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Cardiac Pain

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Abstract
Heart diseases is usually diagnosed based on medical history and physical examination. Diseases are usually confirmed by selected non-invasive and invasive tests. A complete history is crucial for the diagnosis of heart disease and cannot be replaced by any examination. It is necessary to take a detailed family history because many heart diseases are basically hereditary. Cardiac symptoms include chest pain or discomfort, shortness of breath, weakness, fatigue, palpitations, dizziness, presyncope, and syncope. These symptoms occur equally in several forms of cardiac and extracardiac diseases. A complete examination of all organ systems is essential to detect the peripheral and systemic effects of cardiac and extracardiac diseases that may have an effect on the heart.

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Introduction
Pathophysiologic responses and adaptive changes to extensive tissue injuries function to maintain hemodynamics, minimize tissue injury, and promote healing [1]. However, the exact same neural and hormonal catecholamine responses that promote recovery in healthy young adults worsen pain intensity, promote cardiovascular instability and pulmonary dysfunction and increase infection risk in American Society of Anesthesia highrisk patients. Anesthesiologists have traditionally been the physician specialists most at home with pain physiology and pathophysiology and play the key role in initiating highly effective neuraxial, regional, and multimodal analgesia. Findings from randomized controlled trials and meta-analyses suggest that continuous epidural analgesia and regional analgesia can significantly reduce pain intensity scores, sympathoadrenal responses, and pulmonary complications. Although these techniques are more expensive, time-consuming, technically difficult to initiate and need continuous follow-up, their application in high-risk patients has been shown to reduce postsurgical morbidity, mortality, and time to hospital discharge.

The anamnesis is important for several reasons within the patient with chronic pain [2]. Enquiry should initially be made into the patient's general health. other than the worth of this as a screening question to exclude serious morbidity, patients who consider themselves generally healthy may respond differently to a chronic pain condition than those with a history of chronic pathological state.

Serious comorbidity may complicate or perhaps contraindicate some pain treatment options. Particular hazards of systemic drug treatments is also posed by seriously impaired liver or kidney function. Some invasive treatments carry greater risk in patients with an increased bleeding tendency, either from a hemorrhagic disorder (e.g. thrombocytopenia, hemophilia) or anticoagulant treatment. Neuraxial nerve blocks, and a few sympathetic blocks producing large regional vasodilatation, could also be dangerous in patients with impaired cardiac reserve. Potent opioids should be used with caution in patients with severe chronic respiratory disease.

Many patients with diseases related or unrelated to their painful condition are doping up long run which can potentially produce to adverse interactions with pain medication.

Nonpain-contingent causes of disability, e.g. some neurological diseases, may limit attainable objectives of rehabilitation.

Trauma
Surgical trauma is promptly followed by increases in plasma concentrations of epinephrine and norepinephrine [1]. The magnitude and duration of catecholamine release is directly associated with patient related factors like the type of surgery, inherent sympathetic response, patient age, and genetic (inflammatory) polymorphisms. In general, highest elevations in plasma catecholamines are observed following extensive procedures and in younger individuals. The earliest aspects of the catecholamine response reflect pronounced, but transient, increases in adrenal medullary secretion, whereas latter aspects reflect continued release of norepinephrine from sympathetic nerve endings. Pathophysiological changes related to increased sympathetic

tone and altered regional perfusion include the following: [1]. an increased incidence of postsurgical hypertension that ranges from 5% following minor, uncomplicated procedures to approximately 50% in patients recovering from more extensive vascular surgery [2]. Increased peripheral vascular resistance is related to increases in contractility and myocardial oxygen consumption because the organism attempts to take care of or augment cardiac output.

Increases in oxygen consumption may precipitate myocardial ischemia in patients with arteria disease. Enhanced sympathetic tone could also be especially deleterious in patients recovering from peripheral vascular surgery, because elevations in arterial pressure may risk rupture of vascular anastomoses, whereas intense vasospasm may compromise distal graft patency.

