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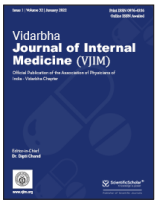

Review Article

Basics of Chronic Pain Management

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ABSTRACT

Approximately one in ten people suffer from chronic pain globally with pain being the most common reason to seek medical help. Despite the long-term prevalence of pain, the practice of pain management and the scientific discipline of pain research are relatively new fields. To relieve suffering from chronic pain in the 'fifth vital sign' era to date remains a 21st-century dilemma for healthcare providers. In the current review article, numerous articles from various pain journals and books for chronic pain management using medical search engines such as PubMed, Scopus, and Google scholar have been reviewed in an attempt to shed light on the approach, evaluation, and management of chronic pain. Along with the same, recent advances in pharmacotherapy and interventional nerve blocks have been discussed in brief.

Keywords: Chronic non cancer pain, Pharmacotherapy, Nerve blocks, Cancer pain

INTRODUCTION

The International Association for Study of Pain defines pain as 'An unpleasant sensory and/or emotional experience associated with, or resembling that associated with, actual or potential tissue damage.' Even if the patient is experiencing pain in the absence of actual tissue damage, it should be accepted as pain. According to a global burden disease study, one in ten adults is diagnosed with chronic pain each year globally.[1]

PAIN PATHWAYS

(1) Transduction, (2) Transmission, (3) Perception, and (4) Modulation. These are the sequence of events that are involved in the neural processing of noxious stimuli [Figure 1].[2-6]

TYPES OF AFFERENT FIBERS THAT CARRY PAIN

- A-Delta fibre– myelinated and fast, with a conduction velocity of 5–15 m/s, transmits sharp, localised, and fast pain
- C-fibres – unmyelinated with slow conduction <2 m/s, carry dull, diffuse, aching, and delayed pain.

Gate control theory of pain

Melzak and Wall proposed historical 'Gate control theory' in 1965. It states that activating larger diameter A-beta fibres leads to inhibition of pain signals transmitted through smaller diameter A delta and C fibres. An inhibitory interneuron acts as physiological gate which is closed by stimulation of A beta fibres. For example, Spinal cord stimulator and TENS for myofascial pain.[6]

AUTONOMIC NERVOUS SYSTEM

Autonomic nervous system plays an important role in different types of pain. For example, pain signals from thoracic or abdominal viscera and intervertebral disc-or vertebral body are carried by different afferent sympathetic fibres.[1,2-4,7]

SOMATIC SYMPATHETIC COUPLING AND MECHANISM

Somatic and sympathetic nerves are well insulated with no cross-talks in between them. However, with pathological conditions such as peripheral nerve injury/neurolysis and complex regional pain syndrome (CRPS), there is a cross-connection between somatic and sympathetic signals. Sympathetic efferent fibres release catecholamines, which instead of acting on sympathetic receptors may stimulate the nociceptors and generate action which is carried by somatic nerves[2]

SYMPATHETIC BLOCKADE AND ITS SIGNIFICANCE IN PAIN MANAGEMENT

Sympathetic blockade results in interruption of transmission by both the efferent and afferent fibres. It does not result in sensory or motor loss as seen in the somatic blockade.[2]

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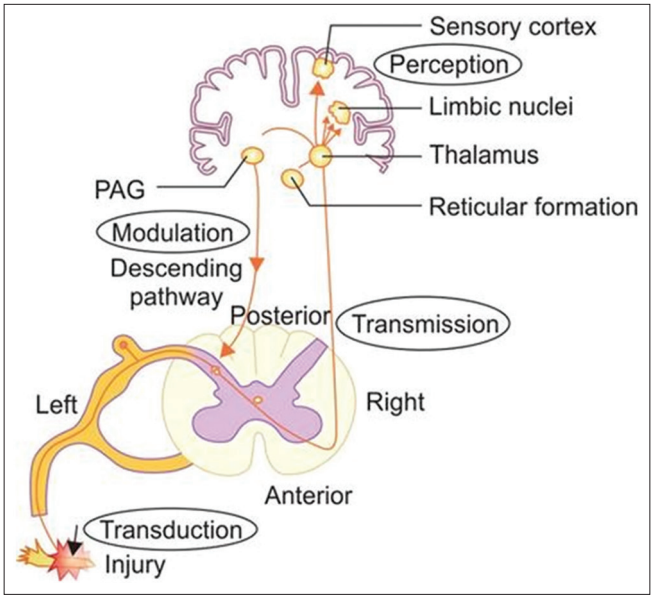

Figure 1: Afferent pain pathway.

