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Management of the Hypertensive Dental Patient

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ABSTRACT Hypertension is a common malady and a harbinger of such diseases as heart attack and stroke. Because millions of Americans are not aware they are hypertensive or it is not adequately controlled, dentists can contribute significantly to national health by screening their patients. Dentists must also be cognizant of the implications high blood pressure has for dental practice. Specific treatment recommendations include limiting dental care in patients with severe hypertension, reducing stress, and periodically monitoring blood pressure.

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Hypertension is the most prevalent systemic disorder in the United States. One-third of American adults, or about 70 million individuals, have high blood pressure.[1] A similar number have prehypertension, which predisposes them to developing clinical hypertension in the future, and still others have one or more risk factors for it. Worldwide, hypertension may afflict as many as 1 billion individuals and be responsible for 7.1 million deaths per year.[2] It is inevitable that most dentists will be called upon to treat hypertensive patients on a weekly basis. The purpose of this review is to provide the practitioner with an understanding of (1) the basic pathophysiology of hypertension, (2) how hypertension and its management can affect dental treatment, and (3) steps the dentist can take to provide optimal care for the hypertensive dental patient.

Hypertension

Hypertension is defined in adults by a mean systolic blood pressure of 140 mm Hg or greater, a mean diastolic blood pressure of 90 mm Hg or greater, or when the individual is taking antihypertensive medication for blood pressure control.[3] Hypertension can be characterized as either primary or secondary. Primary hypertension is the term used for high blood pressure where no specific etiology can be found. When a specific cause is identified, the increase in blood pressure is called secondary hypertension.

The Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure now classifies blood pressure values into four categories (**TABLE 1**).[4] The designation of normal blood pressure as less than 120/80 mm Hg and prehypertension as 120-139/80-89 mm Hg reflects recent data on the progressive development of high blood pressure with age, its profound negative influence on cardiovas-

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TABLE 1

Classification of Blood Pressure for Adults

BP classification	SBP (mm Hg)	DBP (mm Hg)
Normal	<120	And <80
Prehypertension	120-139	Or 80-89
Stage 1 hypertension	140-159	Or 90-99
Stage 2 hypertension	≥160	≥100

SBP: systolic blood pressure; DBP: diastolic blood pressure
From Chobanian, et al.[4]

cular health, and the resultant increased emphasis on preventing its development.

PATHOPHYSIOLOGY

Primary hypertension, also known as essential or idiopathic hypertension, comprises 95 percent of all cases of high blood pressure. It is the default diagnosis when all other causes for the disorder have been excluded. As is the case with obesity, that other scourge of modern life, primary hypertension may not be a disease in the classic sense. It is instead most likely a consequence of homeostatic mechanisms that were appropriate in prehistorical times but are now problematic given the sedentary lifestyle and salt- and calorie-rich diet common to Western nations.[5] Indeed, the term essential hypertension, reflecting the once widespread belief that the condition was necessary to support normal tissue perfusion, may have held some truth in previous epochs.

An increase in arteriolar constriction and total peripheral vascular resistance is frequently observed in primary hypertension.[6] Despite the resultant heightened myocardial afterload, cardiac output remains in the normal range. Increased sympathetic nervous system activity helps explain the arterial vasoconstriction, as well as the augmented myocardial function and activated renin-angiotensin axis that collectively maintain the cardiac output. Over time, the increased hemodynamic load, and some of the mediators supporting it, cause the heart and blood vessels to hypertrophy and become less compliant.

Although the exact cause of primary hypertension remains elusive, a unifying theme has been proposed whereby any one of several inciters — sympathetic nervous system activity, renin-angiotensin axis stimulation, or hyperuricemia — causes renal vasoconstriction.[7] Decreased renal blood flow then activates the body's mechanisms to increase blood pressure and volume. Variables that influence these inciters include genetics, physical and emotional make-up, diet, physical activity, and environmental conditions.

