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THE DIFFERENT APPROACHES TO PAIN AND THE CARE THAT MUST BE TAKEN

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Abstract: Pain is an unpleasant sensory and emotional experience, caused by injury or imminent risk of its development, and its pathophysiology consists of 4 main stages, which are signal transduction, conduction, transmission and perception, each with your particular action. Pain can be classified as acute or chronic, differing mainly in terms of onset, duration, etiology and association with the injury. There are certain more specific situations in which the patient feels pain, such as chest pain, abdominal pain, headache, back and neck pain. Each type of pain has its respective treatment with certain drugs according to the severity, which are decided individually for each case. The following study aims to present, describe and demonstrate the importance of clinical management of acute and chronic pain, differentiating them for better clinical therapy. This is an integrative literature review of a descriptive nature. Finally, the study concludes that there is a great need for health professionals to differentiate the types of pain and understand the changes that encompass this pain, in order to obtain a more punctual and effective management of clinical conditions.
Keywords: Pain. Acute pain. Chronic pain.

INTRODUCTION

Pain can be a repulsive sensory and emotional experience, associated with actual or potential injury, and can be classified as acute or chronic pain. The main differences between acute and chronic pain are their etiology, pathophysiology and duration.

To understand this differentiation, it is extremely important to have knowledge about the anatomy of the pain pathway, this pathway is basically constituted by Aβ, Aδ and C afferent fibers, which can be: unmyelinated, poorly myelinated or very myelinated. The total composition and location of these fibers refers to the way in which the stimulus is produced

and, consequently, to the classification of this type of pain.

Chronic pain, unlike acute pain, is a persistent pain that can remain for years and directly interfere with the individual's daily life. Acute pain, on the other hand, has a less prolonged duration, usually resulting from something momentary in which the body can reconstitute itself over the days without major further harm in which the individual continues his activities normally after it disappears, as an example of cuts, burns and other factors that eventually occur in the Corinthians.

In addition to the fibers and the nervous stimulus, in short, the structures involved in the system to generate the patient's pain are the spinal cord, spinal cord roots - in the horns, neurotransmitters, sensory neurons, thalamus, cortex and neuropeptides. Such structures participate in a scale of events that in the end generate a type of pain.

Regarding the type of pain, the present study clearly highlights the more specific situations in which the patient has pain, in a very detailed way. Among these situations, conditions such as chest pain, heart pain, abdominal pain and headache will be highlighted.

Each of these pains, the study highlighted several aspects that characterize and differentiate them in terms of acute and chronic pain. Among these factors, some of the most important were: the description of the pathophysiology and the neural pathways involved, the main risk detection and characterization tests, the anamnesis and specific physical examination of each one of them that are auxiliary to the diagnosis, in addition to the conducts. performed in each case and the most effective treatments. (Harrison, 2017).

In this aspect, the treatment of different types of pain must take into account different aspects. In view of this, there are different approaches so that there is effectiveness and damage reduction in the treatment for each type of pain.

The present work goes through highlighting the main classes of drugs, mechanisms of action, present routes, doses, recommended administration forms and side effects. All this in association with a global analysis of pain differentiation and its effects on the patient's life, since the main interest in each case is to identify the type of pain so that an effective conduct is carried out, in an individualized way and closely linked to the limitations that pain brings to patients' lives.

GOAL

The present study aims to present, describe and demonstrate the importance of clinical management of acute and chronic pain, addressing the clinical differences of each type, the structures involved, how the sensation of pain is generated, in addition to classifying them according to in order to achieve better clinical therapy.

METHODOLOGY

The present work consists of a qualitative literature review that sought to address results found in research on the subject of pain, in its acute and chronic classification, either in a comprehensive, orderly or systematic way. To carry out the work, the following steps were followed:

1) Selection of the corresponding themes;
2) Selection of samples found and used;
3) Analysis of the characteristics of the original research;
4) Analysis of the results obtained;
5) Conducting the review.

The databases of scientific literature and techniques used in carrying out the review were Google Scholar, Scientific Electronic Library Online (SciELO), Virtual Health Library, Latin American and Caribbean

Literature on Health Sciences (LILACS), using the following search engines: "chronic pain", "acute pain", "ways of approaching pain treatment".

