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Cardiovascular disorders in dental practice

Cardiovascular emergencies are rare events in dental practice. However, they may be life-threatening, and a dentist should therefore be able to recognize them and be prepared to take appropriate action. The diagnosis and treatment of angina pectoris, cardiac arrhythmias, valvular diseases, heart failure, hypertensive crisis and stroke will be reviewed. Since anti-coagulant therapy is frequently used in cardiovascular patients, it will also be discussed.

Medical emergencies in dental practice are rare (1). It takes an average of 0.5 years before the dentist will see the most common complication, vasovagal collapse, in the dental practice (1). Cardiovascular emergencies are even more infrequent. It takes approximately 5.7 years before a dentist observes a patient developing chest pain, which accounted for 11.9 % of the total number of emergency events in a British dental practice (1).

Hypertensive crisis, myocardial infarction and cardiac arrest accounted for 13.8 % of the emergency cases in an Australian survey by Chapman (2). The incidence of angina pectoris was similar to that in the British survey (1,2). However, when a cardiovascular emergency does occur, it can be life-threatening, and therefore a dentist has the responsibility of being properly trained to recognize and deal with cardiovascular emergencies in the practice.

Angina pectoris, myocardial infarction, cardiac arrhythmias, heart failure, hypertensive crisis and stroke are the most common causes of acute cardiovascular disorders in dental practice. The diagnosis and treatment of these situations will be reviewed.

Angina pectoris and myocardial infarction

Coronary heart disease is very common in the general population, and it is therefore likely that a dentist will meet such a patient in clinical practice. Coronary heart disease can manifest itself in many forms, i.e stable and unstable angina pectoris, myocardial infarction and sudden cardiac death.

Angina pectoris originates from an inadequate supply of oxygen to the myocardium which is most often due to coronary atherosclerosis. The pain is typically described as tight, squeezing, like a weight on the chest, or like indigestion; as with any visceral pain, the localisation is vague and there is considerable variation between patients (3). It should not be forgotten that anginal pain is often felt in the mandible, with secondary radiation to the neck and throat. Therefore, the patient may initially suspect the pain to be of dental origin (4). The dental environment increases the likelihood of an anginal attack because of fear, anxiety and pain (4).

The patient's history should be a relevant part of dental practice. A dentist should know about the patient's chronic diseases and medical treatments. The former history may also determine how to deal with an angina pectoris attack in the dental chair.

Angina pectoris can manifest itself as stable or unstable forms which have different treatment strategies. Stable angina is defined as exercise angina, which is typically associated with exercise but can also be caused by emotional stress. The Canadian Cardiovascular Society has classified ischaemic chest pain into four levels (5), which run from I (very mild) to IV (symptoms at rest or during minimal exertion) (Table 1). These levels have been proven to be useful in clinical trials and in clinical practice.

Unstable angina pectoris (UAP) is defined as acute angina at rest (within 48 hours before presentation), subacute angina at rest (within the previous month but not within the 48 hours before presentation), or new onset of accelerated (progressively more severe) angina or angina within two weeks after an acute myocardial infarction (6).

The patient with stable coronary artery disease is usually on continuous treatment with beta-blockers, aspirin, and cholesterol-lowering medications, mostly statins. If they suffer from occasional periods of angina pectoris, they may be treated with long-acting nitrates and short-acting nitrates as well. Short-acting nitrates are available as orally soluble tablets or an oral spray. Patients with stable angina pectoris

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Table 1. *Classification of angina pectoris according to Canadian Cardiovascular Society*

Class I	Ordinary physical activity: Angina with strenuous or rapid or prolonged exertion at work or recreation
Class II	Slight limitation of physical activity: Walking or climbing stairs rapidly, walking uphill, walking on stairs after meals, in cold, in wind, or when in emotional stress, or only few hours after awakening
Class III	Marked limitations of ordinary physical activity: Walking one to two blocks on the level, and climbing more than one flight of stairs in normal conditions
Class IV	Inability to carry any physical activity without discomfort-anginal syndrome may be present at rest

should be encouraged to carry their short-acting nitrates with them when they visit the dentist. If a patient with stable coronary artery disease experiences chest pain during dental treatment, the procedure should be discontinued and a short-acting nitrate should be given. If the acute attack passes with no more than two or three short acting nitrates (tablets or spray), the procedure can be continued after a painless rest of 10–15 minutes. However, if the pain is prolonged and the nitrates do not help, myocardial infarction should be suspected and an ambulance called.