Secondary hypertension accounts for only 5 percent of all cases of high blood pressure. This diagnosis is important, however, because it suggests that effective treatment of the underlying disease process will lead to its cure and because it notifies the clinician of an associated health problem with medical significance.[6] **TABLE 2** lists the leading causes of secondary hypertension along with some of their identifying characteristics. Other instigators of secondary hypertension include pregnancy (preeclampsia), various neurologic disorders (brain tumors, dysautonomias, etc.), and vascular abnormalities (e.g., arteriovenous shunts).

Many prescription and nonprescription drugs and several herbal products can cause secondary hypertension or exacerbate primary hypertension.[4] For example, immunosuppressive agents such as cyclosporine, tacrolimus, and methylprednisolone increase blood pressure in up to 90 percent of solid-organ transplant recipients.[8] Cigarettes and most nicotine-replacement products (but not the transdermal patches), decongestants, amphetamine-like drugs, and other sympathomimetic agents likewise promote hypertension. The hypertensive effects of ephedra (ma huang) caused the Food and Drug Administration in 2004 to place limits on its sale. Nonsteroidal antiinflammatory drugs and cyclooxygenase 2 inhibitors do not cause hypertension by themselves but are capable of destabilizing blood pressure control in patients with hypertension.[9]

CONSEQUENCES OF HYPERTENSION

End-organ damage is a natural consequence of chronic, uncontrolled hypertension. The heart, brain, and kidneys are most at risk. Hypertrophy of arterial muscle and other arteriosclerotic changes in small arteries that occur in response to high blood pressure and related mediators such as angiotensin II begin to impair perfusion of affected tissues. The development of ventricular hypertrophy and atherosclerosis of large arteries further affects blood flow to end-organs by reducing cardiac output and arterial elasticity. In the kidneys, the reduced renal tissue perfusion promotes local ischemia, tubular injury, and interstitial inflammation. Eventually, loss of adequate renal blood flow and associated ischemic changes can reduce glomerular filtration to the point of clinically evident renal insufficiency and end-stage renal failure. As renal failure progresses, there is increased fluid retention and accelerated release of vascular mediators.

TABLE 2

Primary Disorders Associated With Secondary Hypertension

Disorder	Signs and Symptoms
Acromegaly	Enlargement of hands, feet, tongue, jaw; headaches; fatigue; visual problems
Alcoholism	Alcohol-seeking behavior; intoxication; confusion; unsteadiness; skin capillary enlargement
Aldosteronism	Hypernatremia; hypokalemia; fatigue; thirst
Coarctation of the aorta	Decreased or delayed femoral pulses; cold; legs; heart murmur; abnormal chest radiograph
Cushing's syndrome	Weight gain; moon face; dorsal hump; truncal obesity; fatigue; weakness; hirsutism; amenorrhea; purple striae; hypokalemia
Drug and herbal therapy*	Medication history
Hyperparathyroidism	Kidney stones; osteoporosis; depression; lethargy; muscle weakness
Hyperthyroidism	Heat intolerance; weight loss; palpitation; tachycardia; exophthalmos; tremor
Hypothyroidism	Fatigue; muscle weakness; weight gain; alopecia
Obstructive uropathy	Pain; reduced urine output; hyperkalemia
Pheochromocytoma	Headaches; diaphoresis; palpitation; tachycardia
Renal parenchymal disease	Renal insufficiency; edema; elevated BUN, creatinine; proteinuria
Renovascular disease	Systolic/diastolic abdominal bruits; elevated plasma renin
Sleep apnea	Snoring; daytime somnolence; obesity

BUN: blood urea nitrogen

* Drugs and herbs that may increase blood pressure include acetaminophen (long-term use); antidepressants (e.g., bupropion, desipramine, venlafaxine); appetite suppressants (e.g., phentermine, sibutramine); bromocriptine; corticosteroids (e.g., dexamethasone, prednisone); epoetin alpha; ergotamine; immunosuppressants (e.g., cyclosporine, tacrolimus); liquorice; mineralocorticoids (e.g., fludrocortisone); monoamine oxidase inhibitors (with tyramine-containing foods); nicotine; nonsteroidal antiinflamatory drugs and cyclooxygenase (COX)-2 inhibitors (e.g., celecoxib); sex steroids (e.g., estrogen, testosterone); St. John's wort; sympathomimetic amines and related stimulants (e.g., amphetamine, methylphenidate, pseudoephedrine); and yohimbine.