Thus, the present work seeks not only to analyze the interface of pain, but also to differentiate acute pain from chronic pain, whether in the scope of development, pathophysiology and medication, aiming to shed light on an educational path, clarifying and raising awareness on the subject. in question.

DEVELOPMENT

CONCEPT OF ACUTE AND CHRONIC PAIN

The experience of pain is present in the daily life of most people, in which it manifests itself as a particularity of each subject and presents its own sensation with intensity and varied clinical forms.

Acute pain can be characterized as sudden onset pain, usually rapid with short duration, manifestation of up to six months and associated with trauma or injury. Chronic pain, on the other hand, can be characterized as continuous or intermittent pain, long duration, with undefined etiology and manifesting for six months or more.

Chronic pain is a complexity in the patient's life, as most of the time its underlying origin cannot be cured. However, therapy for these individuals must be performed in a multidisciplinary manner, with psychological assessment and behavioral-based treatment models being performed. Examples of diseases that promote chronic pain: arthritis, cancer, chronic daily headaches, fibromyalgia, and diabetic neuropathy (Harrison, 2019).

A guiding principle in the investigation of patients with chronic pain is to assess emotional and organic factors before instituting treatment. The joint analysis of these factors, without having to wait for the exclusion of possible organic causes before considering the emotional aspects, improves the patient's adherence to the treatment, which in part can be explained by the patient's reassurance when he realizes that the psychological evaluation does not it means that the doctor is doubting the validity of your complaint. Even when an organic cause for the patient's pain can be identified, it is still prudent to investigate other factors. For example, patients with cancer and painful bone metastases may also experience pain from nerve damage and be depressed. Optimal treatment requires that each of these factors be investigated and treated (Harrison, 2019).

STRUCTURES INVOLVED IN THE PATIENT'S PAIN SYSTEM

Pain is an unpleasant sensation confined to some part of the body. From this, pain is related to penetrating or tissue-destructive processes (e.g., stabbing, burning, twisting, tearing, compressive) and/or as a bodily or emotional reaction (e.g., dreadful pain)., nauseating, debilitating). Therefore, some structures are involved in this process, they belong to the peripheral mechanisms - the primary afferent nociceptor; central mechanisms - spinal cord and referred pain and ascending pain pathways. (Harrison, 2019).

In the composition of the peripheral nerve, the following structures are present, primary sensory afferents, motor neurons and sympathetic postganglionic neurons.

In primary sensory afferents, they are composed of A-beta, A-delta and C-type fibers. A-Beta fibers have a large diameter, are myelinated, stimulate light touch or movement. On the other hand, type A-delta fibers have a small diameter, are myelinated, and compose the pain stimulus. Finally, type C fibers have a small diameter, are not myelinated and present the stimulus of pain.

PATHOPHYSIOLOGY OF PAIN

The pathophysiology of pain has 4 main stages: Signal transduction, conduction, transmission and perception. We have in transduction the peripheral nerves, which are composed of axons of three different types of neurons: primary afferent sensory neurons, motor neurons and sympathetic postganglionic neurons. The cell bodies of primary afferent sensory cells are located in the dorsal root ganglia of the vertebral foramen. Primary afferent axons have two branches: one projects centrally to the spinal cord and the other innervates tissue peripherally. Primary afferent nerves are classified according to their diameter, degree of myelination, and conduction velocity. The largest diameter afferent fibers, A-beta, are the most responsive to light touch and motor stimuli and are found primarily in the nerves that supply the skin. In normal people, the activity of these fibers does not cause pain. There are two other classes of primary afferent fibers: small diameter A-delta myelinated axons and unmyelinated axons. These fibers are found in the nerves that supply the skin and the deep somatic and visceral structures. Some tissues, such as the cornea, are innervated only by A-delta and C afferent fibers. Most A-delta and C afferent fibers respond maximally only to painful stimuli and, upon electrical stimulation, produce the subjective experience of pain, defining -as as characteristics of primary afferent nociceptors. When conduction through the A-delta and C axon fibers was blocked, the ability to detect painful stimuli was completely lost.

