PAIN MANAGEMENT in Primary Care – Current Concepts

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INTRODUCTION

René Descartes *(1596-1650)* may be considered the first scientist in pain physiology. In his famous book *De Homine* (published posthumously in 1662) he described the transmission of pain signals via the nerves and spinal cord, terminating in the brain ventricles and "pineal organ". The pain processing (nociception) system was conceptualised as a *"hard-wired" pain pathway* which reproduces a pain sensation in direct proportion to the extent and severity of the painful (noxious) insult – this outdated concept has been reinforced over years (and even today!) by many text books and healthcare professionals.

After World War II enormous challenges arose relating to the medical care of the millions wounded during the war. John Bonica (1917-1994) was the foremost pioneer in this field in the United States and he established the first inter-disciplinary pain clinic in 1947 at the University of Washington in Seattle, to treat the pain of war veterans.

The modern *discipline of pain management* was launched by the publication of the first edition of John F Bonica's *Management of Pain* in 1953.[1] He recognised that the complexity of pain management was mostly beyond the knowledge and skills of a particular healthcare provider. He also called for improved pain-education for healthcare providers and his initiative resulted in the foundation of the *International Asso-ciation for the Study of Pain (IASP)* in May 1973. The Pain Society of South Africa, known as *Pain SA* is currently a full chapter of the IASP.

The *gate-control theory* of pain mechanisms published by Melzack and Wall in 1965,[2] had a profound influence in the field of pain research and in the development of various forms of pain therapy. This theory integrates the views of neurophysiology and psychology and states that spinal transmission of pain impulses is continuously modulated by the relative activity in the small *(A-delta and C)* fibres and the large *(A-beta)* fibres and by *descending messages* from the brain that originate in the cerebral cortex and brainstem. Activation of the *large A-beta* fibres through pressure or low-intensity electrical currents may *"close the gate"* and prevent noxious stimuli from reaching the brain. The *descending pain inhibitory* pathway involves the action of endogenous opioids and neurotransmitters, including serotonin and noradrenalin – this system is responsible for such

diverse events as the action of opioids and certain anti-depressants, and the benefits of cognitive-behavioural therapy.[3] The descending pathways are influenced by limbic system input, and a relaxed person is likely to experience less pain than an anxious/depressed individual. In subsequent years the theory has been criticised as an over-simplification and a series of gates at different levels of the cord and in the higher centres have been postulated.

The explosive growth of our knowledge in most aspects of pain in recent years has produced major advances in the classification and management of pain. Despite these advances, repeated sur-veys still reveal that poorly controlled pain remains a widespread problem – according to a 1998 World Health Organisation Survey of 26 000 primary care patients on five continents, 22% reported persistent pain over the past year.[6] Part of the problem lies with health-care providers who have failed to keep up with the advances in pain medicine. The "trial-and-error" subjective approach which often still prevails is based on the *biomedical approach* which regards a specific pathway as the source of pain. In this model, pain is seen as a warning signal of tissue injury, likely to be aggravated by physical activity. If conservative treatment fails, some surgical technique will then be able to "fix" the problem.[7,8] This approach is often enhanced by public expectation, and internet websites.

The modern paradigm of pain management has moved from this *biomedical* to the broader *biopsychosocial approach,* where pain mechanisms now integrate input from sensory, emotional and cognitive systems.

In the *biopsychosocial model* of chronic pain, *bio* refers to the sensory or physical component of pain, *psycho* refers to psychological factors (e.g. anxiety and depression) that impact on pain perception, and *social* recognises the importance of interpersonal relationships, work environment etc, on the pain process.[7,9]

The current *definition of pain* as proposed by the IASP reads: *"Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage"*.[11] This definition identifies the complex and multi-dimensional experience of pain, where the patient's physical, cognitive, emotional and behavioural characteristics mediate the pain experience.[12] Emotional factors may have a profound effect on pain perception and depression may lower the *tolerance threshold* for pain perception.