Examples of sympathetic blockade at various levels:

1. Stellate ganglion and lumbar sympathetic blockade for CRPS of the upper and lower limbs
2. Splanchnic and celiac plexus block for the upper abdominal malignancy and chronic benign pain
3. Superior hypogastric plexus block: Cancer and non-cancer chronic pelvic pain.[8]

TYPES OF PAIN

Based on time

- Acute pain: Pain lasts <3 months, mostly nociceptive
- Chronic pain: Pain persisting for more than 3 months, mostly neuropathic.[9,10]

Based on mechanism

- Nociceptive pain arises from actual or threatened damage to non-neural tissue. For example, acute trauma, postoperative pain, and sickle cell crisis
- Neuropathic pain is caused by a lesion or disease of somatosensory nervous tissue. For example, post-herpetic neuralgia, trigeminal neuralgia, distal polyneuropathy, and CRPS type
- Mixed pain is having both nociceptive and neuropathic components. For example, cancer pain and vascular pain syndromes
- Non-inflammatory/non-nociceptive where the aetiology of the origin of the pain is still confusing. For example, fibromyalgia and irritable bowel syndrome.

ASSESSMENT AND DIAGNOSIS OF PAIN

Pain history

An effective history is vital in making a diagnosis of pain. A pain questionnaire should be based on the following aspects of pain.[9-18]

1. Onset, location, and radiation of pain
2. Character or description of pain:
   - Nociceptive pain – Dull aching, cramp, stabbing knife, shooting and throbbing
   - Neuropathic pain – current-like and tingling pain
   - Mixed–burning pain in the chest
3. Aggravating and relieving factors would explain the possible pathophysiological mechanism.

PAIN ASSESSMENT TOOLS[2,8,18]

1. Visual Analogue Scale (VAS): This is the most commonly used scale. In this, the patient is asked to place a marker on a 100 mm continuous line between no pain and the worst imaginable pain on every visit [Figure 2]
2. Numerical Rating Scale (NRS): The patient directly assigns a number between 0 (no pain) and 10 (the worst pain imaginable) [Figure 2]
3. Verbal Categorical Scale: The patient describes the severity of pain ranging from no pain to 'mild,' 'moderate' and 'severe' [Figure 2].
   - Mild pain: VAS or NRS of 0–3
   - Moderate pain: VAS or NRS of 4–7
   - Severe pain: VAS or NRS of 8–10 or even if the pain is moderate but the patient says I cannot bear it, it can be counted as severe pain.
4. The faces pain rating scale is used mainly for children and mentally impaired patients. The scale depicts six faces of facial features, each with a numeric value of 0–5, ranging from a smiling and happy face to a sad and teary face.
5. Comprehensive Pain assessment Questionnaires:
   - McGill pain questionnaires
   - Brief pain inventory
   - Oswestry pain questionnaire
   - Patient health questionnaire-9 scoring is to rule out depression.

CLINICAL EXAMINATION

General physical examination

Pain diagram

It is essential to draw a pain diagram for assessment of pain as it provides [Figure 3][8]

- Visual confirmation of patient's pain
- It can be stored as a medical record

- It helps to note the change in the area of pain especially if the patient's pain keeps on changing with every visit.

NEUROLOGICAL EXAMINATION

It consists of an examination of all cranial nerves, spinal nerves, and autonomic nervous systems.

Musculoskeletal system examination

It includes:

- Posture, muscle symmetry, and obvious muscle wasting: Muscle spasm occurs in spinal nerve compression in corresponding myotomes. Straightening of lumbar lordosis or canal stenosis occurs due to spasm of erector spinae muscle. For example, stooped gait or 'shopping cart' posture in lumbar canal stenosis
- Range of motion of joints is noted.
- Palpation: Done for soft tissues and bones in affected dermatomyotomes to map areas of pain
    - Oedema, clicks, and crepitus in joints
    - Cord like the feel of muscles that are in spasm can be palpated
    - Tenderness, for example, paraspinal tenderness may be felt in facet joint pain
    - The presence of trigger points like trapezius trigger points can cause pain and numbness of the arm or forearm and mimic pain caused by a cervical herniated disc.

SPINE EXAMINATION

It includes sensory and motor examination of spinal nerves.

Spine symmetry and kyphosis or scoliosis should be noted.