Cardiac problems develop when the heart begins to decompensate from the increased workload and decreased perfusion of the myocardium. The heart enlarges as it increasingly depends on Starling's principle (in which stretching of cardiac muscle up to certain limit increases contraction force). Myocardial perfusion is further impaired by the increased intramural tension and by the accompanying tachycardia, and angina pectoris may occur when the oxygenation of tissues cannot keep up with the demand. Signs and symptoms of left and right congestive heart failure — including dyspnea and ascites — develop as blood backs up, respectively, into the pulmonary and systemic venous vasculatures. Arrhythmias may develop in response to the myocardial enlargement and ischemia. Sudden death from coronary thrombosis is a common terminal outcome.

Vascular pathology affecting the central nervous system includes arteriosclerotic changes and the development of microaneurysms. Occipital headaches are common early manifestations of hypertension. Episodes of dizziness or syncope, representing transient ischemic attacks, may herald the danger of stroke. Hemorrhagic strokes follow the breakage of weakened arteries or microaneurysms; ischemic strokes are the result of atherosclerosis and thrombus formation in, or embolization of, cerebral arteries.

HYPERTENSIVE CRISIS

Hypertensive crisis is a term used to indicate an acute, severe increase in blood pressure and is often defined by a systolic blood pressure of 180 mm Hg or more, and/or a diastolic pressure of 120 mm Hg or more.[3] Because individuals with chronic hypertension can tolerate higher blood pressures than their normotensive counterparts, emergency treatment of a hypertensive crisis is determined more by the rate of increase in blood pressure and its associated signs and symptoms rather than by absolute pressure values.[10] When the acute hypertension is accompanied by ongoing or impending target organ damage, the crisis becomes a true

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hypertensive emergency. In this situation, the acutely elevated blood pressures progressively damage blood vessels, which may then precipitate cerebrovascular accidents (hemorrhagic or ischemic) or myocardial infarction, or lead to acute forms of encephalopathy, congestive heart failure, renal failure, or ocular damage. Signs may include pulmonary edema, acute angina, vision loss, and seizures. Hypertensive emergencies require immediate hospitalization and treatment.

A hypertensive urgency occurs when there is no associated target organ dysfunction. Although the patient may experience severe headache and/or anxiety, shortness of breath, or epistaxis, the terms crisis and urgency are misnomers in the sense that hospitalization is usually not necessary. Instead, early outpatient medical treatment with oral medications and close follow-up by a physician is suitable. In the dental office, activation of emergency medical services is the preferable choice to ensure proper treatment when there is any question about the patient's condition.

Implications for the Dentist

Because only one-third of individuals with hypertension have their blood pressure under control to the degree recommended by the JNC, the average dentist will, perforce, regularly encounter dental patients with uncontrolled hypertension.[4] Almost half of these patients will not even know they have the disorder. Thus, the dentist should take the blood pressure of all new patients regardless of their medical history.[11] The dentist can expect to uncover untreated or inadequately treated hypertension in about one of every five adult patients tested. Because of the almost linear increase in the prevalence of hypertension with age, and the possibility that secondary hypertension may develop at any time, recall patients should also have their blood pressure checked.

Significant benefits accrue from the participation of dentists in screening their patients for hypertension. Current statistics indicate that one death will be prevented for every 11 patients the dentist identifies as hypertensive who then receive treatment sufficient to reduce their mean systolic blood pressure by 12 mm Hg for 10 years.[12] Acutely, identification of the dental patient in hypertensive crisis may be life saving for the patient and protect the dentist against malpractice litigation.

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ORTHOSTATIC HYPOTENSION

Some patients with high blood pressure, especially the elderly, and those with diabetes mellitus or autonomic dysfunction, have an increased risk of developing acute hypotension when they stand after having lain in the dental chair for a length of time.[7,13] In addition, orthostatic hypotension is a known side effect of certain drugs used to treat high blood pressure, including the α-adrenergic blocking drugs, β-adrenergic blocking drugs, and other sympatholytic agents. The use of conscious sedation to reduce operative stress can exaggerate postural changes in blood pressure. Physical injuries in the form of broken bones and facial lacerations are common sequelae of the impaired consciousness and balance caused by the lack of effective cerebral circulation. In most cases, having the patient gradually assume more vertical postures after dental treatment prevents orthostatic hypotension.

