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Review Article

Risks and Complications of Dental Treatment for Cardiac Patients

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Abstract

Treatment of patients with coexisting cardiovascular disease (heart problems) often raises concerns about possible problems during dental treatment. The incidence of Cardiovascular Diseases (CVD) increased from the beginning of the century to the point where it became the leading cause of death in industrialized countries as each country changed its traditional lifestyles. Therefore, an exhaustive bibliographical review was carried out to argue the main risk factors that occur in patients with heart disease in relation to dentistry.

Keywords: Orofacial Manifestations; Risks; Heart Disease; Bleeding; Haemorrhage; Thrombi

Introduction

In the United States this happened in 1920, in Spain it did not happen until the late 50's, in Latin America in the 90's. The Bogalusa (1998) study currently establishes that the precursor elements of adult cardiovascular diseases begin in childhood. Autopsy studies show that coronary atherosclerotic lesions occur early in life and are associated with very low-density lipoprotein cholesterol, systolic and diastolic blood pressure, and obesity, presenting an inverse relationship with cholesterol. of high-density lipoproteins (Harrison, 1949). It is estimated that in 2008 17.3 million people died from this cause, which represents 30% of all deaths registered in the world; 7.3 million of these deaths were due to coronary heart disease, and 6.2 million to strokes (WHO, Cardiovascular Diseases, 2007). Given the growing number of patients with heart disease who need dental treatment, it is essential that the dentist knows or knows how to properly identify the needs to treat this group of patients. This being the most common medical problem found in dental practice. In addition, the increasing survival of children with congenital heart disease makes them a significant proportion of those presenting for dental treatment. These patients are at risk for bleeding, infective endocarditis (inflammation of the inner lining of the heart), drug action and interaction, and a decreased ability of the patient to tolerate the stress and trauma associated with dental treatment, for which a good medical history carried out and supported by evaluations by specialists in cardiology, will reduce these risks in dental practice [1,2].

Objective

Deepen and discuss the main risks of heart disease in dental treatments.

Reference Search Methods

The scientific information was compiled through a search using the following descriptors in English: The Medical Subject Headings (MeSH): "heart disease, oral cavity, Risks, Complication, Head and Neck.

Analysis Strategy

The search was based solely on heart disease conditions of the buccomaxillofacial complex

Developing

Heart disease

Coronary heart disease is a group of disorders that have a common etiology and pathogenesis: blockage of coronary blood flow, which causes various degrees of ischemia in the heart muscle. Cardiopathies are classified into congenital and acquired. The difficulties that derive from the lack of blood supply to the myocardium are also diverse, and can cause death. This set of alterations is the first cause of death in the population of developed countries and in Latin America they also occupy higher places within the most considerable causes of morbidity and mortality. The relationship that exists with age (it tends to appear after 40 years) and the current advances in early detection, treatment and care, means that patients with coronary heart disease come to our office requesting care more frequently. The risk factors for the development of cardiovascular diseases are [1,2].

- Hypertension.
- Age: men older than 55 and women older than 65 years.
- Mellitus diabetes.

- High numbers of low-density (LDL) or total cholesterol, or low numbers of high-density (HDL) cholesterol.
- Glomerular filtration rate less than 60 mL/minute.
- Family history of premature CV disease, before the age of 55 in men and before the age of 65 in women.
- Microalbuminuria.
- Obesity (BMI >30 kg/m2).
- Physical inactivity.
- Positive smoking; especially cigarettes

Classification

They are divided into congenital and acquired. Congenital heart disease congenital heart diseases include structural, valvular, septal alterations and also of the great vessels that leave or enter the heart. Its etiology is varied as well as its clinical manifestations and consequences. There are serious ones that are incompatible with life at birth, others seriously limit life in its duration and quality, a last group allows those who suffer from it to live in a way close to normal, others even disappear during the growth of the person, which allows them to develop normally. Most of them, at present, can be corrected surgically, and correcting the disease early will mark its prognosis. They are the most frequent major anomalies at birth and cause 20% of neonatal deaths. The incidence of these defects in our population is 8.5 per 1000 live births. The perinatal prognosis of critical congenital heart disease improves if its diagnosis is made during the prenatal period (Viñals, 2002). This allows programming the most appropriate perinatal conduct, considering that many times the deterioration of these newborns (NB) is sudden and abrupt. Even RN carriers of treatable defects can be operated [1,3].

Acquired heart disease coronary heart disease is frequently caused by atherosclerosis, a disease that plagues all population groups and is characterized by the storage of lipids in the walls of large and medium caliber arteries. Not only the family history establishes its development, but other biological and social elements such as age and sex also have an influence (it is more frequent in males and in women after menopause). Suffering from arterial hypertension, diabetes mellitus, left ventricular hypertrophy, as well as smoking, obesity, and a sedentary life, are risk factors for atheroma formation. Other items that are considered to be of lower risk are the consumption of alcohol, coffee, and oral contraceptives. Some of these risk factors can be corrected, which makes it possible to reduce the prevalence of coronary heart disease (Castellanos, 2002).

Atherosclerosis

Atherosclerosis is a condition where pathology is still poorly understood. Atherosclerosis is characterized by the formation of patchy plaques (atheromas) in the intima layer surrounding the lumen of medium- and large-sized arteries; the plaques contain lipids, inflammatory cells, smooth muscle cells, and connective tissue. Atherosclerosis is the most common form of arteriosclerosis, which is a general term that includes several disorders responsible for the thickening and loss of elasticity of the arterial walls. Atherosclerosis is also the most serious and clinically relevant form of arteriosclerosis because it causes coronary heart disease and cerebrovascular disease. Atherosclerosis can affect all the large and medium-sized arteries, such as the coronary, carotid, and cerebral arteries, the aorta and its branches, and the main arteries of the limbs. It is the leading cause of morbidity and mortality in the United States and in most developed countries. In recent years, the age-related mortality rate attributable to atherosclerosis has been declining, although in 2016 cardiovascular disease, primarily coronary and cerebrovascular atherosclerosis, still accounted for almost 18 million deaths worldwide (> 30% of all deaths) Risk factors for the development of this disease are dyslipidemia, diabetes, smoking, family history, sedentary lifestyle, obesity, and high blood pressure [4].

anatomo-pathology the initial lesions are represented by lipid streaks that appear in the form of an accumulation of foam cells loaded with lipids in the intima layer of the artery, pale yellow in color, with fuzzy contours, which can measure from millimeters to 2 cm. along the vascular axis. These are related to an accumulation of lipid-filled macrophages. These stretch marks do not produce vascular obstruction or symptoms. They can appear early and disappear secondarily. Plates to advance and develop take 10 to 15 years. Formed atheromatous plaques initially appear in the abdominal aorta, coronary arteries, and carotid arteries and increase with age. They constitute a necrotic center of extracellular lipids covered by a fibromuscular layer. These poles are richly irrigated by newly formed vessels that can easily rupture. This grows from the intima to the vascular lumen because they are voluminous, causing the vascular obstruction that causes the symptoms, such as angina pectoris when it is the coronary arteries. These plaques are the origin of clinical signs by four mechanisms: • Artogenesis: increase in plaque volume. • Thrombi: rupture of the fibrous cover • Vascular tone abnormalities. • and destruction of the middle layer, aneurysm. The mechanisms that lead to complete occlusion of the arteries are associated with heart attacks. Plaque fracture is responsible for acute thrombosis, and intraplatelet hemorrhages are linked to rupture of the vessels that supply the atheroma.

Clinical manifestations. As we have seen, atherosclerosis occurs with an asymptomatic period of variable duration and various clinical manifestations can be presented by patients. Angina results from myocardial ischemia, myocardial infarction from thromboembolic affectations. Myocardial ischemia can affect electrical conduction and cause problems with heart rhythm, which can lead to cardiac arrest. Diagnosis The diagnosis is based on the observation of the electrocardiogram, particularly in the ST segments during a stress test, a depression in the ST segment of less than 1mm on exertion is a classic sign of myocardial ischemia.

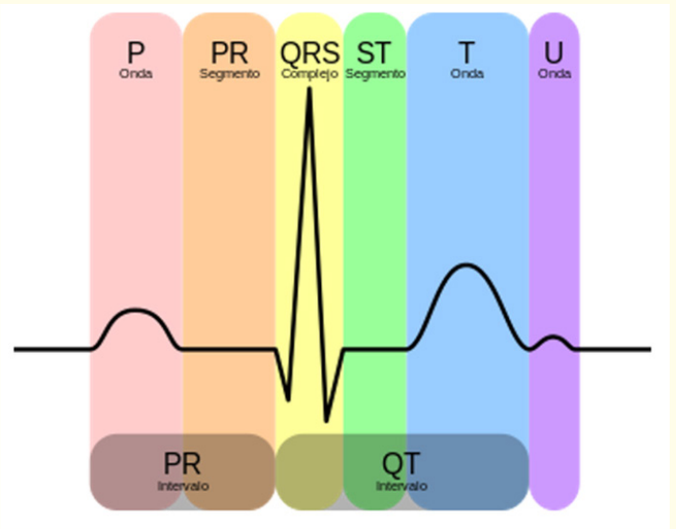

Figure a

Electrocardiogram. Treatment Among the preventive measures that can be taken are a decrease in tobacco consumption and an increase in physical activities. Establish a regimen to reduce the patient's weight and lower blood cholesterol levels. In case of persistent hyperlipidemia, lipid-lowering treatment should be established by the cardiologist. oral manifestations Among those that can occur in severe forms of ischemia that are symptomatic, such as angina pectoris and myocardial infarction, which we will see in the corresponding sections. In dental practice, we must identify patients who present an asymptomatic atherosclerotic cardiovascular condition, a diagnosis or suspicion of it, this identification will be made by anamnesis or clinical questioning. Patients already diagnosed the evolution is intended to specify the severity and know the nature of the treatment. In undiagnosed patients, this evaluation allows evidence. Particular attention should be paid to male patients aged 50 and over and post menopausal patients. The presence of risk factors should be investigated in these groups of patients. In the event that the factors are established such as hypertension, smoking, hyperlipidemia or others such as diabetes, stress, family history, postmenopause, oral contraceptives, obesity, sedentary lifestyle, etc., these are diseases and situations that should alert the dentist. According to the classification of the American Society of Anesthesiology, patients with atherosclerosis belong to its ASA III -IV classification, these patients are considered to have a severe systemic condition requiring care with minimal exposure to the stress generated by the procedure and an evaluation prior to the procedure. same. The ASA IV patient is considered a systemic condition that represents a vital risk. Consultation with the cardiologist is essential and the treatment that requires strict modifications must be carried out in the hospital environment.

Management of the patient in the office Among the general precautions we have the consultation with a specialist will be requested in the following conditions: • In the presence of signs and symptoms suggest that the patient suffers from complications of atherosclerosis. • When even under treatment the patient is symptomatic. • When the patient with atherosclerosis has not consulted in the previous year or when he presents most of the risk factors, he has not been medically evaluated in the last 12 to 18 months. • The treating physician will be consulted in case of doubts about the patient's state of health to know precisely the state of health, its severity of the condition, the nature of the treatment (medications used, dosages, etc.). follow-ups and levels of patient control. • According to the signs presented, eventual modifications of your treatment, as well as other pathologies and comorbidities will be consulted. stress caution • Anxiety and stress reduction must be controlled and this should be the dentist's priority concern, for this a trustworthy doctor-patient relationship must be established, not only in the professional field but also personally. • In a large number of patients, anxiety can be reduced with premedication such as benzodiazepines, which cause a limited depressant effect on the cardiovascular system, constituting the mode of choice within the framework of oral pharmacological sedation. • In patients who will be taken to the operating room, intravenous or inhaled sedation, if not contraindicated, can be performed particularly in severe forms. • The procedures, to the extent possible, should be performed in the morning. However, this factor is currently discussed since dawn is the time when there is a higher incidence of heart attacks (heart attack, stroke, arrhythmias and sudden death). The first thing in the morning and the last thing in the afternoon should be avoided when fatigue and stress may be greater. Freymiller Caution with anesthetics. • Anesthesia in all patients with cardiovascular disease, particularly local anesthetics, should be done with caution. • The use of vasoconstrictore4s is not contraindicated, but it is advisable not to exceed 0.04 mg of adrenaline, which is the equivalent of 2 carpules at 1:100,000 or 4 carpules at 1:200,000. • In all cases, an aspiration at the time of injection should be performed to ensure that injection is not performed intravascularly and is performed slowly. Caution with medications used by the patient. • Hypolipid-

emic drugs have no influence on the patient to be treated. • Coronary patients are usually treated with anticoagulants, by low-dose acetylsalicylic acid. These patients are the most exposed to primary hemostasis problems. Caution to the risks of infection. • Hygiene, asepsis and antisepsis measures must be respected to minimize the risks of bacterial or viral cross-infection. Precaution to take into account in prescriptions. • Paracetamol or any other type of analgesic, with the exception of acetylsalicylic acid and NSAIDs, do not present any contraindication. Caution regarding associated pathologies • The presence of pathologies and/or associated complications such as diabetes, kidney failure, etc. they need other measures that are specific to these pathologies in order for them to be taken into account in the prescriptions to be made. • Among the specific precautions in all types of procedures are an exception, multiple extractions, surgeries with flap lifts, retained teeth can be performed without particular protocols in patients who present a single risk factor and with recent medical evaluation. In any case, the general precautions must be respected.

