Cardiovascular diseases in dental practice. Practical considerations

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Abstract
Coronary heart disease is the principal cause of death in the industrialized world. Its most serious expression, acute myocardial infarction, causes 7.2 million deaths each year worldwide, and it is estimated that 20% of all people will suffer heart failure in the course of their lifetime. The control of risk cardiovascular factors, including arterial hypertension, obesity and diabetes mellitus is the best way to prevent such diseases. The most frequent and serious cardiovascular emergencies that can manifest during dental treatment are chest pain (as a symptom of underlying disease) and acute lung edema. Due to the high prevalence and seriousness of these problems, the dental surgeon must be aware of them and should be able to act quickly and effectively in the case of an acute cardiovascular event. In patients with a history of cardiovascular disease, attention must center on the control of pain, the reduction of stress, and the use or avoidance of a vasoconstrictor in dental anesthesia. In turn, caution is required in relation to the antiplatelet, anticoagulant and antihypertensive medication typically used by such patients.

Key words: *Chest pain, acute myocardial infarction, angor, heart failure, dental treatment.*

Introduction
The motor of cardiac activity is the myocardium, composed of contractile striated muscle fibers that exert the pumping action of the heart. Anatomically, the latter comprises two atria and two ventricles. Physiologically, the heart can be divided into two halves: the right heart receives systemic venous blood from the upper and lower vena cavas, and redistributes it towards the pulmonary circuit through the pulmonary arteries. In turn, the left heart drains oxygenated arterial blood from the pulmonary veins and redirects it to the rest of the body (systemic circulation) through the aorta. This blood trajectory and distribution is possible thanks to the cardiac cycle. The epicardial and intramyocardial coronary arteries supply the myocardium with the necessary oxygen and nutrients. A reduction in blood flow through these arteries is responsible for ischemic heart disease (1)(Figure 1).

The electrical activity of the heart originates in the sinoatrial (SA) node, which is composed of numerous pacemaker cells. Conduction continues along the internodal interatrial bundles to the atrioventricular (AV) node, and then to the bundle of His where the conduction system ramifies towards both ventricles via the so-called Purkinje fibers. Each electrical impulse traveling this circuit generates one harmonious contraction of the heart. Under normal resting conditions, the number of cycles completed per minute is between 60-100. The electrical field generated by such conduction activity can be recorded by means of the electrocardiogram (ECG).

According to the World Health Organization (WHO), in the year 2003, cardiovascular diseases caused a total of 16.7 million deaths. Of these, 7.2 million corresponded to ischemic heart disease, 5.5 million to cerebrovascular accidents (stroke), and 3.9 million to other cardiovascular

disorders - fundamentally associated to arterial hypertension (2).

The present study provides a review of the two most frequent cardiovascular disorders - ischemic disease and heart failure - and discusses the approach to be adopted by the dental surgeon when dealing with patients that suffer such problems.

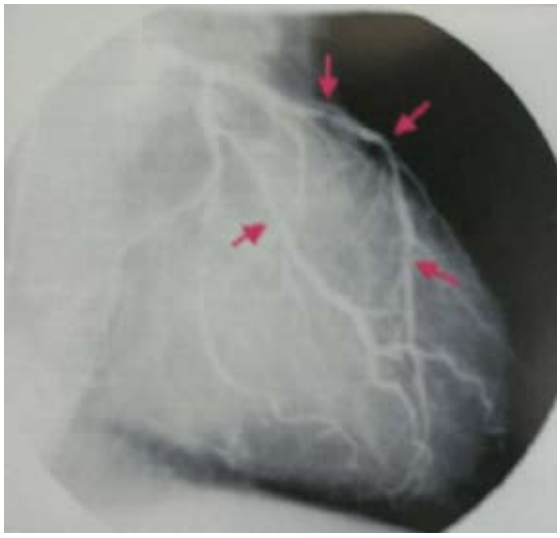


Fig. 1. Arteriographic view of the coronary arteries. The arrows indicate points of stenosis.