XEROSTOMIA

Dry mouth is a common side effect of many drugs, including agents used in the management of high blood pressure. Centrally acting antihypertensives, diuretics, sympatholytics, angiotensin-converting enzyme inhibitors, and calcium-channel blockers have all been identified as causing xerostomia.[14,15] These effects are generally additive, so most patients with clinically significant xerostomia are usually on multiple medications. Oral complications associated with xerostomia include angular cheilosis, altered taste (dysgeusia), candidiasis, difficulty swallowing (dysphagia), painful, burning tongue (glossodynia and glossopyrosis, respectively), thirst, and increased caries. Because there is considerable variation in the degree to which different drugs cause dry mouth in specific patients, a useful strategy for alleviating persistent xerostomia is for the physician to seek an alternative antihypertensive regimen with less inhibition of salivation. Salivary stimulants in the form of gums or tablets containing nonsucrose sweeteners and/or citric acid may be helpful, along with oral moisturizers (plain water, salivary substitutes).[14,16] Sialogogues (pilocarpine, cevimeline) taken before meals can help provide the necessary saliva for mastication; topical fluorides are beneficial in reducing caries.

GINGIVAL OVERGROWTH

Calcium-channel blocking drugs, especially nifedipine, have been associated with gingival overgrowth.[15,17] The reaction occurs most prominently on the labial surfaces of the incisors. The enlargement is not a true hyperplasia but involves proliferation of noncellular

TABLE 3

Antihypertensive Drug Interactions in Dentistry

Antihypertensive	Dental Drug	Possible Effect	Recommended Action
Diuretics (e.g., furosemide, hydrochlorothiazide	NSAIDs (e.g., ibuprofen)	Decreased renal blood flow, loss of antihypertensive effect	Warn patient about possible interaction; use alternate analgesic if hypertensive response
	Epinephrine, levonordefrin	Transient hypokalemia	Consult physician; avoid use if patient is hypokalemic
β-Adrenergic receptor blockers (e.g., propranolol, metoprolol)	NSAIDs (e.g., ibuprofen)	Decreased renal blood flow, loss of antihypertensive effect	Warn patient about possible interaction; use alternate analgesic if hypertensive response
Nonselective β-blockers (e.g., propranolol)	Epinephrine, levonordefrin	Hypertension and bradycardia	Use cautiously; monitor blood pressure
ACE inhibitors (e.g., captopril)	NSAIDs (e.g., ibuprofen)	Decreased renal blood flow, loss of antihypertensive effect	Warn patient about possible interaction; use alternate analgesic if hypertensive response
Centrally acting α_2-adrenergic receptor agonists (e.g., clonidine)	CNS depressants, opioid analgesics	Increased CNS depression	Use cautiously
Peripheral adrenergic neuron blockers (e.g., guanethidine)	Epinephrine, levonordefrin	Increased cardiovascular responses to vasoconstrictor	Use cautiously; monitor blood pressure

ACE: angiotensin-converting enzyme
Modified from Yagiela JA, Turner RN.[21]

connective tissue elements of the gingiva. Local plaque accumulation and inflammation are involved in the etiology of gingival overgrowth, and meticulous oral hygiene after surgical resection of the enlarged tissues is effective in preventing recurrence. Switching to an alternative antihypertensive drug addresses the problem successfully without relying on the patient's skill with the toothbrush.

OTHER REACTIONS

Diuretics, ACE inhibitors, and several β-adrenergic blocking drugs may cause lichenoid reactions.[15,18] These lichen planus-like lesions are best managed by having the physician change the antihypertensive therapy. If that cannot be done, topical corticosteroids can be used to ameliorate the condition. ACE inhibitors have also been associated with loss of taste and a burning sensation in the mouth. More importantly, both ACE inhibitors and angiotensin II receptor blockers have been implicated in causing angioedema of the oral cavity in up to 1 percent of patients taking the drug.[19] Although edema of the tongue, uvula, and soft palate is most common, laryngeal edema is more serious because of the potential for loss of airway. The angioedema does not appear to be dose related; although most reactions occur early in therapy, angioedema has also developed after months of treatment. Epinephrine, antihistamines, and corticosteroids are used in the emergency management of acute reactions; switching to an alternative class of antihypertensives is required to prevent future attacks.