Arterial hypertension Hypertension is a chronic and persistent elevation of blood pressure that, if untreated, is, among other things, the origin of severe cardiovascular complications. Arterial hypertension (AHT) continues to be the leading cause of death in the world. It is the main risk factor for cardiovascular diseases (CVD). Hypertension is a disease of multifactorial etiology, controllable, which decreases the quality and expectancy of life. Hypertension is defined as systolic blood pressure values greater than 140 mmHg and/or diastolic blood pressure greater than 90 mmHg, based on evidence in randomized studies of patients with these blood pressure values and with treatments to reduce the blood pressure. themselves are beneficial (image1). The same classification is used in young, middle-aged and elderly subjects, while in children and adolescents for whom data from intervention trials are not available different criteria, based on percentiles, are adopted.

The 7th report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, introduced in 2003 the category of prehypertension, which is defined as systolic blood pressure between 120 and 139 mmHg and blood pressure between 80 and 89 mmHg. Diastolic (Image 2). Patients with prehypertension have a higher risk of developing hypertension, those with blood pressure values 130-139/80-89 mmHg have a two times higher risk of developing hypertension than those with lower values. Hypertension is a highly prevalent cardiovascular disease, affecting more than one billion people worldwide [2]. Although more than 70% of hypertensive patients are aware of the disease, only 23-49% are treated and fewer (20%) achieve control The prevalence of hypertension varied according to age, race, education, etc. In adults, diastolic hypertension can normally be elevated to varying degrees (mild, moderate, or severe).

Categoría	Presión arterial sistólica (mm Hg)	Presión arterial diastólica (mmHg)
Óptima	<120	<80
Normal	120-129	80-84
Normal-alta	130-139	85-89
Hipertensión arterial grado 1	140-159	90-99
Hipertensión arterial grado 2	160-179	100-109
Hipertensión arterial grado 3	>= 180	>=100

Figure 1: Definitions and classification of office blood pressure levels (mmHg). Taken from Mancia, Fagard, Narkiewicz., et al., 2007.

A systolic blood pressure greater than or equal to 160mm/hg associated with normal diastolic blood pressure defines isolated systolic hypotension. In children and women blood pressure is lower. Thus a reading of 120/80 may indicate abnormal elevation. The incidence of hypertension is higher in elderly subjects and 80% have a family history. Stress, obesity, diabetes and alcohol are considered essential risk factors. Young men are more exposed than young women. In geriatric patients this is reversed. The light form is much more frequent than the severe one. Permanently high blood pressure (BP) affects the blood vessels of the kidneys, heart, and brain, increasing the incidence of heart, kidney, and coronary heart disease and stroke. Hypertension was called the "silent killer" because it often affects target organs (kidney, heart, brain, eyes) before the onset of clinical symptoms.

Etiology and Classification of Hypertension As in all genetic diseases of polygenic inheritance, the more genes are inherited, the greater the possibility of suffering AHT. However, it should be noted that the environment, through obesity, diet, sedentary lifestyle, salt consumption and smoking, has a decisive impact on the development of the disease. Blood pressure (BP) is the result of cardiac output multiplied by peripheral vascular resistance; in turn, this is determined by the circulatory volume, on the one hand, and on the other, both by the contractile force of the left ventricular musculature and by the heart rate. These elements (volume, heart rate, contractile force, peripheral vascular resistance) are regulated by various factors. the release of atriopeptin, activation of the renin-angiotensin-aldosterone system, and sodium retention increase blood pressure by contributing to increasing circulatory volume and, therefore, cardiac output.

With regard to peripheral vascular resistance, represented by the force with which the arterioles oppose the flow of blood, it influences BP due to the vascular capacity to contract or dilate. There are various constricting elements that increase this peripheral vascular resistance; As can be noted, BP regulation is complex

and some of these factors are strongly driven by genetic makeup. Each hypertensive person will especially have some dysfunction in these factors that participate in the disease; for this reason, pharmacological control will have to be individualized. Hypertension is classified into primary or essential hypertension (without organic cause) and secondary hypertension (has a well-established organic cause). 1. Primary or Essential Hypertension (without Organic Cause). Primary hypertension is the term used for medium to high BP over a long time (chronic) without a known cause, which is a very common form of hypertension, comprising about 90-95% of all patients with hypertension.

Secondary Hypertension. Hypertension these have a well-established organic cause, they are the following

- chronic renal pyelonephritis (parenchymal or renal vascular), acute and chronic glomerulonephritis, polycystic kidney disease, renal vascular stenosis or renal infarction, other severe kidney diseases (arteriolar nephrosclerosis), renin-secreting tumors.
- **Endocrine:** Oral contraceptives, adrenal hyperfunction (Cushing's syndrome, primary aldosteronism, congenital or hereditary adrenogenital syndrome), pheochromocytoma, myxedema, acromegaly, thyroid and parathyroid hyperfunction.
- **Neurological:** Psychogenic "diencephalic syndrome", familial dysautonomia (Riley-Day), polyneuritis (acute porphyria, lead poisoning), increased intracranial pressure.
- **Others:** Coarctation of the aorta, increased intravascular volume (excessive transfusion polycythemia vera), polyarteritis, hypercalcemia, drugs (corticosteroids, cyclosporine), sleep apnea, toxemia of pregnancy, acute intermittent porphyria. Pathogenesis of essential (primary) hypertension

From family and epidemiological studies, it is clear that hypertension is the result of a complex interaction between genetic factors and the environment. There are at least 50 known factors that increase blood pressure, among which the most important are age (55+ for men, 65+ for women); family history of premature cardiovascular disease;

- Tobacco use;
- Increased alcohol consumption;
- Sedentary lifestyle.
- Diet high in cholesterol.
- Coexistence of other diseases (diabetes, obesity, dyslipidemia).

Signs and symptoms

Arterial hypertension usually remains asymptomatic until the appearance of complications in the target organs. Dizziness, facial flushing, headache, tiredness, epistaxis, and nervousness do not develop when hypertension is uncomplicated. Severe arterial hypertension (hypertensive emergencies) can cause significant cardiovascular, neurologic, renal, and retinal symptoms (eg, symptomatic coronary atherosclerosis, heart failure, hypertensive encephalopathy, renal failure). Suspicion of HBP: Through questioning, triggering factors and situations for high BP figures can be identified, in the same way that with certain tools the clinical context of the patient can be evidenced from the mental and emotional point of view.

- Family history of kidney disease (polycystic kidney)
- Proteinuria/albuminuria, hematuria, increased creatininemia, oliguria, edema, anemia, upper urinary tract infections (parenchymal kidney disease).
- Episodes of sweating-diaphoresis, headache, palpitations, postural hypotension, paleness, facial flushing (pheochromocytoma)
- Episodes of muscle cramps, arrhythmias, muscle weakness and tetany, diuretic-induced symptoms (primary aldosteronism)
- Rapid weight gain, weakness, fatigue, oligo/amenorrhea, polydipsia, polyuria, arrhythmias, skin fragility, dorsal hump, and skin striae (Cushing's syndrome).
- Fatigue, lethargy, weight gain, hair loss, confusion, muscle weakness (hypothyroidism)
- Heart palpitations, weight loss, dry skin (hyperthyroidism)
- Daytime sleepiness, snoring, morning headaches, circadian rhythm inversion, nocturnal hypertension (OSA) Auscultation of a fourth heart sound is usually one of the earliest signs of hypertensive heart disease. Retinal changes may include arteriolar narrowing, hemorrhages, exudates, and, in patients with encephalopathy, papilledema (hypertensive retinopathy). The changes are classified (according to the Keith, Wagener, and Barker classification) into 4 groups with prognoses of increasing severity:
  - **Grade 1:** Only constriction of the arterioles
  - **Grade 2:** Constriction and sclerosis of the arterioles
  - **Grade 3:** Hemorrhages and exudates in addition to vascular changes
  - **Grade 4:** Papilledema.

Drugs that can cause high blood pressure • Nonsteroidal anti-inflammatory drugs, including Cox 2 inhibitors • Corticosteroids, anabolic steroids • Anorectics, amphetamines, cocaine and other drugs of abuse • Sympathomimetics: nasal decongestants • Oral contraceptives and estrogen replacement therapy • Excessive alcohol consumption • Erythropoietin, cyclosporine • Antidepressants, especially monoamine oxidase inhibitors, serotonin reuptake inhibitors, venlafaxine, clozapine, antiparkinsonian drugs • Herbal products that can cause HTA (cascara sagrada, licorice, etc.). Energy drinks. • Pills and products that contain caffeine (black tea, green tea) • Post-suppression rebound of bromocriptine and clonidine • Antimigraines, ergotamine.

Diagnosis In its early stages, AHT is silent and can only be diagnosed by a BP reading. If symptoms occur, such as occipital head-

ache, shortness of breath, tinnitus, dizziness, phosphenes, nausea or vomiting, it is because it has already been established for a long time. Among other things you must first: • Take multiple blood pressure measurements to confirm hypertension • Urinalysis and ratio between albuminuria and creatininuria; if abnormal, renal ultrasound should be considered • Blood test: fasting lipidemia, creatininemia, kalemia • Renal ultrasound if creatinine concentration is increased • Look for aldosteronism in patients with hypokalaemia • ECG: in patients with ventricular hypertrophy, consider echocardiography • Sometimes measurement of thyroid-stimulating hormone • Look for a pheochromocytoma or a sleep disorder if the elevation of blood pressure is sudden and labile or severe. Arterial hypertension is diagnosed and classified with sphingomanometry. History, physical examination, and other tests help identify and define target organ damage. complications The complications of hypertension are related to the elevation of blood pressure exerted by vascular and cardiac changes such as atherosclerosis, producing specific complications such as: • Myocardial: left ventricular hypertrophy; • Cerebrovascular: cerebral infarction and cerebral hemorrhages; • Renal: nephrosclerosis cause of kidney failure • Aortic and atherosclerotic The natural history of uncontrolled hypertension leads to death from myocardial infarction, renal failure, and cerebral hemorrhage, especially if it is accompanied by other cardiovascular risk factors, such as being male, having uncontrolled diabetes mellitus, disorders in the lipid metabolism, smoking and having a positive family history of hypertension.

Treatment Adopting a healthy lifestyle is essential to prevent high blood pressure. Major lifestyle changes that could lead to lower blood pressure include reduction of body weight in overweight or obese patients, adoption of a low-salt diet rich in potassium and calcium, increased physical activity, moderate alcohol consumption and smoking cessation. The pharmacological treatment of hypertension depends on the stage of hypertension, the associated diseases and the risk factors present. The recommendations are based on the definition and classification of hypertension adopted by the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure in the US in 2003 [2] and the conclusions of the European Society of Hypertension-European Society of Cardiology (ESH-ESC) in 2013 [1]. The selection of drugs is based on several factors. When a drug is given initially for non-black patients, including those with diabetes, initial treatment might be with an ACE (angiotensin-converting enzyme) inhibitor, angiotensin receptor blockers, angiotensin channel blockers, calcium, or a thiazide-type diuretic (chlorthalidone or indapamide). For black patients, including people with diabetes, a calcium channel blocker or thiazide diuretic is initially recommended, unless patients have stage 3 or higher chronic kidney disease. In black patients with stage 3 chronic kidney disease, an ACE inhibitor or angiotensin II receptor blocker is appropriate. When 2 drugs are given initially, a single tablet combination containing an ACE inhibitor or angiotensin II receptor blocker and a diuretic or calcium channel blocker is recommended.

