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Dent Clin N Am 50 (2006) 483–491

THE DENTAL
CLINICS
OF NORTH AMERICA

Ischemic Heart Disease: Dental Management Considerations

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Ischemic heart disease (IHD) is among the most common medical problems in the general population. The Framingham Heart Study revealed that for people who reach the age of 40, 49% of men and 32% of women show clinical manifestations of ischemic heart disease during their lifetime. Although the death rate from IHD has declined since 1975, the number of deaths due to this disease remains high [1]. Since IHD is so prevalent, all health care providers managing dental patients should strive to stay up to date with respect to IHD. Though dentists are not expected to diagnose IHD in patients under their care, heart disease in a patient can jeopardize the safe delivery of dental care. The importance of understanding IHD gains even more significance for oral-maxillofacial surgeons assessing the anesthetic needs of their patients.

This article is designed to update dental professionals with respect to IHD. The article describes risk factors that have been well documented and also discusses factors that have recently been linked to promoting IHD. Information about risk factors helps dentists better determine the risk of cardiac problems, particularly in patients with little or no history of clinically apparent coronary problems.

This article then goes on to discuss the major problems due to IHD, namely angina and myocardial infarction. A brief review of the pathophysiology of each entity is given. Then the article provides the critical steps the dental provider should take to determine the degree of control of each problem. This prepares the dentist for deciding if modifications to routine dental

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doi:10.1016/j.cden.2006.06.002 *dental.theclinics.com*

care are needed and, if so, what those modifications should be. Finally, the discussion of angina and myocardial infarction reviews the protocols for acutely managing each of these problems should they occur in the dental office.

Risk factors for IHD

Dental professionals managing patients with potentially clinically significant IHD are wise to take note of the presence of major risk factors for IHD in their patients [2]. Factors related to family history, gender, and age cannot be modified. Family history of IHD is especially important because the presence of IHD at an early age in a patient's parents significantly increases the risk of IHD in the patient and the risk that IHD will occur at a relatively young age. Also, recent studies show an even stronger correlation of IHD with siblings [3]. So questioning a patient about IHD in their brothers and sisters is useful, particularly once they reach middle age.

Gender as a risk factor has its greatest impact between ages 25 and 64, when men have a three times greater incidence of cardiovascular disease and fivefold higher mortality rate than women of a similar age [4]. This difference decreases dramatically once women are in their 60s and older. Thus, it is particularly important to question male patients as young as age 25 about symptoms of IHD, while for women without other risk factors such questions become more critical as they reach menopausal years.

The most critical modifiable risk factors include total serum cholesterol levels, systolic and diastolic blood pressure (BP), cigarette smoking, and diabetes mellitus [5]. Total cholesterol $\geq$240 mg/dL is closely associated with the development of IHD [6]. Other forms of dyslipidemia that are relevant include elevated low-density lipoprotein cholesterol levels, low high-density lipoprotein (HDL) levels, or increased total-to-HDL cholesterol ratio, hypertriglyceridemia, and increased levels of lipoprotein a [7]. Fortunately, lipid lowering drugs, such as the statins (eg, atorvastatin) and fibrates (eg, fenofibrate), are typically able to partially or fully correct serum lipid abnormalities and stabilize or even reverse coronary atherosclerosis [8]. Therefore, determining a patient's current control of serum lipids is useful.

Similarly, hypertension is a risk factor that can be controlled. Systolic BP of $\geq$140 or diastolic BP $\geq$90 increases the likelihood of IHD [5]. Although usually asymptomatic, high BP is routinely detected in patients who see health care providers on a regular basis and in most cases treatment can be effective in normalizing BP. In this situation the dental professional can easily monitor a patient's BP to determine if there are compliance or other problems causing failure in BP control. For persistently elevated BP, the possibility of IHD increases.

Smoking has long been recognized as a health risk factor. Women who smoke $>$19 cigarettes per day are six times more likely to have IHD and

three times more likely to have a myocardial infarction than women who have never smoked [9]. A dentist should thus determine a patient's smoking history and intervene if a smoking addiction is present.

Diabetes is the other major modifiable risk factor and recent reports confirm that tight control of serum glucose levels decreases the risk of IHD in patients [10]. Thus determining the degree of control of a patient's diabetes is important when assessing risk.

Other IHD risk factors useful for the dental professional are abdominal obesity, lack of regular exercise, lack of regular inclusion of fruits and vegetables in the diet, excessive ethanol use, excess psychologic stress, and laboratory findings of either elevated levels of C-reactive protein, cystatin C, or microalbuminuria [5,11–13].