In acute myocardial infarction the chest pain is usually prolonged, more severe, and coexistent nausea and sweating may occur. The patient should be given 250 mg of aspirin (if not allergic) to chew while waiting for the ambulance. Additionally, inhaled 100 % oxygen helps to increase the oxygen supply to the myocardium and relieves pain as well. If possible, an intravenous cannula should be set to the patient in order to give analgesics and other medications if needed.

A patient with UAP is not a usual occurrence in a dental practice. In general, elective dental treatment has traditionally been contraindicated, except for conservative emergency procedures in patients with UAP and within six months after the onset of an acute myocardial infarction (7). Moreover, patients with UAP belong in the hospital. An early invasive treatment strategy, including percutaneous coronary angioplasty and stenting, or coronary by-pass operation, has proven beneficial in UAP (8). Therefore, a patient with UAP should be immediately referred to a hospital, and any dental procedures should be postponed. Patients with UAP have undergone dental procedures, but in a hospital with continuous ECG and blood pressure monitoring (7).

Cardiac arrhythmias

A cardiac arrhythmia can be described as an abnormality in rate, regularity, or site of origin of the cardiac impulse. In addition, conduction of the impulse within the heart may be abnormal. The severity of arrhythmias ranges from harmless extrasystoles in a healthy heart to severe ventricular arrhythmia, such as ventricular fibrillation in a patient with coronary heart disease, which causes cardiovascular collapse and sudden death.

A specific diagnosis of an arrhythmia during a dental appointment would necessitate continuous ECG monitoring, and good knowledge of interpretation of the abnormalities observed. Thus, in the usual dental setting, patient history, symptoms and palpation of the pulse are the available diagnostic tools.

In a patient with no history of heart disease or arrhythmias, an increase in heart rate before and during dental management is a normal physiological reaction. Palpitations and transient irregularities («jumps») in the rhythm in such patients are generally not alarming. They represent normal reactions to endogenous or exogenous (from local anesthetics) adrenaline. A vasovagal syncope, also called ordinary fainting, resulting from fear or pain is not an indication of significant cardiac abnormality. During such a reaction, the pulse may be weak and slow, but the patient usually recovers totally and rapidly, especially when placed in a supine position. It is very unlikely that a person with a healthy heart would experience any significant arrhythmia during dental treatment.

In patients with pre-existing heart disease the situation may be different. Patients with coronary heart disease may experience angina before or during treatment because of the increase in heart rate. Increase in heart rate increases myocardial oxygen demand and leads to ischemia in patients with compromised coronary circulation. Ischemia, on the other hand, may trigger arrhythmias in such patients. A scar of a previous myocardial infarction may cause electrical instability in the heart and may be the substrate of potentially lethal arrhythmias. The risk of harmful arrhythmias is also increased in patients with cardiomyopathies, heart failure and valvular problems. Such patients should be carefully evaluated by their physician and adequate medication and other measures (such as an implantable cardioverter-defibrillator, see later) should be implemented before extensive dental procedures. A treatment plan, including calling emergency medical services and cardiopulmonary resuscitation skills, should be made and regularly re-evaluated in all dental settings treating such patients. A dentist should be aware of his skills and resources to treat such emergencies and consider referring high-risk patients to adequate treatment facilities.

Should a patient with known heart disease develop arrhythmia during treatment, the treatment should be discontinued, supplemental oxygen considered and the patient status closely monitored. If the patient recovers quickly, continuation of treatment may be considered if the patient wishes. Even a brief loss of consciousness, however, may indicate significant cardiac arrhythmia, and the patient should be referred to medical evaluation. If a patient with heart disease collapses in the chair (loss of consciousness, non-palpable pulse), cardiac arrest should be suspected and emergency medical services activated immediately and cardiopulmonary resuscitation initiated without delay.

The above caveats refer to high risk cardiac patients with severe heart disease with either a history of harmful arrhythmias or poor cardiac function, indicated by angina or dyspnea already during light exercise or even at rest. Many patients with cardiac disease have little or no limitation of physical activity. Additionally, if there is no history of significant arrhythmias, such patients can generally be treated in an ordinary dental office.

It is important to ensure that patients with heart disease have been taking their medication regularly. Beta-blockers are the cornerstone of treatment of many cardiac conditions such as coronary heart disease, heart failure and arrhythmias. Beta-blockers protect the heart by attenuating sudden increases of heart rate or blood pressure, and they also suppress arrhythmias. There is no need, and it may in fact be harmful, to discontinue such medications because of dental treatment.