When intense, repetitive, or prolonged stimulation is applied to injured or inflamed tissue, the activation threshold of primary afferent nociceptors decreases and the firing rate becomes higher for all stimulation intensities. Sensitization occurs at the level of peripheral nerve endings as well as at the level of the posterior horn of the spinal cord. Peripheral sensitization occurs in injured or inflamed tissues when inflammatory mediators activate signal transduction in intracellular nociceptors, driving increased production, traffic, and insertion of chemically activated and voltage-activated ion channels into the membrane. This change increases the excitability of nociceptor terminals and lowers their threshold for activation by mechanical, thermal, or chemical stimulation. Central sensitization occurs when activity produced by nociceptors during inflammation increases the excitability of neurons in the posterior horn of the spinal cord. The application of normally innocuous stimuli can cause pain after an injury that causes allergies. Sensitization is a clinically important process that leads to palpation, tenderness, and hyperalgesia, increased pain intensity in response to the same noxious stimuli, eg tingling that causes severe pain. A notable example of an allergy is sunburned skin.

Most spinal neurons in contact with primary afferent nociceptors send their axons to the contralateral thalamus. These axons form the spinothalamic tract, located in the white matter of the spinal cord, in the lateral pons and midbrain. Spinothalamic pathways are critical for the perception of pain in humans. Disruption of this pathway can lead to permanent deficits in pain and temperature recognition. Spinothalamic tract axons ascend to various regions of the thalamus. Pain signals vary widely from these thalamic locations to different regions of the cerebral cortex involved in different aspects of the pain experience. One of these thalamic projections is to the somatosensory cortex. This projection mediates the purely sensory aspects of pain, that is, the location, intensity, and character of pain. Other thalamic neurons project to cortical regions associated with emotional responses, such as the cingulate gyrus and other regions of the frontal lobe, including

the insular cortex. This pathway to the frontal cortex acts on the unpleasant affective or emotional dimension of pain. The emotional dimension of pain causes distress and exerts strong control over behavior. Because of this dimension, pain is always accompanied by fear.

PAIN CLASSIFICATION

Pain is a sensation that arises from a threat of tissue damage. Therefore, we can divide pain into basically two types: acute, which works as a defense, stimulates the production of substances that inhibit pain and works as a warning that something abnormal is happening, is characterized as a stinging or stinging sensation, sudden onset, associated with injury, and usually lasting up to six months conducted by fiber. The delta associated with the painful throbbing and burning sensation, is usually a pain of a superficial nature, perceives mechanical and thermal painful stimuli, and ends when the damage is repaired. (MINSON, 2015).

And chronic (non-malignant) that is characterized as constant or intermittent, of undefined etiology and lasting more than six months (slow pain), conducted by C fibers, is usually a pain located more deeply (in organs), perceives painful stimuli mechanical, thermal and chemical, the pain usually continues even after the damage is repaired. In the case of chronic pain, in its approach, it is extremely important to focus not only on symptomatic relief, but also on a change in behavior, including the cognitive and affective components. A person with chronic pain may show irritability, insomnia, mental depression, worry, stress, lack of interest in daily activities, and various psychological and other changes that will affect their daily lives and social relationships. Chronic pain is usually caused by pathological processes in somatic or visceral structures, or by prolonged dysfunction of PNS and/or CNS components. It can also result from environmental or psychopathological factors. Some diseases described as chronic pain are: headache, lumbar disc herniation, low back pain, rheumatism, fibromyalgia, and others. Chronic pain can also be divided into primary and secondary. Primary pain is described as pain in one or more regions of the body that persists for more than 3 months. To be called primary, the prerequisite is that the pain is not explained by another chronic condition. For example, chronic generalized pain (eg, fibromyalgia) and chronic visceral pain (eg, irritable bowel syndrome). Secondary ones are linked to other diseases. As an example: chronic post-surgical / post-traumatic pain (pain that persists beyond the expected period) and chronic neuropathic pain (pain caused by injury and/or disease in the somatosensory nervous system). (Minson, 2015).

MORE SPECIFIC SITUATIONS IN WHICH THE PATIENT HAS PAIN

Chest pain is among the most common reasons patients seek medical assistance in emergency rooms or doctors' offices. It is important to structure the initial diagnostic evaluation and screening of patients with acute chest pain into three categories: (1) myocardial ischemia; (2) other cardiopulmonary causes (pericardial disease, aortic emergencies, and pulmonary conditions eg spontaneous pneumothorax); and (3) non-cardiopulmonary causes- Esophageal reflux, Esophageal spasm, Emotional or psychiatric disorders. Electrocardiography is crucial in the assessment of non-traumatic chest pain (Harrison, 2017).