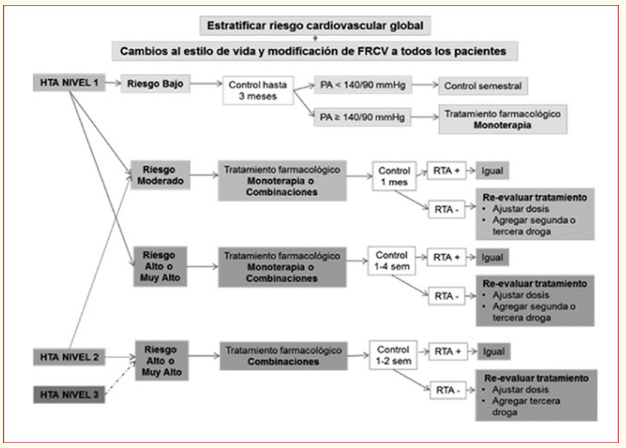

Figure 2

Initial treatment algorithm for essential hypertension. HTA: arterial hypertension. CVRF: Cardiovascular risk factors. PA: blood pressure. RT: Answer. wk: weeks. Taken from Waisman, G. (2013).

Adequate pressure control sometimes requires multiple evaluations and changes in drug therapy. Reluctance to titrate or add drugs to control blood pressure must be overcome. Lack of compliance by the patient, particularly considering that the treatment must be maintained for life, can interfere with adequate control of blood pressure. Education associated with empathy and support are critical to the success of treatment. Oral manifestations caused by the adverse effects of antihypertensive drugs There is a series of potential interactions between the drugs that the hypertensive patient receives as part of their treatment and those that the dentist may prescribe, as well as side reactions that require the implementation of modifications in the dental treatment plan. AHT does not generate specific oral lesions. Perhaps the only ones that could be observed, without being pathognomonic, are those of the hemorrhagic type generated by the sudden elevation of BP and rupture of small blood vessels; however, antihypertensive drugs could produce secondary reactions of inhomogeneous manifestation; that is, they do not occur in all patients and, on occasions, they represent a challenge for diagnosis. Among these oral manifestations we have: Xerostomia. Many antihypertensive medications such as ACE inhibitors, thiazide diuretics, loop diuretics, and clonidine are associated with xerostomia. Its probability increases with the number of concomitant medications. Xerostomia has many consequences, as a result of a deficiency in the availability of immunoglobulin A, present in secretions and which favors periodontal problems, cavities, difficulty chewing, swallowing and speaking, candidiasis and burning mouth syndrome. Sometimes the sensation is transient and the salivary function is adjusted by the patient himself. There are situations in which it is required to change the antihypertensive medication. It is often necessary to treat xerostomia directly with parasympathomimetic agents such as pilocarpine or cevimeline. Other recommendations include frequent sips of water, sugar-free sweets, cutting back on coffee, and avoiding mouthwashes that

contain alcohol. To reduce the risk of caries, topical applications of fluoride are recommended, particularly in the form of gels with high concentrations in impression trays.

Gingival hyperplasia. It can be caused by calcium channel blockers, with an incidence ranging from 6 to 83%. Most cases are associated with nifedipine. The effect could be dose related. Gingival hyperplasia is manifested by pain, gingival bleeding and difficulty chewing. Good oral hygiene greatly reduces its incidence. Changing antihypertensive medication can reverse hyperplasia. Lichenoid reaction. Many antihypertensives (thiazide diuretics, methyldopa, propranolol, captopril, furosemide, spironolactone, and labetalol) are associated with oral lichenoid reactions. The clinical forms differ greatly from lichen planus itself, but without the characteristic of bilaterality and skin involvement observed in this condition. In the pathogenesis of lichenoid reactions of drug origin, it is likely that the immune response that generates keratotic linear white lesions, which have the appearance of lace or mesh, distributed on an erythematous background, participates. The easiest way to treat it is to change the antihypertensive medication, and lichenoid reactions resolve after stopping the offending drug. If the medication cannot be changed, lichenoid reactions are treated with topical corticosteroids. Other undesirable effects. ACE inhibitors are associated with coughing and loss of taste (ageusia) or taste disturbance (dysgeusia). Dysgeusia has also been reported with the use of other antihypertensives, such as β-blockers, acetazolamide, and diltiazem. It has been postulated that dysgeusia may result from a mechanism that affects salivary handling of metal ions such as magnesium. Management of the patient in the office • BP should be taken at each visit if the patient is identified as hypertensive, diabetic, has established renal or coronary disease, since dental care will depend on the values that allow safe management. • Any person not diagnosed as hypertensive, who has BP values greater than 140/90 mm Hg should be referred to a doctor to confirm the diagnosis and provide treatment if necessary. • Those patients classified as prehypertensive should also be referred to a specialist, so that the doctor can provide preventive measures to control metabolic syndrome and compensate or avoid risk factors for hypertension and cardiovascular disease. • The patient should be asked at each visit if they have taken their medications. If the administration was omitted due to forgetfulness, but the figures are normal, the treatment could be continued; however, the patient should be reminded of the importance of the use of drugs in the long term. Precautions with local anesthetics • The recommended dose of epinephrine in a patient at cardiac risk is 0.04 mg, which is equal to that contained in about two cartridges of LA with 1:100,000 with epinephrine or 4 cartridges with 1:200,000 with epinephrine. Although vasoconstrictors can precipitate significant elevations in blood pressure, numerous studies have shown that the use of one or two cartridges of 2% lidocaine with 1:100,000 epinephrine (0.018 to 0.036 mg epinephrine) is of little consequence in most patients. patients with hypertension.

In patients with severe disease, it may be useful to measure BP and heart rate after anesthetic injection. • Slow administration and aspiration can prevent undesirable reactions. • Other contraindications to local vasoconstrictive anesthesia include severe uncontrolled hypertension, refractory arrhythmias, myocardial infarction or stroke before 6 months of age, unstable angina, coronary artery bypass grafting before 3 months, congestive heart failure. and untreated hyperthyroidism. • Due to the higher concentrations of epinephrine (nearly 12 standard cartridges) in gingival retraction cords used for denture impressions and their rapid uptake into the circulation, the use of epinephrine for gingival dislodgement in patients with cardiovascular disease is contraindicated. Stress Precautions • Control of pain and anxiety is very important in patients with high medical risk. Patients with cardiovascular disease are at high risk of complications due to endogenous catecholamines (adrenaline and norepinephrine) released by pain and stress. These catecholamines can drastically increase blood pressure and cardiac output. This effect is reduced by controlling dental pain. • In a large number of patients, anxiety is reduced by sedative premedication. The benzodiazepines that it produces have a limited depressant effect on the cardiovascular system, constituting the drug of choice for the oral route. If the patient is going to be taken to the operating room, intravenous sedation reduces the effects of anxiety in severe hypertensive patients. • As far as possible, procedures should be of short duration and preferably performed during the day, and the patient should be reassessed after the procedure. Precautions at the time of prescription A drug interaction can be defined as a situation in which a substance, which in this case is another prescribed drug, affects the activity of a drug that a patient is already taking. Drug-analgesic interactions are often reported, but only a small number have clinical relevance in dentistry. Knowledge of the importance of these interactions will allow dentists to optimally prescribe analgesics and minimize the potential for adverse reactions. Antihypertensive medication is one of the most prescribed drugs in primary care, representing almost a quarter of all prescribed drugs. 15 The most frequently prescribed drugs for the treatment of hypertension are thiazides and loop diuretics, calcium channel blockers (CCBs), ACE inhibitors, β adrenergic receptor blockers, α adrenergic receptors and angiotensin II receptor antagonists. In addition, on many occasions a single antihypertensive drug is not adequate and complementary antihypertensives are prescribed until hypertension is controlled.

Usually, when two antihypertensive drugs are indicated, an ACE inhibitor or angiotensin II receptor antagonist can be combined with a thiazide or CCB. British Medical Association and the Royal Pharmaceutical Society of Great Britain. British national formulary. 64th ed. London: BMJ Publishing Group. 2012. 17 All of these drugs have different pharmacological methods of action and therefore different potential harmful interactions. In summary, the hypotensive effects of CCBs, ACE inhibitors, β adrenergic receptor blockers, angiotensin II receptor antagonists, and loop diuretics are antag-

onized by the use of NSAIDs. 23 White W B. Cardiovascular risk, hypertension, and NSAIDs. Curr Rheumatol Rep 2007; 9:36–43 NSAIDs interact with diuretics in three different ways: they can cause nephrotoxicity, 24 Swainson C P, Griffiths P. Acute and chronic effects of sulindac on renal function in chronic renal disease. Clin Pharmacol Ther 1985; 37:298–300. they antagonize the diuretic effect and increase the risk of hyperkalemia. 23 The latter could initiate potentially harmful cardiac arrhythmias or even congestive heart failure. Calcium channel blockers are also a relatively common medication for the treatment of hypertension, accounting for 1.7% of all prescribed medications. These drugs have significant potential interactions. One of the most harmful interactions may be with macrolide antimicrobials (eg, erythromycin, clindamycin). Precautions in the attention of a dental emergency. 1. If the patient presents a diastolic BP between 90 and 99, the patient is medically controlled, and is under medical supervision, all types of dental care can be considered, including emergency ones, provided that the established precautions are taken into account.

These patients these acts to tolerate all non-surgical procedures, as well as simple surgical procedures (simple extractions, curettage, gingival plasties, etc.), taking into account the above, particularly with regard to anxiety and doses of adrenaline in the anesthetics Multiple extractions, periodontal, periapical, implant and included tooth surgery will require effective pre-surgical sedation. 2. If the patient has a diastolic BP between 100 and 109, the patient should consult a professional to take the measurements. If the treating dentist considers that dental care would control the TA levels, emergency treatment can be carried out with the necessary precautions. If important care is required, these will be performed under sedation in a hospital setting. As a general rule, all patients with moderate hypertension can undergo non-surgical procedures under the usual conditions. The realization of simple surgical treatments and root canals is preferable to use sedation. Intermediate procedures (multiple extractions, surgeries with flaps and advanced procedures such as extraction of retained teeth, implant surgery should not be performed in the office. They must be associated with effective sedation. These procedures must be performed in a hospital environment or with a doctor. specialist present in case of increased hypertension and development of major bleeding

If the diastolic pressure is greater than 100 mm/hg, no procedure will be performed until a doctor has been consulted and a treatment established or the reassessment of an ongoing treatment. In this type of patient, only non-surgical procedures limited to intraoral clinical examination, oral hygiene instructions, x-rays and dental impressions can be performed. This type of patient should be directed to a cardiology evaluation or more complete reevaluation in other types of medical treatments. 3. If the patient presents malignant hypertension, this is considered a medical emergency and the patient must be immediately evacuated and supported by a medical plan. In the event of a hypertensive emergency (>120/210 mmHg), the emergency service should be activated and furosemide (40 mg, orally) should be administered. If this proves insufficient to restore pressure control, captopril (25 mg orally or sublingually) should be administered. If the pressure does not decrease within 30 minutes of these measures, the patient should be referred to the Emergency Department of the nearest hospital.

Symptomatic ischemic heart disease. angina pectoris. Ischemic heart disease or coronary disease refers to diseases caused by the lack of blood supply (and therefore oxygen and food) to the heart, in most cases as a consequence of the obstruction of the passage of blood through the arteries. coronary. Angina pectoris is a paroxysmal chest pain syndrome produced by coronary ischemia of less than 20 min. This can be caused by obstruction of the caliber of a coronary vessel, it can cause brief pain due to myocardial ischemia, presenting as angina pectoris, if the pain is longer with a necrotic lesion, it degenerates as a myocardial infarction and even sudden death, generally due to arrhythmias. Angina pectoris occurs when cardiac afterload and the resulting myocardial oxygen demand exceed the capacity of the coronary arteries to supply an adequate amount of oxygenated blood. Such an imbalance between supply and demand can occur when arteries become narrow. Lumen narrowing is usually the result of coronary artery atherosclerosis, although it may also be due to coronary artery spasm or, rarely, coronary artery embolism. Acute coronary thrombosis can cause angina if the obstruction is partial or transient, but usually results in an acute myocardial infarction. In addition to exercise, the workload on the heart muscle can increase in the presence of certain diseases such as high blood pressure, aortic stenosis, aortic insufficiency, or hypertrophic cardiomyopathy. In these cases, angina can occur both in the presence of atherosclerosis and without it.