Trauma remains a serious reason behind morbidity and mortality throughout the globe [3]. Medical advances have significantly reduced the mortality related to trauma, which has led to an increased emphasis on secondary outcome measures, like psychological well-being, functional improvement, and vocational and social reintegration. Pain includes a profound impact on all of those variables. The stress response after multi-trauma exceeds that following elective surgery and includes cytokine and acute phase reactant release, altered immunologic response, and elevated levels of catecholamines, cortisol, growth hormone, and adrenocorticotropic hormone. Studies have shown that inadequately treated acute pain increases this response, which might end in higher morbidity. Poorly controlled inflammatory pain also ends up in myriad anatomical and physiological changes within the nervous system (i.e., neuroplasticity), which might manifest as chronic neuropathic pain. Trauma patients with high levels of persistent pain are less likely to return to work, more likely to suffer from depression, posttraumatic stress disorder, and other psychological comorbidities, and report greater disability than trauma victims who report less pain. Even among survivors of severe trauma, the long-term morbidity is significantly higher compared with matched controls, a control which will be partly attributable to the sequelae of chronic pain.

Heart

Despite considerable improvements in anesthetic technique and maintenance of intraoperative hemodynamic stability, cardiac dysfunction secondary to myocardial infarction, cardiac failure and arrhythmia still account for a major percentage of postoperative deaths [1]. In high-risk populations, perioperative ischemia is possibly to occur following surgery, most commonly between postoperative days 1–3. Although a spread of things may contribute to the development of postoperative myocardial ischemia, including hypothermia, anemia, anxiety, and tracheal intubation/suctioning, responses to poorly controlled pain play a prominent role. Catecholamine-induced tachycardia, enhanced myocardial contractility, increased afterload, and hypervolemia, secondary to enhanced release of AVP (arginine vasopressin) and aldosterone, are well-characterized factors responsible for increased oxygen demand. Increased oxygen demand, along with hypervolemia, may precipitate ischemia and acute cardiac failure, especially in patients with poorly compensated coronary artery and/or valvular heart disease.

Despite increased myocardial oxygen requirements, oxygen supply is also diminished due to alterations in pulmonary function (refer to the following section). Pulmonary alterations include atelectasis secondary to pain-induced hypoventilation and pulmonary edema resulting from stress-induced hypervolemia. A second explanation for reduced oxygen supply includes coronary artery occlusion. coronary artery blockage may result from [1] high circulatory levels of catecholamines and increased coronary sympathetic tone, [2] stress-induced increases in plasma viscosity and platelet-induced thrombosis, and [3] coronary vasospasm secondary to platelet aggregation and release of serotonin.

Cardiac chest pain

Cardiac chest pain is traditionally related to autonomic symptoms like nausea, vomiting, sweating and pallor [4]. Transient neurological disturbance, or the loss of pulses within the limbs, suggests aortic dissection. Shortness of breath (dyspnoea) is usually related to chest pain, and it's useful to differentiate dyspnoea that simply occurs in association with chest pain from pleuritic pain that's triggered or increased by inspiration. The latter suggests pathology within the pleura or chest wall, and often gives a subjective impression of dyspnoea; however, the 2 problems may co-exist in some conditions, like pneumonia. Patients with an increased rate of respiration or effort may develop musculoskeletal chest pain over time, and chronic coughing can have the same effect, even to the purpose of causing a 'cough fracture' of 1 or more ribs. This phenomenon is more common within the elderly, or those with pre-existing osteoporosis. Sudden onset of shortness of breath occurs in larger pulmonary embolisms, and will be related to haemoptysis (coughing up blood). However, haemoptysis also occurs in respiratory infections, when the patient's cough may produce purulent sputum. oesophageal reflux can produce an unpleasant taste within the mouth, and a sore throat or cough, particularly when the patient lies down and at nighttime. it should even be related to upper abdominal pain, but this is often not exclusive to gastrointestinal pathology, and abdominal pain can also occur in inferior myocardial infarct and lower lobe pneumonias.