Finally, emerging evidence identifies other factors that point toward increased risk for IHD. These include levels of homocysteine; the presence of left ventricular hypertrophy; collagen vascular diseases, especially rheumatoid arthritis and systemic lupus erythematosus; and the finding of coronary artery calcifications detected by electron beam or multidetector row computed tomography [5,14,15].

Even risk factors that can be controlled, are not always controlled. Failure to control such risk factors can be due to patient compliance problems or lack of proper medical management. Studies show that IHD risk factors are undertreated and under-controlled in many regions of the world, including North America [16].

Once the dental professional has a full picture of a patient's IHD risk factors and how well each is controlled, he or she can better gauge the patient's likelihood of safely undergoing planned dental interventions.

Angina

Angina as a symptom has several common manifestations, the most frequently reported being a substernal squeezing or pressure sensation in the chest. The symptom typically presents when one's heart rate increases to the point at which coronary artery oxygenated blood supply cannot keep pace with myocardial oxygen requirements. This is usually due to atherosclerotic lesions narrowing the caliber of coronary arteries. In rarer cases coronary artery spasms may be the cause (eg, Prinzmetal's angina).

Pathophysiology of atherosclerosis is an insidious process that typically takes decades to worsen to the point of causing signs or symptoms. The term is derived from the Greek words for hardening (sclerosis) and gruel or the accumulation of lipid (athere). The process is localized to the inner wall of arteries with a predisposition to form at locations of "disturbed" blood flow, such as points where arteries branch. Atherosclerotic lesions begin with the deposition of lipoproteins in the intimal layer of the affected artery. The lipoprotein particles, such as low-density lipoprotein, then seem to permit the accumulation of monocytes and lymphocytes in the intimal layer.

The monocytes then differentiate into macrophages, which slowly become lipid-laden foam cells. These cells eventually die and leave a necrotic lipid-rich element behind in the arterial wall. These lipid-containing areas calcify to varying degrees. At the same time, smooth muscle cells in the arterial wall are stimulated to migrate into the intimal layer, where they can proliferate. Meanwhile, microvessels invade the affected area, which can cause intraplaque hemorrhages. A fibrous cap that faces the interior of the artery eventually covers the atherosclerotic lesion.

As atherosclerosis progresses, it enlarges and begins to encroach upon the lumen of the artery. Usually up to 40% of the circumference of the intimal layer of the artery must be involved before the caliber of the lumen begins to be compromised. Eventually, the lesion is large enough to obstruct blood flow. Initially, this obstruction occurs only at times of demand for increased flow, such as when heart rate increases, causing anginal symptoms. Over time, the obstruction can become large enough to compromise blood flow at all times, leading to angina even at rest.

The process of atherosclerosis is slowed or even reversed when levels of high-density lipoproteins are relatively high. This lipoprotein has the ability to help remove lipids from arterial walls. Estrogen protects against the progress of atherosclerosis for reasons that are unclear. The protection may stem from estrogen's effects on high-density lipoprotein levels, but this does not fully explain the benefits of estrogen.

Diabetes mellitus, particularly if poorly controlled, is known to accelerate the atherosclerotic process. This seems to be due to its detrimental effects on lipoprotein profiles [17].

Myocardial infarction, like angina, is related to the formation of atherosclerotic lesions. However, the cause of myocardial infarction differs from that of angina. Angina stems from a fixed partial obstruction, which is why the symptom can come and go without myocardial damage. In contrast, a myocardial infarction stems from an ulceration or rupture of the fibrous cap covering the plaque. The exposed contents of the plaque triggers the deposition of platelets and the clotting of adjacent blood at the site of an atherosclerotic lesion. If this clotting is significant, a thrombus forms. If and when the thrombus becomes large enough, it obstructs blood flow and myocardial tissue downstream from the obstruction begins to suffer ischemia. This occurs no matter what the level of myocardial oxygen demand. The ischemia produces angina. If the ischemia lasts long enough, the affected myocardium begins to infarct, usually beginning in the subendocardial layer that is furthest from coronary capillary blood flow, but not in contact with intracardiac blood. If the thrombus spontaneously dissolves, is mechanically removed by insertion of a stent, or is pharmacologically lysed with thrombolytic agents, the ischemia resolves. At that point, the duration of the ischemia determines the extent of lasting myocardial injury [17–19].

Angina-like symptoms may or may not induce a patient to seek medical attention. So the absence of a history of diagnosed IHD in a patient

does not eliminate the need to query a patient about the presence of angina. This is especially important in patients with one or more IHD risk factors [2]. Obviously, a patient giving a history consistent with angina should be directed to seek immediate medical evaluation before dental care proceeds.