The ECG is critical for identifying patients with impending ischemia as the main reason for their complaint, as well as cardiac complications secondary to other disorders. Professional society guidelines recommend

performing an ECG 10 minutes after patient arrival, with the primary objective of identifying those with ST-segment elevation.

Plain chest radiography is done routinely when patients present with acute chest pain and selectively when those evaluated in the outpatient setting have subacute or chronic pain. Laboratory tests in patients with acute chest pain aim to detect myocardial injury, which may occur due to the presence of circulating proteins released by injured myocardial cells, with cardiac troponin being the preferred biomarker for the diagnosis of MI and must be measured in all patients. arriving with suspected ACS and repeated 3-6 hours later (Harrison, 2017).

Other non-invasive chest imaging tests can be used selectively to obtain additional diagnostic and prognostic information about patients with chest pain. Echocardiography is not necessarily a routine test in patients with chest pain, however, in patients with an uncertain diagnosis, particularly those with non-diagnostic ST elevation. Angiotomography is emerging as a modality for the evaluation of patients with acute chest pain, being a sensitive technique for the detection of obstructive coronary artery disease, this exam can exclude aortic dissection, pericardial effusion and pulmonary embolism. Magnetic resonance imaging (MRI) also allows a highly accurate assessment of aortic dissection, but is used infrequently as a first test because CT and transesophageal echocardiography in general are more practical (Harrison, 2017).

Mechanisms of Cardiac Pain The neural pathways involved in ischemic cardiac pain are poorly understood. Ischemic episodes are thought to excite local receptors sensitive to chemical and mechanical stimuli, which in turn stimulate the release of adenosine, bradykinin, and other substances that activate the sensory ends of sympathetic and vagal afferent fibers. Afferent fibers pass through nerves that connect to the five superior thoracic sympathetic ganglia and the five dorsal thoracic roots of the spinal cord. In addition, cardiac vagal afferent fibers synapse in the nucleus of the solitary tract of the medulla and then descend to the upper cervical spinothalamic tract, and this pathway may contribute to the anginal pain felt in the neck and jaw.

In cases of acute abdominal pain, the diagnosis is readily defined in most cases, while success is less frequent in patients with chronic pain. The most frequent causes of abdominal pain: Perforated appendix or other perforated viscera Pelvic inflammatory disease, Perforated ulcer, Pancreatitis, Small or large bowel obstruction); Cardiothoracic (Acute myocardial infarction, Myocarditis, endocarditis, pericarditis); Metabolic causes (Diabetes, Uremia, Hyperparathyroidism); Neurological and psychiatric causes (Herpes-zoster, irritable bowel syndrome); Toxic causes (poisoning by animals or insects).

A meticulously performed history and physical examination are essential to address the differential diagnosis. Only patients with exsanguinating intra-abdominal hemorrhage (eg, ruptured aneurysm) must be taken to the operating room immediately (Harrison, 2017).

During the physical examination, simple critical inspection of the patient, such as facies, bed position, and respiratory activity, provides valuable clues. Abdominal signs may be practically or totally absent in cases of pelvic peritonitis, so a careful pelvic examination and digital rectal examination are essential in every patient with abdominal pain. Hypersensitivity to pelvic or rectal examination in the absence of other abdominal signs may be caused by surgical treatment pathologies such as perforated appendicitis, diverticulitis, ovarian cyst torsion (Harrison, 2017).

Auscultation of the abdomen is one of the least fruitful aspects of the physical examination of patients with abdominal pain. In general, it is sudden-onset severe chemical peritonitis that is associated with a truly silent abdomen (Harrison, 2017).

Laboratory tests may be important in the evaluation of patients with abdominal pain; however, with few exceptions, they rarely establish a diagnosis. Leukocytosis must never be the only deciding factor in indicating or not surgery. A WBC count > 20,000/μL may be seen in a perforated viscus; however, pancreatitis, acute cholecystitis, pelvic inflammatory disease, and intestinal infarction are conditions that may also be associated with marked leukocytosis (Harrison, 2017).