1. Ischemic heart disease

Myocardial ischemia occurs as a result of diminished coronary blood flow. Such reduction in flow can be partial or total, and is usually attributable to thrombus formation over an atheroma plaque, with occlusion of the coronary vascular lumen. The presence of atheroma plaques (arteriosclerosis) within the coronary arteries is the most common cause of myocardial ischemia (over 90% of all cases). When the plaque ruptures, ulcerates or suffers fissures, primary platelet aggregation takes place. This effect in turn is reinforced by the intervention of agonists (collagen, ADP, serotonin, adrenalin) - leading to the release and production of thromboxane A2, which is responsible for local vasoconstriction (among other actions). The tissue factor exposed to the bloodstream as a result of vascular endothelial damage triggers hemostasia and blood coagulation - the end result of which is the accumulation of platelets and fibrin that conform a thrombus (3).

Ischemic heart disease has two manifestations according to the degree of arterial vascular obliteration and the presence or absence of myocardial necrosis. When obliteration is total (full occlusion) and tissue necrosis occurs, acute myocardial infarction (AMI) results. In the case of partial obliteration of the vascular lumen without myocardial necrosis, angina (chest pain) results (4). The differences between these two conditions are reflected in Table 1. However, it is sometimes difficult to differentiate between AMI and angina in the early stages of the process. As a result, these manifestations are presently classified as acute coronary syndrome (ACS) with ST-segment elevation on the electrocardiogram (which can benefit from emergency myocardial reperfusion treatment) and ACS without ST-segment elevation (where the objective is to stabilize the complicated atheroma plaque)(5).

Chest pain is the initial presentation in approximately 45% of cases, with myocardial infarction in 42%, and sudden death in the remaining 13%.

- *Acute myocardial infarction (AMI)*

Acute myocardial infarction is characterized by acute-onset myocardial necrosis of ischemic origin, secondary to thrombotic occlusion of a coronary artery (6).

Ischemic heart disease is principal cause of death in industrialized countries. In Spain, the incidence is 135-210 infarctions/100,000 males/year, versus 29-61 infarctions/100,000 females/year (7). Despite the difference in incidence between males and females, the latter have a comparatively greater risk of death over the short and long term following AMI. This is because infarction in women typically occurs at later ages, and moreover,

Table 1. Differences between acute myocardial infarction and chest pain (angina, angor pectoris).

	Infarction	**Angina**
Degree of ischemia	Complete	Partial
Cell necrosis	Yes	No
Subsides with rest, sublingual nitroglycerin	No	Yes
Duration of pain	> 30 minutes	< 30 minutes
ECG	ST-segment elevation, pathological Q-waves	Presence or absence of ST-segment alterations

the causes and locations of infarction are different. The co-morbidity characteristics found in women are an additional influencing factor (8). In the United States it is estimated that 500,000 people suffer myocardial infarction each year (4). The mortality is maximum in the first hours after the event, and approximately 50% die before reaching hospital (3).

The cardiovascular risk factors found among the general population have been well known for decades: smoking, arterial hypertension, hyperlipidemia, diabetes mellitus, age, the male sex and genetic factors have all been implicated (9).

Arterial hypertension (AHT) affects 6-8% of the general population. The underlying cause is known in 1-15% of cases (secondary hypertension); consequently, in the great majority of subjects the cause of hypertension is not known (primary, essential or idiopathic hypertension). The condition remains asymptomatic for a prolonged period of time, and subsequently evolves towards arteriosclerosis that ultimately leads to the cardiac problems examined in the present study (among other disorders). In effect, the presence of arterial hypertension implies a three-fold increase in the incidence of coronary disease.

Diabetes mellitus (DM) is a very common disease that affects about 5% of the population in industrialized countries - with important geographical variations. It is a metabolic disorder produced by insulin deficiency in which the affected individual is unable to adequately metabolize carbohydrates. Two types of diabetes can be observed: type I (typical of younger individuals, with a sudden onset and a tendency to produce ketoacidosis), and type II (more common in adults, with a slow onset and no tendency to produce ketoacidosis). Its complications include diabetic macroangiopathy, which is a form of arteriosclerosis characterized by faster progression than in the general population. It is the most frequent cause of death among diabetics. In effect, 49% of all deaths among diabetics in the 35-44 years age range are due to acute myocardial infarction (10).

Clinically, AMI manifests as acute, intense and oppressive pain of retrosternal or precordial location, and sometimes irradiating to the arms, neck, mandible or back. The pain lasts for over half an hour and does not subside with rest. In addition, it is accompanied by profuse perspiration, nausea and vomiting, and the patient typically experiences a sensation of imminent death. AMI can also manifest as a sudden loss of consciousness, confusional state or weakness.