The use of local anesthetics containing adrenaline in cardiac patients continues to be a debated issue. It has been clearly demonstrated that oral injection of local anesthetics (lidocaine) containing adrenaline causes measurable hemodynamic changes in patients with heart

disease. An increase in heart rate, systolic blood pressure and cardiac output has been demonstrated (9). The same study also demonstrated the attenuation of these reactions in patients using beta-blockers. Despite measurable hemodynamic changes, no symptoms or adverse consequences were observed, and use of lidocaine-epinephrine was considered safe.

Other studies have demonstrated increased sympathetic activity (increased blood pressure, heart rate and arrhythmias) in patients *before* any dental procedures (including administration of local anesthetics) reflecting fear and anxiety and the effects of endogenous adrenaline in response to these feelings (10,11). The study by Campbell et al. (11) demonstrated more arrhythmias during the surgical procedure than during injection of the anesthetic. This underlines the importance of adequate anesthesia rather than fear of side-effects. Current consensus seems to favour judicious use of local anesthetics containing vasoconstrictors to ensure profound anesthesia. The amount of vasoconstrictor should be limited to total epinephrine 0.04–0.054 mg per appointment (12). In other words, deep local anesthesia achieved with the aid of vasoconstrictors is probably less harmful than the rise in endogenous adrenaline levels in response to fear and pain caused by inadequate local anesthesia. Other measures to control anxiety (e.g. sedation) should also be considered in cardiac patients.

Atrial fibrillation is the most common chronic arrhythmia in the population. It has been estimated that 5 % of 60-year-old and even 50 % of 80-year-old people have AF (13). Many diseases predispose for AF, such as hypertension, coronary heart disease, diabetes, hyperthyreosis, and rheumatic valvular diseases. In atrial fibrillation the electrical impulses in the atria are chaotic, and there is no synchronous contraction of the atria. The ventricular rate and thus the palpable pulse are irregular because there is no systematic impulse coming from the atria. The ventricular rate is determined by how well the atrioventricular node lets through impulses from the atria and can be controlled by medication (beta-blockers, digitalis, certain calcium channel-blockers).

Patients with chronic atrial fibrillation are usually under medication and generally tolerate the arrhythmia rather well, and the main issue regarding dental treatment is the use of anticoagulants, which is recommended in atrial fibrillation. Atrial fibrillation may be due to valvar problems which may require antibiotic prophylaxis. Both these issues will be addressed later in this article.

New onset atrial fibrillation may occur during dental treatment, and often causes symptoms of palpitation, irregular pulse, chest discomfort and tachycardia. If the patient is familiar with such paroxysmal atrial fibrillation, treatment does not necessarily have to be discontinued, and the arrhythmia may spontaneously end. If the arrhythmia is new to the patient or causes marked symptoms, it may be better to discontinue the treatment and refer the patient to medical evaluation. Some patients with frequently recurring atrial fibrillation may be on anticoagulant treatment.

Cardiac pacemakers and implantable cardioverter-defibrillators

Cardiac pacemakers are increasingly common in the aging population. It has been estimated that worldwide there are over 3.25 million functioning cardiac pacemakers and 180 000 ICD's (14). Almost every practicing dentist is going to meet a patient with a cardiac pacemaker or implantable cardioverter-defibrillator (ICD). Understanding the principles of their function helps to understand, avoid and possibly treat problems arising during dental treatment.

Pacemakers are used to inhibit harmful or symptomatic bradycardias (low heart rate). Such bradycardias usually arise from either

sinus node dysfunction (the sick sinus syndrome) or from varying degrees of atrio-ventricular block. The sinus node normally controls the heart rate, increasing it with exercise and stress and decreasing it at rest. In sick sinus syndrome the sinus node is dysfunctional, leading to a low heart rate and occasionally missed beats, or it may be unable to increase heart rate enough to meet demands during exercise. In atrio-ventricular block, the sinus node usually functions normally, but conduction of the electrical impulse from the atria to the ventricles is blocked which leads to missing heart beats and a low heart rate. In both cases the hemodynamic effect is low cardiac output which causes the symptoms of weakness, dizziness and syncope or presyncope.

Cardiac pacemakers monitor heart rate constantly. If the pacemaker observes normal cardiac rhythm, it does nothing. Immediately when the pacemaker observes slowing of the heart rate below a programmed heart rate, e.g. 70 beats per minute, it begins to give electrical impulses (pace) to the heart, thus keeping the heart rate constantly at or above the programmed lower rate. Modern pacemakers can also increase heart rate to higher rates than the lower rate. Such rate-responsive pacemakers react to many different triggers, most commonly body movements which signify that the person is moving or exercising.