Some tests to aid in the diagnosis of diseases that generate abdominal pain: Plain radiographs of the abdomen in an upright or lateral position can be useful in cases of intestinal obstruction, perforated ulcer and several other disorders. In the absence of trauma, CT and laparoscopy have replaced peritoneal lavage as a diagnostic tool. Ultrasonography has been shown to be useful in detecting an enlarged gallbladder or pancreas, gallstones, ovarian enlargement, or tubal pregnancy. Laparoscopy is particularly useful for diagnosing pelvic conditions such as ovarian cysts, tubal pregnancy, salpingitis and acute appendicitis. Hepatobiliary iminodiacetic acid scintigraphy can help differentiate between acute cholecystitis and biliary colic from acute pancreatitis (Harrison, 2017).

Mechanisms of pain of abdominal origin inflammation of the parietal peritoneum The pain caused by inflammation of the parietal peritoneum has a constant and uncomfortable character, being located directly on the inflamed area, making it possible to establish its exact reference, since it is transmitted by the somatic nerves that innervate the parietal peritoneum.

Headache is among the most common reasons patients seek medical care, accounting, on a global level, for more disability than any other neurological problem (Harrison, 2017).

The classification system characterizes headache as primary or secondary, with primary headaches being those in which the headache and its associated manifestations constitute the disorder itself, often resulting in considerable disability and reduced quality of life for the patient, while secondary headaches are those caused by exogenous disorders. Mild secondary headache, such as that seen in association with upper respiratory tract infections, is common but rarely of concern. Primary headache: Tension headache, Migraine, idiopathic stabbing, exertion and cluster headache. Headache secondary to: Systemic infection, Head trauma, vascular disorders and Subarachnoid hemorrhage (Harrison, 2017).

Relatively few cranial structures generate pain; they include the scalp, middle meningeal artery, dural sinuses, falx cerebri, and proximal segments of the great arteries of the pia mater (Harrison, 2017).

In severe, recent-onset headache, the likelihood of finding a potentially serious cause is much greater than in recurrent headache. Patients with recent onset of pain require prompt assessment and appropriate treatment. Serious causes to consider include meningitis, subarachnoid hemorrhage, extradural and/or subdural hematomas, glaucoma, tumor, and purulent rhinosinusitis (Harrison, 2017).

If there are no warning signs, a reasonable approach is to treat when a diagnosis is established. As a rule, investigation must focus on identifying causes of concern for headache or gaining confidence if no diagnosis of primary headache can be made.

After treatment is started, follow-up care is essential to identify whether progress has been made against the headache complaint. Not all headaches will respond to treatment, but in general, troubling headaches will evolve and it will be easier to identify them (Harrison, 2017).

Pain usually occurs when peripheral nociceptors are stimulated in response to tissue injury, visceral distention, or other factors; in these situations, pain perception is a normal physiological response mediated by the healthy nervous system. Pain can also occur when the peripheral or central nervous system (CNS) pain production pathways are injured or overactivated. Headache can originate from one or both of these mechanisms (Harrison, 2017).

PAIN TREATMENT

Anti-inflammatories

These are drugs from a heterogeneous group, which present an individual variation that is not predictable in the response, and one of them may provide a better effect for a given patient. They are the most used analgesics, alone or in association with other medications or techniques. They have a "ceiling" effect, above which their analgesic action does not increase, with only an increase in side effects. The action of these medications occurs at a peripheral and central level, causing inhibition of the formation of prostaglandins, leukotrienes and the release of substance P. The central effect is associated with mechanisms involving opioids, serotonin and nitric oxide. Dipyrone and paracetamol have no anti-inflammatory effect, but are classified in this class and have mainly central action. (Treaty of Anesthesiology, 2001)

Gastric and renal side effects are related to cyclooxygenase-1 (COX-1) inhibition, as anti-inflammatory drugs block both COX-1 and COX-2. The anti-inflammatory drugs celecoxib and rofecoxib have predominantly action on COX-2, having fewer side effects than the others. At the gastric level, they can cause ulceration, erosion and bleeding. When used postoperatively, the incidence of these effects is low, as the treatment period is relatively short. Renal changes can occur in the glomeruli and tubules, with acute tubular necrosis, interstitial nephritis, and nephrotic syndrome. They result from reactive metabolites, cytotoxicity and ischemia. (Treaty of Anesthesiology, 2001)