Signs and symptoms

Angina pectoris can present with indefinite pain of little concern or in a short time become intense and severe precordial compression. Rarely does the patient describe it as pain. The discomfort is most often felt below the sternum, although the location varies. The pain may radiate to the left shoulder and down the inside of the left arm, including to the fingers, to the back, throat, jaw and teeth, and occasionally to the inside of the right arm. It can also be felt in the epigastrium. The discomfort caused by angina is never felt above the level of the ears or below the navel. The person who suffers from it has the sensation of imminent death, puts his fist to his chest (Levine's Sign) and adopts a position of self-protection. Any anginal attack lasting longer than 20 minutes should be considered a myocardial infarction. The patient during the attack adopts a fixed position and usually brings his fist to his chest. It usually occurs between 45 and 65 years of age, especially in men whose profession is often considered highly stressful. Women are affected to a lesser extent. The diagnosis of angina is suspected in patients with typical chest discomfort triggered by exertion and relieved by rest. The presence in the history of significant risk factors for coronary artery disease (CAD) adds weight to the reported symptoms. When chest discomfort lasts > 20 minutes or occurs at rest,

or if patients present with syncope or heart failure, tests should be ordered to identify an acute coronary syndrome. Chest discomfort can also be caused by GI disorders (eg, gastroesophageal reflux, esophageal spasm, indigestion, cholelithiasis), costochondritis, anxiety, panic attacks, hyperventilation, and other heart conditions (eg, aortic dissection, pericarditis, prolapsed mitral valve rupture, supraventricular tachycardia, atrial fibrillation), even despite lack of compromise of coronary blood flow. Angina typically occurs during exercise or intense emotion, generally lasts no more than a few minutes, and subsides with rest. The response to exercise is often predictable, but in some patients well-tolerated exercise on one day may trigger an angina episode on another day due to variations in arterial tone. The symptoms are exaggerated when exertion or exercise is carried out after a meal or in cold weather; for example, a walk against the wind or the first contact with cold air when leaving a warm room can generate a crisis. Symptom severity is usually classified by the degree of exertion required to elicit the angina episode (see table Canadian Cardiovascular Classification System for Angina). ECG is always done. More specific tests include stress testing with ECG or myocardial imaging (eg, echocardiography, scintigraphy, MRI) and coronary angiography. In the first place, the indication of non-invasive tests is considered. Treatment The treatment of angina pectoris responds to one of the following approaches: • Reduction of risk factors • Medication management • Revascularization and Coronary Angioplasty • Surgery.

Reduction of risk factors They essentially consist of eliminating tobacco use, controlling diabetes and hypertension. 2 years after quitting, the risk of heart attack drops to that of people who never smoked. Intensive reduction of total cholesterol and that associated with low-density lipoprotein (LDL) (with diet and statins) decreases the rate of progression of coronary disease, can promote the involution of some lesions and improves endothelial function and, in consequently, the arterial response to stress. An exercise program with an emphasis on walking often improves a sense of well-being, decreases the risk of acute ischemic events, and increases exercise tolerance. drug control These are intended to reduce the demand for oxygen by the myocardium to increase its contribution. • Antiplatelet drugs inhibit platelet aggregation. Aspirin irreversibly binds to platelets and inhibits cyclooxygenase and platelet aggregation. • Beta-blockers limit symptoms and prevent heart attack and sudden death more than other drugs. Beta-blockers block cardiac sympathetic stimulation and reduce systolic blood pressure, heart rate, contractility, and cardiac output, thereby decreasing myocardial oxygen demand and increasing exercise tolerance. • Long-acting nitrates (oral or transdermal) are indicated when symptoms persist despite receiving a maximum dose of beta-blockers. If anginal episodes occur at predictable times, a nitrate should be administered to prevent these episodes at predetermined times. Oral nitrates are isosorbide dinitrate and isosorbide mononitrate (the active metabolite of dinitrate). Both drugs act in 1 to 2 hours and their effect lasts 4 to 6 hours. • Calcium channel blockers can be used if symptoms persist despite nitrate administration or if the patient does not tolerate nitrates. Calcium channel blockers are particularly helpful in those with high blood pressure or coronary spasm. Different types of calcium channel blockers have different effects. Revascularization, either with percutaneous coronary intervention (eg, angioplasty, intravascular stenting) or surgically with intracorporeal pump CABG, should be considered if angina persists despite drug therapy and worsens quality of life as a result of the presence of anatomical lesions (identified on angiography) that increase the risk of patient death. The selection between percutaneous coronary intervention and myocardial revascularization surgery depends on the extent and location of the lesions, the experience of the surgeon and the medical center, and, to some extent, the preferences of the patient. oral manifestations. Oral manifestations are of two types: painful, and the other is a consequence of medication. The pain associated with angina can radiate to the head and neck, in such cases, the locations are essentially dental and maxillofacial. In certain circumstances, patients report a burning sensation at the level of the tongue and hard palate. All pains are generally simultaneous with chest pain.

The manifestations associated with the medications are mainly burning upon the administration of nitrates. The phenomena of gingival hyperplasia and fibrosis are induced by calcium inhibitors. Precautions in dental practice. A medical consultation will be demanded • In the presence of signs or symptoms suggestive of angina pectoris, those already treated and symptomatic patients. • Patients who have suffered angina pectoris and have not consulted in a year present more risk factors, those that have not been evaluated in the last 12 to 18 months. The treating physician will be consulted • in case of uncertainty about the patient's state of health or the patient's diagnosis and to know it precisely, the severity of the condition, the nature of the treatment and the levels of controls. • To define according to the table presented and the possible modifications concerning the treatment, as well as to know about other present pathologies if their follow-up is multidisciplinary. Stress Precautions • A condition that closely characterizes brain-heart connections is angina pectoris (hereinafter referred to as angina). • The severity of angina is more strongly associated with psychological variables, such as pre-existing health beliefs, depression, and anxiety. • Anxiety control and stress reduction are the dentist's top concerns. These precautions require excellent trust with the patient, not only from the professional side but also from the personal side. In a large number of patients, anxiety can be reduced with preoperative sedation. • Inhalation or intravenous sedation is a way to reduce anxiety, but it must be managed in a hospital setting. anesthetic precautions • As in all patients with cardiovascular disease, the administration of anesthetics, particularly local ones, should be done with caution. • The use of a vasoconstrictor is not contraindicated, but the recommended dose of epinephrine of 0.04 mg is recommended, which is equal to that contained in about two cartridges of lidocaine with 1:100,000 epinephrine or 4 cartridges

with 1:200,000 epinephrine. Both the American Heart Association and the American Dental Association state that 0.2 mg epinephrine and 1.0 mg levonordefrine can be used as the maximum dose of these vasoconstrictors; Even so, caution is recommended in those who have suffered coronary diseases, so no more than three cartridges should be used per appointment. • In patients with severe disease, it may be useful to measure BP and heart rate after anesthetic injection. • Slow administration and aspiration can prevent undesirable reactions. This accident produces systemic symptoms derived from the anesthetic drug, not from the vasoconstrictor. For a similar reason the use of injections is contraindicated. • Intraligamentary or intrapulpar, as well as the use of tissue retraction cord containing adrenaline. In the latter case, it is not possible to control the amount of adrenaline that can enter the bloodstream from the gingival crevice where the thread is placed to make the final impressions on fixed prostheses. • The use of vasoconstrictors will be excluded in uncontrolled patients or patients with heart rhythm problems, in the case of 3% mepivacaine will be used. • High doses of local anesthetics without a vasoconstrictor are contraindicated in any patient, especially those with cardiovascular disease, since the risk of toxicity from the local anesthetic is greater. The vasoconstrictor contributes to a slow absorption of the anesthetic and therefore decreases its toxicity and favors the duration and depth of anesthesia.

Caution in patients following treatment Certain medications, due to their side effects, may interfere with the symptoms, for example, those derived from nitrates may cause hypotension and syncope; • Vasodilators derived from nitrates can be the cause of headaches or tachycardia; • beta-blockers can cause bradycardia and fatigue and can mask hypoglycemia; • Calcium inhibitors can cause headaches. Caution to the risks of infection. • Hygiene, asepsis and antisepsis measures must be respected to minimize the risks of bacterial or viral cross-infection. Caution when prescribing • The prescription of paracetamol or other analgesics such as NSAIDs does not present any contraindication. Cardio depressant effects, barbiturates and narcotics must be avoided. Caution with concomitant pathologies • The presence of pathologies and associated complications (diabetes, kidney failure, etc.) require more specific precautions for these diseases. Dental precaution in case of specific angina light angina • Surgical and non-surgical procedures must all be performed respecting general precautions. Moderate angina. • Only diagnostics (oral examination, x-rays, dental impressions), orthodontics and simple dental surgery can be considered without a particular protocol. • Simple surgical procedures (simple extraction, flapless periodontal surgery and dental implants), more complex dental operations such as scaling and curettage will not be performed unless nitroglycerin (0.3-0.4mg) is administered sublingually before the procedure. procedure, other general precautions must be taken into account. • Regarding the other surgical acts: multiple extractions, surgeries that require flaps for access, the same precautions must be taken and depending on the complexity they can be performed in a hospital setting. severe angina • Only diagnostics will be carried out under normal conditions. But any other procedure must have a specialist doctor present. • Simple operations can be carried out scrupulously with the general precautions already expressed. • Simple surgical procedures (simple extraction, periodontal surgery without flaps and dental implants), more complex dental operations such as periodontal curettage will be performed in a hospital environment and will be discussed with the attending physician. • Particular attention must be paid to patients who are under a large dose of beta-adrenergic antagonist (160 mg of propranolol) depending on the importance of the procedure and the type of anesthesia to be used. This medication must be administered intravenously and a doctor present is Strongly recommended in case the patient presents unstable angina pectoris, this condition will be treated until the symptoms stabilize.

Management of the patient before an attack of angina pectoris. The management of the patient who presents with a PA attack during dental treatment should be as follows: • Discontinue dental treatment. • Place the patient in a position that is comfortable for him (45°), since he tends to sit down and it is not recommended to force him to remain in a supine position. • Administer a short-acting coronary vasodilator (nitroglycerin, isosorbide, nitroglycerin aerosol) and wait for it to act between 2 and 5 minutes. • Take vital signs. • If oxygen is available, administer it through a nasal cannula at a dose of six liters per minute. • Give a second dose of the coronary vasodilator if no response is obtained within five minutes. • If pain relief is not achieved within 10 minutes give a third dose and seek medical help. • Accompany the patient in the ambulance to the hospital If a patient with no history of heart disease presents paroxysmal chest pain during dental consultation, the clinician should treat the condition as a myocardial infarction and request help to transfer the patient to a hospital, since the chances of survival will increase if he receives specialized medical help. SYMPTOMATIC ISCHEMIC HEART DISEASE. MYOCARDIAL INFARCTION Myocardial infarction is myocardial necrosis secondary to a sudden reduction in coronary blood flow to an area of the myocardium. The infarcted tissue develops permanent dysfunction, although there is an area adjacent to the infarcted tissue with potentially reversible ischemia. Myocardial infarction primarily affects the left ventricle, but the lesion may extend to the right ventricle or atria. The patient with ischemic heart disease and especially the one who has suffered an acute myocardial infarction has a high incidence of suffering a reinfarction or serious arrhythmias. Etiology Acute myocardial infarction occurs in patients with ischemic heart disease. In most cases it results from complete occlusion of one or more of the main coronary arteries. This occlusion is produced by the formation of an atheromatous plaque found inside a coronary artery. This plaque can ulcerate or rupture, causing the obstruction of that vessel. The factors that have been related to the rupture of the atheromatous plaque and the appearance of thrombosis are of two types: histological and rheological. A heart attack can also be caused by the existence of arteriosclerosis in a blood vessel that causes a narrowing of the lumen of said vessel. This fact facilitates

the development of a thrombus that ends up clogging the vessel. The clot can produce from a thrombosis to an embolism (in this case the clot is transported from its place of origin to another more distant by the bloodstream), thus triggering a myocardial infarction. There are a number of factors associated with myocardial infarction. These factors are the same as those associated with arteriosclerosis, since it is the main cause of heart attacks. The main risk factors are: arterial hypertension, old age, male sex, smoking, hypercholesterolemia, diabetes mellitus, obesity and stress. Another of the risk factors that can trigger a heart attack, although it is less frequent than the previous ones, is periodontal disease. Oral health is an index to predict coronary disease, as well as previous history of myocardial infarctions and diabetes, since patients who suffered acute myocardial infarction had periodontitis and poorer oral health compared with healthy patients. Cocaine use and other causes of coronary spasm can culminate in a myocardial infarction. Spasm-induced myocardial infarctions can be detected in both normal and atherosclerotic coronary arteries. There are triggering factors for a myocardial infarction such as strenuous exercise, stress, large meals and sometimes it can occur with the subject at rest. pathophysiology The initial consequences vary according to the size, location, and duration of the obstruction, ranging from transient ischemia to infarction. Measurement of newer and more sensitive markers indicates that the patient is likely to experience some degree of cell necrosis even in the mildest forms; consequently, ischemic events can manifest with a spectrum of signs and symptoms, for which their classification into subgroups, although useful, is somewhat arbitrary. The sequelae of the acute event depend above all on the mass and type of infarcted cardiac tissue. This is a medical emergency that requires immediate hospitalization in an intensive care unit (ICU); more than 50% of deaths occur within the first 2 hours of symptom onset, and most are often from ventricular fibrillation. climatic demonstrations. The symptoms of acute coronary syndrome depend to some extent on the extent and location of the obstruction and are quite variable. Painful stimuli from the thoracic organs, including the heart, can cause discomfort described as tightness, tearing, distension with an urgent need to belch, indigestion, heartburn, dull ache, stabbing pain, and sometimes sharp needle-prick pain. a needle. These can be prolonged (more than 30 minutes), resistant to sublingual administration of nitroglycerin, and become more intense and acute.