Spinal flexibility – Pain on certain movements of the spine could indicate possible diagnosis in spine pain patients like pain in the neck or back on forward bending will indicate discogenic pain. On the contrary; pain on backward bending will point toward facet arthritis or muscular spasm [Table 1].

Palpation

Spinous process tenderness might be present in discogenic pain, vertebral body fractures, or spine metastasis.

PHARMACOTHERAPY IN PAIN MANAGEMENT

The most common method of pain management is the use of drugs. Nociceptive pain responds well to simple analgesics such as anti-inflammatory agents, but neuropathic pain responds better to coanalgesics such as anticonvulsants and anti-depressants.[2,8]

Drugs should have

- Rapid onset, long duration, and effective pain relief
- Minimal side effects on long-term use
- Easily self-administered.

WHO STEP LADDER

The World Health Organisation 1986 devised a step-wise approach popularly known as the 'WHO step ladder' approach for the management of chronic pain and cancer pain patients. In 2016, with the advent of newer modalities and interventions, this original analgesic ladder was upgraded to the current modified four-stepladder approach [Figure 4].[2]

Drugs used in pain management can be categorised under two broad headings: Analgesics and Coanalgesics.

- Analgesics are anti-inflammatory, non-selective COX – inhibitors like NSAIDs, and selective COX-2 inhibitors. Acetaminophen/Paracetamol and Opioids
- Coanalgesics or adjuvant group includes anticonvulsants, antidepressants, local anaesthetics, steroids, muscle relaxants, botulinum toxins, and NMDA receptor antagonists. Others such as Alpha 2, agonists, calcium channel blockers, vitamins, and nutritional supplements.

ANALGESICS

Anti-inflammatory, COX –inhibitors: NSAIDs, COX -2 inhibitors

Commonly used NSAIDs for pain management include Ibuprofen, Diclofenac sodium, Nimesulide, Ketoprofen, Naproxen, Piroxicam, and Mefenamic acid. COX-2 selective inhibitors such as etoricoxib and celecoxib have better gastrointestinal safety profiles and least effect on platelet function.[2,19,20]

These are useful in inflammatory conditions such as rheumatoid arthritis, osteoarthritis, and ankylosing spondylitis where inflammation is the main pathology.

OPIOIDS

Opioids can be classified according to their receptor activity or duration of action (long acting and short acting) or their analgesic and ceiling effects (weak and strong opioids). Opioid receptors are G protein-coupled receptors. mu is the major receptor responsible for analgesia. Pure agonist like morphine is preferred in moderate-to-severe chronic pain like cancer pain. Opioids with agonist-antagonist have a ceiling effect for analgesia. Weak opioids are tramadol, tapentadol, and codeine. Strong opioids are morphine, fentanyl, and buprenorphine. Long-acting opioids are good to treat baseline pain whereas short-acting opioids are useful in breakthrough pain. If a trial of opioids is considered, then: [Table 2][8]

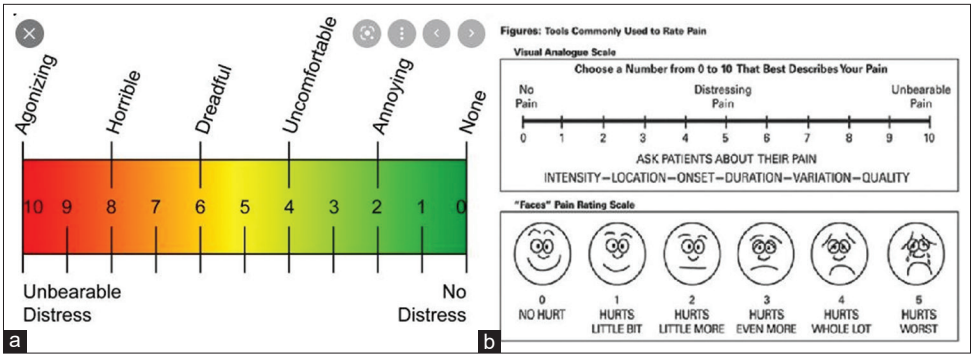

Figure 2: (a) Visual analogue scale (VAS) and (b) Combined pain assessment tool showing numerical rating scale, VAS, Verbal categorical scale, and Wong – Baker fascial pain rating scale.