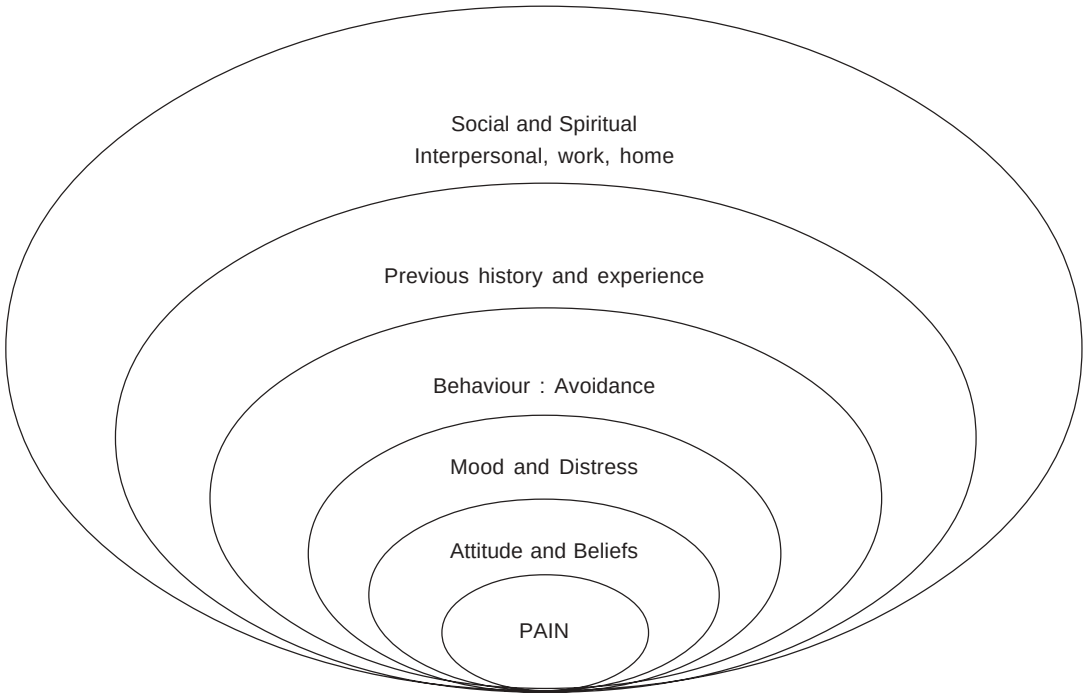
With *acute pain* of known cause, there may only be a minor contribution from the emotional and cognitive dimensions. In patients with a *chronic pain* disorder for many years the emotional (e.g. depression) and cognitive (e.g. negative thoughts) dimensions may play a major role in pain perception.[13]

Pain transmission from the nociceptors in the periphery takes place via the *myelinated A-delta* fibres (rapid trans-mission, sharp pain) and the *unmyelinated C-fibres* (slow transmission, dull pain).

These *afferent fibres* transmit the painful stimuli to cell bodies in the *dorsal root ganglion* and then to the *dorsal horn* in the spinal cord. From here, the stimuli reach the *somato-sensory region* in the postcentral parietal cortex, via the spinothalamic tract, where the sensory dimension of pain is processed. Connections between the cingulate cortex in the limbic system and the frontal lobe are thought to be responsible for the emotional dimension of pain.[13,14]

However, the nociceptive system is very complex and is continually influenced by emotional and cognitive processes. Functional imaging over recent years has

Biopsychosocial model of pain[7,9,10]



provided new insights in the multitude of brain regions activated following a pain stimulus, known as the *"pain matrix".* Rather than registering the pain signal to produce pain perception, the *brain matrix* constructs the pain experience by integrating multiple inputs, including biological (sensory) factors, present and past psychological events and socio-cultural influences.

ACUTE PAIN

Acute pain is a *normal biological* response to injury / tissue trauma and a signal of ongoing or impending tissue damage e.g. post-operatively. It contributes to survival by protecting the organism from injury and promotes healing when injury has occurred. Acute pain is a symptom that must be treated or its cause eliminated.[14,15]

There is increasing recognition that *long-term changes* may occur within the peripheral and central nervous system following the noxious input of painful stimuli. Even brief intervals of untreated acute pain can induce long-term neuronal remodelling and *sensitisation* ("plasticity") and chronic pain.[2,14,15,16] Studies have indicated that the biological foundation for long-term persistent pain – which is due to the neuronal expression of new or dormant genes – could be in place within hours of the initial injury.[16] This *"plasticity" of the nervous system* then alters the body's response to further sensory input and it becomes more sensitive to pain impulses.