Many patients deny that they are in pain, insisting that it is just a "nuisance." Except when the infarction is massive, it is difficult to recognize the magnitude of the ischemia based on the symptoms alone. Diagnosis Although there may be asymptomatic myocardial infarction, most of them will give symptoms that include paroxysmal chest pain that persists for more than 20 to 30 minutes. This is the basis for the diagnosis of said condition. The positive diagnosis is established with the association of at least two of the following criteria: • typical pain • Typical electrocardiogram modification • Elevation of cardiac enzymes. The electrocardiogram is an important diagnostic tool because it allows the physician to identify the site and size of the area of necrosis; and after stress tests, an ambulatory ECG (Holter), stress perfusion scintigraphy with Thallium 201, exercise echocardiography, ambulatory monitoring of ventricular function, and cardiac catheterization. Biomarkers that may be useful after acute myocardial infarction. These markers are released into the bloodstream after myocardial cell necrosis. The markers appear at different periods after injury and their concentration declines at different rates. Sensitivity and specificity for detecting myocardial cell injury vary significantly between these markers. These biomarkers are creatine phosphokinase (CK) and its isoenzyme CK-MB. The latter is sensitive and quite specific, reaching its highest figures 14 to 36 hours after the infarct and returning to normal in 2 or 3 days. Lactodehydrogenase (LDH) is later with a maximum peak between 2 to 3 days later and with normal levels at 10-14 days. We must consider that the elevation of CK and LDH indicate necrosis, but are not specific to the myocardium. complications After the acute event, many complications can occur. They tend to compromise • Electrical dysfunction (eg, conduction defects, arrhythmias) • Myocardial dysfunction (eg, heart failure, ventricular septal or free wall rupture, ventricular aneurysm, pseudoaneurysm, mural thrombus formation, cardiogenic shock) • Valvular dysfunction (typically mitral regurgitation) • Hypertension • Hypotension Treatment The treatment of the infarction is oriented to the restoration of the perfusion of the myocardium (thromboembolic treatments) and to reduce the oxygen consumption by the myocardium. The orientation of the treatment consists of: • Implement emergency actions (artificial ventilation, external cardiac massage) in case of ventilatory and/or circulatory failure. • hospitalize the patient in an intensive care unit. • Monitor vital signs. • Place a venous access line and oxygen administration. • Control pain. In the acute phase the following measures should be carried out: • Administer morphine or its derivatives • Administer acetylsalicylic acid • Intravenous thromboembolism if not contraindicated. • Emergency angioplasty in case thromboembolism is contraindicated. The reduction of oxygen consumption is essential for pain control. Once the pain is controlled, the levels of circulating catecholamines are reduced with morphine (1-4mg) and this is repeated every 5 to 10 minutes until the pain or the pain is controlled. side effects (nausea, hypotension, respiratory depression), meperidine is a alternative to morphine. Sublingual (0.15-0.6mg) or intravenous (25-50 µg) nitroglycerin helps with ischemic pain, sedation with anxiolytics is beneficial in the early days. Reperfusion of ischemic myocardium is the second orientation of treatment. Thrombolytic and angioplasty treatment constitute the classic management. Precautions in dental practice. A medical consultation will be requested when: • the signs and symptoms suggest that the patient was exposed to a heart attack • Patients who are under treatment and symptomatic patients • Patients with a history and who have not made a medical consultation in the previous year or who present more risk factors, and who have not been recently evaluated by the cardiologist. The treating physician will be consulted when: •

In case of uncertainty about the patient's state of health or when the already diagnosed patient does not precisely know her state of health, the severity of the problem, the nature of the treatment and the levels of control of the disease. • To define according to the table presented and the possible modifications concerning the treatment, as well as to know about other present pathologies if their follow-up is multidisciplinary. Stress Precautions • Anxiety control and stress reduction are the dentist's top concerns. These precautions require excellent trust with the patient, not only from the professional side but also from the personal side. In a large number of patients, anxiety can be reduced with preoperative sedation. • Inhalation or intravenous sedation is a way to reduce anxiety, but it must be managed in a hospital setting. • Dental treatments, if possible, will be of short duration and carried out during the course of the day (the reasons are explained in the section on angina pectoris). anesthetic precautions • As in all patients with cardiovascular disease, the administration of anesthetics, particularly local ones, should be done with caution. • Concerning the performance of local anesthesia, the rules for angina pectoris are the same as for a patient with a history of heart attack. The use of vasoconstrictors is not contraindicated, but the recommended dose of epinephrine of 0.04 mg is recommended, which is equal to that contained in two cartridges of lidocaine with 1:100,000 with epinephrine or 4 cartridges with 1:200,000 of epinephrine. Both the American Heart Association and the American Dental Association state that 0.2 mg epinephrine and 1.0 mg levonordefrine can be used as the maximum dose of these vasoconstrictors; Even so, caution is recommended in those who have suffered coronary diseases, so no more than three cartridges should be used per appointment. • In patients with severe disease, it may be useful to measure BP and heart rate after anesthetic injection. • Slow administration and aspiration can prevent undesirable reactions. • The use of vasoconstrictors will be excluded in uncontrolled patients or patients with heart rhythm problems, in the case of 3% mepivacaine will be used. Caution in patients following medical treatment • With the exception of anticoagulated cases, these do not require particular modification. • As in cases of angina pectoris, the prescription (vasodilators, beta-blockers etc.) can interfere with the procedures, and these must be taken into consideration due to their side effects.

Caution in anticoagulated patients • A concern of the dentist will be the management of the patient who requires some oral surgical procedure and receives oral anticoagulants such as sodium warfarin or coumadin. • Before proceeding to ask the doctor for any modification to the dose of the drug, the type of oral procedures to be performed must be planned, since activities such as the initial clinical examination, taking X-rays or models, prophylaxis, surgical procedures or prosthesis, root canal treatment, activation of orthodontic or orthopedic appliances, do not require modifications to oral anticoagulant management. • For patients who require one or more extractions, implants or periodontal surgery, an INR (International Normalized Ratio) should be requested to determine the degree of anticoagulation. In no case is it recommended to perform surgical procedures with figures greater than three, due to the high risk of bleeding. Given an INR result of ≥ 3, the doctor should be asked to reduce the dose to have an INR less than 2.5. In patients undergoing anticoagulant therapy, extensive surgical procedures, involving more than one sextant or multiple extractions, should be avoided, as there is a greater risk of bleeding due to greater exposure of the bone surface. • Other anticoagulant To perform any elective surgery in patients receiving heparin, it is indicated to carry out the procedure the day after stopping the medication, since by then it will have been metabolized and eliminated. This drug has a very short half-life (within 6 hours of stopping it, it has been metabolized almost completely). Caution to the risks of infection. • Hygiene, asepsis and antisepsis measures must be respected to minimize the risks of bacterial or viral cross-infection. Caution when prescribing • The prescription of paracetamol or other analgesics such as NSAIDs does not present any contraindication. Cardiodepressant effects, barbiturates and narcotics must be avoided. Caution with concomitant pathologies • The presence of pathologies and associated complications (diabetes, kidney failure, etc.) require more specific precautions for these diseases. Dental precaution in the event of a heart attack in the previous month. • Only intraoral examination, hygiene instructions. Dental impressions and emergency dental operations can be performed according to what the treating physician recommends. • All other types of treatment will be postponed beyond one month. In emergency cases, treatments should be as conservative as possible. If a surgical treatment is presented (simple extraction of a tooth) the treating physician should be consulted. To the extent possible, procedures will be performed in a hospital environment. Dental precaution in the event of a heart attack more than a month after it occurred. • The dentist must consult with the treating physician, to find out about his treatment plan and have confirmation of the treatments carried out by the patient and to know the possible problems associated with the history of heart attack. • The intraoral examination, hygiene instructions and dental impressions can be carried out without any particular protocol. • Non-surgical cases, as well as simple surgical procedures (simple extractions, curettage, etc.) can be performed after consulting the attending physician. In the cases of procedures respecting the general precautions described above. • For certain patients it is preferable to defer the most aggressive surgical procedures (multiple extractions, surgeries that require a flap for access, extraction of impacted teeth) and perform them 12 months after the infarction. Patients who have had a heart attack for more than a year. • Non-surgical procedures and simple procedures (simple extractions and curettages) can be performed. • The most complex procedures will not be performed before consulting with the treating physician. The procedures will be performed in an all hospital environment especially if a general anesthesia is provided. • If during treatment the patient with a history of coronary disease fatigues or presents changes in pulse levels (frequency and rhythm), the procedure will be immediately interrupted

and the patient will be placed under monitoring. Heart attack in the dental chair. • In the event of myocardial infarction at the time of treatment, an emergency medical team should be called. • The vital functions will be monitored and an oxygen administration will be practiced. • In case of cardiac and/or ventilatory arrest, cardiac massage and artificial ventilation will be put into practice. HEART FAILURE Heart failure is a complex and heterogeneous syndrome that occurs as a consequence of impaired ventricular relaxation or contraction, which disables this organ to meet the metabolic and oxygen demand in the rest of the organs, and is associated with symptoms and typical signs such as dyspnea, fatigue, and pulmonary and peripheral edema. It is responsible for a large burden of morbidity and mortality and a growing public health problem throughout the world. Surgical and dental procedures can aggravate pre-existing heart failure. These patients should be considered as risk patients due to the severe medical complications that can manifest in the heart such as infections, cardiac arrests, excessive bleeding, cerebrovascular accidents and myocardial infarctions. The dentist plays an essential role in the detection of these patients. Most use medication intended for their treatment which can interfere with dental treatments. Etiology Heart failure has multiple etiologies, they are related to a decrease in myocardial contractile activity prior to ischemic heart disease, myocardial infarction, chronic hypertension. followed by a myocardial infarction. Symptoms and signs Heart failure can be ongoing (chronic) or it can start suddenly (acute). Some of the signs and symptoms of heart failure, although they are not specific, can be:

Difficulty breathing (exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea, or dyspnea at rest)

- Fluid retention and edema
- Abdominal pain due to liver congestion
- Normal vital signs, but presence of tachycardia, hypotension, and reduced pulse pressure.
- Jugular venous distension (used to assess right atrial pressure)
- Cold extremities and cyanosis.
- Wheezing, rales, or snoring on chest auscultation
- Additional heart sounds (third heart sound or gallop rhythm)

Diagnosis A patient presenting for dental treatment with symptoms of dyspnea, fatigue, and edema should increase the index of suspicion for the presence of heart failure and immediately consult the patient's physician, who will perform a clinical history and physical examination (vital signs, cardiac and pulmonary auscultation), depending on the signs and symptoms, an electrocardiographic evaluation, chest x-ray, echocardiography, ventriculography and cardiac catheterization help the diagnosis, all of these carried out by a specialized doctor. Orofacial manifestations: distention of the external jugular veins and coagulation and bleeding problems Treatment The treatment of heart failure is managed according to etiology and its symptoms. Medical treatment It is beyond the scope of this chapter to discuss in detail the evidence for the various medical and surgical treatments for heart failure. However, the dentist must be aware of the range of treatment options and the drugs used. Figure 1 illustrates the new classification and staging system and the appropriate therapies for each stage of heart failure. Precautions in dental treatment Signs and symptoms consistent with heart failure should be further investigated and consultation with a physician or cardiologist should be sought. Dental treatment for asymptomatic patients can be performed unless other correlated diseases such as hypertension or diabetes are poorly controlled. Drugs used by patients with heart failure may be associated with certain dentally important side effects such as xerostomia, lichenoid reaction, and orthostatic hypotension.