Predisposing factors are dehydration, previous renal impairment and concomitant use of other medications that cause renal damage, especially in elderly patients. Functional changes with oliguria, edema and increased blood volume may occur due to prostaglandin inhibition. These analgesics cause changes in platelet adhesion irreversibly or not. Spinal cord depression and agranulocytosis are rare complications. Hepatotoxicity appears to be dose-dependent and associated with prolonged use of anti-inflammatory drugs, and is generally asymptomatic. Other side effects are: alteration of platelet aggregation, urinary retention, vomiting, dizziness, tinnitus, vertigo and confusion. (Treaty of Anesthesiology, 2001)

Drugs, doses, intervals and routes:
— AAS (Aspirin, Bufferin): 325-1000mg/4-6h;
— Dipyrone (Novalgina, Conmel): 500-1000mg/6h (10-20mg/kg); Vias: IV, IM, VO, VR
— Paracetamol (Tylenol, Dorico): 500-1000mg/6h (10-15mg/kg), orally
— Diclofenac (Voltaren, Cataflan): 50-75mg/8h (0.5-2mg/kg); Vias: IM, VO, VR
— Aceclofenac (Proflam): 100-150mg/12-24h; IM, VO
— Indomethacin (Indocid): 10-50mg/8h

(2.5mg/kg/day); IM, VO, VR
— Tenoxicam (Tilatil): 20-40mg/day; IV, IM, VO
— Ketoprofen (Profenid): 50-100mg/6h; IV, IM, VO
— Ibuprofen (Artril, Motrin): 200-800mg/4-6 h orally
— Piroxicam (Feldene, Inflamene): 20mg/day: VO, IM, SL
— Naproxen (Naprozyn, Flanax): 250-500mg/8-12h; GRANDMOTHER
— Nimesulide (Scaflam): 50-100mg/12h (2.5mg/kg/12h); VO, VR
— Meloxicam (Movatec, Meloxil): 7.5-15mg/day; IM, VO
— Lysine Clonixinate (Dolamin): 125-200mg/6-12h; IM, IV, VO
— Celecoxib (Celebra): 100-200mg/day; GRANDMOTHER
— Rofecoxib (Vioxx): 25mg/day; GRANDMOTHER
— Flurbiprofen (Targus): patch/12h

Opioids

Opioids act by binding to μ (mu), delta and kappa opioid receptors, inhibiting pain transmission in the spinal cord, activating descending inhibitory mechanisms and altering the emotional reaction to pain. Regarding binding to receptors, opioids are agonists when they have an affinity for the receptor and the ability to trigger an endorphin-like action. Antagonists have an affinity for the receptor, but binding does not result in any pharmacological action. Most opioids in use are agonists with predominant action on the μ (mu) receptor (morphine, codeine, tramadol, fentanyl, meperidine). (Treaty of Anesthesiology, 2001)

Opioids can be administered by various routes (oral, intravenous, intramuscular, subcutaneous, transmucosal, transdermal). The oral route is not very useful in the immediate postoperative period, when only a small percentage of patients can use it. The intramuscular route is widely used, but it has the disadvantage of providing high initial doses (above the analgesic plasma concentration) and plasma concentration drops below the analgesic level. The sublingual route promotes rapid absorption of the opioid, being an alternative for the treatment of postoperative pain. The subcutaneous route is a simple option for intermittent or continuous administration of opioids. Through intravenous injection, better control of plasma concentrations of opioids is achieved. Side effects of opioids are: nausea, vomiting, sedation, itching, euphoria, urinary retention, respiratory depression, increased bile duct sphincter tone, dizziness, confusion, and hallucination. Normally, opioids are used concomitantly with anti-inflammatory drugs to obtain analgesia through different mechanisms. It can also be associated with regional locks. (Treaty of Anesthesiology, 2001)

Drugs, doses, intervals and routes:
— Codeine (Codein): 30-60mg/4h (0.5-1mg/kg); GRANDMOTHER
— Tramadol (Tramal, Sylador): 50-100mg/4h (1-2mg/kg); VO, IM, IV, SC
— Nalbuphine (Nubain): 10-20mg/4h (0.1mg/kg); IV, IM, SC
— Buprenorphine (Temgesic): 0.2-0.4mg/6h; SL, IV
— Morphine (Dimorph): 15-60mg/4-8h (0.1mg/kg); GRANDMOTHER
(Dimorph): 1-10mg/4-8h; SC, IM, IV
— Meperidine (Dolantine, Demerol): 20-50mg/4h (0.75mg/kg); IM, IV
— Fentanyl (Fentanyl): 10-100mcg (0.5-2mcg/kg); IV

Antidepressants

Antidepressants are indicated for neuropathic pain, migraine, low back pain,

fibromyalgia, neck pain, cancer, etc. The mechanisms of action are:

- 1- Inhibition of serotonin and noradrenaline reuptake;
- 2- Activation of descending opioidergic pathways;
- 3- Inhibition of NMDA receptors;
- 4- Blockade of sodium channels;
- 5- Action on H1 receptors.