The mechanism through which these changes take place, is known as *central sensitisation* and occurs in the dorsal horn of the spinal cord following tissue injury. It may be defined as *hyperexcitability of the CNS* neurons in response to peripheral noxious stimuli, leading to an exaggerated response to painful stimuli (hyperalgesia) and persistent pain. This is a complex process involving many neurochemical and molecular changes, and explains the concept of chronic pain in the absence of ongoing tissue damage (e.g. persistent pain long after breast surgery).[16,17] *Central sensitisation* is induced by the release of neuropeptides such as *substance P* and *glutamate* by the central terminals of C-fibres, which then activate the NMDA-receptor-complex. The subsequent influx of Ca^{++}-ions initiate a cascade of intracellular events leading to persistent neuronal changes and chronic pain.[15,16,17] It should be noted that *supraspinal input* from different brain areas is also dynamically involved in this process.

Acute post-operative pain is followed by persistent pain in 10-50% of individuals after common procedures, such as inguinal hernia repair and breast surgery.[18] *Central sensitisation* after tissue injury and altered gene transcription is also believed to be the mechanism for this phenomenon. Risk factors include intensity of post-operative pain, genetic susceptibility and psycho-social factors.[18] Current data therefore support a comprehensive and multi-modal approach to post-operative pain management.[19] It is therefore fundamental to *assess* pain routinely, just as one monitors other vital signs, and to treat acute pain as early as possible, using non-drug and drug interventions.[16]

In conclusion, early and appropriate acute pain management will reduce the intensity of acute pain and may also lesson the likelihood of changes in the spinal cord and brain that are involved in the transition to chronic pain.

PRIMARY TYPES OF PAIN[14,15]

• Nociceptive pain (e.g. trauma, surgery)

Nociceptive pain occurs when intact peripheral nerve endings *(nociceptors)* are stimulated by noxious stimuli which may be mechanical, thermal, chemical or inflammatory. Tissue damage generates the release of peptides and other components of the *inflammatory soup* with eventual *peripheral sensitisation.* Blocking of nociceptive messages at these first stages may prevent some of the central alterations associated with central sensitisation.[15]

• Neuropathic pain

While nociceptive pain is the result of stimulation of the nervous system, neuropathic pain is due to a lesion in the peripheral or central nervous system, e.g. in patients with diabetic or AIDS polyneuropathy, post-herpetic neuralgia, nerve damage after trauma / compression, spinal cord injury, etc.

• Dysfunctional pain

There is a large group of chronic pain patients where no peripheral abnormality or neurological deficit can be detected. The mechanism of pain is *abnormal sensory processing* of non-painful stimuli – once the central nervous system has become sensitised, minimal peripheral pain stimuli will be perceived as being painful *(pain amplification)* and this is known as hyperalgesia.[14,17,20] These include the *idiopathic pain disorders* such as irritable bowel syndrome, chronic headaches, post-whiplash disorders, fibromyalgia syndrome and others. There is growing evidence that the onset of these disorders is associated with both physical and emotional triggers that initiate pain in individuals with a *genetic susceptibility*.[21]

Both neuropathic and dysfunctional pain may be present spontaneously in the absence of any peripheral stimulus or "organic cause". The increased sensitivity of the nervous system may thus lead to pain in the absence of noxious stimuli, and it is wrong to assume that these patients are only "psychological" or "hysterical".[14,20,21] The pain in these patients may also be evoked by light touch and this phenomenon is known as *allodynia.*

• Mixed pain

This include patients with *cancer pain* and *low back pain* (in particular low back pain following surgery, or *failed back surgery syndrome*) where neuropathic, nociceptive and myofascial components may contribute to the patient's pain experience.