DENTAL DRUG THERAPY

Perhaps the most controversial issue regarding dental treatment of the hypertensive patient is the use of vasoconstrictors in local anesthetic solutions. (Opinion is fairly unanimous that gingival retraction cord impregnated with epinephrine should not be used in these patients.) The primary concern is the possibility of hypertensive crisis in uncontrolled hypertensive individuals and in hypertensive patients taking drugs that interact with vasoconstrictors. A systematic literature review of the cardiovascular effects of epinephrine during dental treatment found little evidence of risk in uncontrolled hypertensive patients, with mean maximum increases in systolic blood pressure of 15.3 mm Hg when 2 to 4.5 mL of lidocaine with epinephrine was injected, and 11.7 mm Hg when anesthesia without vasoconstrictor was used.[20] Corresponding values for normotensive individuals were 5.0 and 5.0 mm Hg. The diastolic blood pressure was little affected in

any group, nor were there any cases of adverse cardiovascular events.

These results suggest that one to three cartridges of 2 percent lidocaine with 1:100,000 epinephrine or its equivalent can be used safely in patients with uncontrolled hypertension. They are consistent with the fact that low concentrations of epinephrine decrease peripheral vascular resistance by selectively stimulating vasodilatory β_2-adrenergic receptors.[21] Doses beyond three cartridges, which are more likely to activate vasoconstrictive α-adrenergic receptors, have not been well studied. Because epinephrine once absorbed into the circulation is quickly converted to inactive metabolites, injections can be separated over time (e.g., by 30 minutes) to avoid cumulative effects of larger aggregate doses. Little information exists regarding the cardiovascular effects of levonordefrin in uncontrolled hypertensive patients; this fact, plus the greater tendency for levonordefrin to increase peripheral vascular resistance, argues against its use in these patients.

Specific drug interactions involving antihypertensive medications and drugs the dentist may use or prescribe are outlined in **TABLE 3**.[22] Interactions with epinephrine generally occur acutely after administration. In the case of hypertensive reactions (which may result in reflex bradycardia), injecting a small amount of epinephrine (e.g., the equivalent of 1 mL 1:100,000 solution), and monitoring the blood pressure five minutes later will assist the dentist in determining the safety of subsequent doses. Interactions with NSAIDs prescribed by dentists are slower to develop. Here, advising the patient to monitor their blood pressure at home will help uncover the rare case in which substitution with alternative analgesic therapy is warranted.

Management of the Hypertensive Patient

As a matter of course, dentists must provide for the dental needs of their hypertensive patients, including those with uncontrolled high blood pressure, associated diseases (cardiovascular and otherwise), and complex therapeutic regimens. In addition to the specific issues discussed previously regarding these patients, the dentist must be able evaluate these patients, deliver care safely, and respond appropriately to any episode of acute hypertension that might develop.

HYPERTENSIVE CRISIS as a result of invasive dental care gains clinical relevance with Stage 2 hypertension.

PATIENT EVALUATION

Patients with hypertension are often identified by their medical history. Hypertension is obviously an important medical disorder and should be specifically addressed in the medical history. Hypertension may also be identified by the medicines the patient reports taking. (Some patients with controlled hypertension, reasoning they are normotensive with treatment, do not indicate they have high blood pressure.) Medical diseases that are causative for secondary hypertension or increased cardiovascular risk should also be considered in evaluating the patient for hypertension. Consultation with the physician should be sought whenever the dentist needs clarification about the patient's physical status and/or ability to tolerate stress.

