDROMORPHONE (Dilaudid®)	6	3-4

Alternative Routes of Administration. If the oral route is impossible or results in side effects such as nausea, then the rectal route using suppositories should be considered. Intramuscular and intravenous routes should be confined to short term usuage only.

a) Subcutaneous Administration. This route, by continuous syringe pump infusion, offers several advantages. Administration is by simple small diameter (23 or 25 gauge) butterfly needle easily placed subcutaneously. It may be readily changed every 3-7 days with little technical knowledge. Since the dose is continuously administered, troughs and peaks in serum levels are avoided, allowing a lower total dose for the same pain control. The pump itself is portable, being very light with preset or variable programs, allowing it to be attached to clothing and permitting full ambulation where possible. Home care is facilitated requiring health care attention infrequently, at the most, once in 24 hours.

b) Epidural Administration of Narcotics. Like the foregoing, it is used where other routes are unsuitable. However, it may allow greater pain control in some instances, especially lower half of body pain with considerably lesser doses. The spinal cord has opiate receptors and it is at these sites with this route of administration, that the narcotics work. Unwanted side effects of sedation, nausea and vomiting are often avoided. Technically, however, they are more involved to set up, but once in operation they can be as easy as the subcutaneous route to manage.

Analgstic adjuvants and Co-Analgesics. The non-steroidable anti-inflammatory analgesics (NSAIDS) are particularly helpful in bone pain especially because of their ability to inhibit prostagladin release. They may be used alone or in addition to narcotics.

Tri-cyclic anti-depressants in low dose (up to 75 mg daily) can be efficacious in improving pain tolerance and in deafferentation pain syndromes. The neuro-suppressive drugs, Tegretol® and Dilantin,® may also be indicated in certain neuropathic pain states.

Other drugs include steroids such as Decadron® orally, or methylprednisolone epidurally. These agents reduce pressure caused by tissue reaction and local edema adjacent to the tumours, as well as membrane stabilization.

Physical Adjuvants. Transcutaneous electrical nerve stimulation (TENS) may have a place for localized pain syndromes, but there is limited place for acupuncture or hypnosis. Physiotherapy can play an invaluable part where joint stiffness, chest problems and contractures occur.

Nerve Conduction Blockade. This has a role both in the diagnostic arena, using reversible local anesthetics and in the more permanent, therapeutic destruction of nerves. Of the neurolytic agents, alcohol (50-100%), aqueous phenol (6.5%) and phenol in glycerine (5-10%) are most commonly used. They are particularly useful in areas such as coeliac plexus block for carcinoma of the pancreas, stomach, biliary tree or liver. Lumbar sympathectomy may be managed in the same way, while epidural and subarachnoid block with these agents may also be occasionally invaluable.

Neurosurgery. Other neuro ablative procedures also have a place in cancer pain control. Pituitary ablation can be very helpful in certain hormonally dependent tumours such as carcinoma of breast and ovary. Open or percutaneous cordotomy, cutting the ascending pain pathways may be beneficial in some intractable pain states, especially those affecting one side of the body, below the level of the neck. These procedures, however, have limited duration of action of up to 6 or 9 months before pain recurs.

Other forms of palliative surgery include decompression, bypassing of obstruction, stabilization of fractures, and all have a place in certain cases.

Radiotherapy will offer similar palliation by helping to reduce the activity of or swelling associated with metastatic disease.

CONCLUSION

The foregoing has been an overview of pain relief in cancer sufferers. Better understanding and knowledge regarding the use of pain and symtom control can only help to contain suffering in these unfortunate people.

As a model, the health care programs need to concentrate on a home-based service. This should consist of visiting nursing staff, social, occupational and volunteer services, supported by hospital based outpatient and inpatient facilities. The hospital or free-standing hospice unit provides a place for more intensive symptom control, stabilization and treatment with the aim of rehabilitation to the home, if possible. It would also provide a haven for those no longer able to be at home and assurance should be given to patients in the program of access to the unit whenever necessary.

This type of program is developing in Nova Scotia but examples of established programs can be seen elsewhere as at Edinburgh, Scotland, Oxford and London, England and elsewhere in the world.

Myths, fears, lack of knowledge and understanding still stand firmly in the way of achieving the objectives of a pain controlled, dignified existence during the final days of many cancer suffers. Awareness education and resources are improving but much has to be done yet. □

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RENAL FAILLURE DUE TO SARCOIDOSIS

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Dogs look up to men, cats look down on them, but pigs just treat us as equals.

— Sir Winston Churchill (1874-1965)

APPOINTMENT NOTICE

MARITIME TEL & TEL

John D. Prentice, M.D.

Ivan Duvar, President and Chief Executive Officer of Maritime Telegraph and Telephone Company Limited announces the appointment of Dr. John D. Prentice as Medical Director.

Dr. Prentice is a native of Springhill. He received his medical degree from Dalhousie University and holds degrees in occupational medicine from McMaster University and John Hopkins University.

Prior to joining MT&T Dr. Prentice was the Health Unit Director of the Nova Scotia Department of Health and a consultant in occupational health.

He is Chairman of the Nova Scotia Medical Society Committee on Occupational Health, a Director of the Occupational Medical Association of Canada and is a member of a number of Federal–Provincial Advisory Committees on health issues.

Renal Failure Due to Sarcoidosis

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Sarcoidosis is a relatively uncommon cause of renal disease, but its recognition is important because appropriate therapy often results in normalization of renal function. We have recently had the opportunity to treat two patients with renal insufficiency due to sarcoidosis. We report these examples in order to increase clinical awareness of this reversible form of nephropathy.