Not all patients with chronic pain have similar mechanisms, therefore a specific analgesic will not be equally effective for all different pain disorders. Identification of the mechanism involved in a particular pain disorder, will allow for a more *mechanism-specific* pharmacological approach, rather than the current empiric approach where analgesics are classified by the severity of pain (*mild, moderate* and *severe*).[14]

CHRONIC PAIN

The IASP has defined chronic pain as *"pain that persists for longer than the time expected for healing, or pain associated with progressive, non-malignant dis-*

ease", usually taken to be three months.[22]

Chronic pain often persists long after the tissue trauma that triggered its onset, has resolved and may be present in the absence of identified ongoing tissue damage or a previous history.[23] Chronic pain is a dysfunctional response which mostly does not warn the individual of underlying disease or injury.[15,23] However, it may cause severe suffering and reduced quality of life and has often been labelled a *"disease in its own right".*

Chronic pain may be associated with an underlying chronic disease such as arthritis. However, the largest group of chronic pain patients in the current epidemic in developed countries, comprises the chronic pain syndromes of unknown etiology (e.g. the vast majority of patients with *chronic low back pain*).[15] Many patients with chronic pain suffer from clinical syndromes for which there are no confirmatory laboratory studies and are diagnosed on the basis of clinical criteria, e.g. the headache syndromes, neuropathic pain, irritable bowel syndrome, fibromyalgia, myofascial pain syndrome etc.[24]

Chronic pain has been recognised as one of the most frequent reasons why people seek medical attention and a Canadian study found a chronic pain prevalence of 27% for men and 31% for women.[25] Based on USA data, chronic pain costs society more than heart disease and cancer combined.[15]The tendency to consider chronic pain as either psychological or physical, implies a false dichotomy – both play a role in most chronic pain disorders, although the balance between organic pathology and psychosocial contributions may differ in different pain disorders.[26,27]

Emotions and beliefs will influence and modify (not cause) the perception of pain, regardless if the pain is associated with cancer[27] or not. Neoplastic disease has a very negative connotation with images of death, fear of the unknown, loss of independence, anxiety and depression etc. There is evidence that most of the same emotions are also prevalent in patients with all *persistent pain syndromes*.[27] Negative beliefs and an outlook of hopelessness may generate abnormal or maladaptive *illness behaviour* with increased help-seeking and painreporting.[26]

The emotional component of pain is complex and is influenced by past experiences, patient-beliefs and fears.

PAIN ASSESSMENT[28,29,30,31]

Pain is a subjective, complex and personal phenomenon and can only be assessed indirectly by patient report.

Methods used for *acute pain* screening are insufficient to provide a comprehensive view of the multidimensional impact of *chronic pain* on the patient. In a patient with chronic pain, assessment should not be limited to *pain severity*, but at least also include pain-related *functional interference* and the *emotional impact* of the pain.[29] (There is often not a direct relationship between these 3 variables.)
Pain assessment is critical to monitor the clinical condition over time and to analyse response to treatment. The *pain history* should include present and past therapies, medical and psychiatric history, social / physical disability and reinforcing factors, behavioural responses to pain, pain beliefs and mood-status.

Uni-dimensional pain scales assess *pain intensity* include verbal rating scales, numerical rating scales, visual analogue scales and picture scales *(facial expressions).*

Multidimensional pain scales assess the effect of pain on mood, activities and quality of life, and include the McGill Pain Questionnaire (very time-consuming for patients and health professionals to fill in) and the short and more practical Brief Pain Inventory.

A full *clinical examination* may provide clues to the causes of pain and also demonstrate associated features such as disuse-atrophy, sympathetic over-activity and neurological deficits.

Formal *psychological evaluation,* (when indicated) includes assessment of emotions / mood, cognitive function, maladaptive and adaptive pain behaviour, coping behaviours *(e.g. fear avoidance)* and assessing for somatoform pain disorder.

MANAGEMENT OF CHRONIC PAIN SYNDROMES

A critical factor underlying suboptimal care in chronic pain patients has been the perpetuation of the reductionistic *biomedical pain model,* – where pain is defined as a sensory event that results from underlying organic disease or tissue damage.[32] The biomedical approach has traditionally promised a cure by cutting or blocking the pain pathways pharmacologically or surgically.