Implantable cardioverter-defibrillators (ICD) are used to treat harmful, often life threatening tachyarrhythmias. Such arrhythmias usually arise in the ventricles, thus called ventricular tachyarrhythmias. In ventricular tachyarrhythmias the heart rate becomes too fast (e.g. over 200 beats/minute) to keep up sufficient cardiac output (the heart beats very fast but the beats are ineffective) leading to symptoms of dizziness, palpitations, syncope or presyncope. A ventricular tachycardia may lead to ventricular fibrillation (chaotic electric activation in the ventricles) during which there is practically no cardiac output. Such a situation causes syncope and if not treated, sudden cardiac death. An ICD continuously monitors the heart for such arrhythmias and treats them either by overdrive pacing or with an electrical shock, called cardioversion or defibrillation.

A pacemaker (and ICD) consists of a generator and lead(s). The generator contains the battery and electrical circuits, the «brains» of the pacemaker. It is a small metal box implanted subcutaneously usually in the chest wall on either side below the clavicle. They are occasionally implanted in the abdominal wall. The generator can usually be felt under the skin, even through normal clothing. Another part of the pacemaker system contain the lead or leads, usually one or two. The leads are thin wires attached to the pacemaker. They pass into the venous circulation and into the heart, either the right atrium, right ventricle, or both (in two-lead systems). The leads both monitor the heart rate and deliver the pacing impulse. In an ICD, the ventricular lead also provides the cardioversion/defibrillation current.

Problems with pacemakers that a dentist might expect to encounter are: infection related to bacteremia, and interference of electrical appliances used in dental treatment of the pacemaker or ICD patient.

Bacteremia is quite common in dental procedures, especially those causing gingival bleeding (scaling, extraction, gingivectomy) (15). As a pacemaker system represents foreign material in the circulation, there is a theoretic risk of endocarditis. An implanted pacemaker or ICD, however, is classified as a negligible risk factor for endocarditis, and thus antimicrobial prophylaxis is not recommended for such patients (16).

Modern pacemakers are rather well protected against external electric interference. Still, electric devices that generate electromagnetic fields can interfere with pacemaker function. There are several

such machines in the dental office. Data on the effects of different devices are sparse, but in an *in vitro* setting Miller et al. (17) tested fourteen dental devices in conditions mimicking a patient with a pacemaker. The electrosurgical unit, ultrasonic cleaning bath and magneto-restrictive ultrasonic scalers caused marked interference. In contrast, the amalgamator, electric pulp tester, composite curing light, dental handpieces, electric toothbrush, microwave oven, dental chair and light, ENAC ultrasonic instrument, radiography unit and sonic scaler caused no interference (17). Unipolar pacemakers are more sensitive to external interference than bipolar. The mechanism of interference is that the pacemaker observes external impulses which it erroneously interprets as heart beats and thus inhibits itself from giving pacing impulses. If the patient is pacemaker-dependent (very slow or no intrinsic rhythm) and the pacer is inhibited, asystole and syncope may follow. The pacemaker, however, is not easily damaged and usually resumes normal operation immediately when external interference ends.

In practice, the most common cause of problems is the electrosurgical unit or diathermy. Diathermy is best avoided in patients with pacemakers. If diathermy must be used, bipolar diathermy is preferred. If unipolar diathermy must be used, the ground pad should be placed so that the pacemaker or its leads do not lie within the electric field (between the ground pad and the instrument). Theoretically, electricity can «escape» into the low-resistance leads and cause a burn injury at the site on electrode-myocardium contact. Such an injury can cause pacing problems. The generator may also suffer.

Electromagnetic interference may be erroneously interpreted as a rapid arrhythmia by an ICD, followed by (inappropriate) defibrillation which may be rather painful to the conscious patient. Defibrillation is not dangerous to persons nearby, but may cause the patient to twitch, bite or move suddenly, which may cause problems during treatment (12). Thus, it is commonly recommended that if diathermy is to be used, ICD devices should be programmed off immediately prior to surgery and on again postoperatively (18).

Consultation with the cardiological unit responsible for the follow-up of the patients pacemaker or ICD is recommended in case problems are anticipated or observed.

Valvular disease

Valvular heart disease is the result of one or more pathological processes, including congenital heart defects (e.g. bicuspid aortic valve), ischemic heart disease, rheumatic fever, connective tissue disease (e.g. systemic lupus erythematosus, SLE), Marfan's syndrome, infective endocarditis and several others. In the industrial world incidence of rheumatic fever is decreasing, while incidence of degenerative valvular disease, associated with aging, is increasing.