The patient must be placed in a semi-supine position in a chair, with control of body movements (which must be slow), to avoid orthostatic hypotension. In case of emergency and after contacting the emergency department, the patient should be placed in a sitting position with their legs lowered, and receiving nasal oxygen at a rate of 4-6 liters/minute. Sublingual nitroglycerin tablets (0.4-0.8 mg) are indicated, and the dose can be repeated every 5 or 10 minutes if blood pressure is maintained1. For symptomatic patients with moderate exertion, symptomatic patients with mild exertion, and symptomatic patients at rest, the dentist should consult with a cardiologist who can perform diagnostic testing and evaluation, medication review, evaluation for cardiac resynchronization therapy or an implantable defibrillator, and In general, improve the patient's condition as much as possible before dental treatment. Left ventricular ejection fraction provides a quantitative marker of systolic ventricular dysfunction. It has been reported that patients undergoing surgery with an ejection fraction >55% had a low risk of myocardial complications in the postoperative period, while those with an ejection fraction >35% had a 75% incidence of postoperative myocardial infarction. However, others report that there is no such correlation between ejection fraction and postoperative cardiac complications. Dental treatment of a patient with malcompensated heart failure can be complicated by difficulty breathing and precipitation of pulmonary edema when the patient is placed in the supine position. Any history of dyspnea on exertion, orthopnea, or paroxysmal nocturnal dyspnea provides a useful indicator of this and should alert the dentist. If patients in the asymptomatic or moderately symptomatic stages of heart failure have been medically optimized, dental treatment can proceed relatively unimpeded.

Those with active and advanced-stage symptoms should be managed with caution in a hospital setting. Precautions against stress Controlling anxiety and reducing stress should be one of the dentist's concerns. In a large proportion of patients, anxiety can be reduced by sedative premedication such as benzodiazepines which, with their limited depressant effect on the cardiovascular system, are a means of oral pharmacological choice. Intravenous sedation or procedures with general anesthesia must be performed in a hospital setting. The procedures, as far as possible, should be of short

duration and preferably performed in the morning. anesthetic precautions Local anesthesia with epinephrine can be used, although any intravascular injection that produces tachycardia can cause decompensation and acute pulmonary edema with sudden onset of shortness of breath. It is recommended to restrict the total dose of epinephrine to a maximum of 0.04 mg (two standard cartridges with 1:100,000 epinephrine) in patients with ischemic heart disease. Precautions against haemostasis and coagulation problems.

These precautions apply to patients with heart failure taking aspirin or other anticoagulant drugs. In general, evidence-based guidelines published by the American Academy of Neurology recommend that antithrombotic medication remain unchanged in patients with ischemic cerebrovascular disease. Individual patient characteristics are an important consideration in assessing risk and benefit. Patients with ischemic heart disease who did not undergo coronary stenting belong to the low-risk group, while patients with concomitant prosthetic heart valves and atrial fibrillation are considered to be at high risk. Patients undergoing coronary artery stenting should continue their antiplatelet prescription for up to 12 months. General principles of dental management for patients taking antithrombotic agents recommend following standard procedures to avoid causing bleeding. If the antithrombotic medication is not prescribed as a lifelong medication, dental treatment may be delayed until after the medication is stopped. For patients with metal prosthetic heart valves or coronary stents, direct written instructions from the cardiologist are required to modify their antithrombotic medications. Analgesic medications such as aspirin, ibuprofen, and diclofenac can exacerbate the patient's risk of bleeding and should be prescribed carefully. The most useful hemostasis is local mechanical arrest, such as tamponade of open alveoli with hemostatic material and suture. There is no reason to discontinue antithrombotic medication if the patient is taking aspirin alone, because there is no evidence of a higher risk of bleeding in these patients than in non-antithrombotic patients.

Scheduling elective procedures early in the day and early in the week is recommended for patients taking antithrombotic agents to allow time for bleeding control, in case of bleeding episodes. If the patient is only taking aspirin, it is recommended to limit the initial treatment site, ie extract only one tooth, or limit subgingival scaling to three teeth, so that bleeding can be assessed before further procedures. For procedures with more anticipated bleeding complications, staged treatment with active involvement of local hemostatic measures such as suturing and tamponade should be considered. Currently, the most widely used dual antiplatelet drug is the combination of aspirin with clopidogrel. In such a case, hemostasis may be delayed by up to one hour, which will justify limiting the initial treatment site, for example, a single extraction or limiting subgingival scaling to three teeth before subsequent procedures. For procedures with an increased risk of postoperative bleeding, staged treatments at separate visits and the active involvement of local hemostatic measures are recommended. The key recommendation for a patient on warfarin is no disruption of vitamin K antagonist medication with an INR below 4 If the patient's INR is stable, INR verification within 72 hours prior to dental procedure is acceptable, while the INR should be checked no more than 24 hours before when the INR is unstable. It should be noted that stable INR means that INR measurements have not been above 4 in the last two months. If the INR is greater than 4, the dental procedure should be delayed until corrected by anticoagulation management by medical personnel. In addition, initial treatment site limitation or staging treatment at separate visits should be considered for procedures with increased risk of bleeding. Infectious risks caution In patients with an underlying valvular condition, antibiotic prophylaxis should be administered when the actions to be performed tend to cause a bacterial infection. Cross infection by the dentist should be minimized. CARDIAC ARRHYTHMIA The normal heart beats in a regular and coordinated manner thanks to the transmission of electrical impulses generated by and scattered through cardiomyocytes (which have unique electrical properties), triggering a sequence of organized myocardial contractions. A defined cardiac arrhythmia is simply a variation from the normal heart rate and/or rhythm that is not physiologically justified.16 pathophysiology.

Rhythm disturbances are the result of disturbances in impulse formation or conduction, or both steps. Bradycardias are due to decreased intrinsic pacemaker function or conduction block, primarily within the AV node or His-Purkinje system. Most of the tachycardias are caused by reentry mechanisms, some are the result of an increase in normal automatism or abnormal mechanisms in the generation of automatism. Signs and symptoms Arrhythmias and cardiac conduction disorders may be asymptomatic or cause palpitations (feeling of missing or increased beats), symptoms of hemodynamic compromise (eg, dyspnea, chest discomfort, near syncope, syncope), or cardiac arrest. Occasionally, the patient experiences polyuria due to secretion of atrial natriuretic peptide during prolonged supraventricular tachycardia. Pulse palpation and cardiac auscultation can determine the ventricular rate and its regularity or irregularity. Examination of the jugular vein pulse waves could aid in the diagnosis of AV blocks and tachycardias.

For example, in the presence of complete AV block, the atria contract intermittently as the AV valves close, generating large a (gunshot) waves in the jugular vein pulse. There are few other physical findings of arrhythmias. oral manifestations The first stage of management of patients with cardiac arrhythmia is the collection of anamnesis. According to Vitalievna (2021). The following oral effects have been reported: hyperemia of the oral mucosa (36.2%), discomfort in the oral cavity (29.5%), burning sensation in the oral mucosa (14.3%), pain in the oral mucosa (22.9%), swelling of the tongue (12.4%), bite of the mucous membrane of the cheeks and/or tongue (16.2%) DENTAL MANAGEMENT Many antiarrhythmic drugs have side effects such as gingival hyperplasia or xerostomia.

Short visits in the morning are preferable. Patient monitoring,

with pulse recording, is indicated before starting treatment. It is very important to limit the use of vasoconstrictor in local anesthesia, with the administration of no more than two capsules. The planned treatment should not be too long or complicated. According to Becker (2009) although modern pacemakers are more resistant to electromagnetic interference, caution is required when using electrical devices (for example, ultrasound and electric scalpels) that may interfere with pacemakers, particularly older models, since the Most of these devices were developed in recent years. 30 years are bipolar and are generally not affected by the small electromagnetic fields generated by dental equipment. If arrhythmia develops during dental treatment, the procedure should be discontinued, oxygen should be provided, and the patient's vital signs should be assessed. The patient should be placed in the Trendelenburg position, with vagal maneuvers when necessary (Valsalva maneuver, massage in the region of the carotid pulse, etc.). Patients with arrhythmias can be managed with electronic devices that emit electrical signals (cardiac pacemakers and implantable cardioverter-defibrillators). These devices have been shown to be sensitive to electromagnetic signals produced by dental instruments such as electrosurgical unit, electric pulp tester, electronic apex locator, etc. Although the newer ones (electromagnetic shielded bipolar devices) are generally unaffected by the small electromagnetic fields generated by dental equipment, one should be aware.

Caution when operating ultrasonic scalers, ultrasonic cleaning systems, curing lights in close proximity to persons who have pacemakers or implantable cardioverter defibrillators. BRADYCARDIA Bradycardia is a slower than normal heart rate. The heart of adults at rest usually beats between 60 and 100 times per minute. If you have bradycardia, your heart beats less than 60 times per minute. Bradycardia can be a serious problem if the heart rate is very slow and the heart cannot pump enough oxygen-rich blood to the body. If this happens, you may feel dizzy, very tired or weak, and short of breath. Sometimes bradycardia does not cause symptoms or complications. A slow heartbeat doesn't always cause concern.

For example, a resting heart rate of between 40 and 60 beats per minute is quite common during sleep and in some people, particularly healthy young adults and trained athletes. If the bradycardia is severe, a pacemaker may need to be implanted to help the heart maintain a proper rhythm, to better understand the causes of bradycardia, it can help to know how your heart normally beats. The normal heart has four chambers: two upper chambers (atria) and two lower chambers (ventricles). Inside the upper right chamber of the heart (right atrium), there is a group of cells called the "sinus node." The sinus node is the heart's natural pacemaker. It produces the signal that starts each heartbeat. Bradycardia occurs when these signals are delayed or blocked. The causes of bradycardia can be the following

- Damage to heart tissue related to aging
- Damage to heart tissue from heart disease or heart attack.
- Heart disorder present at birth (congenital heart defect)
- Inflammation of the heart tissue (myocarditis)
- A complication after heart surgery
- An underactive thyroid gland (hypothyroidism)
- Imbalance of chemicals in the blood, such as potassium or calcium
- Repeated pauses in breathing during sleep (obstructive sleep apnea)
- Inflammatory disease, such as rheumatic fever or lupus,
- Medications, including sedatives, opioids, and drugs used to treat heart rhythm disorders, high blood pressure, and certain mental health disorders.

Clinical manifestations

Symptoms Bradycardia can be asymptomatic. Heartbeats that are slower than normal (bradycardia) can cause the brain and other organs to not get enough oxygen, which can cause the following signs and symptoms

- Chest pain
- Confusion or memory problems
- Dizziness or lightheadedness
- Tired quickly during physical activity
- Fatigue
- Fainting (syncope) or near fainting
- Shortness of breath Oral manifestations

The oral manifestations result from the effects of antiarrhythmic drugs that may be associated with the use of pacemakers.

Treatment

In general, asymptomatic bradycardia does not require treatment. this is the case of a young and active patient. Patients who present symptoms do require treatment. Bradycardia treatment includes lifestyle changes, medication changes, or an implanted device called a pacemaker. If an underlying health problem (such as thyroid disease or sleep apnea) is causing the slower-than-usual heart rate, treating that condition may correct the bradycardia.

General risk factors

Bradycardia is generally associated with damage to heart tissue caused by some type of heart disease. Anything that increases the risk of heart problems can increase the risk of bradycardia. Risk factors for heart disease include:

- Advanced age
- High blood pressure
- Smoke
- Excessive alcohol consumption
- Consumption of illicit drugs
- Stress and anxiety Lifestyle changes or medical treatment can help lower your risk of heart disease. The stress developed during dental treatment also results in cardiovascular changes, possibly the most common factor in arrhythmia. Roman., *et al*. (2009) observed various electrocardiographic changes

during dental implant surgeries under local anesthesia in healthy patients. There was tachycardia during anesthesia, incision and, above all, bone perforation.