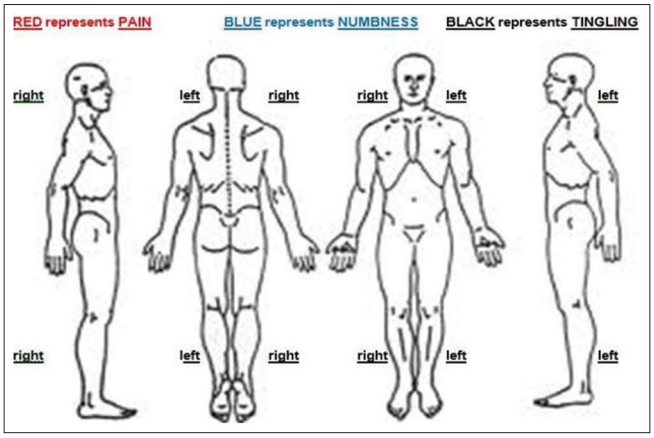

Figure 3: Pain diagram.

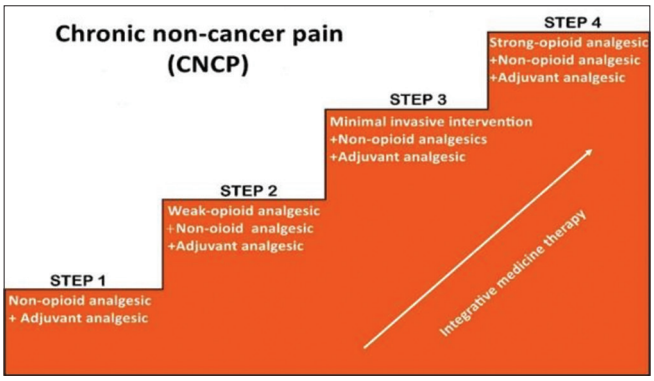

Figure 4: Modified four-step ladder.

- The patient should be explained the benefits and risks of opioids use
- The WHO analgesic ladder to be followed
- Strong opioids like morphine should never be used as SOS drug
- Side effects should be explained and majors to be taken to correct them if possible

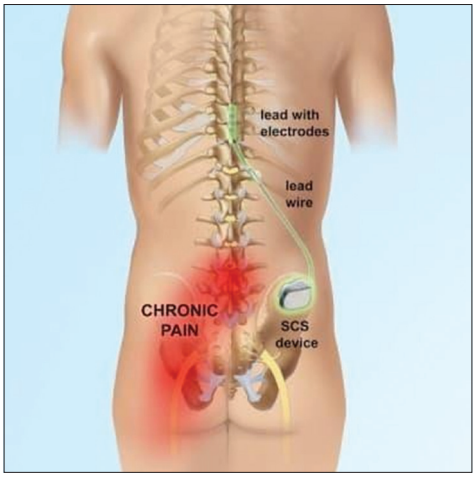

Figure 5: Spinal cord stimulator.

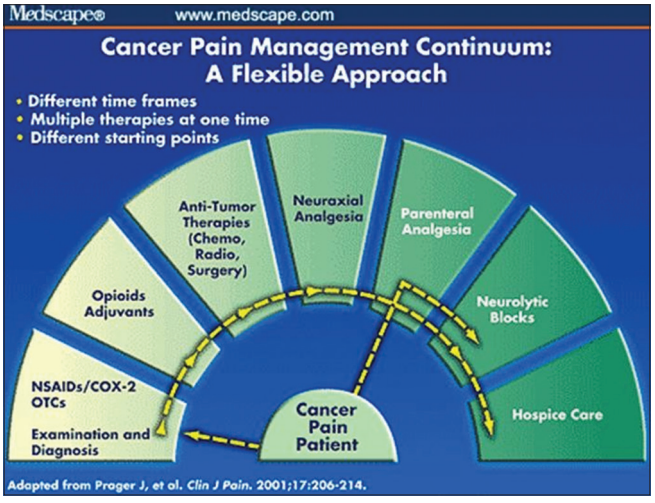

Figure 6: Cancer pain management continuum: A flexible approach.

- For elders 'start low and go slow', instructions for follow-up and emergency should be provided.