Dental patients known to have angina or a history of myocardial infarction are approached in a similar manner. The dental professional needs to determine the level of each patient's IHD control. Stable angina has a predictable quality. The activities necessary to cause an anginal event are usually known. For example, a patient usually knows how many stairs he or she can climb before angina occurs. In addition, once a patient's angina appears, it usually stops once the patient ceases the activity that triggers it. Also, if nitroglycerin is required to relieve the angina, the amount is similar with each event. If the patient's angina is predictable in these ways, then the patient's angina is considered stable. One additional check needed before proceeding is to determine any change in IHD risk factors. Thus, a patient with probable stable angina needs to be questioned on control of lipids, blood pressure, and diabetes. A useful and commonly used system for classifying angina is the Canadian Cardiovascular Society Angina Classification System (Table 1). This classification system improves communication among treating doctors [18].

The most dangerous circumstance for a dental provider to face in a patient with known IHD is unstable angina. A patient's angina is considered unstable if it is changing for the worse in some parameter. Thus, if angina is now occurring more frequently, appears at lower levels of exertion than in the past, requires larger doses of nitrates for relief, or relief from angina takes longer than in prior episodes, the patient's angina is considered unstable. This form of angina can imply that atherosclerotic lesions have worsened, the oxygenation of the patient's blood has become compromised, or that the myocardial baseline oxygen demand has increased, as in the case of a new dysrhythmia, such as atrial fibrillation, or worsening congestive

Table 1
Canadian cardiovascular society angina classification system

Class	Common description of when angina is present
I	Ordinary physical activity does not cause angina. Only occurs with strenuous or prolonged exertion.
II	Slight limitation of ordinary activity by angina. Walking uphill or climbing >1 flight of stairs brings on angina.
III	Marked limitation of ordinary physical activity by angina. Angina occurs when walking on level ground or climbing <1 flight of stairs.
IV	Unable to carry on any physical activity without anginal symptoms and may be present at rest.

Data from Firriolo FJ, Hupp JR. Angina pectoris. In: Hupp JR, Williams TP, Firriolo FJ, editors. Dental clinic advisor. St. Louis: Mosby Elsevier; 2006. p. 20.

heart failure or hypertension. The presence of angina at rest is also considered unstable angina and may even be a prodrome to an impending coronary artery thrombosis and subsequent myocardial infarction. In any circumstance where a dentist feels a patient's angina is unstable, immediate referral to the patient's physician is indicated.

A patient with stable angina can usually undergo routine dental care safely. The important factors to remember are the usual triggering event to angina and how to manage the angina should it occur. The major instigator of angina in the dental setting is tachycardia provoked by fear or pain. Most dental professionals work hard to reduce fear of dental work and pain on all patients. However, for patients prone to angina, reducing fear and pain is even more important. Thus, angina-prone patients who experience greater than normal stress from the thought of dental work benefit from the administration of oral anxiolytics or nitrous oxide. Dentists trained to administer intravenous sedation might also consider its use for these patients. All other forms of anxiety control should be considered as well. Similarly, pain control is critical for lessening the chances of angina in IHD patients. In most circumstances, this means producing and maintaining profound local anesthesia in the surgical area. Again, this is typically important to all patients, but in patients subject to angina, the unexpected sensation of sharp pain causes the endogenous release of epinephrine. This causes tachycardia, which may lead to angina. Therefore, the anesthesia drug selected needs to be of sufficient duration so that dense anesthesia is maintained even if the planned procedure goes longer than expected. This can be done via the use of longer-acting anesthetics, such as bupivacaine, or by using an anesthetic containing a vasoconstrictor. The commonly used vasoconstrictors, such as epinephrine and neocobefrin, can both cause a rise in heart rate. It is thus prudent to use vasoconstrictors in concentrations of 1:100,000 and 1:20,000, respectively, or less. It is also important to avoid intravascular administration so careful aspiration before any injection is a sound practice. Also, small amounts of these vasoconstrictors are absorbed from extravascular deposits. This is why some experts advise limiting the amount of vasoconstrictor to no more than 0.04 mg of epinephrine or about two cartridges of an anesthetic containing epinephrine at the 1:100,000 concentration [17]. The request by some physicians to eliminate all use of vasoconstrictors in the local anesthetic given to angina-prone patients ignores the important role that vasoconstrictors have in prolonging the effects of the local anesthetic. If the local anesthesia wears off prematurely and a patient suddenly experiences pain, the release of endogenous epinephrine will far exceed the amount and rate of absorption from anesthetic injection sites. Part of the confusion here may be that physicians routinely use epinephrine in its 1:1000 concentration and in most settings this concentration would not be safe for angina patients. But the 100-fold decrease in concentration of vasoconstrictors used in dental anesthetics totally changes the situation.