The triggering stimuli are emotional stress, intense physical exercise, some background disease, or surgical intervention. Silent infarctions, i.e., those without pain, are more common among elderly individuals, in women, and in diabetic patients. It should be mentioned that other processes such as pulmonary thromboembolism, aortic dissection, pneumothorax, costochondrosis or simple episodes of a psychic nature can manifest with pain of similar characteristics (3,4).

The diagnosis of AMI is based on the clinical findings, the ECG tracing and the determination of myocardial necrosis markers (11). The ECG reflects the impact of sudden and acute ischemia upon the electrical activity of the heart, in the form of ST-segment elevation (Figure 2).

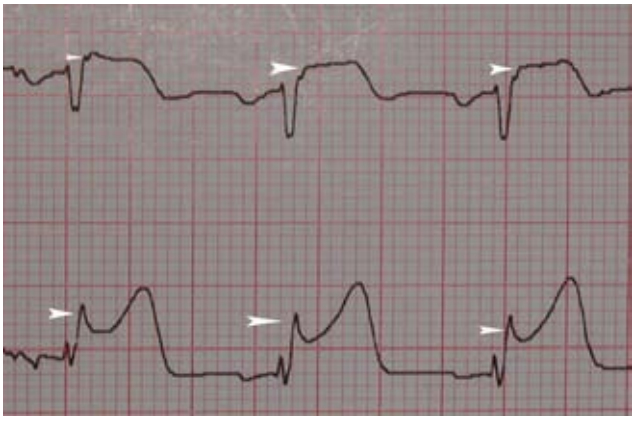


Fig. 2. ECG tracing of a patient with acute myocardial infarction. The arrows indicate ST-segment elevation.

The appearance of pathological Q-waves is synonymous of myocardial necrosis. The determination of serum markers has grown in importance and diagnostic usefulness over the years. In this context, evaluations are made of serum creatine phosphokinase and its isoform MB (CPK and CPK-MB), troponin T and I, and myoglobin. Troponin I has displaced CPK-MB in terms of relevance (12). Other complementary diagnostic techniques are chest X-rays, two-dimensional (2D) echocardiography, gammagraphy and cardiac catheterization.

The objective of in-hospital treatment in acute phase AMI is to ensure immediate myocardial reperfusion (13), based on pharmacological or surgical means. Fibrinolysis plays a particularly important role in the first hours after infarction. Its principal complication (from which the contraindications of such therapy are derived) is the appearance of bleeding. A possible option in this sense is to add aspirin as coadjuvant treatment, at a dose of 75-325 mg/day (14), thanks to its antiplatelet action, and heparin. Other drugs used for the treatment and secondary prevention of AMI comprise beta-blockers (which reduce oxygen consumption and cardiac cycle frequency and contractility), calcium antagonists, and angiotensin converting enzyme inhibitors (ACEIs).

The complications that may manifest after AMI can be divided into three categories: mechanical, electrical and ischemic. The most important complication in terms of frequency and severity is heart failure, followed by atrial or ventricular fibrillation, conduction block, postinfarction angina and reinfarction.