Case 1: A 33 year old woman with a history of inflammatory bowel disease well-controlled for five years with sulfasalazine presented with a creatinine of 507 μM/L. Six months before, the serum creatinine was 75 μM/L. One month prior to presentation, the patient had complained of fatigue, nausea, vomiting, and headache, and had been treated with naproxen and metoclopramide. Naproxen intake was only one tablet per day. There were no other symptoms.

Physical examination was normal, and urinalysis revealed only 1+ protein, with a few granular casts on microscopic examination. Serum calcium was normal, but the alkaline phosphatase was elevated at 135 U/L (normal > 40). The hemoglobin was 104 g/L. A chest x-ray was normal. Renal ultrasound revealed normal sized kidneys.

Renal biopsy (Fig. 1) revealed normal glomeruli, but there was an extensive interstitial lymphocytic infiltrate and many non-caseating granulomas containing Langhans-type multinucleate giant cells. A liver biopsy revealed a few small granulomas in portal areas. A diagnosis of sarcoidosis was made.

The patient was treated with prednisone 40 mg daily, and within three weeks the serum creatinine had dropped to 177 μM/L. Steroids were discontinued slowly over six months and renal function remained stable until one year after her initial presentation. At that time the patient, temporarily lost to follow-up, returned to hospital with a serum creatinine of 1100 μM/L and uremic symptoms. A second renal biopsy (Fig. 2) revealed one sclerotic glomerulus, with other glomeruli surrounded by fibrosis. There was extensive granuloma formation in the interstitium, featuring epithelioid cells and multinucleated giant cells. Tubules and vessels were unremarkable.

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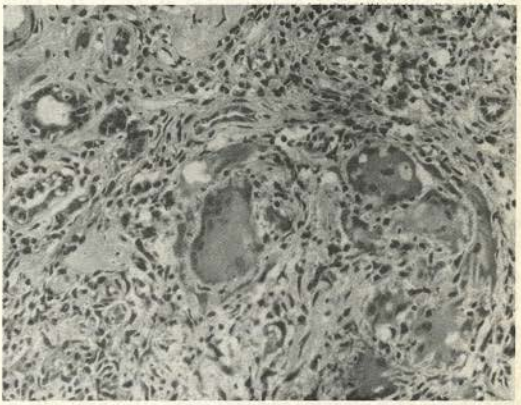

Fig. 1. There is evidence of a large non-caseating granuloma at centre-field, containing several multinucleate giant cells.

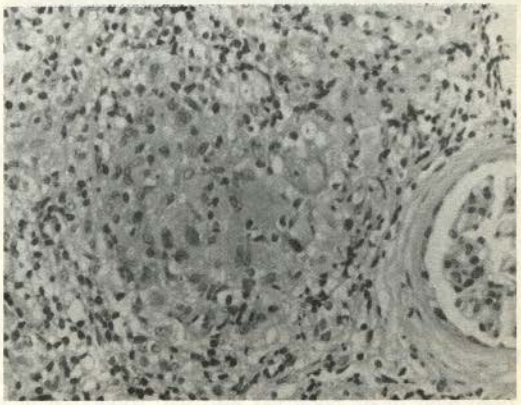

Fig. 2. On the right is a glomerulus surrounded by periglomercular fibrosis, while at centre-field is a large granuloma.

Prednisone therapy was restarted at 40 mg per day, and the serum creatinine slowly fell to 330 μM/L over three months. The patient's renal function remains stable on 10 mg of prednisone daily.

Case 2: A 34 year old male was referred with hypertension, a normochromic normocytic anemia of 109 gm/L, and renal insufficiency (serum creatinine of 600 μM/L). He had noted fatigue and headaches for three months, and had complained of ill-defined diffuse leg discomfort for one month. On repeated questioning, he admitted to mild wheezing dyspnea and a non-productive cough for two months.

Physical examination revealed a blood pressure of 154/104, diffuse bilateral rhonchi in the chest, and splenomegaly. Urinalysis revealed trace proteinuria, with a few red cells per high power field and an occasional granular cast.

Chest X-ray demonstrated a diffuse interstitial infiltrate, without obvious hilar adenopathy. His alkaline phosphatase was elevated at 273 U/L. The serum calcium was repeatedly normal in a peripheral hospital, but ten days later at the Victoria General Hospital it was reported at 3.50 mM/L (normal 2.19-2.54). Renal size was normal by ultrasonography. Pulmonary function studies showed a very mild restrictive impairment.

Renal biopsy (Fig. 3) showed normal glomeruli but extensive tubular and interstitial microcalcification (i.e. nephrocalcinosis). On recut tissue blocks, a single non-caseating granuloma was noted. A transbronchial lung biopsy demonstrated extensive pulmonary granulomas typical of sarcoidosis. In addition a small erythematous plaque on the patient's left shoulder was noted by the pulmonary consultant. Skin biopsy of this lesion also revealed granulomas.

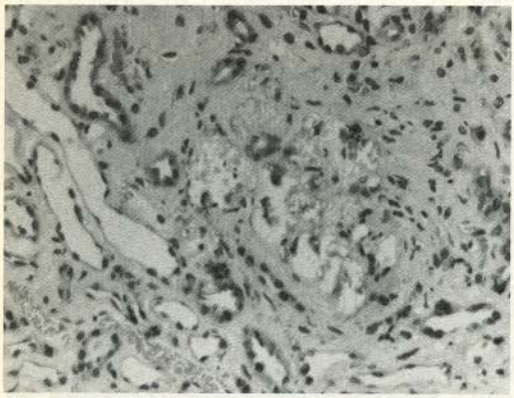

Fig. 3. At centre-field is an area of calcification. The surrounding interstitium shows extensive fibrosis.