The routine use of oxygen in patients subject to angina makes little sense physiologically since the room air that patients breathe usually has hemoglobin oxygen saturation close to 100%. Thus, supplement oxygen is only indicated in patients who have pulmonary conditions that impair blood oxygenation or are having dental work at high altitudes.

Since angina is a symptom, a patient in the ambulatory setting should not be put into a sedated state that impairs his or her ability to report angina. In addition to reminding the patient to report the appearance of angina should it occur, the dentist should regularly check the patient's heart rate and BP during long appointments. Any significant rise in either may indicate the need to conclude dental care on that day and reschedule another appointment.

Angina patients may be on various drugs to manage their IHD and related problems. So protocols to manage patients with diseases such as hypertension, congestive heart failure, and diabetes, discussed elsewhere in this article, should be implemented. In addition, many patients with more serious problems associated with angina or with angina due to coronary artery spasm may be on long-acting nitrates or calcium channel blockers, both of which can precipitate postural hypotension.

In the event that a patient reports a feeling of discomfort in the chest, left shoulder, arm, or neck, and IHD is a reasonable possibility based on risk factors, all dental care should cease. The patient with a prior history of angina can usually tell if the discomfort feels like angina. If the patient has no history of angina or if the patient identifies the pain as different from the angina he or she normally experiences, the dentist should have an assistant check the patient's vital signs. The patient should be placed in a semireclined position. A heart with an impaired oxygen supply will usually underperform, so blood pressure may fall even though the heart rate is normal or rises to compensate for the falling pressure. If the diagnosis of angina is clear or the dentist has strong suspicions angina is present, a dose of nitroglycerin should be given. If a spray form is available, give two metered sprays intraorally. If nitroglycerin in tablet form is being used, a 0.3- to 0.6-mg tablet must be dissolved under the patient's tongue [20]. The patient's response to the nitroglycerin should be monitored. If angina is still present after 5 minutes and the patient's BP is above 90/50, another dose of nitroglycerin is indicated. Again, monitor the patient's response for 5 minutes. If BP is still above 90/50 and the patient still reports angina, a third and final nitroglycerin dose should be administered.

In addition to monitoring the patient, emergency assistance from emergency medical technicians should be summoned, particularly after failure of the patient to improve after the first nitroglycerin dose. The search for oxygen to give to a patient experiencing angina should not distract from providing nitroglycerin. However, if oxygen is available, it can be given via nasal or oral-nasal mask. The administration of oxygen will not stop angina, but may assist in filling the lungs with oxygen-enriched air should the patient suffer a cardiorespiratory arrest.

If the prior measures fail to stop the patient's angina, the dentist should presume the patient is experiencing a myocardial infarction. The concurrent appearance of nausea, marked bradycardia, or hypotension are signals a myocardial infarction may be occurring. Since successful recovery from an acute myocardial infarction usually depends upon immediate coronary artery stenting or the administration of thrombolytic agents, immediate transfer to a facility able to offer these therapies is critical [21].

Myocardial infarction

The dental management of a patient with a prior history of a myocardial infarction differs little from that used for patients prone to angina. The ability to stop or limit the damage caused by the coronary artery thrombosis that produces a myocardial infarction has changed the approach to such patients. In the past when myocardial infarctions caused areas of the heart to fully infarct, become necrotic, and then, if the patient survived, scarify, physicians sought to limit noncardiac surgical interventions on these patients for at least 6 months. This was due to the finding that by 6 months after a myocardial infarction patients had recovered as well as they could, so any further delay in elective surgical care was meaningless. Nowadays, if a patient's myocardial infarction is recognized early and rapid interventions are successful, myocardial damage can be minimal and there is little reason to delay even elective surgical procedures, including dental procedures. However, it is now inherent in the evaluation of the patient with a myocardial infarction history for a dental provider to learn enough about the circumstances surrounding the myocardial infarction to decide when care can commence. In most cases, a simple call to the office of the patient's physician will suffice. Unless the patient suffered serious myocardial damage, there is little need to delay needed dental care.