Incidence of cardiogenic chest pain from myocardial infarction (MI), angina, pulmonary embolism (PE), and heart failure is estimated to be 50% of patients presenting to the emergency department [5]. Comparatively chest pain seen within the outpatient setting is often caused by stable coronary artery disease (CAD), musculoskeletal conditions, gastrointestinal disease, pulmonary disease, or psychiatric disorders. within the outpatient setting, the incidence of pain

secondary to pulmonary disease is 5%, gastrointestinal disease is 19, musculoskeletal conditions is 36%, and psychiatric disorders is 8%. Differential for cardiovascular causes of chest pain is extensive consisting of coronary artery disease (CAD), MI, angina, aortic dissection, pericarditis, cardiac tamponade, PE, heart failure, or cardiomyopathies. Pulmonary causes of chest pain include but aren't limited to pneumonia, pneumothorax, COPD, or lung cancer. gastroesophageal reflux disease (GERD) is that the most typical reason behind gastrointestinal disease but may include other ulcer disease, esophagitis, esophageal rupture, esophageal spasms, pancreatitis, splenomegaly, biliary colic, or cholecystitis. Musculoskeletal pain will be caused by common conditions like costochondritis, traumatic injuries, myofascial pain, or more rare conditions like Tietze syndrome. Psychiatric causes include generalized anxiety disorder, panic disorder, major depressive disorder, illness anxiety disorder, posttraumatic stress disorder (PTSD), or substance use disorders (cocaine, methamphetamines, or alcohol). Seventy percent of panic disorders have chest pain as a symptom. Up to 15 of chest pain is determined to be nonspecific without a definitive diagnosis.

MCD

Pharmacotherapy employed in patients with microvascular coronary dysfunction (MCD) is targeted at their underlying mechanism for pain [6]. Therapies utilized in patients with coronary artery disease like nitrates, b -blockers, calcium channel blockers, and statins have also been found to be bene fi cial in those with MCD. Other agents found to have promising effects on anginal symptoms includes ranolazine and angiotensin converting enzyme inhibitors. Hormone therapy in women with MCD improves quality of life, although improvement in myocardial ischemia remains to be determined. In patients with cardiac syndrome X, small trials are performed showing either beneficial or indeterminate results with the employment of other pharmacologic agents like tricyclic medications, L-arginine, xanthine derivatives, n-3 polyunsaturated fatty acids, nicorandil, and trimetazidine. Nonpharmacologic therapies within the management of chronic angina also play a very important role. Lifestyle modification, exercise, and cognitive behavioral therapy have shown to improve angina and exercise capacity in those with MCD. the utilization of neurostimulation, including transcutaneous electrical nerve stimulation and spinal cord stimulation, may also improve symptoms in those with chronic angina. Incorporating both pharmacologic and non-pharmacological therapies can result in the effective management of chronic angina in patients with MCD.

Cardiovascular Disease

Stress, depression, anxiety, social isolation, inadequate social support, social conflict, and other psychosocial difficulties can have indirect effects in causing cardiovascular disease by fostering adverse health behaviors, like smoking, poor nutrition, and inadequate use of health services [7]. Additionally, psychosocial factors can have a right away impact in producing cardiovascular disease by influencing neuroendocrine and platelet activation.

Both pre-existing vulnerability to cardiovascular disease, e.g., hypertension, obesity, and sedentary lifestyle, and major stressors can produce cardiac arrhythmias and/or plaque rupture, leading to death. However, more research is required to clarify the link between psychosocial stress and therefore the etiology of cardiovascular disease.

A number of pathophysiological mechanisms may explain the psychosocial effects on cardiovascular disease. The hypothalamicpituitary-adrenal axis, hypertension and cardiovascular reactivity, inflammatory markers, platelets, coagulation factors, fibrinogen, lipids, and glucose metabolism is also involved during this process.