Prednisone 60 mg per day was started. After ten days of steroid therapy, the serum calcium was 2.50 mM/L and the serum creatinine had fallen to 240 μM/L. The patient was asymptomatic save for mild dyspnea on exertion, which was improved since admission to hospital. Follow-up continues, with plans to reduce steroid therapy slowly once the creatinine stabilizes.

DISCUSSION

These cases demonstrate the two major reported types of sarcoid-related renal disease: case 1 exemplifies granulomatous normo-calcemic nephropathy, while case 2 includes hypercalcemic nephropathy as a feature. Of note, neither patient initially complained of pulmonary symptoms, despite the common perception of sarcoidosis as a predominantly pulmonary disease.

In both cases the clinical pattern of renal disease was non-specific, in that marked reductions in glomerular filtration rate were accompanied by minimal proteinuria and unimpressive urinary sediment. Of note, in both cases, renal ultrasonography detected normal-sized kidneys, suggesting an acute course without evidence of loss of renal mass due to scarring. Renal biopsy was required to establish a diagnosis in both patients, and in both cases steroid therapy promptly reversed much of the renal insufficiency.

These clinical features are similar to those reported by others. Table I lists the described forms of sarcoid nephropathy, with references. The most common cause of renal failure in the patient with sarcoid is hypercalcemic nephropathy, sometimes with concomitant renal stone disease, while granulomatous interstitial nephritis represents a much less common presentation. A few patients with glomerulonephritis in association with sarcoidosis have been reported but this is such a rare situation that it may represent a chance occurence.

TABLE I

RENAL EFFECTS OF SARCOIDOSIS

Hypercalcemia related	
	nephrolithiasis[1,2]
	hypercalcemia- associated acute renal failure[3]
	nephrocalcinosis[1,2,4]
Non-hypercalcemic interstitial renal disease	
	granulomatous interstitial nephritis[5,6]
	chronic insterstitial fibrosis[5]
Glomerular disease	
	membranous nephropathy[7]
	proliferative glomerulonephritis[7,8]

The favourable response to steroids seen in our cases is the typical experience, highlighting the importance of considering this diagnosis in patients with unexplained acute renal dysfunction, especially in the setting of hypercalcemia. Also important is the necessity for close follow-up and the need for gradual reduction of steroids, since recurrences (as noted in case 1) have been reported by others. Although reintroduction of steroid therapy resulted in a second response with improvement of renal function, biopsy under such circumstances often shows evidence of irreversible chronic scarring in the kidney.[5] Such scarring represents irreversible loss of renal mass, highlighting the need for care in monitoring patients. □

References

1. Maycock RL, Betrand P, Morrison CE *et al.* Manifestations of sarcoidosis. *Am J Med* 1963; **35**: 67.

Continued on page 154.

Current Topics in Community Health

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

PUBLIC HEALTH PHILOSOPHY

"Disease is largely a removable evil. It continues to affect humanity, not only because of incomplete knowledge of its causes and lack of adequate individual and public hygiene, but also because it is extensively fostered by harsh economic and industrial conditions and by wretched housing in congested communities. These conditions and consequently the diseases which spring from them can be removed by better social organization. No duty of society, acting through its governmental agencies, is paramount to this obligation to attack the removable causes of disease. The duty of leading this attack and bringing home to public opinion the fact that the community can buy its own health protection is laid upon all health officers, organizations and individuals interested in public health movements. For the provision of more and better facilities for the protection of the public health must come in the last analysis through the education of public opinion so that the community shall vividly realize both its needs and its powers".

Source: Winslow, G.E.A. The Life of Herman M. Biggs, Philadelphia: Lea & Febizer, 1929. p. 146.

THE HEALTH OF NOVA SCOTIA CHILDREN

While vital statistics data show that Nova Scotia adults score poorly on health status indices, Nova Scotia children are also at risk of assuming their parents' unhealthy lifestyles.[1,2] Preventive programs are required to correct this inheritance.

The 1984-85 Canada Health Attitudes and Behaviour Survey of 9, 12, and 15 year olds was designed to identify the health related attitudes and behaviours of young Canadians.[2] Nova Scotia children were close to Canadian averages on the percentages of young people consuming the four food groups. However, the province was above average with respect to the proportion of 9 and 15 year olds in the high category of high sugar content foods consumed. Also the province was above average in the proportions of the all three age groups in the high category of high salt foods consumed. Fibre consumption was about normal, but white bread, crackers and buns were consumed more often by Nova Scotian 15 years olds than other Canadian children.

The proportion of Nova Scotia's 12 year olds classified as overweight and slightly overweight was higher than the national norm for the two categories combined. Obesity at any age carries a high risk of psychological stress and a wide range of behavioural and social correlates have been defined. There is an increased risk of obesity for persons who are overweight or obese at younger ages.

Relative weight and skin fold thickness "track" from age to age as do other measures of physical development. Obesity in childhood is also related to hypertension in childhood (although less so than in adults).

Turning again to results of the Health Attitudes and Beahviour Survey, physical activity levels were approximately the Canadian norm. All ages were low in the proportion of children taking daily physical education classes in school or classes two or three times a week. The province had the highest proportion taking physical education classes less than once a month. Nova Scotia's young people at all three ages were among those young Canadians who watched the largest amount of television.

Nova Scotia was similar to the national norms for cigarette smoking by 12 and 15 year olds. The province had relatively low proportions of them drinking alcohol. The percentages of young Nova Scotians at all age levels who wore seat belts most of the time were lower than most other provinces.