As mentioned previously, once the dentist decides to go forward with dental care, the same considerations should be taken for the myocardial infarction patient as are taken for angina patients. In addition, because most patients with a prior history of myocardial infarction are placed on potent platelet inhibitors, such as clopidogrel, extra care needs to be taken when doing surgery likely to cause significant bleeding. In those circumstances, extra measures should be taken to promote local hemostasis. Local hemostasis can be promoted by, for example, using procoagulant materials, such as collagen or topical thrombin; applying direct pressure for longer than usual; injecting vasoconstrictor containing local anesthetic directly into the surgical site; and using sutures to close the wound even in situations where such sutures would normally not be required.

IHD is a common enough problem that most dental professionals who manage adult patients will see IHD patients on a daily basis. Fortunately, for those patients whose diseases and risk factors are well controlled, dental

care can proceed safely, as long as the straightforward steps covered in this article are followed.

References

[1] Lloyd-Jones DM, Larson MG, Beiser A, et al. Lifetime risk of developing coronary artery disease. Lancet 1999;353(9147):89–92.

[2] Glick M, Greenberg BL. The potential role of dentists in identifying patients' risk of experiencing coronary heart disease events. J Am Dent Assoc 2005;136(11):1441–6.

[3] Murabito JM, Pencina MJ, Nam BH, et al. Sibling cardiovascular disease as a risk factor for cardiovascular disease in middle-aged adults. JAMA 2005;294(24):3117–23.

[4] Jousilahti P, Vartianinen E, Tuomilehto J, et al. Sex, age, cardiovascular risk factors, and coronary heart disease. Circulation 1999;99(9):1165–72.

[5] Wilson PW, Culleton BF. Overview of the risk factors for cardiovascular disease. Up To Date 2005;14.1:1–29.

[6] Khot UN, Khot MB, Bajzer CT, et al. Prevalence of conventional risk factors in patients with coronary artery disease. JAMA 2003;290(7):898–904.

[7] Ashen MD, Blumenthal RS. Low HDL cholesterol levels. N Engl J Med 2005;353(12): 1252–60.

[8] LaRosa JC, Grundy SM, Waters DD, et al. Intensive lipid lowering with atorvastatin in patients with stable coronary disease. N Engl J Med 2005;352(14):1425–35.

[9] Prescotte E, Hippe M, Schnohr P, et al. Smoking and the risk of myocardial infarction in women and men: longitudinal population study. BMJ 1998;316(7137):1043–7.

[10] DCCT/EDIC Study Research Group. Intensive diabetes treatment and cardiovascular disease in patients with type I diabetes. N Engl J Med 2005;353(25):2643–53.

[11] Danesh J, Wheeler JG, Hirschfield GM, et al. C- reactive protein and other circulating markers of inflammation in the prediction of coronary heart disease. N Engl J Med 2004; 350(14):1387–97.

[12] Shilipak MG, Sarnak MJ, Katy R, et al. Cystatin C and the risk of death and cardiovascular events among elderly persons. N Engl J Med 2005;352(20):2049–60.

[13] Yan LL, Daviglus ML, Liu K, et al. Midlife body mass index and hospitalization and mortality in older age. JAMA 2006;295(2):190–8.

[14] Pai JK, Pischon T, Ma J, et al. Inflammatory markers and the risk of coronary heart disease in men and women. N Engl J Med 2004;351(25):2599–610.

[15] Hoffmann MH, Shi H, Schmitz BL, et al. Noninvasive coronary angiography with multislice computed tomography. JAMA 2005;293(20):2471–8.

[16] Bhatt DL, Steg PG, Ohman EM, et al. International prevalence, recognition, and treatment of cardiovascular risk factors in outpatients with atherothrombosis. JAMA 2006;295(2): 180–9.

[17] Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. N Engl J Med 2005;352(16):1685–95.

[18] Kolodgie FD, Gold HK, Burke AP, et al. Intraplaque hemorrhage and progression of coronary atheroma. N Engl J Med 2003;349(24):2316–25.

[19] Libby P. The pathogenesis of atherosclerosis. In: Kasper DL, Fauci AS, Longo DL, et al, editors. Harrison's principles of internal medicine. 16th edition. New York: McGraw-Hill; 2005. p. 1425–30.

[20] Malamed SF. Angina pectoris. In: Medical emergencies in the dental office. 5th edition. St. Louis (MO): Mosby; 2000. p. 437–51.

[21] Selwyn AP, Braunwald E. Ischemic heart disease. In: Kasper DL, Fauci AS, Longo DL, et al, editors. Harrison's principles of internal medicine. 16th edition. New York: McGraw-Hill; 2005. p. 1434–44.