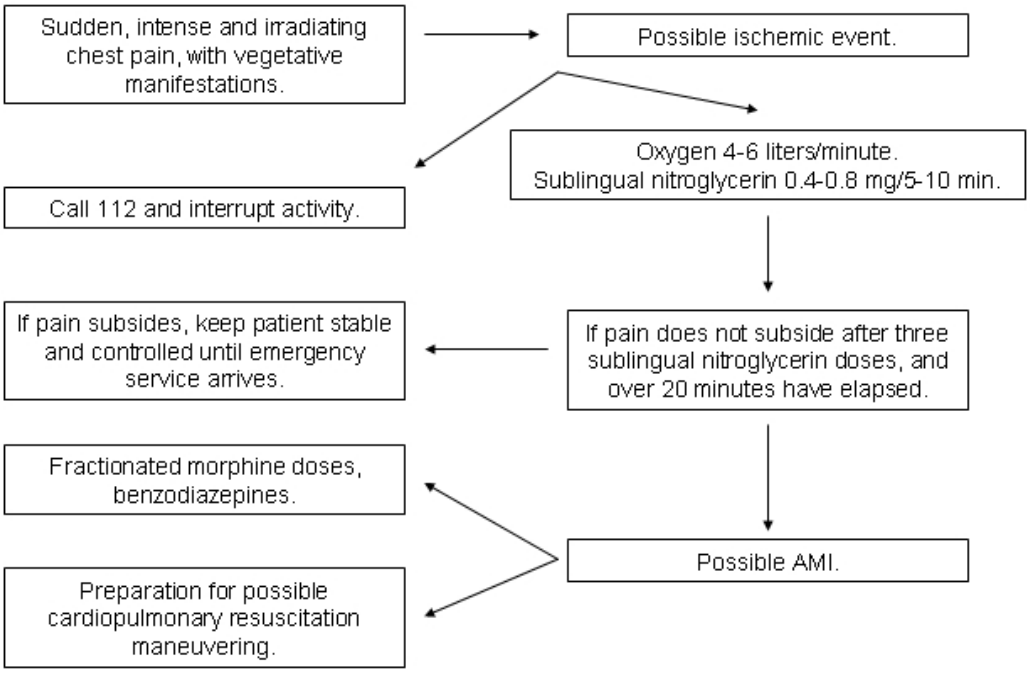
- *Angor pectoris – chest pain*

Angor pectoris, angina or chest pain is the result of a partial and reversible reduction in myocardial perfusion, of thrombotic origin, in which no cell necrosis occurs. Stable angina is preceded by physical exertion or emotional stress, and is characterized by pain of the same location and characteristics as pain in AMI, though of lesser duration (in the order of minutes). It also subsides with rest and/or with the administration of sublingual nitroglycerin. In contrast, unstable angina manifests under resting conditions, the pain is characteristically longer lasting and more intense (though without exceeding 20-30 minutes), and is less responsive to nitrates. In one-half of all cases of angina the ECG tracing is normal, while in the rest of cases ST-segment alterations are evidenced. Stable angina can evolve towards unstable angina, or the latter may lead to AMI or (in the worst of cases) to sudden death. Patients with a history of chest pain receive treatment in the form of antiplatelet drugs (aspirin, ticlopidine, clopidogrel), nitrates (nitroglycerin), beta-blockers and calcium antagonists - a condition that must be taken into account by the dental surgeon, as will be seen below.

Approach of the dental surgeon to emergency situations

In the event of chest pain with the above mentioned characteristics, location and triggering stimuli inherent to ischemic episodes, the approach is the same regardless of whether angina or AMI is involved. The associated presence of dyspnea, syncope, hypotension, intense hypertension or tachyarrhythmia suggests a potentially fatal disorder. The emergency service therefore must be contacted immediately (telephone number 112), and the patient should be administered oxygen with a mask or nasal cannula at a rate of 4-6 liters/minute. This is to be followed by sublingual nitroglycerin tablets (or using an aerosol formulation) at a dose of 0.4 - 0.8 mg. Dosing can be repeated every five minutes. The patient should be instructed to bite the tablet and place it under the tongue. The effect should be noted after approximately two minutes, and the dose is to be repeated every 10 minutes up to 3-4 times if improvement is not obtained. It should be taken into account that the administration of nitroglycerin can cause side effects with some frequency, in the form of headache, dizziness and tachycardia (15). It is also possible to administer 325 mg of aspirin, in view of its antiplatelet effects, though taking into account that the drug also increases the blood levels of nitroglycerin, and therefore can increase the toxicity of the latter. The administration of aspirin in the first hours following infarction reduces mortality 20%. Consequently, as soon as infarction is suspected, a dose of aspirin is recommended. If the pain persists for more than 20 minutes, fails to subside with rest, and does not respond to three doses of sublingual nitroglycerin, the diagnosis is very likely AMI. In this case, emergency care must center on the provision of pain relief as quickly as possible, administering morphine in fractionated doses of 5 mg via the intramuscular or intravenous route (½ ampoule of generic formulation containing 1 ml of 1% solution). The dose can be increased to 30 mg in the first 20 minutes, or alternatively pentazocine can be provided at equivalent doses if the patient presents background lung disease (½ ampoule containing 30 mg via the intramuscular or intravenous route every 5 minutes). Sedation is to be provided with oral benzodiazepine in the event of vegetative manifestations, with the application of cardiopulmonary resuscitation maneuvers where required (16)(Figure 3).