Nova Scotia had pronounced differences on self-esteem and mental health items. The province had relatively low proportions of students at all three grade levels who indicated that they had confidence in themselves. Proportions of Nova Scotian 15 year olds who responded that they felt good about how they looked and that they made friends easily were low in comparison with those of 15 year olds across Canada. This age group was above the national average in the proportion who agreed that they wished their complexions were better. The province was above the national average in the proportion of 12 year olds who felt they needed to lose weight. More young Nova Scotians at all age levels compared to other Canadians said they could not sleep worrying about things. In general, the self-esteem and mental health of Nova Scotia's young people was deemed an area for concern in the report.

The Need for Child Health Promotion in Nova Scotia

The brief analysis presented of the risk factors and

poor health status of Nova Scotians, both young and adult, underscores the necessity of health promotion programs in the province. This is best accomplished through community-oriented risk factor reduction techniques and a wellness approach to health improvement and maintenance.

References

1. White F: The Health of Nova Scotians. *NS Med Bull* 1985; **64:** 65-68.
2. Social Program Evaluation Group. Canada Health Attitudes & Behaviour Survey, 9, 12, 15 years, 1984-1985, Nova Scotia Report.

Source: Dr. Lynn McIntyre, Hospital Epidemiologist; IWK Hospital for Sick Children, and Department of Community Health and Epidemiology, Dalhousie University, Halifax.

PROTECTIVE EFFECT OF PHYSICAL ACTIVITY ON CORONARY HEART DISEASE

Many studies have suggested that physical activity helps prevent coronary heart disease (CHD), but several others have shown no such association. Thus, evidence to support a beneficial association has been considered weak or questionable, primarily because physical activity is difficult to measure and assess.

An extensive review of studies on the possible association between physical activity and CHD focused on the quality of the measures and methods used. The results of that review indicate that physical activity does help prevent CHD.

A systematic review of the literature yielded 43 studies in English that provided relative risks or multiple regression coefficients of the association between physical activity and CHD. For each of these studies (36 cohort, three mortality, and four case-control studies), the reviewers used specific criteria to assess the quality of the physical activity measure, the CHD outcome measure, and the epidemiologic methods.

The seven criteria used in evaluating the physical activity measures were: 1) clarity of the definition of physical actity; 2) reliability and validity of the measure; 3) assessment of individual physical activity rather than of group activity; 4) use of frequency, intensity, and duration of physical activity to characterize the behavior; 5) measurement of lifetime patterns of activity; 6) adherence to an activity pattern over time; and 7) systematic collection of the measure (usually via self-report surveys).

The four criteria used in assessing the CHD outcome measure were: 1) specifically established diagnostic criteria; 2) objective diagnosis; 3) equal opportunity for diagnosis of CHD; and 4) systematic collection of CHD information.

The eight criteria used in evaluating the epidemiologic methods were: 1) the temporal sequence of physical activity before CHD; 2) statistical control of other CHD risk factors; 3) representativeness of the sample; 4) whether subjects from the cohort studies who were lost to follow-up were located later or at least compared with the other subjects; 5) if random selection methods were used for placing subjects in active and inactive groups; 6) whether cases and control were identified via predetermined selection criteria; 7) if they were equally subjected to exclusionary criteria; and 8) if neither subjects nor data abstractors were informed of the hypothesis being studied.

Primarily on the basis of these criteria, the authors considered 40% of the physical activity measures, 2% of the CHD outcome measures, and 30% of the epidemiologic methods to be unsatisfactory (Table I).

These 43 studies reported 96 comparisons of the association between physical activity and CHD. The reviewers eliminated those comparisons that could not be interpreted (n=3), that focused only on angina (n=10) or women (n=15), that reported information on extra subpopulations or extra physical activity measures (n=5), and that reported multiple CHD outcomes for a given study (n=16). A total of 47 comparisons remained, and all of these were used to draw inferences about men.

Of these 47 comparisons, 32 (68%) showed a statistically significant inverse association between physical activity and CHD. Further, the reviewers' ability to detect such an association increased as the quality of the measures and methods improved (Table II). For example, among the studies using unsatis-

TABLE I

Percentage of 43 epidemiologic investigations of the association between physical activity and coronary heart disease, by the quality of the measures and methods used

	Percentage of Studies, by Quality Category					
Measure/Method	**Unsatisfactory**		**Satisfactory**		**Good**	
Physical activity measure*	40	(17/42)	40	(17/42)	19	(8/42)
Coronary heart disease outcome measure	2	(1/43)	58	(25/43)	40	(17/43)
Epidemiologic methods	30	(12/43)	35	(15/43)	35	(15/43)

*In one study, the method used for measuring leisure-time physical activity was satisfactory and that for measuring work-time activity was unsatisfactory.

TABLE II

Percentage of 47 comparisons from 43 epidemiologic investigations reporting significant inverse associations between physical activity and coronary heart disease, by the quality of the measures and methods used

Measure/Method	Percentage of Comparisons, by Quality Category					
	Unsatisfactory		Satisfactory		Good	
Physical activity measure*	50	(9/18)	76	(16/21)	88	(7/8)
Coronary heart disease outcome measure	100	(1/1)	64	(18/28)	72	(13/18)
Epidemiologic methods	60	(9/15)	61	(11/18)	88	(12/14)

factory physical activity measures, the reviewers noted that 50% showed significant associations; among those using satisfactory measures, 76%; and among those using good physical activity measures, 88%. Similar trends were noted for CHD measures and epidemiologic methods.