The *biopsychosocial approach* views pain as a dynamic interaction between physical, psychological and social factors, and more realistic treatment goals for patients include[24]:

- The reduction, mostly not elimination, of pain
- Improvement in physical / social functioning
- Improvement in mood and associated symptoms such as sleep cycle
- Development of active coping style and self-management-skills
- A return to work
- Reduction in utilisation of medical services

The treating professional often has to reconcile a patient in severe suffering with little or no evidence of tissue pathology. Disbelieving a patient may damage the therapeutic relationship, prevent the delivery of optimal therapy, but it may also promote exaggeration and *abnormal pain behaviour.*[34]

Evidence increasing lends support to the use of an *interdisciplinary approach* where multiple therapies are provided in a *co-ordinated* manner,[24,34] and where there is *active interaction* and a *common philosophy* that promotes *active patient involvement*, between participants. (In the traditional multi-disciplinary model each contribution often stands on its own and there are often no shared objectives in the management of the patient.)[24,34]

The *core-team* that is involved in the *primary care management* of chronic pain patients, will differ from area to area and also depends on the availability of resources and the complexity of a patient's problem. In the South African context, a *core-team* may consist of a *pain management physician* (mostly a primary care

doctor with a special interest in pain management) and a *physiotherapist*. After initial screening the core-team will then decide which additional members will be needed for the initial assessment / management of the patient, e.g. an occupational therapist, a psychologist, an anaesthesiologist with interventional skills etc.

The comprehensive interdisciplinary evaluation will lead to recommendations for treatment depending on the needs and expectations of the individual. For some individuals education and intermittent pharmacotherapy may suffice, others may need an intensive rehabilitation programme, including formal cognitive behavioural therapy.

The roles of team members may also overlap and the physiotherapist may also be responsible for education, the exercise programme and to implement the principles of cognitive behavioural therapy,[24] therefore, communication between team members is essential.

A functional restoration programme may take place over 4-8 weeks, depending on the severity and chronicity of the patient.

It has been shown that interdisciplinary management, which emphasises *functional restoration* produces the best outcomes.[35]

Education is the starting point of management and reinforcement of initial explanations is mostly necessary. It is very important to *validate* the patient's pain complaint and to *explain* why they have pain. Factors that have initiated the pain problem are often different from those that maintain it.

The *gate-control theory* of pain provides an explanation for the direct influences on a person's perception of pain and provides a model to illustrate *mind-body interaction* in chronic pain. Fears, apprehension and bad memories of pain may increase the pain experience.

The patient should be informed about the goals of the treatment programme and certain *chronic pain myths* should be dispelled, including:[36]

- Search long enough, you will find the cause and the cure
- Abnormal scans validate and explain your pain
- Only organic pain is real
- You have to learn to live with it
- Let pain be your guide, rest when it hurts
- Hurt is equal to harm

The outcome of management is closely determined by what the doctor, therapist and patient expect – whatever the cause of pain, patients can learn techniques to make the pain less intense.[36] Access to *appropriate resources* provides motivation and inspiration. Unfortunately, some of the available books, internet-websites and in particular pain-support groups *reinforce* self-defeating thought patterns and *illness-behaviour*.

Physiotherapy, exercise and occupational therapy

Physiotherapists practise a movement and rehabilitation based profession and have frequent contact with patients in pain – they therefore have the *"potential to revolutionise the management of pain and alleviate much suffering"* – in the words of Sir Patrick Wall.[33] The role of the physiotherapist is broad and includes education on pain mechanisms and self-management, goal-setting and a graded activity programme, pacing and helping patients to acquire problem solving skills.[33]

The most important element in physical rehabilitation addresses improvement in function through *therapeutic exercises* designed individually to increase functional activity. Exercises include range of motion and stretching, strengthening, specific exercises, general aerobic fitness and relaxation exercises.

Passive manual methods are de-emphasised in modern physiotherapy and should be integrated in a more comprehensive programme. Physiotherapists who are too somatically focussed e.g. on the "degenerated disc" may reinforce beliefs and perpetuate the problem.

Although the physiotherapist doesn't delve into the "whole psychology" of the patient, he/she should be informed on the cognitive and behavioural components of the pain presentation and assist in addressing inappropriate pain-behaviour.