Fig. 3. Approach to chest pain of possible ischemic origin.



2. Heart failure

Heart failure comprises the global clinical manifestations associated with ventricular dysfunction, heart valve defects or ventricle loading conditions, reflected as a reduction in cardiac output (volume pumped per minute) and deficient tissue distribution. In 2003, in Spain, heart failure caused 20,000 deaths out of a total of 385,000 cases (i.e., a 5.2% mortality rate), and accounted for 15.3% of all deaths of cardiovascular origin. It is estimated that 20% of all people will suffer heart failure in the course of their lifetime (17). The survival of these patients is limited due to the intervention of aggravating factors and the presence of concomitant diseases.

Ventricle function is regulated by four factors: myocardial contractility, ventricular preloading and postloading, and heart rate. Ventricular postload is the most relevant parameter, as it reflects the strength of myocardial contraction, and is the principal determinant of ejection volume. When any one of these four factors fails, the heart becomes unable to supply the required blood flow in relation to the existing venous return flow and the tissue and organ needs.

Failure can be acute, as a result of exposure to cardiotoxic drugs or secondary to coronary occlusion, or it may be chronic and associated to a patient history of arterial hypertension and ischemic heart disease. The chronic form of heart failure is the most common presentation, and is the form referred to below. As has already been mentioned, the principal cause is coronary disease. Over 45% of all cases of AMI are complicated by heart failure (18). Other causes are dilated myocardiopathy, valve disease, alcoholic heart disease, cor pulmonale and hypertrophic and restrictive cardiomyopathy. Arterial hypertension is considered to be the most important risk factor, particularly when concomitant to coronary atherosclerosis. Another factor to be taken into account is diabetes mellitus, which increases the risk of chronic heart failure 2.5-5 times, due to the known metabolic alterations and increased cellular oxidative stress levels involved (19). Chronic heart failure, resulting from deficient left ventricle function, is characterized by lung congestion (clinically manifesting as important dyspnea, cough, hemoptysis and nocturia) and tissue hypoperfusion (manifesting as asthenia, muscle weakness, palpitations, lessened tolerance of physical exercise, mental alterations and oliguria). In the case of right ventricle failure, the resulting manifestations comprise peripheral venous congestion with the appearance of ankle and foot edemas, with a positive fovea sign.

Surgical management is based on heart transplantation. Due to the complexity of the condition, other techniques have also been developed, such as mitral valve replacement or repair, left ventricle reconstruction, total or partial mechanical replacement of the ventricle, the implantation of a pacemaker, cardiomyoplasty, or coronary revascularization (20). Pharmacological treatment centers on neurohormonal block with ACEIs (captopril, enalapril, lisinopril) and beta-blockers (atenolol, bisoprolol, propanolol); aldosterone antagonists (spironolactone) and angiotensin receptor antagonists are also used (losartan, valsartan)(21).

Acute heart failure, either primary or as an exacerbation of the chronic form of the disease, tends to manifest as acute lung edema. The most frequent triggering factors are severe and prolonged arterial hypertension, valve disease, ischemic heart disease (AMI) and severe pericardial disorders. Lung edema is characterized by the displacement of liquid from the capillary space into the interstitial and alveolar compartments when pulmonary capillary pressure exceeds the oncotic pressure of plasma. Affected patients often present some background disorder or a history of progressive dyspnea, dry cough, asthenia, etc. Edema manifests with sudden onset or progressive dyspnea, cough with abundant pink colored expectoration, and a worsened general condition involving cyanosis, cold skin, intense perspiration and a sensation of extreme illness. The patient experiences suffocation and laryngeal irritation, and tends to adopt the sitting position to facilitate breathing. The condition may simulate an asthma episode. The clinical history of the patient and the absence of rales in asthma help to establish the differential diagnosis (Figure 4).