The reviewers also examined the potential causal effect of physical activity on CHD by using six criteria: consistency of findings: strength of the association; appropriate temporal sequence; dose-reponse relationship; plausibility; and experimental evidence. A consistent statistically significant association between physical activity and CHD was found for more than two-thirds of the studies. The strength of the association between physical inactivity and CHD (median relative risk = 1.9 for the 47 comparisons) was of similar magnitude as that for several commonly accepted risk factors previously reported in the Coronary Pooling Project, which was based on five studies. In those studies, the median risk ratios were 2.1 for high systolic blood pressure (>150 millimetres of mercury [mm Hg] versus < 130 mm Hg), 2.4 for serum cholesterol (>268 milligrams per decilitre [mg/dl] versus < 218 mg/dl), and 2.5 for smoking (>1 pack of cigarettes/day versus no smoking). Most of the 43 studies reviewed showed that the activity assessment predated the CHD outcome, demonstrating an appropriate temporal sequence. More than two-thirds of the studies demonstrated a dose-response relationship, with lower levels of physical activity leading to more instances of CHD. There are plausible and coherent mechansism whereby physical activity could exert a beneficial influence on CHD. Although no experimental evidence exists in the form of a randomized, controlled clinical trial, better studies (i.e., those in which the measures and methods used were judged to be good or satisfactory) were more likely to report a significant inverse association. On the basis of these criteria, the authors concluded that a causal inverse association exists between physical activity and CHD.

Source: *Morbidity and Mortality Weekly Report* 1987; **36:** 426-430. (adapted)

Editorial Comments: A copy of the original article (including editorial note and reference) is available on request.

□

OBITUARIES

Dr. Ralph W.M. Ballem, (66) of Dartmouth, N.S. died on August 11, 1987. Born in Sydney he received his medical degree from Dalhousie Medical School in 1944. He worked as an anaesthetist at the Victoria General Hospital before becoming a family practitioner in Shubenacadie Medical Clinic. He was an avid outdoorsman and active in the Boy Scouts organization. He is survived by his wife, two daughters, two sons, and four grandsons. The *Bulletin* extends sincere sympathy to his family.

Dr. Robert Marsden Caldwell, (80) of Yarmouth, N.S. died on September 5, 1987. Born in New Brunswick he graduated from Dalhousie Medical School in 1932. He decided to take an extra year of internship before starting general practice in Yarmouth. He served in the Royal Canadian Army Medical Corps in the Second World War and obtained the rank of major. He was made Senior Member of The Medical Society of Nova Scotia in 1979. He is survived by his wife, two daughters and two grandchildren. The *Bulletin* extends sincere sympathy to his family.

Dr. Walter James Fisher, (98) of Halifax, N.S. died on September 8, 1987. Born in Germany he received his medical degree at the University of Breslau in Germany. His specialty was psychiatry which he practised in his later years in the Halifax-Dartmouth area. He was the author of several scientific publications and was a member of The Nova Scotia and Canadian Psychiatric Associations. He is survived by his wife, a daughter and three grandchildren, to whom the *Bulletin* extends sincere sympathy. □

Appreciations

DR. DONALD MAXWELL NICHOLSON

Dr. Donald Maxwell Nicholson passed away on June 24, 1987 at the age of 52.

Don was born in Summerside, P.E.I. He received his B.Sc. degree from Mount Allison in 1955 and graduated in Medicine in 1960 from Dalhousie.

While at both universities he was an outstanding athlete, participating in English rugby, Canadian football, and track and field. He played Canadian football for Dalhousie during his five years of medical school and was one of the very best running backs developed in the Maritime Provinces. His athletic career was characterized by superb physical conditioning, an intense desire to do his best, and a strong sense of honesty and fairness and dealing with both teammates and opposition. He carried these characteristics forward through the remainder of his life.

On completion of medical school, he began a residency in Surgery at Dalhousie University. He was an R.S. McLaughlin Travelling Fellow at Radcliffe Infirmary in Oxford in 1964-65, and took further training in thoracic surgery at Baylor University Medical Centre in Dallas. He received his Fellowship for the Royal College of Surgeons of Canada in 1966, and joined the active staff of the Victoria General Hospital at that time.

He was a member of the Thoracic Surgery Division at the Victoria General Hospital. He subsequently received his fellowship for the American College of Surgeons in 1970 and his Certificate in Thoracic Surgery in 1979. He became a Fellow of the American College of Chest Physicians in 1981. In 1980, he was appointed Chief of Thoracic Surgery at the Victoria General Hospital and continued with that appointment until his death.

He was Past President of the Surgical Section of The Medical Society of Nova Scotia and a former member of the Fee Committee of the Society.

Don took great pride in his family which includes his wife June, his daughters Joanne and Donna, and son John. He enjoyed the outdoors and was an avid horseman and gardener. Don was a highly respected member of the Department of Surgery and the Victoria General Hospital Medical Staff. His integrity, devotion to duty and surgical skills were of such a level that a major void has been created which will be very difficult to fill.

In our younger years we travelled many roads together, some rocky and others smooth. There never could be a better travelling companion, particularly for the rocky roads, than Don Nicholson.

Brian M. Chandler, M.D.

DR. RALPH WILLIAM McKEEN BALLEM

It is with regret that we announce the death of Dr. Ralph William McKeen Ballem on August 11th, 1987, as the result of a boating accident.

Ralph Ballem was born and educated in Sydney, Cape Breton. He attended Dalhousie Medical School where he attained his M.D. in 1944. Following three years service with the R.C.A.F. he returned to his Alma Mater and started his career in Anaesthesia. After one year in the Victoria General Hospital, he pursued further graduate training in Anaesthesia in Hartford, Connecticut, attaining his Fellowship in the Royal College of Physicians, Canada, in Anaesthesia and returned to serve for 33 years in the Department of Anaesthesia, at the Victoria General Hospital. He was a Fellow of the American College of Anaesthesiology, and Assistant Professor on the teaching staff of Dalhousie University.