Table 1 : Spine-specific tests.[8]

Name of the tests	Analyse	Remarks
SLR or Lasegues test	Leg pain at an angle between 40 to70 will be positive	Positive means L4, L5, S1, and S2 are having pain on stretching the leg
Reverse SLR	Anterior thigh pain on the stretch will be positive reverse SLR	Positive meaning L2, 3, 4 could be getting stretched causing pain
Cross SLR	Positive leg pain on the affected side will indicate stretch of L4,5, S1nerveroots	More specific than SLR
Fabre test for sacroiliac joint pain	Pain on the affected side will indicate strain of the SI joint capsule and ligaments	Not specific but hints at SI joint pathology

Table 2: Commonly available opioids in India.[8]

Drug	Route	Starting dose and frequency	Onset of action
Codeine	Oral	30 mg 3–6 hrly	30–45 min
Morphine	Oral	10 mg 3–6 hrly	30–60 min
Morphine Parenteral	SC/IM/IV	5 mg 3–6 hrly	Subcutaneous10–30 min. IM 10–20 min; IV2–5 min
Fentanyl Trandermal	Transdermal	25 ug once in 3 days	10–12 hrs
Fentanyl Parenteral	Transdermal	50 ug 1–4 hrly	SC 10–20 min; IM 10–20 min IV 2–5 min
Fentanyl Lozenges	Transmucosal (Lollipop)	200 ug	5–10 min
Pentazocine	SC/IM/IV	30 mg 3–6hrly	SC 10–30 min; IM 10–20 min IV 2–10 min
Buprenorphine (Transdermal)	Transdermal	5 mg once in 7 days	12–24 h
Buprenorphine Parenteral	SC/IM/IV	150 ug 8–12 hrly	SC 10–30 min; IM 10–20 min IV 2–10 min
Tramadol	Oral	50 mg 6–8 hrs	30–60 min
Tramadol	SC/IM/IV	50–100 mg 6–8 hrly	10–30 mins
Tapentadol	Oral	50 mg, 100 mg OD to 4 times a day	10–30 min

Table 3: Commonly used anticonvulsants.

Drug Name	Dose	Mechanism of Action	Side Effects
Gabapentin	Starting dose 100–300 HS. The usual dose is 900–3600 (max) mg in three divided doses	Membrane stabiliser by binding at alpha2delta subunit of L type calcium channel.	Dizziness, somnolence, fatigue, Peripheral oedema
Pregabalin	Starting dose 50 mg/day Max dose 300 mg/day		Dizziness, somnolence, fatigue, peripheral oedema, ataxia
Carbamazepine	Starting dose 100 mg/day Max dose1800 mg/day	Sodium channel blocker Inhibit pain via a central and peripheral mechanism	Aplastic anaemia, Agranulocytosis, leukop enia, sedation, gait alteration.
Oxcarbazepine	Starting dose 300 mg/day Max dose 1200– 2400 mg/day	Maybe modulating voltage-activated calcium current	Risk of hyponatremia in first few months
Topiramate	Starting dose 50 mg/day Max dose 200 mg/day	Enhances action of GABA, Inhibit AMPA Type glutamate	Sedation, may predispose glaucoma and renal calculi
Lamotrigine	Starting dose 20–50 mg/day Max dose 300–500 mg/day	Prevent the release of glutamate	Rash

Table 4: Commonly used antidepressants in pain practice.

Drug Name	Oral dose in mg/duration	Clinical consideration
Amitriptyline (TCA)	10–25/12–24 h	Caution in elderly male Urinary retention
Nortriptyline (TCA)	10–25/12–24 h	Better tolerated than amitriptyline
Duloxetine (SNRI)	20–60/12–24 h	DOC IN diabetic peripheral neuropathy & fibromyalgia.
Milnacipran (SNRI)	50–100/12–24 h	FDA-approved drug for fibromyalgia
Venlafaxine (SNRI)	37.5–112.5/12–24 h	SNRI better tolerated
Desipramine (TCA)	10–25/24 h	Better tolerated TCA in elderly

TCA: Tricyclic antidepressant, SNRI: Serotonin norepinephrine re-uptake inhibitors

The oral route is the preferred root of administration. Other routes are subcutaneous, IM, IV, transdermal patches, and intrathecal pumps.

Common side effects are nausea, vomiting, itching, drowsiness, euphoria, constipation, urinary retention, respiratory depression, tolerance, dependence, sexual dysfunction, etc.

COMMONLY USED ANTICONVULSANTS IN PAIN PRACTICE

These are mainly either sodium or calcium channel blockers which raise the threshold for nerve depolarisation and thus suppress abnormal neuronal discharge [Table 3].