CAN

Cardiovascular autonomic neuropathy (CAN) is defined because the dysfunction of the autonomic control of the cardiovascular system [8]. Clinical predictors of CAN include hyperglycemia, presence of DPN, nephropathy, retinopathy, hypertension, obesity, smoking, and dyslipidemia. CAN is closely correlated with mortality and morbidity like myocardial ischemia, stroke, and diabetic nephropathy progression. The assessment of CAN is additionally clinically useful for classifying cardiovascular risks as indicators so they will then be used for more intensive pharmacotherapeutic and lifestyle management of comorbid conditions. Cardiovascular reflex tests, which are the gold standard for assessing CAN, have good sensitivity, specificity, and reproducibility. Relatively simple tests that are widely used to assess CAN include heart rate variability to deep breathing, a Valsalva maneuver, and blood pressure changes in response to posture. The optimal period for CAN assessment could also be at the time of the type 2 diabetes diagnosis and at 5 years after diagnosis of type 1 diabetes in patients with risks of CAN thanks to long-term hyperglycemia, cardiovascular risks, DPN, and angiopathic diabetic complications.

Surgery

Thoracic epidural analgesia allows specific blockade of nociceptive reflex arcs and will reduce or eliminate stressinduced alterations of organ dysfunction [1]. Untoward sympathetic effects on atherosclerotic vessels are suppressed and blood flow to at risk areas of myocardium is improved. Understanding the pathophysiology of pain and providing optimal management has become important in cardiac surgery. The utilization of thoracic epidural anesthesia following coronary artery bypass graft surgery, although controversial from a safety point of view, has been shown to improve hemodynamic stability, reduces the release of troponin and also the incidence of supraventricular arrhythmia and allows earlier extubation. Epidural analgesia with local anesthetics plus opioids, but not opioids alone, blocks noxious impulses to and from the sympathetic ganglia and attenuates activation of the sympathoadrenal axis. Such

suppression helps to clarify why a recent analysis of thoracic epidural analgesia continued for over 24 hours was found to reduce mortality and postoperative myocardial infarct.

Catecholamines released in response to surgical stress and poorly controlled pain incite vasospastic, vasoconstrictive, and thrombotic occlusive complications. Vasospasm as a results of high plasma concentrations of epinephrine and locally released norepinephrine may compromise distal graft potency in patients recovering from vascular surgery and increase risk of deep venous thromboses in other varieties of lower extremity procedures. Compared with general anesthesia, epidural anaesthesia followed by continuous epidural analgesia maintains fibrinolysis, reduces the danger of arterial thrombosis, and is related to a lower incidence of reoperation for inadequate tissue perfusion. Although local anesthetics directly inhibit platelet aggregation and have antithrombotic effects it remains unclear whether local anesthetics absorbed from peripheral or epidural sites of administration have clinically significant effects at the positioning of vascular surgery.

Catheterization

Cardiac catheterization is completed to substantiate and define the extent of coronary artery disease, evaluate cardiac function, and perform interventional procedures to treat coronary artery disease and other cardiac disorders [9]. Angioplasty is taken into account the gold standard of care. The most important advantage is that it avoids a serious surgical treatment, scar, and long postoperative recovery period.

Right heart catheterization is typically done via femoral venous access but alternate venous access sites are often used, including the internal jugular veins, subclavian vein, or brachial veins. Once the superior or inferior vein is reached, the catheter is advanced through the right atrium, right ventricle, and into the pulmonary artery. Pressures are recorded and O_2 saturations obtained when indicated. Contrast dye will be injected for imaging of the right atrium, right ventricle, or pulmonary artery. cardiac output is determined using the thermodilution technique. In some procedures, like catheter-based mitral valve repair or closure of a patent foramen ovale, the left atrium is accessed via transseptal puncture through the atrial septum.

A sheath is usually cannulated into the femoral artery under fluoroscopy. Procedural sedation and analgesia are given for the procedure. Once the procedure is over, the sheath are going to be removed. Bleeding times are assessed prior to removal. The sheath removal is usually uncomfortable for the patient and a few may even report it as painful. Patient education on what's to be expected may assist in allaying their fears. Clinical judgment is employed in determining the best analgesic method for the patient-IV opioid analgesia and/or a brief acting local injected round the site so as to reduce pain and avoid a vagal reaction because the sheath is being pulled.