Occupational therapists work closely with physiotherapists in activity planning according to set goals and in assessing domestic and workplace circumstances. The patient who leaves the workplace and stops living is unlikely to recover – patients need a reason to recover, not just a desire to be rid of the pain.[36]

William Fordyce said "People don't hurt as much if they have something better to do."[36,37]

Principles of pharmacological therapy[32,38,39]

- The goal of pharmacotherapy should be to improve *pain intensity and functioning* (sleep, mood and exercise tolerance), while avoiding cognitive impairment and organ toxicity.[32]
- Many patients don't present with pure nociceptive or neuropathic pain, but rather have a *mixed pain syndrome,* therefore *rational polypharmacy* that targets key peripheral and central mechanisms and modulating pathways often produces best outcomes.[32]
- The poor efficacy of current treatment for *neuropathic pain* reflects that, even in single disease entities, only some patients respond to pharmacotherapy.[14]
- The World Health Organisation (WHO) *analgesic three-step ladder* for the rational use of analgesics in cancer pain, has also been applied for *non-cancer pain* for many years, in particular for nociceptive pain. There is a move away from this *empirical therapeutic* approach for chronic pain, to one that is targeted specifically at the particular mechanism of the pain experienced by the patient.[14]
- Start with low doses and titrate carefully upwards to optimise pain relief while managing side effects.
- For *continuous analgesia* consider long-acting medication on a regular basis, rather than *"as needed".* Additional short acting analgesia may be required ±30 minutes before pain inducing activities, e.g. physiotherapy.[38]
- Analgesics are generally more effective for *nociceptive pain*, less effective for *neuropathic pain*.

Non-opioid analgesics

Paracetamol is still recommended as first-line therapy for osteoarthritis of the

hip or knee.[40] It causes little or no GI irritation and its effect on renal function is minimal.[40]

Non-steroidal anti-inflammatory drugs (NSAIDs) may be combined with paracetamol or opioids. Inhibition of COX-1 is responsible for common side-effects such as GI irritation / peptic ulceration and inhibition of platelet aggregation. *COX-2 specific agents* (e.g. *celecoxib*) reduce these risks, but are also (similarly to older NSAIDs) associated with renal dysfunction and potential cardiovascular side-effects, e.g. hypertension and coronary vascular disease. These side-effects are in particular important in older patients on long-term medication.

Opioid analgesics

Weak opioids

Codeine phosphate is a very weak analgesic and has almost no analgesic effect by itself, except at doses above 90mg.[39] It is often used in combination with paracetamol, however, its role in chronic pain management is very limited (if any).

The use of *polycomponent codeine combinations* (containing caffeine, meprobamate and others) is strongly discouraged in chronic pain. Their potential for nephrotoxicity is greater, they are often associated with rebound pain and are addictive.

Tramadol is an opioid of moderate strength and also inhibits noradrenaline and serotonin re-uptake from nerve endings. A number of studies have demonstrated the efficacy of tramadol in chronic pain conditions such as neuropathic pain, osteoarthritis, fibromyalgia and low back pain.[39,40] It has a proven synergy with paracetamol and is not associated with peptic ulceration, renal dysfunction or cardiovascular side-effects.[39,40] It has a very low addictive potential (less than 1/100 000 users).

Strong opioids

Current evidence supports the use of strong opioids in a carefully selected subset of patients with *chronic non-cancer pain*.[41,42,43]

Strong opioids should be reserved for patients with *resistant nociceptive and / or neuropathic* pain with the aim of improving physical and social function. A detailed assessment should be performed by an experienced *pain management physician.* Strong opioid treatment should not be considered life-long treatment and be limited to *sustained – release opioids*, e.g. *transdermal fentanyl* and *sustained release oral morphine*. *Addiction is rare* if used appropriately and guidelines exist for appropriate use.[43]

Adjuvant drugs[24,32,44]

Neuropathic pain is mostly treated with medications that influence neurotransmitters, e.g. antidepressants and epileptic drugs, and opioids are reserved for patients with refractory neuropathic pain.

Antidepressants

- *Tricyclic antidepressants (e.g. amitriptyline)* are effective for neuropathic and non-neuropathic pain and its analgesic effect occurs at lower doses than its antidepressant effect. However, these drugs have the potential for bothersome anti-cholinergic side-effects and life-threatening cardiovascular effects.
- *Selective serotonin re-uptake inhibitors (SSRIs)* are predominantly serotonergic drugs and are mostly ineffective in treating chronic pain.
- *Serotonin and norepinephrine re-uptake inhibitors (SNRIs) e.g. venlafaxine* and *duloxetine* have balanced inhibition of serotonin and norepinephrine without blocking other receptors that are responsible for the typical tricyclic side-effects. They have proven effectivity in patients with neuropathic pain.