COMMONLY USED ANTIDEPRESSANTS PAIN PRACTICE

These drugs have a direct analgesic effect at doses much lower than that required for antidepressant action with the added advantage of sedation, diminished anxiety, muscle relaxation, and a restored sleep cycle. These drugs act on descending inhibitory pain pathways. Depending on the mechanism of action antidepressants are-Tricyclic antidepressants, selective serotonin reuptake inhibitors, serotonin norepinephrine re-uptake inhibitors [Table 4].

COMMONLY USED MUSCLE RELAXANTS IN PAIN PRACTICE

Muscle relaxants are used in addition to rest, physical therapy, and other measures to relieve discomfort. They are typically prescribed for short-term use to treat acute, painful musculoskeletal conditions [Table 5].

Table 5: Commonly used muscle relaxants in pain practice.

Drug Name	Dose (Max) Po Mg/Duration	Special Points
Baclofen	5–10 (80)/8–24 h	Abrupt withdrawal may predispose to seizures, hallucinations and increased flexor spasm
Tizanidine	4–8 (36)/12–24 h	More effective in reducing muscle spasm but less consistent in decreasing muscle tone. Careful administration when used with other antihypertensive
Diazepam	2–4 (20)/12–24 h	Concomitant use of other centrally acting drugs may potentiate side effects

Steroids

Glucocorticoids are extensively used in pain management for their anti-inflammatory and possibly analgesic actions. Although epidural and intra-articular steroids are frequently used in pain management, scope of oral steroid is limited. Particulate steroids are preferred over soluble preparations. Commonly used preparations are triamcinolone acetate and methylprednisolone.

NMDA receptor antagonist

NMDA receptors are involved in 'windup' phenomenon, modulation of pain pathway and hyperalgesia. Thus, inhibition of these can have potent analgesic effect. Ketamine is popular one but has no oral preparation. Parental ketamine is used in central conditions such as fibromyalgia, CRPS and opioid-induced hyperalgesia.

Alpha agonists

These drugs have sympatholytic effect and they also alter calcium and potassium conductance at spinal cord level. Commonly used is Clonidine (dose 0.1 mg/day orally). It is useful in sympathetically mediated neuropathic pain conditions like CRPS.

Local anaesthetics

Local anaesthetics drugs such as lignocaine and bupivacaine are used to block nerves as a diagnostic or therapeutic

Table 6: Commonly performed interventions for chronic pain management.

Interventions	Indication	Special Points	Imaging Required
Trigger Point Injection	Muscle Spasm,	Lignocaine and normal saline injections of steroids may be added hydrodilatation of muscles	Blind Or USG Guided
Intraarticular Steroid Injections	Osteoarthritis	Repeated steroid injections may lead to damage to cartilage, osteopenia	USG, fluoroscopic guided
Intraarticular platelet-rich plasm (Prp) Injections	Osteoarthritis	Promising results	Blind, USG or fluoroscopic guided
Lumbar and steroid cervical injections Lumbar Transforaminal injections	Lumbar or cervical disc prolapse, nerve rootirritation, sciatica, lumbar canalstenosis	Very effective in pain relief, can be repeated 3–4 times in a year risk of infection minimal	Mostly fluoroscopic guided recently USG guided
Gasserian Neurolysis	Trigeminal Neuralgia	Radiofrequency ablation is preferred Effective In Primary Trigeminal Neuralgia Glycerol Phenol 6% p	Fluoroscopic Or CT guided
Thoracic Epidural Steroid Injection	Post-herpetic Neuralgia	Intercostal nerve block with steroid need to be combined in a thoracic dermatomal distribution	Fluoroscopic guided
Ulnar Nerve Or Median Nerve Hydrodilattion	Cubital Tunnel or Carpal tunnel Syndrome	Steroid injection or hydro dilatation	USG guided
Sympatholytic Blocks			
Stellate Ganglion Block	CRPS Hand Reynauld's Disease Peripheral Vascular Disease	Steroid (Triamicinolone) is preferred Needs repetition 2-3 Times No Alcohol or Phenol Or Radiofrquency Ablation as it may lead permanent toHorner's Syndrome	Fluoroscopic Or USG guided USG guided blocks has better results
Lumbar Sympathetic Blocks	Burgers' Disease Pvd Diabetic Neuropathy Phantom Limb	Steroid Injection Alcoho80% Phenol 6% Radiofrequency Ablation	Fluoroscopy Guided USG Guided for Patient Who Cannot Lie Prone
Splanchnic Nerve Blocks Celiac Plexus Block	Chronic Pancreatitis Upper GI Malignancy GB Malignancy Irritable Bowel Syndrome	Steroid Injection Alcohol 80% PHENOL 6% Radiofrequency ablation	Fluoroscopy guided CT guided
Superior Hypogastric Plexus Block	Chronic Pelvic Pain Carcinoma Of Uterus, Bladder, Vagina, Cervix	Alcohol 80% Phenol 6% Radiofrequency Ablation	Fluoroscopy guided USG guided patients who cannot lie prone
Ganglia Impar	Coccydynia Ca Anal Canal Ca Vagina	Steroid Alcohol 80% Phenol 6% Radiofrequency Ablation	Fluoroscopy guided

