CAPM NEWSLETTER

This first edition of the newsletter for 2009 will focus on several relevant clinical as well as administrative areas.

We have a plethora of great pain information sites and organizations available to all of us. Our challenge is to make the CAPM newsletter more relevant to you.

It is important to quantify who we are - and why we are different that other organizations concerned all about pain. We are not only family physicians, anesthesiologists, and medical specialists but we are also psychologists, physiotherapists, chiropractors, nurses, pharmacists and occupational therapists, concerned about the better management of our pain patients.

Though our membership draws mainly from the medical sciences at present, our outreach is to all health providers interested in working in a multidisciplinary environment (whether on one site or many) to better integrate services for our patients in pain.

Because of time and space constraints, this first edition of our 2009 newsletter will focus only on the following areas:

I. Update from the CAPM Executive: Dr. Eldon Tunks

II. Pain Initiatives from other health disciplines - The Canadian Physiotherapy Association: Gloria Gilbert III. Specialty Article: Dr. G.D. Gale on 'Interventional Management for Chronic Non-Malignant Pain (CNMP), including the use of Palliative & Therapeutic Nerve Blocks'

Our next newsletter will begin a series of articles on improving our communication skills – both verbally and by the use of questionnaires. Information has been received to date by Dr. Kevin Rod at the Toronto Polyclinic.

Members of all health disciplines are welcome to share with CAPM their own useful assessment and treatment procedures. In particular, your experience using intake questionnaires is appreciated.

The Editor also encourages all members to provide ideas for future newsletter topics, articles and meeting notices. Please forward submissions to: **gloria@downtownclinic.ca**

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Yours sincerely,

Gloria Gilbert BSc(PT) MSc Secretary CAPM Member of AAPM



Update from the Executive

An initial telephone conference was held in December between Dr. E. Tunks of CAPM and OMA Section of Pain Physicians, and Dr. Rocco Gerace, Registrar of the College of Physicians & Surgeons of Ontario. The purpose was to inform the CPSO of OMA and CAPM activities aimed at reducing barriers to referral to pain clinics, and to promote a consensus on physician preparation for this field. Dr. Gerace offered his encouragement as well as some helpful recommendations and caveats.

Meetings are also planned with the Ontario Deputy Minister of Health and Long-Term Care in Ontario as well as with officials of O.H.I.P (Ontario Medicare), dealing with the same issues. That meeting will be led by Dr. Howard Jacobs, Chair of OMA Section of Pain Physicians.

Your executive is seeking discussion on:

1. Improvement in the access to Pain Clinics when the referral is initiated by a physician in primary care to a Pain Clinic that is staffed by primary care physicians

2. Removal of barriers for referring primary care physicians.

3. The possible role of credentialing in pain management

Yours sincerely,

Eldon Tunks MD FRCPC President CAPM Diplomate of CAPM Member and Diplomate of AAPM

Update from the Professions

Pain Science Division, Canadian Physiotherapy Association (www.physiotherapy.ca)

In May 2008 at the annual meeting of the Canadian Physiotherapy Association (CPA), the Pain Science Division (PSD) was born! Membership is currently at 350 (and growing)!

Starting as an informal meeting of 'pain interested physios' on line, the group developed into a chat line and then consolidated calling themselves the Canadian Pain Sciences group. These active members have produced a monthly on line newsletter called 'Nocioception' which has had up to 1000 'hits' each month.

It is well acknowledged that physiotherapists are integral members of the health provider teams involved with pain management,. Plans are underway to develop a more interactive website for physios that will include more resources for their members.

The Executive has also collaborated with CPA to develop an online Virtual Pain Symposium, which took place in early November. Physiotherapist members paid a fee to register for the three session 'educational experience'.

Many plans for the future development of the PSD are underway...stay tuned for details.

Yours sincerely,

Gloria Gilbert

Specialty Article by Dr. G.D. Gale

<u>INTERVENTIONAL MANAGEMENT FOR</u> CHRONIC NON-MALIGNANT PAIN (CNMP)

<u>INCLUDING THE USE OF</u> <u>PALLIATIVE AND THERAPEUTIC NERVE BLOCKS</u>

Chronic Pain in Canada:

Moulin, *et al* (2002) found the incidence of chronic pain in Canada to be 29%. It has major social and economic impact (Jovey 2002, page 113) and is under-treated in Canada (Moulin, *et al* 2002).