Risk factors in patients with pacemakers

When other treatments are not possible and the symptoms of bradycardia are severe, it is necessary to use a device called a pacemaker to control the heart's rhythm. Pacemakers are cardiovascular implantable devices (CIEDs), including implantable cardiac pacemakers, implantable cardioverter defibrillators (ICDs), cardiac resynchronization devices, and implantable cardiac monitors. Pacemakers work only when needed. When the heart beats too slowly, the pacemaker sends electrical signals to the heart to speed up the heartbeat. It is important to note that most patients with pacemakers generally fall into a higher risk stratification in relation to physical status.

As with any proposed procedure/surgery and anesthetic plan, a detailed and focused physical examination and medical history should be performed. For those patients with CIED, special attention should be paid to obtaining a complete cardiovascular history with full details on historical events and/or surgical interventions. Activity tolerance should be documented. Depending on the nature of the procedure and the skill level of the sedation/anesthesia provider, the patient's physical condition may simply preclude in-office treatment due to complexity, resources, and expert consultation recommendations. electrocautery Extensive studies investigating the use of monopolar cautery for electrosurgery have shown a low risk of initiating harmful electromagnetic interference for pacemakers. However, most advise that bipolar electrosurgery be used instead of monopolar devices, and that electrosurgery dispersion electrode pads (grounding pads) be placed as far away from the pacemaker as possible. There are specific recommendations for each pacemaker model and manufacturer, and similarly, relevant information on the type of electrosurgical device should be investigated in the preoperative phase.

Common dental devices and equipment Electronic and piezoelectric dental scalers, apex locators, light-curing units, except electrosurgery, produce minimal electromagnetic interference in dental practice. Take care to keep potential sources of electromagnetic interference as far away from the pacemaker device as possible. Radiotherapy Exposure to 10 Gy ionizing radiation can damage the random access memory of pacemakers used in programming and interrogation.

Digital images with regard to typical dental diagnostic radiographs, clinicians are also urged to consult with the treating cardiologist and verify the directives and precautions with patients presenting with the pacemaker patient.

Dental radiographs, and digital dental radiography specifically, result in ionizing radiation exposure orders of magnitude less than that seen in radiation oncology. Of the relatively few diagnostic and imaging precautions related to pacemakers, MRI is contraindicated in patients with non-MRI-compatible pacemakers. The high-frequency pacemaker has been triggered by exposure to the MRI field leading to deaths. anesthesia A thorough preoperative evaluation and consultation should be considered for any patient with a pacemaker. Commonly used vasoactive drugs can have considerable effects on pacemaker sensing and function without prior modification of anesthetic techniques.

Consider treating the patient in a hospital setting or deferring treatment if the possibility of cardiac events or irregular pacemaker activity is anticipated.

Rheumatic valve diseases

Rheumatic heart disease and valvular involvement is the result of an inadequate immune response to Group A streptococcal infections, especially during childhood. Up to 65% of hospitalizations for cerebrovascular events are due to a valvular cause and up to 10% of hospital discharges in developed countries are due to a valvular condition. It is estimated that, worldwide, between 15 and 20 million people suffer from rheumatic heart disease, and each year this figure grows with approximately 300,000 new cases. These figures worldwide are highly influenced by elements such as human overcrowding, access to health resources, climate, the availability of programs for the prevention, detection and treatment of bacterial pharyngoamigdalitis and an element that is added particularly by the rise of the end-stage chronic kidney disease, detection and treatment of bacterial endocarditis, mainly in acute valvular disorders. Mention should also be made of congenital heart disease which, during adult life, can also develop a valvular condition and the cases increase, and with them all the elements as previously reviewed. General Risk Factors As already mentioned, the prevalence and etiology of valve disease is influenced in the first place by the level of development of the country. Degenerative causes are the first cause in first world countries, while rheumatic fever is the main element involved in developing countries.

Likewise, risk factors have a lot to do with it; while age is the main factor for degenerative causes, in underdeveloped countries there are multiple elements that together lead people to valve disease; elements that include both socioeconomic and environmental aspects such as malnutrition, overcrowding and the level of that, interacting with each other, affect the magnitude and severity of rheumatic fever and rheumatic heart disease, mainly due to constant exposure to group A streptococcus strains Favored by unhygienic conditions and malnutrition, which favors high prevalence of rheumatic heart disease.

Pathophysiology

In general, it is accepted that an inadequate response of humoral and cellular components of the host to group A Streptococcus exposure, generally in upper respiratory tract or skin infections, leads to the subsequent development of complications, rheumatic heart disease is the most serious complication of rheumatic fever,13 developing 4 to 8 weeks after initial exposure in 30 to 45% of patients. The streptococcal bacterium contains a protein similar to that found in certain tissues of the body. The body's immune system, which normally targets infection-causing bacteria, attacks its own tissues, particularly the tissues of the heart, joints, skin, and central nervous system. This immune system reaction causes tissue swelling (inflammation). Factors related to the microorganism It is currently known that any strain of group A strectococcus can cause this entity and even those of group C and G.

Host-related factors the characteristics of the host regarding the susceptibility to the development of rheumatic heart disease have a high hereditary component, with a concordance of up to 44% in monozygotic twins, in most cases related to immunological elements and type II human leukocyte antigen (HLA II).) and others related to tumor necrosis factor α (TNF-α). Factors Related to the Immune Response It is in these factors where the concept of molecular mimicry has its implications, in which the immune response normally directed against streptococcal antigens is also recognized in human tissues by similarities in protein composition. Cross-reacting antibodies bind to endothelial cells in the heart valve, activating adhesion molecules VCAM-1 thereby attracting activated lymphocytes and lysing endothelial cells in the presence of complement, releasing peptides which in turn activate reacting T lymphocytes crossed and invade the heart and amplify the damage. Signs and symptoms the symptoms of rheumatic fever vary. You may have few or many symptoms, and the symptoms may change during the course of the disease. The onset of rheumatic fever usually occurs about two to four weeks after a strep throat infection. Caused by inflammation of the heart, joints, skin, or central nervous system, signs and symptoms of rheumatic fever may include

- Fever
- Painful and tender joints, most often the knees, ankles, elbows, and wrists
- Pain in one joint that migrates to another joint
- Red, hot, or swollen joints
- Small, painless bumps under the skin

Chest pain

- Heart murmur
- Fatigue
- Flat or slightly raised, painless skin rash with an irregular border
- Jerky, uncontrollable body movements (Sydenham's chorea), most often in the hands, feet, and face
- Outbursts of unusual behavior, such as inappropriate crying or laughing, that accompany Sydenham's chorea Diagnosis For a comprehensive approach to valvulopathies, the European Society of Cardiology suggests the organization of a comprehensive clinic (20) in which the evaluation of patients, like any other disease, includes an important order 16 that begins with an exhaustive examination. medical history and personal history, physical examination and complementary tests, both invasive and non-invasive. In imaging studies, the echocardiogram cannot be missing as the first and most feasible option to establish the etiology, morphology and cardiac function. It can be complemented with imaging studies such as cardiac resonance and computed tomography. Within the non-invasive studies, the stress test allows evaluating the patient's capacity, ventricular function and the appearance of signs or symptoms during the effort. Special caution should be taken in patients with concomitant coronary artery disease due to the risk of low coronary flow during exertion and that may cause added ischemic events.

Non-rheumatic valve diseases

Valvular heart conditions, may be of acquired or congenital origin, are clinically manifested by respirations that result in valve opening and closing problems. Valvular affections are of two types: stenotic and regurditive. Stenotic Valve Condition: Aortic Stenosis. Origin It can be of degenerative origin (senile calcified aortic stenosis), congenital (congenital aortic stenosis) or rheumatic. pathophysiology Pathophysiologically, aortic valve stenosis produces a pressure overload in the left ventricle due to the increased pressure that must be generated to force blood through the stenotic valve. This pressure causes compensatory concentric hypertrophy of the left ventricle. Clinical manifestations

- Clinically, angina (due to limited oxygen supply due to impaired coronary blood flow reserve), syncope, and heart failure are the usual symptoms.
- The following clinical signs are observed: hard and harsh systolic ejection murmur, reduction or abolition of B2. Diagnosis The diagnosis is based on the electrocardiogram (left ventricular hypertrophy), in the presence of calcium objectified by fluoroscopy. in Doppler echocardiography and possibly cardiac catheterization. Treatment Treatment is palliative, by prescription of digitalis, diuretics, or curative by aortic valve replacement.

Stenotic valvar conditions: mitral stenosis

Origin In adults, mitral stenosis is secondary to cardiac rheumatism. Most mitral stenosis is found in women. pathophysiology from a pathophysiological point of view, mitral valve stenosis interferes with left ventricular filling. An increase in pressure appears in the left atrium, it is transmitted to the lung, and pulmonary venous congestion occurs.

Clinical manifestations

- **Clinically:** exertional dyspnea, orthopnea, and left-sided deficiency paroxysmal nocturnal dyspnea develop. In the presence of pulmonary hypertension, the right ventricle may become deficient, producing edema, ascites, and fatigue. Elevated pressure at the level of the left atrium may be at the origin of anastomosis ruptures between the bronchial and pulmonary systems at the origin of hemoptysis. Finally, a systemic embolism can occur.
- **The physical signs are the following:** arrhythmia, pulmonary rales, burst of B1, splitting of B2, with mitral opening click, diastolic rocking of variable intensity with presystolic reinforcement.

Diagnosis

The diagnosis is based on the electrocardiogram. chest x-ray. Doppler echocardiography and cardiac catheterization. Treatment the treatment is: - or doctor, then it is reserved for stricture patients with mild to moderate symptoms. It is based on the prescription of diuretics to control pulmonary congestion and limit dyspnea and orthopnea; digitalis in patients with atrial fibrillation and anticoagulants to reduce the incidence of systemic embolism in patients with coexistent mitral stenosis and atrial fibrillation; - or curative, by percutaneous or surgical mitral commissurotomy or mitral valve replacement. Valvular Regurgitation: Aortic Regurgitation Origin Hypertension may be the cause of dilatation of the proximal root of the aorta. Infectious endocarditis. Marfan syndrome. aortic dissection, syphilis, and collagen pathologies (spondylitis and lupus erythematosus) can also cause aortic regurgitation.

Pathophysiology

From a pathophysiological point of view, part of the left ventricular blood volume ejected during systole is regurgitated during diastole. Clinical manifestations.

Clinical symptoms are dyspnea, orthopnea, and paroxysmal nocturnal dyspnea (resulting from left ventricular deficiency), syncope due to impaired cerebral perfusion following a reduction in systemic diastolic blood pressure, and angina due in part to decreased circulation of the left ventricle. coronary blood flow. The hyperdynamism of the maximum shock and the peripheral vessels and the diastolic murmur represent the main signs. Diagnosis Diagnosis, again, is based on electrocardiographic investigations (left ventricular hypertrophy), radiographs (dilatation of the proximal aorta, cardiac enlargement), echocardiography, and aortography during cardiac catheterization. Treatment If aortic regurgitation is severe, aortic valve replacement is usually required. However, the injury can be tolerated for several years, making surgical planning difficult. Careful monitoring is necessary to detect early signs of decompensation. In the presence of such signs, it is necessary to replace the valve. If surgery is not considered, symptomatic medical treatment based on digitalis, diuretics and vasodilators are placed.

Valvular regurgitation

Mitral Regurgitation Origin Mitral valve prolapse, rheumatic heart disease, coronary artery disease, and infective endocarditis are the main causes of mitral regurgitation.

- Surgery is recommended in chronic forms even in the presence of mild symptoms. Valvular Regurgitation Tricuspid Regurgitation Origin Endocarditis is a common cause of tricuspid regurgitation. Other causes are: right ventricular deficiency. cardiac rheumatism and right coronary artery occlusion with ventricular infarction. pathophysiology Pathophysiologically, tricuspid valve dysfunction during systole causes blood to regurgitate into the right atrium, causing systemic venous congestion and venous hypertension. Clinical manifestations
- The symptoms are those of the right deficiency (edema and ascites). In severe and acute cases, liver congestion is the cause of right upper quadrant pain.
- **Physical signs are:** holosystolic murmur, jugular pulsation during systole, and signs of right heart failure. Diagnosis Diagnosis is based on Doppler echocardiography. Treatment Effective treatment of left deficiency results in a reduction in right ventricular pressure. This reduction may be appropriate to reduce the size of the right ventricle and thus restore valvular competence. If the tricuspid regurgitation is due to organic valve disease, surgery may be required.