USG: Ultrasonography, CT: Computed tomography

procedure. In central desensitisation procedure, it acts as a membrane stabiliser.

Botulinum toxin

Pain associated with spasticity, myofascial pain, cervical dystonia, and some headache well responds to therapeutic botox injection. It acts on the neuromuscular junction, analgesic action probably mediated by blockade of substance P, glutamate, and calcitonin gene-related peptide.

Topical agents

NSAIDs gel, Capsaicin gel, Lignocaine gel, and EMLA cream are found to be useful in pain management.

Interventional pain procedures

An interventional pain procedure is interruption of signals traveling along a nerve by injecting steroids or neurolytic agent on the nerve or using radio-frequency ablation of the nerve [Table 6].

COMMONLY PERFORMED INTERVENTIONS FOR CHRONIC PAIN MANAGEMENT

Regenerative therapy

Is rapidly emerging with extensive biomedical research over the past decade.[2,8,12]

- Platelet-rich plasma
- Growth factor
- Stem cells
- Prolotherapy.

SPINAL CORD STIMULATOR

Spinal cord stimulation (SCS), also known as neurostimulation, utilises an implant and electrodes to deliver mild electrical pulses to the nerves around the spinal column usually the dorsal column of spinal cord, to stimulate the nerves and block or lessen pain signals that are being sent to the brain [Figure 5].[21,22]

The major benefit of SCS is that it provides targeted pain relief up to 60% with improved mobility, allowing the patients to return to their daily activities. It minimises the need for invasive surgeries to correct spinal conditions and majority of the times remains the last resort for treating chronic back pain.

CANCER PAIN MANAGEMENT: SPECIAL POINTS

Cancer pain affects more than 9 million people worldwide annually. An estimated one-third (24–60%) and two-thirds (62–86%) of cancer patients suffer from pain, with more than one-third having moderate-to-severe pain. Pharmacotherapy constitutes the mainstay of treatment and approach for pain management as per the WHO analgesic ladder. Indications for interventions are unacceptable side effects and well localised pain syndromes in which pain may get relieved by nerve blocks or a comprehensive trial of pharmacologic therapy fails to provide pain relief. Interventional techniques for pain management include [Figure 6][23-27]

- Regional Infusions
- Neuraxial analgesia (Intrathecal pump)
- Neurodestructive procedures
- Neuromodulation
- Percutaneous vertebroplasty/kyphoplasty
- Radiofrequency ablation.

CONCLUSION

Pain treatment goals should include improved functioning and pain reduction. Along with pharmacological treatment, non-pharmacological therapies including integrative medicine therapies such as yoga, physiotherapy, and acupressure which widen the horizon of pain medicine should be routinely considered.

Knowledge of interventional nerve blocks and recent advances such as regenerative therapies and spinal cord stimulators has revolutionised the management of pain in CNCP and cancer patients. Thus, adequate control of chronic pain can be achieved in nearly all patients in a way that adequately balances benefits and potential harms.