Interventional Treatment:

Lerich, a French surgeon, first identified chronic pain as a disease state (1939) and described the treatment of causalga and reflex sympathetic dystrophy (RSD). Livingston described the pain mechanism in Causalga (1943).

Therapeutic nerve blocks were described as early as 1921. Scholl (1921, 1922) reported lesser occipital nerve blocks with Procaine, which relieved headache and confirmed a diagnosis of neuralgia. Woodbridge (1930) used 0.5% Procaine in paravertebral blocks to relieve pleuritic pain, which was less severe when the pain recurred. Woodbridge (1930), Ruth (1934), Mandle (1938) and Rovenstein and Wertheim (1941) popularised diagnostic and therapeutic nerve blocks for pain control. The latter authors and Bonica (1951) described nerve blocks in the management of pain as diagnostic, prognostic and therapeutic.

Nerve block clinics were described by Apgar (1948), Ruben (1951), Dittrick (1950) and Alexander (1978). Bonica described the role of the anaesthesiologist in the management of intractable pain (1951) and the management of intractable pain with analgesic blocks (1952).

Regional anaesthesia techniques were first used by Dr. J. Alfred Lee in the British Eighth Army in 1941-43 and were subsequently taught by him and Dr. R. Atkinson in the British National Health Service. I was fortunate enough to be able to learn regional anaesthesia from them in 1969.

Ablative Procedures:

Alcohol was used to prolong the blocks in coccydynia and trigeminal blocks (Woodbridge, 1930), but because of the development of de-afferentation pain the use of neurolytic and ablative techniques has more recently not been recommended, (Fields, 1987; Gildenberg and DeVaul, 1985), but is still considered acceptable for trigeminal neuralgia (Pawl, 2002) as also is rhizolysis of the medial branch nerve after a positive result with diagnostic nerve blocks.

Spinal Cord Stimulation:

This has been used since Shealy, *et al* and Wall and Sweet in 1967 described pain relief by direct spinal cord stimulation. In practice this has proved very difficult to obtain in Toronto, but there may be a useful role in the future for this therapeutic modality.

Diagnostic Nerve Blocks and Rhizolysis:

Radiographically guided nerve blocks of the medial articular branch of the posterior primary ramus are used for the diagnosis of zygapophysial joint pain. If this is positive, then rhizolysis is indicated to reduce joint pain. However, joints are not the only cause of spinal pain, and even if the diagnostic block is positive the success rate generally accepted for rhizolysis is only 66% and is time-limited to an average of nine months, so this procedure is limited in both applicability and if successful, in time. However, cases that do not respond to diagnostic blocks may still respond to palliative blocks, also known as therapeutic nerve blocks because the pain may be from structures other than just the zygapophysial joints.

Ultrasound-Guided Regional Block:

Loubert, *et al* (2008) described an ultrasound-guided axillary block which resulted in an intravascular injection of local anaesthetic. This report illustrates the fact that imaging techniques do not guarantee the avoidance of misplaced local anaesthetic.

Palliative Nerve Blocks:

Palliative (also known as therapeutic nerve blocks) have been successfully performed for over 70 years, since reports by Woodbridge (1930), Ruth (1934) and with paravertebral blocks by Mandle (1938). The performance of these blocks requires a good knowledge of anatomy and physical landmarks. The efficacy of palliative nerve blocks has been well-documented by Bovim, *et al* (1992), Gawel and Rothbart (1992 x2), Rothbart (1992, 1996) and Rothbart, *et al* 2000. Dr. Rothbart and I have reviewed the use of palliative nerve block treatment in "Cranio-Cervical Pain: Medical Management" in a chapter in the cranio-cervical syndrome: mechanisms, assessment and treatment, Editor Howard Vernon (Rothbart and Gale, 2001).

Despite advances in pain management with intradiscal electrical therapy (IDET), microdiscectomy or disc decompression, diagnostic nerve blocks and rhizolysis not all chronic pain conditions are suitable or amenable to these forms of interventional treatment. Furthermore, in a study in which palliative nerve blocks were compared with cognitive therapy, the majority of patients showed a preference for the palliative nerve blocks (Gale, *et al* 2002). The chronic pain patients' preference for palliative nerve blocks appears to be physiologically-based, probably by reversing both the peripheral effects and the central effects of central sensitization described in chronic pain (Rome and Rome 2000).

The pain relief with palliative block is unlikely to be a placebo effect or the cerebral effect of vascular absorption of local anaesthetic effect because the musculoskeletal pain relief occurs rapidly and the local anaesthetic blood levels usually remain low.