Valvular disease may involve any of the cardiac valves, i.e. tricuspid, pulmonary, mitral or aortic valve. The left sided valves, however, are more often involved, mainly because of the higher pressures on the left side. Valve dysfunction may result in obstructed flow through the valve, called stenosis, or inadequate closure of the valve leading to regurgitation. A calcified valve may be both stenotic and regurgitant. Mild, nonprogressive valvular disease usually causes little or no strain to the heart. More severe progressive stenosis and/or regurgitation of a cardiac valve causes an increase in the workload of the heart, which eventually leads to heart failure if not properly treated. Such treatment is often surgical, either repair of the damaged valve or replacement of the valve by a biological or mechanical valve prosthesis.

The dental patient with valvular heart disease faces several risks: heart failure, arrhythmias and the risk of endocarditis associated

with the dental procedure (12,19). In addition, patients with valvular heart disease are often anticoagulated.

Heart failure will be assessed later in this article. A patient with heart failure due to valvular heart disease is at high risk of an adverse cardiac event. Such a patient should be referred to expert medical care before major dental procedures. On the other hand, patients planned for operative valvular treatment are often referred to a dentist to identify and treat possible oral infectious foci. Such patients, however, have been evaluated by a cardiologist and optimal medical treatment has usually been implemented.

Arrhythmias associated with valvar disease are common. Mitral valve disease is often associated with atrial fibrillation, which has been assessed previously. Chronic, medically controlled atrial fibrillation does not substantially increase the risk of complications during medical treatment.

Infective endocarditis is a serious bacterial infection involving the inner layer (= endocardium) of the heart and valves. Endocarditis usually requires two pathological processes:

- 1) a defect in the endocardium where bacteria can adhere multiply, and
- 2) bacteria in the blood, i.e. bacteremia.

While a healthy endocardium is relatively resistant to bacterial adhesion, a damaged valve may serve as a site for invasion. The situation is the same in many congenital heart defects. Bacteremia, on the other hand, is common in many dental procedures (15). Guidelines with detailed description of both the heart defects and bacteremia causing procedures and need for antibiotic prophylaxis have been published and should be followed (16). A review of these guidelines is beyond the scope of this article.

In summary, any significant valve defect increases the risk of bacterial endocarditis. Previous endocarditis and a valve prosthesis carry a high risk. Congenital heart defects, even surgically corrected, also carry a high risk. Significant bacteremia can be caused by any dental procedure with gingival bleeding. Especially dental extraction, scaling and gingivectomy carry a high risk. Patients at risk should be given prophylactic antibiotics, usually amoxicillin, before dental procedures. For details, see Dajani et al. (16).

Anticoagulated patients

Temporary or permanent anticoagulant treatment is commonplace in cardiac patients. Severe heart failure, atrial fibrillation, valvular prostheses, ischemic heart disease, stroke, deep vein thrombosis and pulmonary embolism are among the most common indications for anticoagulant treatment. Warfarin is most commonly used and its dosage and efficacy is monitored by the international normalized ratio (INR) measured from regular blood samples. For a healthy person without anticoagulant treatment, the INR = 1. Anticoagulant treatment usually targets to an INR between 2.0–3.0. In certain high-risk situations (e.g. a mechanical mitral valve prosthesis), higher INR values 2.5–3.5 (–4.0) may be required.

Most evidence indicates that dental surgical procedures, such as extractions or limited periodontal surgery can be performed without modifying INR levels except in extreme circumstances (12,19,20–22). Local hemostatic measures to control bleeding in anticoagulated patients should be used. These include atraumatic surgical technique, adequate wound closure, pressure application and topical clotting agents (12). Oral rinsing with tranexamic acid can also be used.

The indication for anticoagulation should be known. Many indications allow brief discontinuation of anticoagulant treatment without a substantial increase in the risk of thrombotic events (e.g.

chronic atrial fibrillation) should it be considered necessary. On the other hand, anticoagulant treatment should in general not be discontinued in patients with mechanical valve prostheses. Close collaboration with the patients physician is recommended in these matters.

Warfarin treatment may interact with several drugs, causing derangement of anticoagulant treatment. Many antibiotics such as erythromycin, metronidazole, cephalosporins; antifungal agents, such as miconazole and fluconazole may cause an increase in anticoagulation, leading to the risk of serious bleeding. A decrease in anticoagulative effect has been demonstrated with other antibiotic agents. When prescribing medication to a patient using warfarin, it is recommended that a national drug formulary is referred to, and extra INR measure taken if needed.