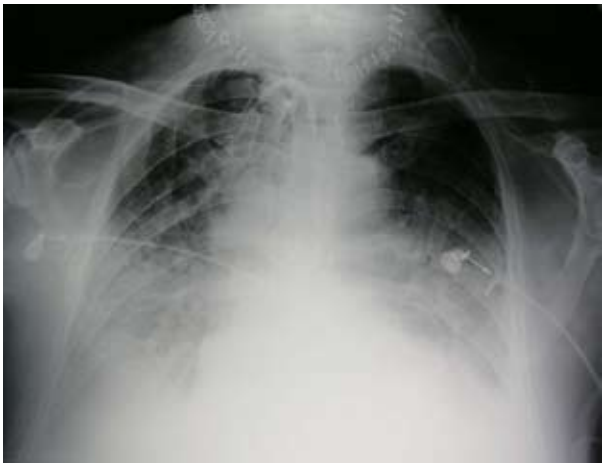


Fig. 4. Chest X-ray view of a patient with acute lung edema.

- *Approach of the dental surgeon to acute lung edema*

After contacting the emergency service (telephone number 112), the patient is to be seated with the legs relaxed, and oxygen is to be administered by means of a mask or nasal catheter or cannula (4-6 liters/minute). Sublingual nitroglycerin can be provided (0.4-0.8 mg), with repetition of the dose every 5 or 10 minutes if blood pressure is maintained. The administration of intravenous morphine (5 mg in an initial dose, followed by 1-2 mg every 5 minutes for a maximum of 15-20 mg in 30 minutes) helps reduce restlessness, heart and breathing rate, and blood pressure (i.e., cardiorespiratory function is globally improved). However, due to the risk of respiratory depression in patients with

prior lung disease, the use of such medication requires extreme caution in patients of this kind, and morphine antagonists must be available in case morphine reversion proves necessary. In such situations, an initial intravenous dose of 0.4-2 mg is to be administered, repeating the dose every 2-3 minutes - without exceeding 10 mg. If possible peripheral catheterization should be carried out to administer loop diuretics (furosemide 40-100 mg iv)(22,23).

3. Considerations applicable to dental practice

In patients with cardiovascular disease it is advisable to minimize the stress of visiting the dentist, and to provide effective pain treatment. The controversy as to whether or not to use a vasoconstrictor (adrenalin or levonordephrine) with the local anesthetic solution is due to the effect of the former upon arterial pressure. In patients with heart disease it is common to use beta-blockers as antiarrhythmic and antihypertensive medication. Some of these drugs are non-selective, inhibiting both beta-1 and beta-2 adrenergic receptors (propanolol, nadolol, timolol, pindolol), as a result of which they lower heart rate and cardiac output, and moreover exert a marked hypotensive effect - although vascular beta-2 adrenergic block facilitates the predominance of alpha-1 adrenergic tone in certain territories, with an increase in peripheral resistance and therefore of diastolic blood pressure. On the other hand, selective beta-blockers with alpha-1 antagonistic action avoid this problem of increased peripheral vascular resistance (labetalol (Trandate®), carvedilol (as generic formulations, Coropres®)). Cardioselective beta-blockers preferentially exert their effects upon the beta-1 adrenergic receptors (metoprolol (Beloken®), atenolol (as generic formulations, Blokium®), esmolol (Brevibloc®)), reducing heart rate, conduction velocity in the atrioventricular node, and cardiac output (15 The joint administration of adrenalin, which acts mainly upon the beta-2 adrenergic receptors, can give rise to arterial hypertension and bradycardia - this being more likely to happen with non-cardioselective beta-blockers, by interfering with their action upon these receptors. Moreover, it must be taken into account that beta-blockers can delay peripheral plasma clearance of the local anesthetic, and that the prolonged use of nonsteroidal antiinflammatory drugs (NSAIDs) can reduce the antihypertensive effects.

A visit to the dentist generates anxiety in itself, with the release of endogenous catecholamines in amounts that may exceed those administered with the local anesthetic solution. The association of a vasoconstrictor therefore should be limited, taking care not to exceed 0.04 mg of adrenalin (i.e., 2 carpules containing 1.8 ml of anesthetic with adrenalin 1:100,000). The use of retraction thread with adrenalin is not advised (24).

During the first 6 months after AMI, dental treatment should be reserved for emergency situations destined to provide odontogenic pain relief: tooth extractions, the drainage of abscesses, and pulpectomies - preferably carried out in the hospital setting. After this period of caution, treatment decision is conditioned by the individual situation and medical condition of the patient (25).