Dental patients with hypertension are also often identified by measurement of their blood pressure. Recording of a blood pressure in excess of 139 mm Hg systolic or 89 mm Hg diastolic in the dental office does not automatically mean the patient is hypertensive. The blood pressure recording may be elevated for technical reasons, which is discussed later, or because the patient is fearful or otherwise emotionally stressed in the dental office. Physician referral for all patients with high blood pressure in the dental office is necessary to help ensure they have the opportunity get their blood pressure under control if it is truly high. The actual diagnosis of hypertension will be made by the physician, often after multiple blood pressures have been measured over several weeks to months. Patients who suffer from white coat syndrome, a condition in which their blood pressures are consistently increased in the medical or dental office but not elsewhere, may have to keep a log of blood pressures taken during the course of daily life for an accurate diagnosis to be made.[23]

In general, the dentist should consider the severity of high blood pressure and the existence of other cardiovascular risk factors in making treatment decisions. Although Stage 1 hypertension poses serious cumulative danger to the patient over the course of years to decades, it is usually of little immediate consequence, and regular dental care is appropriate, with the possible addition of steps to reduce operative stress. Hypertensive crisis as a result of invasive dental care gains clinical relevance with Stage 2 hypertension. **TABLE 4** provides reasonable guidelines for treatment decisions based on the patient's blood pressure and the presence of related medical risk factors.[18,24]

TABLE 4

Dental Treatment Recommendations According to Severity of Hypertension

SBP	DBP	MRF	Recommendation
120-139	80-89	Yes/no	Routine dental care OK; discuss BP guidelines
140-159	90-99	Yes/no	Routine dental care OK; consider stress reduction protocol; refer for medical consult
160-179	100-109	No	Routine dental care OK; consider stress reduction protocol; refer for medical consult
160-179	100-109	Yes	Urgent dental care OK; consider stress reduction protocol; refer for medical consult
180-209	110-119	No	No dental treatment without medical consult; refer for prompt medical consult
180-209	110-119	Yes	No dental treatment; refer for emergency medical treatment
≥210	≥120	Yes/no	No dental treatment; refer for emergency medical treatment

MRF: medical risk factor (e.g., history of myocardial infarction, angina pectoris, high coronary disease risk, recurrent stroke prevention, diabetes mellitus, renal disease).
Modified from Merin RL,[23] after Herman WW, et al.[18]

BLOOD PRESSURE RECORDING

Accurate blood pressure recordings are fundamental to management decisions based on the patient's blood pressure. Although they are simple to obtain, attention to certain details is required. The mercury sphygmomanometer is the gold standard for measuring blood pressure, but concerns about mercury contamination have led to a greater use of (1) aneroid sphygmomanometers, which need calibration every six months to ensure accuracy, and (2) electronic devices, some of which are inexpensive but not very accurate, and others that are both expensive and accurate. Each of these devices requires a properly sized and fitted cuff. Size matters, because a cuff that is too small for the extremity will yield falsely high readings and oversized cuffs will give results that are too low. The length of the cuff bladder should equal at least 80 percent of the circumference of the extremity.[4] Although other recommendations have suggested that the cuff width equal 40 percent of the extremity circumference, studies have shown that this criterion does not produce accurate results and that a long cuff length is more important than a correct cuff width in providing accurate results.[25,26] Cuffs that are placed loosely or below the level of the heart will overestimate the blood pressure; the opposite occurs with cuffs that are positioned above the heart.

Falsely high blood pressures may be obtained if the patient has had exercise, nicotine, or caffeine within the past 30 minutes or is not allowed to sit quietly for five minutes before the recording is taken.[4] A quick estimate of systolic blood pressure can be obtained by feeling for the return of an arterial pulse as the cuff is quickly deflated from a high initial pressure. Then, the cuff can be reinflated to a value about 25 mm Hg above the estimated systolic blood pressure and then slowly deflated. Adequate precision is achieved with a bladder deflation rate of 2 to 3 mm Hg/second.