Oral manifestations

Oral manifestations associated with valvular disease are related, depending on the type of disease, with hypoxemia and compensatory polycythemia. In addition, in the case of congenital valve diseases, malformations are observed.

- Hypoxemia is the cause of cyanosis, which is manifested, among other things, by a particularly visible bluish discoloration of the buccal mucosa, lips, and earlobes. In the presence of compensatory polycythemia, the patient's face appears reddish.
- The malformations are fundamentally cleft lip, abnormal growth and eruption of the first dentition. Valuation in Dental Practice
- As part of oral care and due to the risk of infective endocarditis in patients with valvular disease, it is essential to question the patient to look for such pathology. In case of suspicion on the part of the doctor or uncertainty in the patient's words, an evaluation or re-evaluation will be required by the doctor. This evaluation also aims to specify the presence of associated pathologies (following associated valve disease). The nature of the treatments carried out (surgical: past or planned; medical: nature of the medications and doses) and the current stability of the patient.
- According to the classification developed by the American Society of Anesthesiologists (ASA), patients with valvular disease (including patients with prosthetic valves) belong to class III/IV. Remember that patients classified as ASA III

are considered to have a serious systemic condition that requires precautions during care, minimal exposure to stress, and medical consultation. Patients classified as ASA IV are considered to have a debilitating, life-threatening systemic condition. They are patients for whom a consultation is required and in whom the treatment, which requires taking strict precautions, must be carried out in a hospital environment.

Precautions to take in dental practice

- The physician's primary concern is to prevent infective endocarditis in patients with valvular heart disease. However, there are other precautions. general and specific to consider.
- In all cases, no surgical or non-surgical procedure will be considered in the symptomatic patient without prior consultation with the treating physician. Consultation and medical information
- A consultation will be requested: - in the presence of signs or symptoms suggesting that the patient has a valvular disorder; - when still under treatment, the patient is symptomatic.
- The treating physician will be consulted: -in case of doubts about the state of health of the patient or in the diagnosed patient to know precisely the state of it (the health and nature of the treatment followed (prescriptions and dosages): - define, in accordance with the intended treatment, any modification relating to the treatment; - when other pathologies are present and/or when the patient is polymedicated. stress precautions.
- Due to the manifestations associated with valvular disease, anxiety control and stress reduction should be part of the physician's precautions.
- As with all cardiovascular conditions, anxiety can be reduced by effective sedation, which may include benzodiazepine premedication. However, sedation by inhalation of nitrous oxide remains the best means of intraoperative anxiolysis. If necessary, intravenous sedation will be performed. Once the type of sedation is chosen, it is obviously based on the general state of the patient and the presence or absence of other systemic conditions. Precautions to take during anesthesia.
- Depending on the nature of the associated conditions (heart failure, hypertension, etc.), it is the rule to administer reduced levels of vasoconstrictors. The dose should generally be limited to 0.04 mg epinephrine and the slow injection should be given after aspiration.Patient Treatment Precautions.
- The precautions to be taken (regarding contraindications, side effects, toxicity, etc.) depend on the drugs administered to the patient: diuretics and antiarrhythmics in case of aortic stenosis; diuretics, digitalis and anticoagulants in cases of mitral and digital stenosis, diuretics and vasodilators in case of aortic or mitral regurgitation.
- In addition, certain medications, due to their side effects, may interfere with care. For example, vasodilators can cause headaches and/or tachycardia; Digitalis can cause poisoning.
- In all cases, if changes in treatment should be considered, they will be agreed with the treating physician.
- Precautions should also be taken in patients who have undergone surgical correction or prosthetic valve placement. Precautions to be taken in this context are presented in chapters 12 and 13. Haemostasis and coagulation disorder precautions
- These precautions refer to patients receiving treatment with vitamins K, acetylsalicylic acid or ticlopidine in case of mitral stenosis. The procedures to follow in relation to the performance of surgical procedures in anticoagulant patients are established in the context of disorders of haemostasis and coagulation (see chapter disorders of haemostasis). If procedures are more substantial or bleeding is unpredictable, the patient will be referred for possible dosage modification. In fact, depending on the case: - nature of the acts to be carried out and precise indication of the prescription - anticoagulants will be maintained, reduced or relieving with heparin.

The decision is the responsibility of the patient's treating physician (general physician or hematologist) who will define, in accordance with the information provided by the dental surgeon on the nature of the intervention, the procedures to be followed, including the possible performance of procedures in a hospital setting. In patients taking acetylsalicylic acid or ticlopidine, bleeding time should be monitored. If it is extended, the procedure to be followed is the one presented in chapter 25.

- In all cases, if changes in treatment should be considered, they will be agreed with the treating physician. Precautions against risk of infection
- Due to the risk of endocarditis associated with heart valve disease, all the acts at the origin of a bacteraemia will be carried out under prophylaxis anti-infective The methods (choice of anti-infective agent, methods administration. dosage, etc.) of this prophylaxis are the prevention of infective endocarditis. They are presented in Chapter 12.
- Note that in patients who are on long-term anti-infective treatment. the choice of prophylactic prescription should be for a different antibiotic than the one that is the subject or that has been prescribed. For example. if it is a penicillin, the antibiotic that will then be prescribed for prevention will be pristinamycin or clindamycin which are also listed as the antibiotic of choice for endocarditis prophylaxis.
- Patients who have undergone surgical correction are also exposed during the healing phase. The absence of a cardiac prosthesis appears to reduce this risk. The precautions to be taken and the procedures to be followed in these patients undergoing surgical correction are presented in chapter 13.

- On the other hand, universal hygiene and asepsis measures must be observed to minimize the risk of cross-transmission of bacterial and/or viral infectious diseases. Precautions to take into account when prescribing
- The prescription of paracetamol or any other analgesic, as well as non-steroidal anti-inflammatory drugs does not present a contraindication except in anticoagulant patients in whom NSAIDs should be avoided.
- Because of its cardiodepressant effects. Barbiturates and narcotics should be avoided. Precautions regarding concomitant pathologies and/or associated complications
- The presence of pathologies and/or complications, associated with valvulopathies or of any other origin, requires additional measures to be taken in addition to the precautions inherent to these pathologies and/or complications, as well as against the prescriptions that are part of their treatments. Precautions to take in the context of urgent care
- As with elective care, the same procedures must be followed for urgent care.
- If, in the context of a real emergency that requires care treatment at the origin of the bacteraemia, a medical consultation cannot be obtained and there is doubt about the presence of valve disease, the patient will be considered to have congenital heart disease. Next, anti-infectious prophylaxis will be carried out. heart murmurs Breathing is a non-generic given to all the sounds that are produced at the level of the cardiocirculatory system. Here only heart murmurs will be considered. Heart murmurs are vibrations with a duration longer than the sound classically emitted by cardiac function. To facilitate dialogue with the treating physician and guide whether or not it is appropriate (the precautions to take, some reminders about murmurs are presented here). General Reminders Murmurs result from turbulence of blood flow. In general, these phenomena are caused by an increase in flow, a change in stickiness, or abnormal valves (as a result of congenital or acquired heart defects).

Murmur classification

According to its appearance during the cardiac cycle in systolic murmur. diastolic or continuous: - depending on where they are most noticeable: - depending on whether they are localized or irradiating: - according to its intensity, for which different degrees are defined.

- Grade 1: barely perceptible breathing.
- Grade 2: Explosion of intermediate intensity.
- Grade 3: Loud murmurs without tremors.
- Grade 4: strong murmurs with tremors.
- Grade 5: Very loud murmur but audible with a stethoscope.
- Grade 6: audible murmur without a stethoscope.

Clinical manifestations Murmurs can be: - functional (innocent, physiological, inorganic or benign), when they occur in the absence of pathology; - organic (or pathological), when they result from a cardiac anomaly. functional murmurs

- These are temporary murmurs, most commonly heard during systole.
- They are common in children. during pregnancy, in the presence of anemia or febrile episodes. In children, they probably result from an amplification linked to the combination of increased blood flow and a thorax whose thickness is still limited. In pregnant women, the presence of a murmur results from an increase in blood flow. They disappear very quickly after delivery. organic puffs
- They are permanent murmurs. more frequent during diastole. Diastolic murmurs are always organic.
- Are caused by a congenital heart defect, valve disease, infection (infective endocarditis), or autoimmune disease (lupus).
- They are of regurgitant and/or stenotic origin. The valves on the left side of the heart are the most frequently involved.
- Stroke murmurs are generated by abnormalities within the aortic or pulmonary structures, or by increased flow within them. Mitral regurgitation due to mitral valve prolapse may be associated with a late systolic murmur that is often preceded by a systolic click.
- Diastolic murmurs are heard in aortic or pulmonary regurgitation. Aortic regurgitation may be due to valvular leakage or be secondary to valve annulus dilation (after aortic dissection, for example). Pulmonary regurgitation may be valvular or secondary to valvular annular dilatation. •A diastolic thud is heard when there is narrowing of the mitral or tricuspid valve.
- Continuous murmurs can be caused by arteriovenous fistulas or a patent ductus arteriosus. Some metal prosthetic valves are the source of loud opening and closing noises that can be heard without a stethoscope. Pig valves can be completely inaudible. As long as there is a persistent gradient across any prosthetic valve, a systolic murmur is heard across the prosthetic aortic valves. Diagnosis Murmurs are detected by auscultation with a stethoscope. A physical exam. associated with an electrocardiogram, echocardiography, X-rays and blood tests will help determine the functional or organic origin of the murmur, revealed by cardiac auscultation. Treatment
- Functional murmurs do not require treatment.
- On the other hand, in the presence of organic explosions. medical attention and monitoring is required. They depend on the origin and nature of the symptoms. The treatment generally consists of the restriction of physical activity and the prescription of medications (digitalis, vasodilators, etc.). In the presence of heart failure or embolism, surgical correction (commissurotomy, placement of prosthetic valves, etc.) will be considered. oral manifestations the oral manifestations depend on the origin of the breath. In the presence of valvular involvement that compromises cardiac function, labial and mucosal cyanosis, evidencing hypoxia.

Valuation in dental practice

- In the context of oral care and due to the risk of infective endocarditis in patients with organic murmur. any suspicion and/or history of murmur requires evaluation. In fact, to prevent endocarditis of infectious origin. it is essential to define the nature of a breath that is either suspected or reported by the patient.
- The fact that the dental surgeon is not able to detect and evaluate heart murmurs. It will be necessary to contact the treating physician. This approach will be applied both in case of suspicion (including functional murmur) only when the patient declares to have a murmur or when he provides a history.
- In addition, this assessment will be based on a medical examination aimed both at finding out the patient's medical problems (history of rheumatic fever, cardiac rheumatism, congenital heart disease, etc.) and the nature of the treatment followed (medications, dosage, etc.).
- According to the classification developed by the American Society of Anesthesiologists (ASA), patients with functional murmur belong to class I/II. Patients presenting with an organic murmur belong to class III/IV according to the nature of the associated pathology.
- Remember that patients classified as ASA I are subjects without systemic disease in whom no special precautions are required during treatment. Patients classified as ASA II are considered to have mild to moderate systemic disease with associated risk factors and are medically stable. These are patients whose care requires stress reduction and taking small precautions during care. Patients classified ASA III are considered to have a severe systemic condition that requires precautions during care, exposure minimal stress, as well as a medical consultation. Patients classified as ASA IV are considered to have a debilitating systemic condition that immobilizes them and represents a vital risk. These are patients in whom a medical consultation is necessary and in whom there is treatment. require the taking of strict precautions, they must be performed in a hospital setting. Precautions to Take into Account in Daily Practice
- Precautions are of interest to patients presenting with an organic murmur because if the murmur is functional, no change in the nature of care should be considered. Consultation and medical information
- A consultation will be requested: - in the presence of signs or symptoms that suggest that the patient has an organic murmur: - when still under treatment, the patient is symptomatic.
- The treating physician will be consulted - in case of doubt (s) about the nature of the breathing (suspected or evoked by the patient) or to know precisely in the diagnosed patient his state of health and the nature of the treatment followed (prescriptions and dosages).

To be defined, according to the planned treatment. any changes in processing: - when other pathologies are present and/or when the patient is polymedicated [1-33].

Conclusion

The main clinical characteristics were described, as well as the most frequent risks from the dental point of view. Patients with these diseases are mainly at risk of bleeding and infections.

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