Despite a busy anesthesia practice, he managed to command the 38th Halifax Scout Troop for 10 years, was a keen yachtsman and flier.

On his retirement from the 'Anaesthesia Department' of the V.G.H. he entered family practice in Shubenacadie, Nova Scotia, where he was a much loved and popular physician until his untimely death.

He is survived by his wife, the former Ruth Graham, two sons, Ronald at home, Hugh of Montreal, two daughters, Leslie of Cincinnati, Laura of Halifax, five grandsons and brother Henry Clare of Miami.

He will be sadly missed by his family, friends and colleagues.

C.K. Bridge, M.D.

Chance favours only the mind which is prepared.

— Louis Pasteur (1822-1895)

Personal Interest Notes

A VERY PERSONAL INTEREST NOTE

Dr. Aden C. Irwin, Associate Editor of this journal has recently retired from his position as Professor in the Department of Community Health and Epidemiology, Faculty of Medicine, Dalhousie University. We understand that he will still work part-time in that Department, and of course, we hope, with the staff of *The Nova Scotia Medical Bulletin.* This milestone of retirement, however, could not pass without some formal recognition of his value to this journal.

Dr. Irwin has been associated with the *Bulletin* for 12 years, and in that time he has earned the appreciation and gratitude of many editors.

Al Capp of "L'il Abner" fame once drew a cartoon strip about a cartoon writer, probably satirizing himself. The cartoon writer, who took all the credit, kept a man imprisoned in a closet and fed him bread crumbs while his captive really produced all the work necessary for the publication of the cartoon. I cannot help but remember this story as I write about Aden; as we know he eats more than bread crumbs, not because we support him in any generous way, but his office does remind one of the closet mentioned above.

Organized confusion in Dr. Irwins' Office.

His office consists of a collection of books, papers, envelopes and boxes piled high in all directions and provides a uniqueness that is in definite contrast to the obsessiveness he shows in his proofreading. Dr. Irwin strives for perfection which can easily be explained as his special interest is statistics in medicine.

His thanks have come from the editors, routinely praising him in annual reports, but mostly I think his satisfaction comes from producing a quality journal. He understands that we are not to be compared with the *New England Journal of Medicine,* but his efforts have led to a high standard that is mostly to his credit. His tasks have been many including editing, proofreading, reviewing, correcting and writing and offhandedly critizing the editor; all of which he has done well.

Dr. Irwin was named Professor of the Year by the Graduating Class of the Faculty of Medicine at Dalhousie University in 1985 — an honour he well deserves. During the years he has encouraged many students to publish in our *Bulletin,* and through patience and hard work he has often retrieved articles from the waste basket that consisted of good material but that were poorly written.

For relaxation Dr. Irwin plays the clarinet and has been an active member of the Tupper Band for many years. He also has a long-standing interest in history and has spent many holidays on historical tours throughout Europe. The *Bulletin* wishes Aden many happy years to enjoy these hobbies now that he has more time on his hands.

While smiling at his office, the office inhabitant merits our thanks and appreciation. We hope he will continue his work with the *Bulletin,* for as long as it takes to catalog the material in his office, and for many years thereafter.

Dr. John R. Robertson, President of The Canadian Dental Association presented **Dr. James Douglas McLean** of Bedford, N.S. with the Association's Distinguished Service Award at their recent Annual Meeting. Dr. McLean served as the Dean of the Faculty of Dentistry, Dalhousie University from 1954 to 1975. Earlier this year he was honoured with an Honorary Doctor of Laws Degree at Dalhousie's Spring Convocation. This "innovator in dental education" deserves our congratulations.

Dr. Nuala Kenny has been elected as Chief of Pediatrics at the Izaak Walton Killam Hospital for Children, her appointment takes place Jan. 1, 1988. Dr. Kenny is no stranger to Halifax, having formerly worked as a physician and Acting Director of the IWK's Outpatient Department. She received her medical degree from Dalhousie University in 1972. Currently she is Chief of Pediatrics at Kingston General and Hotel Dieu Hospitals in Kingston, Ontario, as well as Chairman of the Department of Pediatrics, Queens University, Toronto. □

Notice Re: By-Law Amendments

The By-Laws of the Medical Society stipulate that amendments to them may be proposed at an Annual Meeting of the Society provided they are published in the Bulletin at least one month prior to the Annual Meeting.

The following amendments will be presented by the By-Laws Committee at the 1987 Annual Meeting of the Society.

(Where possible, changes/additions in the proposed are indicated by **BOLD** type.)

EXISTING	PROPOSED
5 SECTIONS	**5. SECTIONS**
5.2 Application:	**5.2 Application:**
5.2.1 Such formal application for recognition of a Section shall be sponsored by not less than ten members of the Society and the application, together with such information as may be required, shall be presented to the Executive Committee not less than three months before the Annual Meeting.	**5.2.1** Application for recognition as a Section of the Society must be presented to the Executive Committee not less than three months before the Annual Meeting, and shall be published in the Bulletin at least one month prior to the Annual Meeting.
	5.2.2 The application shall include: (a) The name of the proposed Section. (b) The names of ten or more Society members sponsoring the proposed Section. (c) The names of the interim chairman and secretary of the proposed Sec tion. (d) Reason why the formation of a new Section will benefit both the mem bers of the proposed new Section and the Society as a whole.