Many patients with vascular disease also use acetosalicylic acid and/or clopidogrel. These agents, especially if used together, may increase bleeding risk. The indication for medication should be known, and again, consultation with the patients physician is recommended if discontinuation of the medication is planned.

Hypertension

High blood pressure is a key risk-factor for cardiovascular disease and stroke as well as end-stage renal disease (23). Hypertension is defined as a systolic blood pressure of 140 mmHg or greater and/or diastolic blood pressure 90 mmHg or greater in subjects who are not taking antihypertensive medication (23). Diagnosis is based on average values obtained from at least two readings obtained on separate visits after an initial baseline measurement. Hypertension is classified into different categories, and when a patient's systolic and diastolic pressure fall into different categories, the higher category should apply (23) (Table 2).

Hypertension is treated, according to its severity, with lifestyle changes and/or drug-therapy. Previously identified hypertensive patients should have their blood pressure taken at each visit. Individuals with grade III hypertension should only receive elective dental procedures until the blood pressure is controlled (12). Emergency dental treatment should be as conservative as possible for the uncontrolled or untreated hypertensive individual. There are no contraindications, however, to provide dental care for the well-controlled patient (12).

Under most circumstances, the use of epinephrine in combination with local anesthetics is not contraindicated in the hypertensive patient unless the systolic pressure is over 200 mm Hg and/or the diastolic pressure is over 115 mm Hg (12). Individuals with blood pressure readings within normal range, but who are on anti-hypertensive medication, should also be carefully monitored (12).

Table 2. Definitions and classification of blood pressure levels (mm Hg)

Category	Systolic	Diastolic
Optimal	>120	<80
Normal	>130	<85
High-normal	130-139	85-89
Grade 1 hypertension (mild)	140-159	90-99
Subgroup: borderline	140-149	90-94
Grade 2 hypertension (moderate)	160-179	100-109
Grade 3 hypertension (severe)	180	110
Isolated systolic hypertension	140	<90
Subgroup: borderline	140-149	<90

Hypertensive crisis is divided into hypertensive emergency and hypertensive urgency. Hypertensive emergency is classified as hypertensive acute elevation of blood pressure with ongoing or impending target organ damage (24). These include hypertensive encephalopathy, acute aortic dissection, acute pulmonary oedema with respiratory failure, acute myocardial infarction/unstable angina, acute renal failure and microangiopathic hemolytic anemia. A hypertensive emergency requires immediate blood pressure reduction to prevent or limit target organ damage. Hypertensive urgencies are situations in which it is desirable to reduce blood pressure within a few hours. A hypertensive crisis is not defined by, nor is its clinical course predicted by the magnitude of blood pressure elevation (25). In a study by Zampaglione et al. (26) all patients with hypertensive crisis had a diastolic blood pressure of 120 mm Hg or more. Patients with long-standing hypertension, however, may tolerate systolic blood pressures of 200 mm Hg or diastolic pressures of 150 mm Hg, whereas a systolic pressure > 169 mm Hg or a diastolic pressure > 109 mm Hg in a pregnant woman is considered a hypertensive emergency requiring immediate pharmacologic treatment. Oral/sublingual therapy with short-acting nifedipine has been widely used in the management of hypertensive emergencies. However, the hypotensive effects of nifedipine are difficult to control and it should therefore not be used in blood pressure control of patients with hypertensive crisis. Whenever a dentist meets a patient with hypertensive crisis, the dental procedure should be postponed and the patient should be immediately sent to a hospital.

Congestive heart failure

Congestive heart failure is defined as inability of the heart to pump oxygenated blood to meet the metabolic needs of the body (12).

The most common diseases behind heart failure are hypertension, coronary artery disease, valvular heart disease, and cardiomyopathy.

Dyspnoea and fatigue associated with physical exercise are typical symptoms of left-sided heart failure. The patients may feel shortness of breath in the supine position. Heart failure can also cause nocturnal dyspnoea or non-productive cough. Right-sided heart failure, in turn, causes peripheral oedema and hepatomegaly.

The use of ACE-inhibitors has greatly lowered the rates of mortality, myocardial infarction and hospital admission for heart failure in patients with left ventricular dysfunction (27). Enalapril, lisinopril, and ramipril are examples of widely used ACE-inhibitors in patients with left ventricular heart failure. Diuretics, such as hydrochlorothiazide, furosemide and spironolactone are used in order to avoid the fluid retention and oedema associated with heart failure. Hydrochlorothiazide and ACE-inhibitors are used often as combination treatment. The use of beta-blockers (bisoprolol, metoprolol) or a combined beta- and alpha-blocker (carvedilol) is also indicated in left ventricular dysfunction.