Two recently published guidelines discuss therapeutic of palliative nerve blocks:

1. <u>The CPSO reference Guide for CNMP (2000).</u>

In November 2000, The College of Physicians and Surgeons of Ontario (CPSO) facilitated a Reference Guide for Clinicians of Evidence-Based Recommendations for Medical Management of Chronic Non-Malignant Pain. The report indicated (page 5) that it only addressed nerve blocks in a limited way and that the level of evidence for it using the system of McQuay and Moore (1998) was Class III. Class III evidence was described as: evidence from well-designed trials without randomisation, single group prepost, cohort, time series, or matched case-controlled studies. This report stated that the evidence for the injection of (local) anaesthetic (drugs) into painful soft tissues or facet joints is usually based on level III evidence. This does not mean that patients should not receive a trial of injection therapy, but if patients show lack of clear progress using injection therapy, there is no evidence that would support continuation of the injection treatment. Two reasons may account for the evidence only being Class III: one is that it is difficult to randomise an injection therapy because it may be considered unethical to inject a placebo. The other reason is that the use of therapeutic nerve blocks largely predated the popularity of randomised controlled trials (RCT's).

2. <u>The Wisconsin Guidelines.</u>

The guidelines for the assessment and management of chronic pain developed by the Wisconsin Medical Society Task Force on Pain Management (WMJ 2004 [103]3 pages 13-42) addressed the use of therapeutic nerve blocks (page 27) as follows:

a) by providing anaesthesia, therapeutic blocks may facilitate the application of mobilisation techniques.

b) local anaesthesia combined with steroids may be useful in treating specific pain syndromes, e.g.

radicular pain, rotator cuff injury, tendonitis, bursitis.

c) many therapeutic blocks may be useful diagnostically; examples:

i. trigger point injections may reduce pain and improve movement.

- ii. selective epidural steroid injections may reduce radicular pain and dysesthsia.
- iii facet or medial branch blocks may ameliorate certain types of spinal pain.
- iv. sympathetic nerve blocks may reduce sympathetically-mediated pain.

Local Anaesthetics for Palliative Nerve Blocks:

Chemistry:

Local anaesthetic agents consist of an aromatic residue, an amino residue and a link. Being lipophilic the aromatic residue determines the ability of the agent to cross lipid membranes. The amino residue is a weak base that determines the solubility of the agent and the proportion available in the active form. The link between the aromatic and amino residues is formed by either an amide or an ester bond.

Mechanism of Action:

Local anaesthetics bind with intracellular sodium channels and prevent the normal sodium influx that occurs during membrane depolarisation. If sufficient numbers of sodium channels are blocked the nerve impulse is halted and conduction along the nerve fibre ceases. The dual hydrophilic/lipophilic nature of local anaesthetics is critical for this action. The acid (ionised) form of the local anaesthetic is required to bind the intracellular sodium channel, yet only the base form of local anaesthetic is capable of crossing the lipophilic nerve membrane and reaching this intracellular binding site.

The State of the Nerves:

Local anaesthetics have a higher affinity for open channel states (activated/inactive) than for closed channel states (deactivated/resting). This may explain why local anaesthetics have a longer duration of action in neuropathic pain (Arner, *et al* 1990), than the duration of action of neural blockade in non-chronic pain states.

Differential Nerve Blocks:

Larger diameter nerve fibres (deep touch pressure, motor) require higher concentrations

of local anaesthetic to achieve a given degree of block compared with small myelinated fibres (nociceptive afferents). Myelinated nerves are more easily blocked than unmyelinated nerves since only the Nodes of Ranvier need to be blocked in myelinated nerves as opposed to an entire length of an unmyelinated nerve. As the block proceeds, different sensory modalities are lost in this order: pain, temperature, touch, deep pressure, and motor function. (Baker, *et al* 2007).

It therefore follows that low concentrations of local anaesthetic may be effective blocking pain, but leaving the other functions intact. It is therefore not necessary to have motor paralysis to have pain relief and the pain relief may last a longer time in the chronic pain patient because the sodium channels are in an open state because of chronic pain and are therefore more sensitive to blocking, which can be achieved with lower concentrations of the local anaesthetic.

The Toxicity of Local Anaesthetics:

The toxic effects of local anaesthetics are produced by conduction blockage within the central nervous system (CNS) and the cardiovascular system (CVS). These effects are related to the potency of the offending agent, the total dose delivered and the rate in rise of plasma and the site of injection.