Sphygmomanometry estimates systolic and diastolic blood pressures by listening for the Korotkoff sounds, which result from turbulent blood flow within an artery squeezed by the inflated cuff. Systolic pressure is detected by the initial Korotkoff sound, which appears as blood first squirts past the cuff bladder, and the diastolic pressure is detected by the final Korotkoff sound, which heralds disappearance of discrete auditory pulsations. The disadvantage of this method is its inherent subjectivity. Error can occur because of deficiencies in sound transmission and hearing by the recorder. It is recommended that at least two recordings be taken and then averaged to determine the patient's blood pressure.[4]

STRESS REDUCTION

Hypertensive patients receiving dental care can benefit from treatment modifications designed to reduce physical and psychological stress.[27] Psychological support is especially important for individuals with significant dental fear. Treatment appointments should be scheduled so that the patient will not have to wait in the reception area and the dentist is free to give the patient undivided attention. Pharmacologic anxiety relief in the form of oral (e.g., with diazepam or triazolam) or inhalation sedation (with nitrous oxide and oxygen) should be considered. If fasting guidelines are to be employed, patients should be specifically told to take their antihypertensive drugs as normally scheduled.

Reducing the number of procedures to be performed automatically lowers stress. For example, performing periodontal surgery on two quadrants versus four should, on average, reduce by half the amount of local anesthetic solution that must be administered (and thus the number of injections), the duration of surgery, and the extent of postopera-

tive pain. Controlling pain is an essential treatment goal. Hypertensive patients should be instructed to advise the dentist if they are experiencing discomfort during the procedure, and that they can ask for a "time out" should they so desire. Responding to the patient's needs without delay is vital for the nervous patient to develop a sense of control over the situation.

Blood pressure recordings can provide an independent measure of the stress a patient is feeling and the effectiveness of strategies used to ameliorate it. A baseline blood pressure should be taken each time the hypertensive patient is about to undergo treatment. Subsequent recordings should be taken whenever the dentist suspects the patient's condition may have changed, such as when the patient requests a time out from treatment.

EMERGENCY MANAGEMENT

Acute elevations in blood pressure — for example, to a systolic blood pressure of 180 mm Hg or more or a diastolic blood pressure of 110 mm Hg or more — warn that the patient is experiencing some form of difficulty. Commonly, the underlying problem involves pain, intraoral, or otherwise. The patient may also be anxious, possibly, but not necessarily, as a result of discomfort. Other reversible causes of acute hypertension in the dental office include respiratory depression (in oversedated patients), a full urinary bladder, and vasoconstrictor drug interactions. The dentist should suspend the procedure as soon as the acute hypertension is discovered and seek treatable causes. It may be advisable to terminate the procedure if the blood pressure remains elevated. The patient should not be discharged home, however, until the pressure increase has abated or the patient's physician is consulted.

If a hypertensive crisis, as defined previously, develops, the dentist must stop working and devote full attention to the patient's physical status. Signs and symptoms indicative of a hypertensive emergency should be sought, and the blood pressure should be taken at frequent intervals (e.g., every three to five minutes). Emergency medical services should be requested if the dentist suspects a true emergency may be occurring. Telephonic consultation with the physician can be helpful in making this decision. With no evidence of developing organ damage, the patient may be referred for immediate follow-up care by the patient's physician, if available, or a local emergency medical clinic.

Unless the dentist is formally trained in the pharmacologic management of acute hypertensive reactions, as part of an oral surgery or dental anesthesiology residency, he or she should refrain from administering antihypertensive drugs without consultation from the patient's physician because of the dangers inherent in their use, including tissue ischemia caused by an excessive or too rapid reduction in blood pressure. In one exception to this rule, the dentist should administer sublingual nitroglycerin if the patient's symptoms includes anginal chest pain and there is no specific contraindication to its use.[21,27]

CONCLUSION

Hypertension is a common disorder that must be considered when developing an optimal dental treatment plan. With appropriate treatment modifications — including medical consultation when indicated, effective stress reduction, intraoperative blood pressure monitoring, careful local anesthetic and analgesic use, attention to intraoral manifestations associated with antihypertensive drugs, and avoidance of positional changes that may predispose the hypertensive patient to orthostatic hypotension — all but the most extremely hypertensive patients may be safely treated in the dental office. Detecting hypertension in undiagnosed patients and helping to ensure effective blood pressure control in all patients with hypertension are life-saving contributions that can be made by the treating dentist. ■■■■

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