The possible side effects of the various medications are: drowsiness, insomnia, weight gain, dry mouth, sweating, urinary retention, constipation, blurred vision, tachycardia, postural hypotension, hallucination, increased temperature, triggering of glaucoma, alteration of cardiac conduction, urinary retention, disorientation, hallucination, anxiety, tremor, thrombocytopenia and memory impairment. (Treaty of Anesthesiology, 2001)

Medications:

— Amitriptyline (Tryptanol): 12.5-50mg/day
— Imipramine (Tofranil): 12.5-50mg/day
— Clomipramine (Anafranil): 10-50mg/day
— Nortriptyline (Pamelor): 10-50mg/day
— Maprotiline (Ludiomil): 25-50mg/day
— Mianserin (Tolvon): 10-30mg/day
— Fluoxetine (Daforin, Prozac, Eufor, Fluxene): 10-20mg/day
— Paroxetine (Aropax): 20mg/day
— Citalopram (Cipramil): 10-20mg/day
— Sertraline (Zoloft): 25-50mg/day
— Mirtazapine (Remeron): 30mg/day
— Venlafaxine (Efexor): 37.5-75mg/day
— Moclobemide (Aurorix): 100mg/day
— Tranylcypromine (Parnate): 10mg/day
— Nefazodone (Serzone): 100mg/day

Anticonvulsants

It has a membrane stabilizing effect by blocking sodium channels. They decrease synaptic transmission and suppress spontaneous activity. They potentiate the inhibitory action of the neurotransmitter GABA, increasing its synthesis, inhibiting its metabolism, increasing the release of GABA into synapses, decreasing reuptake from synapses and potentiating GABA-A receptors. They increase glutamate metabolism and decrease its release from nerve endings. Used for trigeminal neuralgia, peripheral neuropathies, post-herpetic neuralgia, central pain, complex regional pain syndrome etc. As side effects they can cause nausea, vomiting, ataxia, vertigo, neutropenia, pruritus, lethargy, headache, blurred vision, pancytopenia, drowsiness and confusion. (Treaty of Anesthesiology, 2001)

Medications:

— Carbamazepine (Tegretol): 200-1200mg/day
— Oxycarbamazepine (Trileptal): 300-1200mg/day
— Hydantoin (Hydantal): 200-300mg/day
— Clonazepam (Rivotril): 0.5-2mg/day
— Gabapentin (Neurontin, Progresse): 900-1200mg/day

Anxiolytics

They decrease anxiety, promote muscle relaxation and improve insomnia. Benzodiazepines act by binding to specific receptors, which complex with GABA-A receptors. This binding causes increased effect of the inhibitory neurotransmitter GABA. This, in turn, is responsible for the opening of chloride channels, with a decrease in membrane potential and neural conduction. Benzodiazepines facilitate the inhibitory action of GABA. Diazepam exerts an additional direct effect on muscle contraction by altering calcium flux. Anxiolytics are given to patients with pain associated with anxiety and muscle tension and spasticity. Benzodiazepines can cause physical dependence, although rarely

at therapeutic doses. These drugs must be used for short periods and must be avoided for alcohol and other drug addicts. Side effects are: sedation, amnesia, intellectual dysfunction, impaired coordination and sleep, and depression. (Treaty of Anesthesiology, 2001)

Medications:
— Diazepam (Diazepam, Diempax): 5-10mg/day
— Lorazepam (Lorax): 2-3mg/day
— Bromazepam (Lexotam): 3-6mg/day
— Buspirone (Buspar): 15-30mg/day

CONCLUSION

Pain is a common circumstance in the most varied situations that cover health, from birth to death, whether inside hospitals or even outside them, affecting people of any age and economic group. To be sure, getting sick is an impact, often with an unpleasant and very traumatic emotional experience. Therefore, it is essential that we have an understanding of the topic related to acute pain, including its warning signs, such as neurovegetative alterations, and also about the alterations and factors that involve chronic pain, such as emotional, cultural, socio-affective, psychic factors, among others. others. Thus, assessing pain is an arduous task for the appropriate conduct, in order to improve the functionality and physical and psychosocial repair of the patient. Therefore, whether the pain is acute or chronic, the cause is known or not, there will always be a variable psychological factor from organism to organism. This is because each person suffers a different interference, be it cultural, social, economic or even environmental. There are individuals who, despite experiencing severe pain, have complete control over themselves. Others, on the other hand, face the disarray caused by pain in an atypical way.