The New York Heart Association divides congestive heart failure into four functional subclasses (Table 3). Increasing dyspnoea during minimal exercise or even at rest is a symptom of decompensated heart failure. In such patients elective dental procedures should not be started (NYHA Class III or IV). In dental emergencies of such patients, only conservative measures should be taken (antibiotics, analgesics). Uncompensated heart failure requires medical consulting.

In the well-compensated patient (NYHA Class I or II) dental care can be considered without prior medical consultation. However, appointments should be short and the dental chair should be kept in a half-sitting position (12). Appropriate sedation should be given to the anxious patient, and supplemental oxygen should be readily available (12).

Table 3. The New York Heart Association Classification of heart failure according to its symptoms

NYHA Class	
I	No limitation of physical activity
II	Slight limitation of physical activity. Fatigue, palpitations, and dyspnoea with ordinary physical activity, but no symptoms at rest
III	Marked limitation of activity. Fatigue, palpitations and dyspnoea with less than ordinary physical activity
IV	Above mentioned symptoms are present at rest and any physical effort worsens them

Pulmonary oedema is the most emergent manifestation of left ventricular heart failure. The patient rapidly develops severe dyspnoea. Wet rales are typically heard from the lungs, even without a stethoscope. The patient is often tachycardic. Without proper treatment, the patient's condition rapidly deteriorates, cough, fear and confusion will follow with pink bloody pulmonary oedema foaming from the mouth. Central cyanosis may occur as a sign of decreased oxygenation of tissues.

The patient should be put in a half-sitting position if not in a shock. Additional oxygen should be provided. If possible, oral nitrates should be given. An ambulance should be called and the patient moved to a cardiac care unit.

Stroke

Strokes can be primarily divided into ischemic or hemorrhagic strokes. Ischemic strokes result from embolic or thrombotic processes in small or large vessels. They cause long-lasting and significant disabilities. Hemorrhagic stroke results from a ruptured vessel. Intracranial hemorrhage accounts for 10% of strokes and has a very high mortality rate.

A transient ischemic attack (TIA) has similar symptoms as stroke, but the symptoms last less than eight hours, sometimes only 15–60 minutes (28). The major risk factors for ischemic stroke are hypertension, diabetes, smoking, hypercholesterolemia and atrial fibrillation.

The primary prevention of stroke is to recognize and treat these risk factors. A good control of hypertension and hyperlipidemia is a cornerstone of primary prevention of stroke. The patient is also advised to give up smoking. Patients with atrial fibrillation are anticoagulated with warfarin if they belong to the high-risk group (i.e. previous thromboembolism, hypertension, left ventricular dysfunction). In the high-risk group, the annual risk of stroke may be as high as 12% (29).

Sudden unilateral weakness and numbness or paralysis of the face, arm or leg are primary symptoms of stroke (28). The patient may have difficulties of formulating words or to understand talk. There can be difficulty in breathing or swallowing. Some patients may lose control of their bowel or urinary bladder.

In hemorrhagic stroke, sudden onset of a severe headache is a typical symptom. The first symptom may also be a sudden loss of consciousness, or even sudden death.

The patient with stroke should be monitored for vital signs (breathing, pulse, blood pressure). If the patient is unconscious, the patient should be put in the supine position. An ambulance should be called immediately, since in certain cases thrombolytic therapy can be used in an ischemic stroke, and it is therefore important to move the patient to the hospital as soon as possible.