Bupivacaine is highly protein bound and highly potent and therefore may not give the earlier CNS warning signs of toxicity given by Lidocaine, which is both less proteinbound and less potent than Bupivacaine. Slow injection of incremental doses with repeat aspiration, also add to safety and minimize the risk of intravascular injection.

Allergic Reactions:

The allergic reactions to amide local anaesthetics are rare and are thought to be in the order of 1:100,000. (Baker, *et al* 2007)

Commonly Used Agents:

Lidocaine:

Lidocaine is the standard agent against which all other local anaesthetics are compared (Wildsmith, 2003). All the general features of the amides apply to it and it has no unusual properties. It has been used safely for all types of local anaesthesia and is also a standard anti-arrhythmic agent (Wildsmith, 2003). This author recommends 0.5% for skin infiltration and 1% for minor nerve blocks. Maximum dose recommended 5 mg per kg (Baker, *et al* 2007).

Bupivacaine:

Bupivacaine is more long acting than Lidocaine when used for surgical regional anaesthetic blocks. However, cardio-toxicity may occur before neurotoxicity in both man and animals and may therefore be less safe than Lidocaine (Wildsmith, 2003). Maximum dose 2 mg per kg (Baker, *et al* 2007).

Ropivacaine:

This is a chemical analog of Mepivacaine and Bupivacaine. It was designed to retain the desirable properties of Bupivacaine while decreasing cardiac toxicity. It is less potent than Bupivacaine, but requires concentrations up to 1%. It appears to block nerve fibres involved in pain transmission (A delta and C fibres) to a greater degree than those controlling motor function (A beta fibres). It therefore appears to have a differential sensory/motor block with some motor sparing. Maximum dose 3 mg per kg (Baker, *et al* 2007).

Choice of Local Anaesthetic Palliative Nerve Blocks:

The information reviewed above and my own experience was taken into consideration in choosing the anaesthetic agent for palliative nerve blocs. Considerations of safety, the avoidance of adverse effects and satisfactory results with pain reduction resulted in the decision to use Lidocaine as the drug of choice. It was also decided to follow Wild-

smith's advice that 0.5% Lidocaine provided adequate analgesia for skin incision, (and therefore for blocking of pain impulses), and 1% Lidocaine for minor nerve blocks. This has proved satisfactory in practice with Chronic Non-Malignant Pain patients.

Summary:

Therapeutic (or palliative) nerve blocks have been performed since the 1920's and 1930's. Attempts to prolong the period of pain relief with neurolytic and ablative techniques were initially popular, but were curtailed in more recent years by the recognition of de-afferentation pain and are no longer recommended (Fields 1987, Gildenberg and DeVaul 1985), except for trigeminal neuralgia (Pawl 2002) and also of the medial articular branch after a positive result with diagnostic nerve blocks. Woodbridge (1930) found that after therapeutic paravertebral blocks for pleuritic pain, when the pain returned, it was less severe. Bonica (1951) reported pain relief with therapeutic nerve blocks in musculoskeletal disorders including low back and shoulder pain and with occipital, post-herpetic and atypical face neuralgias. Arner, et al (1990) found prolonged pain relief of neuralgia after regional anaesthetic blocks. These effects may be due to the palliative effect of the blocks reducing both peripheral and central effects of central sensitisation described in Chronic Pain (Rome and Rome, 2000). This may be the reason for the pain reduction seen in chronic musculoskeletal pain with palliative nerve blocks. Moreover, repeated palliative nerve blocks were found to reduce pain levels, anxiety and depression and increase quality of life and improve activities of daily living in chronic musculoskeletal pain patients (Rothbart, et al 2000).

Conclusion:

In a multidisciplinary pain management clinic, palliative (or therapeutic) nerve blocks may be used in many chronic pain patients to reduce pain levels, anxiety and depression and improve quality of life and activities of daily living. This treatment is to be considered as an adjunct to other therapies including exercise, cognitive therapy, infrared therapy, medication use (analgesics, antidepressants and anti-seizure medication) and alter-

native therapies including Botox, acupuncture, chiropractic, hypnosis and relaxation. In the last four years I have seen 800 patients who have been investigated for precise diagnosis of spinal and other pain conditions and where appropriate, diagnostic nerve blocks have been performed and if positive, the patients were referred for rhizolysis. No epidural spinal stimulators have been obtained for any of these patients because of an apparent lack of availability in Toronto. It is concluded that palliative (or therapeutic) nerve blocks remain a valuable adjunct treatment in the management of chronic pain in a multidisciplinary pain management clinic.



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