Opoids

Opioids can be given by a large type of routes [10]. These include oral, intranasal, transbuccal (sublingual), transdermal, and rectal routes of administration. More common methods of opioid administration for acute pain, especially within the perioperative setting, are intramuscular, intravenous, and neuraxial (intrathecal and epidural). These methods offer rapid onset and better titratability. Emerging technologies for sublingual (sufentanil) and transdermal (fentanyl) administration appear promising. Opioid agonist analgesics are indicated within the treatment of mild, moderate, or severe acute pain. Mild acute pain is treated with oral opioids like hydrocodone, oxymorphone, and oxycodone. These drugs are frequently given after moderate to severe pain symptoms have subsided and discharge from the recovery room or facility is anticipated. they're often combined with an NSAID (Nonsteroidal anti-infl ammatory drugs) like aspirin or acetaminophen and their dosing is usually limited by the nonopioid content. Oral opioids are subject to extensive first-pass effect within the liver and don't seem to be a first-line choice for moderate to severe acute pain because their bioavailability is low. Intramuscular injections (morphine, hydromorphone) are a preferred route of administering opioid analgesics. Serum concentrations of opioids may vary greatly with this modality as uptake is erratic and dependent on perfusion of the positioning. Despite these drawbacks, intramuscular injections of opioids is considered in select situations (lack of IV access). Intravenous opioids (morphine, hydromorphone, fentanyl) are commonly used perioperatively and in intensive care units to treat moderate to severe acute pain. The sedation related to morphine typically precedes its analgesic effect. this can be a crucial clinical consideration to avoid "stacking" doses which can end in oversedation and respiratory depression. Morphine is conjugated (metabolized) within the liver with glucuronic acid into morphine-3-glucuronide (M3G) and morphine-6-glucuronide (M6G) before renal excretion. M6G may be a potent mu receptor agonist, whereas M3G is pharmacologically inactive. the accumulation of M6G may produce respiratory embarrassment in patients with renal disease. Hydromorphone may be a logical choice for renal patients because its metabolism doesn't produce (M6G). Hydromorphone metabolism generates a vigorous metabolite (hydomorphone-3-glucuronide) which will exhibit excitatory properties. Patient-controlled analgesia (PCA) allows patient titration of the opioid against their own pain requirements and eliminates the drawbacks related to PRN dosing like staff availability and subjective staff interpretations of patient's pain. PCA requires patient cooperation and thus appropriate selection of candidates for PCA therapy is indicated. Patient acceptance of PCA has been high, and studies demonstrate less total drug consumption with improved postoperative respiratory function compared to patients receiving conventional as needed or scheduled dosing by trained staff. Continuous ("basal rate") PCA infusions are shown to supply a better incidence of respiratory depression particularly in opioid-

naïve patients, and their use during this group isn't recommended. Morphine, hydromorphone, fentanyl, and sufentanil are all common choices for intravenous PCA. Fentanyl and sufentanil don't have any active metabolites and are used successfully in patients receiving intravenous PCA. Sufentanil provides better analgesia with less respiratory depression than fentanyl when used for intravenous PCA. Intrathecal and epidural opioids provide excellent analgesia and rapid onset. Morphine, fentanyl, and sufentanil are commonly used for this purpose. Morphine's lack of lipid solubility provides extended analgesia for 12–24 h. This property makes one-time dosing or repeat dosing through an epidural catheter with morphine convenient. Fentanyl and sufentanil provide analgesia for about 2 h when administered neuraxial. they're commonly given along with a local anesthetic (ropivacaine, lidocaine) to speed onset of spinal analgesia. Their short duration of effect compared with morphine limits their usefulness as primary modalities for postoperative analgesia when administered as a single-shot injection; however, epidural PCA with either sufentanil or fentanyl via an epidural catheter has been used successfully in patients requiring postoperative analgesia.

Conclusion

The location, duration, nature and type of pain, and the factors that cause the onset and cessation of pain are important. Previous heart disease, the use of medications that can cause coronary artery spasm, and the existence of risk factors for coronary artery disease or pulmonary embolism may be important. The presence or absence of risk factors for coronary heart disease changes the likelihood of heart disease as the cause of these problems, but does not help to identify the cause of acute chest pain. Pain may intensify during breathing, movement, or both in serious and less serious illnesses; these pain-enhancing factors are not specific. Short (<5 seconds), sharp, intermittent pain rarely occurs as a result of serious illness.

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