Declaration of patient consent

Patient's consent not required as there are no patients in this study.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Available from: http://www.iasp.pain.org/taxonomy#pain [Last accessed on 2022 Jul 16].
2. Sharma K, Das G. Painpathways. In: Basics of Pain Mangement. 2nd ed. CBS Publishers: New Delhi; 2019. p. 3-8.
3. Dostrovshy J, Craig A. Ascending projection systems. In: McMahon SB, Koltzenburg M, editors. Wall and Melzack's Textbook of Pain. 5th ed. Elsevier Churchill Livingstone; 2006. p. 187-203.
4. Gold MS, Gebhart GF. Peripheral Pain Mechanisms and Nociceptor Sensitization In: Fishman SM, Ballantyne IC, Rathmell IP, editors. Bonica's Management of Pain. 4th ed. Baltimore, MD: Lippincott, Williams and Wilkins; 2010. p. 24-34.
5. Available from: https://www.physio-pedia.com/epidemiology_of_pain [Laast accessed on 2022 Jun 25].
6. Melzack R, Wall P. Pain mechanisms: A new theory. Science 1965;150:971-9.
7. Griffin RS, Fink E, Brenner JG. Functional neuroanatomy of the nociceptive system In: Fishman SM, Ballantyne IC. Rathmell JP, editors. Bonica's Management of Pain. 4th ed. Baltimore, MD: Lippincott, Williams and Wilkins; 2010. p. 98-119.
8. Jain J. Handbook of Pain Management a Case based Approach. 1st ed. New Delhi: Jaypee Distributors; 2018.
9. Randich A, Ness T. Modulation of spinal nociceptive processing. In: Fishman SM, Ballantyne IC. Rathmell JP, editors. Bonica's Management of Pain. 4th ed. Baltimore, MD:

Lippincott, Williams and Wilkins; 2010. p. 48-60.
10. Merskey H, Bogduk N. Part III: Pain terms, current list with definitions and note son usage. In: Classification of Chronic Pain. 2nd ed. Seattle: IASP Task Force on Taxonomy, IASP Press; 1994.
11. Gatchel RJ, Peng YB, Peters ML, Fuchs PN, Turk DC. The biopsychosocial approach to chronic pain: Scientific advances and future directions. Psychol Bull 2007;133:581.
12. Das G. Clinical Methods in Pain Medicine. New Delhi: CBS Publishers and Distributors; 2015. p. 2-17.
13. Khedr EM, Kotb H, Kamel NF, Ahmed MA, Sadek R, Rothwell JC, *et al*. Long-lasting analgesic effects of daily sessions of repetitive transcranial magnetic stimulation in central and peripheral neuropathic pain. J Neurol Neurosurg Psychiatry 2005;76:833-8.
14. Kaki AM, El-Yaski AZ, Youseif E. Identifying neuropathic pain among patients with chronic low back pain: Use of the Leeds assessment of neuropathic symptoms and signs pain scale. Reg Anesth Pain Med 2005;30:422-8.
15. Bennett MI, Smith BH, Torrance N, Potter J. The S-LANSS score for identifying pain of predominantly neuropathic origin: validation for use in clinical and postal research. J Pain 2005;6:119-58.
16. Meyer RA, Ringkamp M, Campbell JN, Raja SN. Peripheral mechanisms of cutaneous nociception. In: McMahon SB, Koltzenburg M, editors. Wall and Melzack's Textbook of Pain. 5th ed. London, United Kingdom: Elsevier, Churchill Livingston; 2006. p. 3-34.
17. Serpell M. Oxford Handbook of Pain Management. London: Correct Medicine Group; 2008. p. 1-35.
18. Melzack R, Katz J. Pain assessment in adult patients. In: McMohan SB, Koltzenburg MZ, editors. Wall and Melzacks Textbook of Pain. 5th ed. London: Elsevier; 2006. p. 291-304.
19. Burkey DR, Fleisher LA, editors. Regional Anesthesia and Pain Management. 1st ed. Philadelphia, PA: Saunders; 2009. p. 103-22.
20. Waldman SD, editor. Pain Management. 2nd ed. Philadelphia, PA: Saunders; 2011. p. 878-945.
21. Verrills P, Sinclair C, Barnard A. A review of spinal cord stimulation systems for chronic pain. J Pain Res 2016;9:481-92.
22. De La Cruz P, Fama C, Roth S, Haller J, Wilock M, Lange S, *et al*. Predictors of spinal cord stimulation success. Neuromodulation 2015;18:599-602.
23. Texted LH. Intrathecal pumps for managing the cancer pains. Am J Nurs 2016;116:36-44.
24. Kalso E, Edwards JE, Moore AR, McQuay HJ. Opioids in chronic non cancer pain: systematic review of efficacy and safety. Pain 2004;112:372-80.
25. Prager J. Cancer pain management continuum: A flexible approach. Clin J Pain 2001;17:206-14.
26. Joshi M. Cancer pain. In: Joshi MI, editor. Textbook of Pain Management. 3rd ed. Hyderabad: Paras; 2014. p. 392-405.
27. Benzon HI, Rathmell JP, Wu CL, Turk DC, Argoff CE, Hurley RW, editors. Practical Management of Pain. 5th ed. Philadelphia, PA: Elsevier; 2014.

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