Pharmacologically, special mention should be made of the antiplatelet drugs (aspirin, clopidogrel, ticlopidine, dipyridamole), the anticoagulants (antivitamin K or the coumarins), and the beta-blockers (already commented above). The interruption of thrombolytic medication for the provision of dental treatment is risky, due to the possibility of repeat thrombosis. If the suspension of thrombolytic medication proves necessary, the decision to provide dental treatment must be taken in coordination with the physician supervising the patient medication (Table 2)(26,27). When antiplatelet medication cannot

Table 2. Antiplatelet and anticoagulant drugs commonly used in cardiovascular disease.

Generic name	Dose	Brand name	*Withdrawal interval (26)*
Dipyridamole	75-100 mg/day	Miosen® Persantin®	*10-14 days*
Ticlopidine	250 mg/12h	Ticlopidine GDP Ticlodone® Tiklid®	*10-14 days*
Clopidogrel	75 mg/day	Iscover® Plavix®	*10-14 days*
Acetylsalicylic acid	100-300 mg/day	Aspirin® Adiro® Tromalyt®	*10-14 days*
Acenocoumarol ***Warfarin***	*1-10 mg/ day* *2-10 mg/day*	*Sintrom®* *Aldocumar®* *Tedicumar®*	*No withdrawal* *INR ≤ 4*

be interrupted on occasion of invasive dental treatment, and a risk of bleeding is anticipated, local hemostatic measures must be applied such as bone wax, sutures, gelatin of animal origin (Gelfoam®), regenerated oxidized cellulose (Surgicel®), collagen, plasma rich in platelets, thrombin (Thrombostat®), fibrin sealants (Tissucol®), and the use of an electric or laser scalpel (28). The use of antifibrinolytic agents is also advised, such as tranexamic acid (Amchafibrin®) or epsilon-aminocaproic acid (Caproamin®). The intravenous route is not usually used in dental practice; rather, the oral route is employed: epsilon-aminocaproic acid as a dose of 4 grams (1 ampoule) every 4 or 6 hours, and tranexamic acid 1-1.5 g 2-3 times a day in tablets or ampoules (15). Tranexamic acid is also used in mouthrinse format.

In the case of anticoagulated patients, it is essential to determine the INR (International Normalized Ratio) before invasive dental treatment, and without the need to interrupt the medication. The INR compares the prothrombin time (PT) of the patient versus a control or standard PT adjusted according to the International Sensitivity Index (ISI). It is accepted that oral surgical procedures are to be carried out with an INR of ≤ 4 (Table 2), applying the aforementioned hemostatic measures (29).

In patients carrying a pacemaker, interferences may occur with the use of pulpmeters, ultrasound for supragingival tartar removal, and the electrical scalpel (22).