Antiepileptic drugs

Antiepileptic drugs act at several sites that are relevant to pain and are believed to enhance central inhibition and limit neuronal excitation.

Of the *first generation agents, carbamazepine* is indicated for trigeminal neuralgia and has shown modest efficacy in patients with diabetic neuropathy and post-herpetic neuralgia.[44,45]

Second generation antiepileptic drugs are better tolerated and have fewer central nervous system side effects. *Gabapentin* has repeatedly demonstrated analgesic efficacy and improvement in mood and sleep in several studies,[46] but is not registered for pain management in South Africa.

Pregabalin inhibits ectopic discharges from injured nerves by inhibiting the calcium-channels pre-synaptically. It has better bio-availability than gabapentin and effectivity has been proven in several trials in diabetic neuropathy and post-herpetic neuralgia.[46,47] (It was recently approved by the US Food and Drug administration.)

The efficacy of antidepressants and antiepileptics in neuropathic pain is similar, therefore initial drug selection is based on side-effects, contra-indications, co-morbidities and cost.[45,47]

Principles of behavioural therapy[24,26,36,48,49]

Several behavioural approaches may lead to long-term reduction in pain intensity and improvement in physical and social functioning in chronic pain patients. In some chronic pain patients, the *patient's belief* about the pain and its effects is a better predictor of suffering and disability than the actual disease process and or tissue damage.

Cognitive therapy aims to help patients identify maladaptive thinking patterns and develop the ability to challenge these thoughts. This is often a delicate and prolonged process, because most cognitive activity takes place below the level of awareness.[33]

Errors of thinking include:

- I will never get better
- There is nothing I can do
- I am afraid to move
- The situation is hopeless because the pain is incurable
- No one can work out what is wrong

Simple principles such as explanation and clarification are powerful and often neglected interventions. Engaging in pleasurable, stimulating and distracting activities are also powerful means to limit disability. Relaxation techniques and imagery are often of great value.

Primary objectives in a programme of cognitive behavioural therapy include:

- Change view of pain from overwhelming to manageable

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- Change from passive and helpless to active and competent
- Be aware of the association between negative thoughts and maladaptive automatic behaviour
- Teach specific coping skills
- Anticipate problems pro-actively

Cognitive behavioural therapy has 4 basic components:[24]

- Education
- Skills acquisition
- Cognitive and behavioural rehearsal
- Generalisation and maintenance

Interventions

Less invasive methods[24,32,50]

- *Myofascial trigger point infiltration therapy* provides good pain relief and facilitates patient participation in active physical therapy, if it is correctly performed in well selected patients with *myofascial pain syndrome*.[51,52,53]
- *Nerve block therapy* is useful to allow patients to participate in active rehabilitation. It is also used diagnostically to determine the pain generator e.g. lumbar *medial branch blocks*.[50] *Sympathetic nerve blocks* such as at the stellate ganglion and celiac plexus are particularly effective in visceral pain states (e.g. abdominal malignancy) and in sympathetically maintained pains.[24,50]
- *Epidural steroid injections* may provide pain relief in patients with radicular low back pain.
- *Radiofrequency facet rhizotomy* is sometimes performed after successful nerve block therapy.

More invasive methods

- These include *surgical procedures* such as microvascular decompression for trigeminal neuralgia, joint-replacement surgery for severe osteoarthritis, spinal surgery after strict selection, micro-DREZ-otomy after plexus brachialis avulsion injury and palliative neurosurgery for the pain of malignancy (e.g. cordotomy).
- *Spinal cord stimulation* has evolved as a reversible, non-destructive and low-morbidity technique for chronic intractable pain. It is widely used in Europe in the treatment of low back pain and the ischaemic pain of peripheral vascular disease.
- *Epidural* and *intrathecal drug delivery systems* have been used successfully over many years in a group of patients with intractable pain when other therapies have failed.

There has been a shift in emphasis in the management of chronic pain to the interdisciplinary biopsychosocial approach. This include educational interventions (e.g. the patient's active role in management), cognitive behavioural and supervised exercise therapy. Pharmacological treatments are important for pain relief, preferably for short-term and/or intermittent use. The use of invasive therapy should be carefully and conservatively considered after bio-psychosocial assessment and performed by appropriately trained and skilled healthcare providers.❐

Sixty-seven references available on request.