EXISTING	PROPOSED
	Renumber 6.15.6 to read 8.1.2. (Section 8 - Meetings)
11.6 Duties of the Treasurer:	**11.6 Duties of the Treasurer:**
11.6.1 The Treasurer shall be the custodian of all monies, securities and deeds which are the property of the Society. He shall pay by cheque only; such cheques shall be signed by two persons authorized by the Executive Committee. All such cheques are to be covered by voucher. He shall provide an annual financial statement audited by a chartered accountant. He shall furnish a suitable bond for the faithful discharge of his duties, the cost of such bond to be borne by the Society. He shall be responsible for the annual review of all salaries of the secretariat an bring recommendations to the Executive Committee.	**11.6.1** The Treasurer shall be the custodian of all monies, securities and deeds which are the property of the Society. He shall pay by cheque only; such cheques shall be signed by two persons authorized by the Executive Committee. All such cheques are to be covered by voucher. He shall provide an annual financial statement audited by a chartered accountant. He shall furnish a suitable bond for the faithful discharge of his duties, the cost of such bond to be borne by the Society. He shall be responsible for the annual review of all salaries of the secretariat an bring recommendations to the Executive Committee. **He shall be Chairman of the Finance Committee.**
12. COMMITTEES	**12. COMMITTEES**
12.1 Appointment of Statutory Committees: Statutory Committees shall be: The Nominating Committee The Executive Committee	**12.2 Appointment of Statutory Committees:** Statutory Committees shall be: The Nominating Committee The Executive Committee **The Finance Committee**
12.2.1 The Nominating Committee and the Executive Committee shall be elected at the Annual Meeting of the Society.	**12.2.1** The Executive Committee shall be elected at the Annual Meeting of the Society.
12.3 The Nominating Committee:	**12.3 The Nominating Committee:**
12.3.1 The Nominating Committee shall be composed of one member from each Branch Society. This member shall be the immediate Past President of the Branch Society. Each Branch Society is entitled to appoint an alternate member who shall be the Chairman thereof. In the absence of the President the Committee shall elect its own Chair-	**12.3.1** The Nominating Committee shall be composed of one member of each Branch Society, who shall be the immediate Past-President of the Branch Society or his designated alternate. The chairman of the Nominating Committee shall be the President of the Society. In the absence of the President the Committee shall elect its own chairman.

EXISTING	PROPOSED
12.3.2 Duties and Powers of the Nominating Committee:	**12.3.2 Duties and Powers of the Nominating Committee:**
12.3.2.1 At the Annual Meeting following its appointment the Nominating Committee shall place in nomination the Statutory Committees of the Society as follows:	**12.3.2.1** At the Annual Meeting the Nominating Committee shall place in nomination the **Executive Committee** of the Society as follows:
Executive Committee	**Executive Committee**
i)Officers: A President A President-Elect An Immediate Past President A Chairman of the Executive Committee A Vice-Chairman of the Executive Committee A Treasurer An Honorary Secretary; and A Member-At-Large (when appointed)	i)Officers: A President-Elect A Chairman of the Executive Committee A Vice-Chairman of the Executive Committee A Treasurer An Honorary Secretary A Member-At-Large (When Required) In the event that the Presidency will not, for whatever reason, be taken by the President-Elect, then the Nominating Committee will nominate a President.
12.3.4 Election of Officers and Executive Committee:	**12.3.4 Election of Officers and Executive Committee:**
12.3.4.1 When the report of the Nominating Committee has been received by the Annual Meeting other nominations may also be received from the floor. Any such nomination for any of the offices named in Chapter 10.1.a shall be placed in writing in the hands of the Executive Director not later than one week prior to the Annual Meeting. Such nominations must be signed by ten members of the Society in good standing and such nominations must be accompanied by the written consent of the nominee to serve together with his Curriculum Vitae. A ballot shall be taken for each of the offices in turn as well as for the elected membership of the Executive Committee.	**12.3.4.1** When the report of the Nominating Committee has been received by the Annual Meeting other nominations, **as delineated in 12.3.2.1 (i)**, may be received from the floor, provided such nominations are placed in writing in the hands of the Executive Director not less than one week prior to the Annual Meeting. Such nominations must be signed by ten members of the Society in good standing and such nominations must be accompanied by the written consent of the nominee to serve, together with his Curriculum Vitae. A ballot shall be taken for each of the offices in turn as well as for the elected membership of the Executive Committee.

EXISTING	PROPOSED
12.5 Discipline Committee:	**12.5 delete Discipline Committee:**
12.5.1 Terms of Reference. The Discipline Committee members are the Medical Society President (Chairman), Immediate Past President and President Elect. The Committee is charged with the responsibility of investigating charges of unprofessional conduct or conduct unbecoming to a member of the Medical Society. In conducting the proceedings of the Discipline Committee the principles of natural justice shall be observed. Proceedings of the Discipline Committee may only be instituted by written complaint following which a hearing or due inquiry shall ensue. Full and reasonable notice of any such inquiry shall be communicated to the member, or his counsel, to permit him the opportunity to question the complainant and any other witnesses and to argue as to the merits of the complaint. The proceedings shall be recorded by a competent and duly-sworn stenographer. The decision shall be reserved, then rendered in writing with reasons, a copy being forwarded to the accused, but not to the complainant. The Executive Committee is required to review all decisions of hearings of the Discipline Committee.	**12.5.1 delete complete sub-section**
FINANCE COMMITTEE **(Not previously listed in By-Laws as Committee is not a Statutory Committee as yet)** **Terms of Reference** - The Finance Committee shall consist of the Treasurer and two other members of the Society appointed by the Executive Committee. This Committee shall: (a) check all expenditures of funds to ascertain that they are properly made, and bring to the attention of the Executive Committee any likely over-expenditures, misappropriation, misuse or discrepancy in the Society's funds;	**12.5 The Finance Committee** **12.5.1 Terms of Reference** - The Committee shall consist of the Treasurer as Chairman and four voting members of the Society approved by the Executive Committee. The Committee shall meet at least twice yearly. Two voting members of the Committee including the Chairman shall constitute a quorum. The Committee shall be charged with a special duty of studying the immediate and long-term financial needs of The Society, including the establishing of reserve funds, and shall present in its Annual Report to Council appropriate fiscal policies to meet these needs. The Committee shall follow Society Investment Policy in managing Society investments,