References

- Girdler Nm, Smith DG. Prevalence of emergency events in British dental practice and emergency management skills of British dentists. *Resuscitation* 1999; 41: 159–67.
- Chapman PJ. Medical emergencies in dental practice and choice of emergency drugs and equipment: a survey of Australian dentists. *Aust Dent J* 1997; 42: 103–8.
- Davies SW. Clinical presentation and diagnosis of coronary artery disease: stable angina. *Br Med Bull* 2001; 59: 17–27.
- Jowett NI, Cabot LB. Patients with cardiac disease: considerations for the dental practitioner. *Br Dent J* 2000; 189: 297–302.
- Campeau L. Grading of angina pectoris. *Circulation* 1975; 54: 522.
- Yeghiazarians Y, Braunstein JB, Askari A, Stone PH. Unstable angina pectoris. *New Eng J Med* 2000; 342: 101–14.
- Niwa H, Sato Y, Matsuura H. Safety of dental treatment in patients with previously diagnosed acute myocardial infarction or unstable angina pectoris. *Oral Surg Oral Med Oral Pathol* 2000; 89: 35–41.
- ACC/AHA 2002 guideline update for the management of patients with unstable angina and non-ST-segment elevation myocardial infarction. Summary article: a report of the American College of Cardiology/American Heart Association task force on practice guidelines (Committee on the Management of Patients With Unstable Angina). *J Am Coll Cardiol* 2002; 40: 1366–74.
- Niwa H, Sugimura M, Satoh Y, Tanimoto A. Cardiovascular response to epinephrine-containing local anesthesia in patients with cardiovascular disease. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2001; 92: 610–6.
- Vanderheyden PJ, Williams RA, Sims TN. Assessment of ST segment depression in patients with cardiac disease after local anesthesia. *J Am Dent Assoc* 1989; 119: 407–12.
- Campbell JH, Huizinga PJ, Das SK, Rodrigues JP, Gobetti JP. Incidence and significance of cardiac arrhythmia in geriatric oral surgery patients. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1996; 82: 42–6.
- Academy report. Periodontal management of patients with cardiovascular diseases. *J Periodontol* 2002; 73: 954–68.
- Kottkamp H, Hidricks G, Breithard G. Atrial fibrillation. Epidemiology, etiology and symptoms. In: Sauoud N, Schoels W, El-Sherif N, editors. *Atrial flutter and fibrillation*. New York: Futura Publishing Company Inc. Armonk; 1998. p. 133–52.
- Chua JD, Wilkoff BL, Lee I, Juratii N, Longworth DL, Gordon SM. Diagnosis and management of infections involving implantable electrophysiologic cardiac devices. *Ann Intern Med* 2000; 133: 604–8.
- Burden D, Mullally B, Sandler J. Orthodontic treatment of patients with medical disorders. *Eur J Orthodont* 2001; 23: 363–72.
- Dajani AS, Taubert KA, Wilson W, Bolger AF, Bayer A, Ferrieri P, et al. Prevention of bacterial endocarditis. Recommendations by the American Heart Association. *Circulation* 1997; 96: 358–66.
- Miller CS, Leonelli FM, Latham E. Selective interference with pacemaker activity by electrical dental devices. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1998; 85: 33–6.
- Eagle KA (Chair). ACC/AHA guideline update for perioperative cardiovascular evaluation of noncardiac surgery-executive summary. *J Am Coll Cardiol* 2002; 39: 542–53.
- Sirois DA, Fatahzadeh M. Valvular heart disease. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2001; 91: 15–9.
- Blinder D, Manor Y, Martinowitz U, Taicher S. Dental extractions in patients maintained on continued oral anticoagulant. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1999; 88: 137–40.
- Souto JC, Oliver A, Zuazu-Jausoro I, Vives A, Fontcubetas J. Oral surgery in anticoagulated patients without reducing the dose of oral anticoagulant: a prospective randomized trial. *J Oral Maxillofacial Surg* 1996; 54: 27–32.
- Jowett NI, Cabot LB. Patients with cardiac disease: considerations for the dental practitioner. *Br Dent J* 2000; 189: 297–302.

23. Guidelines Subcommittee. 1999 World Health Organization – International Society of Hypertension guidelines for the management of hypertension. *J Hypertens* 1999; 17: 151–83.
24. Varon J, Marik PE. The diagnosis and management of hypertensive crisis. *Chest* 2000; 118: 214–7.
25. Blumfeld JD, Laragh JH. Management of hypertensive crises: the scientific bases for treatment decisions. *Am J Hypertens* 2001; 14: 1154–67.
26. Zampaglione B, Pascale P, Marchisio M, Cavallo-Perin P. Hypertensive urgencies and emergencies: prevalence and clinical presentation. *Hypertension* 1996; 27: 144–7.
27. Flather MD, Yusuf S, Kober L, Hall A, Murray G, Torp-Pedersen C, et al. for the ACE-inhibitor Myocardial Infarction Collaborative. Long-term ACE-inhibitor therapy in patients with heart failure or left-ventricular dysfunction: a systematic overview of data from individual patients. *Lancet* 2000; 355: 1575–81.
28. Nunn P. Medical emergencies in the oral health care setting. *J Dent Hyg* 2000; 74: 136–51.
29. August M. Cerebrovascular and carotid artery disease. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2001; 92: 253–6.

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