References

1. DeWood MA, Spores J, Notske R, Mouser LT, Burroughs R, Golden MS, et al. Prevalence of total coronary occlusion during the early hours of transmural myocardial infarction. N Engl J Med. 1980 Oct 16;303(16):897-902.
2. WHO (website). Geneva; 2003 [access Dec. 12, 2006]. Cardiovascular disease: prevention and control (WHO Global Strategy on Diet, Physical Activity and Health). Site: http://www.who.int/dietphysicalactivity/publications/facts/cvd/en/.
3. Hayoz D, Mazzolai L. Endothelial function, mechanical stress and atherosclerosis. Adv Cardiol. 2007;44:62-75.
4. Antman EM, Anbe DT, Armstrong PW, Bates ER, Green LA, Hand M, et al. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction; A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1999 Guidelines for the Management of patients with acute myocardial infarction). J Am Coll Cardiol. 2004 Aug 4;44(3):E1-E211.
5. Salguero R, Sánchez R, Matía R, Sáez de la Calzada C. Cardiopatía isquémica. Concepto. Aspectos epidemiológicos. Etiopatogenia. Clasificación biológica y clínica. Implicaciones en la calidad de vida. Medicine 2005; 9: 2571-9.
6. Cannon CP. Evolving management of ST-segment elevation myocardial infarction: update on recent data. Am J Cardiol. 2006 Dec 18;98(12A):10Q-21Q.
7. Medrano Alberto MJ, Boix Martínez R, Cerrato Crespán E, Ramírez Santa-Pau M. Incidence and prevalence of ischaemic heart disease and cerebrovascular disease in Spain: a systematic review of the literature. Rev Esp Salud Publica. 2006 Jan-Feb;80(1):5-15.
8. Koek HL, De Bruin A, Gast F, Gevers E, Kardaun JW, Reitsma JB, et al. Short- and long-term prognosis after acute myocardial infarction in men versus women. Am J Cardiol. 2006 Oct 15;98(8):993-9.
9. Amar J, Chamontin B. Cardiovascular risk factors, atherosclerosis and pulse pressure. Adv Cardiol. 2007;44:212-22.
10. Donahoe SM, Stewart GC, McCabe CH, Mohanavelu S, Murphy SA, Cannon CP, et al. Diabetes and mortality following acute coronary syndromes. JAMA. 2007 Aug 15;298(7):765-75.
11. Yang EH, Brilakis ES, Reeder GS, Gersh BJ. Modern management of acute myocardial infarction. Curr Probl Cardiol. 2006 Dec;31(12):769-817.
12. Eriksson S, Wittfooth S, Pettersson K. Present and future biochemical markers for detection of acute coronary syndrome. Crit Rev Clin Lab Sci. 2006;43(5-6):427-95.
13. Henry T. Contemporary challenges in the management of acute myocardial infarction: ST-elevation myocardial infarction guidelines and the real world. Am Heart J. 2006 Jun;151(6 Suppl):S11-6.
14. Hennekens CH, Sechenova O, Hollar D, Serebruany VL. Dose of aspirin in the treatment and prevention of cardiovascular disease: current and future directions. J Cardiovasc Pharmacol Ther. 2006 Sep;11(3):170-6.
15. Catálogo de Medicamentos. Colección Consejo Plus. Madrid. Consejo General de Colegios Oficiales de Farmacéuticos, 2006.
16. Chapman PJ. Chest pain in the dental surgery: a brief review and practical points in diagnosis and management. Aust Dent J. 2002 Sep;47(3):259-61.
17. Muñiz García J, Crespo Leiro MG, Castro Beiras A. Epidemiology of heart failure in Spain and the importance of adhering to clinical practice guidelines. Rev Esp Cardiol. 2006 Jul;6 Suppl F:2-8.
18. Weir RA, McMurray JJ. Epidemiology of heart failure and left ventricular dysfunction after acute myocardial infarction. Curr Heart Fail Rep. 2006 Dec;3(4):175-80.
19. De Flines J, Scheen AJ. Diabetes mellitus and congestive heart failure: physiopathology and treatment. Rev Med Suisse. 2006 Aug 23;2(76):1893-6; 1898-900.
20. Goldstein DJ, Smego D, Michler RE. Surgical aspects of congestive heart failure. Heart Fail Rev. 2006 Jun;11(2):171-92.
21. Howard PA, Cheng JW, Crouch MA, Colucci VJ, Kalus JS, Spinler SA, et al. Drug therapy recommendations from the 2005 ACC/AHA guidelines for treatment of chronic heart failure. Ann Pharmacother. 2006 Sep;40(9):1607-17.
22. Rogers RL, Feller ED, Gottlieb SS. Acute congestive heart failure in the emergency department. Cardiol Clin. 2006 Feb;24(1):115-23.
23. Peacock WF, Allegra J, Ander D, Collins S, Diercks D, Emerman C, et al. Management of acute decompensated heart failure in the emergency department. Congest Heart Fail. 2003 Sep-Oct;Suppl 1:3-18.
24. Silvestre FJ, Miralles-Jorda L, Tamarit C, Gascon R. Dental management of the patient with ischemic heart disease: an update. Med Oral. 2002 May-Jun;7(3):222-30.
25. Miloro M. Congestive heart failure. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2000 Jul;90(1):9-11.
26. Hupp JR. Ischemic heart disease: dental management considerations. Dent Clin North Am. 2006 Oct;50(4):483-91.
27. Jeske AH, Suchko GD, ADA Council on Scientific Affairs and Division of Science; Journal of the American Dental Association. Lack of a scientific basis for routine discontinuation of oral anticoagulation therapy before dental treatment. J Am Dent Assoc. 2003 Nov;134(11):1492-7.
28. Daniel NG, Goulet J, Bergeron M, Paquin R, Landry PE. Antiplatelet drugs : is there a surgical risk. J Can Dent Assoc. 2002 Dec;68(11):683-7.
29. Fitzmaurice DA, Blann AD, Lip GY. Bleeding risks of antithrombotic therapy. BMJ. 2002 Oct 12;325(7368):828-31.