Therefore, even with pain being something so subjective and at the same time multidimensional, adequate and efficient treatment is necessary. This is because, when the treatment is not taken seriously, the quality of life of individuals is reduced, which can result in insecurity, irritation, discouragement, anxiety, depression. Therefore, these feelings only increase the degree of painful sensation and consequently generate greater expenditure on health care consumption. Thus, it is possible to conclude that there is a need for health professionals to comprehensively understand pain and its subtypes, in order to obtain an effective management aimed at reducing damage to the life of this patient and clinical improvement in the face of the different situations that involve pain and its possible changes.

REFERENCES

1. CUNHA, Lorena Lourenço; MAYRINK, Wildete Carvalho. Influência da dor crônica na qualidade de vida em idosos. **Revista Dor**, v. 12, p. 120-124, 2011.

2. GOSLING, Artur Padão. Mecanismos de ação e efeitos da fisioterapia no tratamento da dor. **Revista Dor**, v. 13, p. 65-70, 2012.

3. HARRISON, T.R. **Medicina Interna**. Rio de Janeiro: Guanabara Koogan, 2012.

4. HENNEMANN-KRAUSE, Lilian. Dor no fim da vida: Avaliar para tratar. **Revista Hospital Universitário Pedro Ernesto**, v. 11, n. 2, 2012.

5. JAMESON, J L.; FAUCI, Anthony S.; KASPER, Dennis L.; et al. **Medicina interna de Harrison** - 2 volumes. Artmed: Grupo A, 2019. Disponível em: https://integrada.minhabiblioteca.com.br/#/books/9788580556346/. Acesso em: 12 mai. 2022.

6. KLAUMANN, Paulo Roberto; WOUK, A. F. P. F.; SILLAS, Thiago. Patofisiologia da dor. **Archives of veterinary science**, v. 13, n. 1, 2008.

7. KRELING, Maria Clara Giorio Dutra; CRUZ, Diná de Almeira Lopes Monteiro da; PIMENTA, Cibele Andrucioli de Mattos. Prevalência de dor crônica em adultos. **Revista Brasileira de Enfermagem**, v. 59, p. 509-513, 2006.

8. MINATTI, Sueli Pinto. O psicanalista no tratamento da dor. **Revista Latinoamericana de Psicopatologia Fundamental**, v. 15, p. 825-837, 2012.

9. MINSON, Fabiola P.; MORETE, Marcia C.; MARANGONI, Marco A. Dor. Barueri-SP, Editora Manole, 2015. Disponível em: https://integrada.minhabiblioteca.com.br/#/books/9788578682057/. Acesso em: 03 mai. 2022.

10. PIMENTA, Cibele Andrucioli de Mattos; CRUZ, Diná de Almeida Lopes Monteiro da; SANTOS, Jair Lício Ferreira dos. Instrumentos para avaliação da dor: o que há de novo em nosso meio. **Arq. bras. neurocir**, p. 15-24, 1998.

11. PINHEIRO, Ana Lúcia Uberti et al. Avaliação e manejo da dor aguda: revisão integrativa. **Journal of Nursing and Health**, v. 4, n. 1, p. 77-89, 2014.

12. SAESP. **Tratado de Anestesiologia**. 5ª ed. p.1165-1173. São Paulo: Atheneu, 2001.

13. SALVETTI, Marina de Góes et al. Impacto da dor aguda e adequação analgésica em pacientes hospitalizados. **BrJP**, v. 3, p. 333-336, 2021.

14. VALENTE, Rui Ferreira et al. Recomendações da Sociedade Portuguesa de Anestesiologia para o Tratamento da Dor Aguda 2018. **Revista da sociedade portuguesa de anestesiologia**, v. 28, n. 2, p. 102-109, 2019.