EXISTING	PROPOSED
(b) make recommendations to the Executive Committee with reference to: (i) the raising of funds; (ii) the disbursement of funds; (iii) the allotment of funds to special or trust accounts. (c) prepare an annual budget for consideration by the Executive Committee; (d) be responsible to the Executive Committee for classification of positions, salaries, management and general welfare of the staff of the Society; and (e) on behalf of the Executive Committee present an audited Financial Statement of the Society including all receipts, vouchers and all other supporting or evidentary documents relating thereto.	and may seek professional investment counsel in directing the policy. When directed the Committee shall assess the financial implications of programs and projects and make recommendations to the Executive Committee. The Committee shall prepare for approval by the Executive Committee a budget for the ensuing year indicating distribution of monies to be made available for all purposes. This Committee shall report regularly to the Society's Executive Committee. The Committee shall report annually to Council. Its report shall cover all financial activities of the Society during the previous calendar year and shall indicate the revenues necessary for the subsequent year. The adoption by Council of the annual reports of the Finance Committee and of the Auditor shall validate all financial transactions of the Executive Committee, the Finance Committee and other Committees of the Society during the year covered. The Committee shall recommend the appointment of Auditors to the Council annually. The Committee shall check all expenditures of funds to ascertain that they are properly made, and bring to the attention of the Executive Committee any likely over-expenditures, misappropriation, misuse, or discrepancy in the Society's funds. The Committee shall be responsible to the Executive Committee for classification of positions, salaries, management and general welfare of the staff of the Society.
12.7 Standing Committees: **12.7.1** The Executive Committee shall have power to establish Standing Committees, to vary their number from time to time and to discontinue their activities. The Chairman of Committees designated by the Executive Committee as Standing Committees shall be appointed by the Executive Committee which, in addition to the duties	**12.7 Standing Committees:** **12.7.1** The Executive Committee shall have power to establish Standing Committees, to vary their number from time to time and to discontinue their activities. The Chairman of Committees designated by the Executive Committee as Standing Committees shall be appointed by the Executive Committee which, in addition to the duties provided in 12.8 of this chapter, shall also provide

EXISTING	PROPOSED
provided in 12.8 of this chapter, shall also provide or vary their terms of reference. The Standing Committees shall report to the Annual Meeting of the Council after submitting copies of their reports to the Executive Committee at such time as the Executive may require. The Chairman of any Standing or Special Committee shall not serve for more than a maximum tenure of three years. After one year's absence from office he shall become eligible for re-election.	or vary their terms of reference. Standing Committees shall report to the Executive Committee when requested to do so, and to the Annual Meeting after prior submission of that report to the Executive Committee. The Chairman of any Standing-Committee shall not serve for more than three consecutive years, after one year's absence he shall become eligible for re-election.

RULES AND REGULATIONS
of
THE MEDICAL SOCIETY OF NOVA SCOTIA

3 STANDING COMMITTEES	3 STANDING COMMITTEES
3.1 Recognized as of the date of the adoption of these By-Laws are the following Standing Committees. This list may be varied by the Executive Committee as it may determine.	**3.1** Recognized as of the date of the adoption of these By-Laws are the following Standing Committees. This list may be varied by the Executive Committee as it may determine.
Committees on:	Committees on:
Allied Health Disciplines	Allied Health Disciplines
Annual Meetings	Annual Meetings
Archives	Archives
Awards Committee	Awards Committee
By-Laws	By-Laws
Cancer	Cancer
Child Health	Child Health
Community Health	Community Health
Drug & Alcohol Abuse	Drug & Alcohol Abuse
Editorial	Editorial
Ethics	**Environment**
Finance (Treasurer)	Ethics
Hospital & Emergency Services	**Horizons**
	Hospital & Emergency Services

EXISTING	PROPOSED
Maternal & Perinatal Health	**Liaison Committees**
Medical Education	**(i) Faculty of Medicine**
Membership Services	**(ii) Minister of Health**
Nutrition	**(iii) Registered Nurses' Assoc.**
Occupational Medicine	**(iv) Workers' Compensation Board**
Pharmacy	Maternal & Perinatal Health
Physical Fitness	Medical Education
Presidents' Committee	Membership Services
W.C.B. Liaison	Nutrition
	Occupational **Health**
	Pharmacy
	Physical Fitness
	Presidents' Committee
	Professionals' Support Program

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"The Benefits to YOU"

a special presentation

The Membership Services Committee will present an afternoon seminar, Thursday, November 26th, on the benefits of membership in the Medical Society. Selected vendors will be present to provide details of the new services being offered exclusively as a result of your membership. A few special speakers will outline the major benefits of association, including your group insurance and investment opportunities. There will also be an open session designed to allow input from the members so that new directions can be identified for membership services.

The 134th Annual Meeting

The Medical Society of Nova Scotia

November 26-28, 1987

World Trade and Convention Centre, Halifax

"MIXING BUSINESS WITH PLEASURE"