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Dental Management of Patients with Hypertension

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Surprisingly, there is little if any data to indicate that treating a patient with hypertension alone increases the risk for adverse outcomes or complications. Most dentists, however, realize that hypertension often leads to cardiovascular disease, renal disease, and strokes, which are conditions that increase the risk for complications, both during and after dental care. Oral and systemic side effects may also arise from the medicines used to treat hypertensive patients. This article reviews the current thought on the pathogenesis, diagnosis, and treatment of hypertension, and provides guidance on how best to treat patients with this common medical problem.

Physiology

Blood pressure (BP) is determined by how much blood the heart pumps (ie, cardiac output) and by the resistance to blood flow in the vascular system. Cardiac output in turn is determined by how often the pump contracts (ie, heart rate) and by the amount of blood ejected during each beat (ie, stroke volume). High blood pressure, therefore, results from either narrow inflexible arteries, an elevated heart rate, increased blood volume, more forceful contractions, or any combination of the above. BP is never constant; it peaks right after the ventricles contract (systole) and reaches its low point as the ventricles fill (diastole). Mean arterial pressure (MAP) is calculated by multiplying the diastolic BP by two, adding the systolic BP, and dividing by three. Diastolic BP is multiplied by two as, on average, the heart spends roughly twice the amount of time in diastole as in systole.

The long-term regulation of BP is controlled predominantly by the kidneys through their variable release of the enzyme renin. Renin goes on to

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cleave angiotensinogen to angiotensin 1, which is converted by angiotensin converting enzyme (ACE) to angiotensin 2. Angiotensin 2 causes vasoconstriction (ie, elevated vascular resistance) and stimulates the release of aldosterone, an enzyme that increases sodium resorption in the kidney. Increased sodium resorption raises blood volume, again elevating the BP.

In response to fear (such as impending extraction), exercise, and other environmental stimuli, the autonomic nervous system (ANS) activates its "fight or flight" response and can, within seconds, raise the blood pressure. The sympathetic arm of the ANS releases norepinephrine, which acts on the beta-1 receptors of the heart to increase the rate and force of contractions. This is the positive chronotropic and inotropic effect. Norepinephrine release also triggers the alpha-1 receptors on the vasculature to initiate vasoconstriction, again raising pressure.

In summary, over a given day or week, MAP readings are primarily under control of the kidneys, while fear and stress can provoke the ANS to quickly and dramatically raise values.

Interestingly, some patients respond to severe psychologic stress (such as a dental injection) by first activating the sympathetic arm of the ANS, but then have an exaggerated parasympathetic response. The acetylcholine transmitter of the parasympathetic system causes the heart to slow, leading to a dramatic fall in BP and a resultant syncopal event, such as fainting. These patients usually regain consciousness after being placed in a supine position.

Diagnosis

Many common medical conditions now have guidelines to aid in their diagnosis and treatment. These guidelines are typically formulated by recognized experts from multiple disciplines. In 2003, the National High Blood Pressure Education Program promulgated their latest recommendations for hypertension. This was the seventh revision by the Joint National Committee on the Prevention, Detection, Evaluation and Treatment of High Blood Pressure and is known as the JNC-7 Report [1]. Their latest BP classification is summarized in Table 1.

Table 1
Adult classification

Classification	Systolic BP	Diastolic BP
Normal	<120	<80
Prehypertension	120–139	or 80–89
Stage I hypertension	140–159	or 90–99
Stage II hypertension	>160	or >100

Note that a patient with a "normal" systolic blood pressure (less than 120) would be classified with Stage I hypertension if the diastolic blood pressure is 95.

According to these guidelines, there are now only four BP classifications for the adult patient. The correct diagnosis and classification is predicated on the accurate recording of BP, something often determined incorrectly. As classically taught, the patient should be sitting in a stress-free environment for at least 5 minutes before assessment, and should not have smoked, exercised, or eaten for the previous 30 minutes [2]. For many patients, stress-free and dental care are mutually exclusive, and these patients may have elevated blood pressure secondary to fear and anxiety. This is sometimes called ''white coat'' hypertension. Such patients (and their BP) may respond well to a calm, reassuring chair-side manner (iatrosedation) or to pharmacological sedation itself. Patients with labile ''white coat'' hypertension are best diagnosed with BP readings taken over 24 hours. When pressures are taken over this period and averaged, many ''hypertensives'' are found to be normal, eliminating the need for treatment. Interestingly these 24-hour ambulatory readings sometimes reveal ''masked'' hypertension. That is, those patients actually have low BP in the doctor's office, but when BP is measured over a 24-hour period, the patients demonstrate that, in fact, they have hypertension [3].

Common operator mistakes leading to faulty BP readings involve using improper-size cuffs or applying the cuff too loosely or too tightly. The inflatable bladder portion of the cuff should encircle approximately 80% of the arm and the cuff should be appropriately secured and centered over the brachial artery. A cuff too small or applied too loosely will give a falsely elevated reading. Conversely, a cuff that is too large or applied too tightly will yield spuriously low values. Another possible validity issue involves the use of electronic, ''stethoscope-free'' arm or wrist devices. These are certainly convenient and used by many dentists, but their accuracy should be frequently calibrated against the gold standard mercury sphygmomanometer used in conjunction with the bell of a well-made stethoscope [4]. Note that the units for BP are still in millimeters of mercury. This element still has uses, therefore, even in ''mercury free'' dental offices.

There are several good reasons why dentists should take BP readings on their patients. The first is that many patients are unaware that they have hypertension. Thus, the dentist may be first to detect the problem. It is well known that many patients avoid medical or dental care unless serious symptoms arise and dental pain often prompts the first visit to a health care provider in years. The dentist is thus in the position of improving the public's health by informing patients that they have hypertension and referring them for evaluation and treatment. It is estimated that 30% of all patients have undiagnosed high BP [2]. Early detection and treatment reduces the likelihood of the serious consequences of hypertension, namely cardiovascular disease, retinopathy, renal disease, and strokes. Hypertension by itself is asymptomatic. That is why it is known as the ''silent killer'' with symptoms resulting only when such organs as the eyes, heart, or kidneys become damaged. Why and how chronic elevated BP causes damage is a most interesting

question. Untreated, hypertension simply makes the heart work harder, yielding eventually to congestive heart failure. Hypertension, via mechanisms not well understood, also can induce atherosclerosis and the analogous problem in the kidney, nephrosclerosis. These vascular problems correlate with increased risk of heart attack, stroke, loss of vision, and renal failure [5,6].

Hypertensives, when compared with normotensives, are less sensitive to painful stimulation, including electric pulp testing. The more hypertensive the patient is, the less he or she reacts to painful stimulation [7]. Discovering the physiologic link between the two may help researchers favorably modulate the variables for both BP and pain perception.

Besides simplifying BP classification to four categories, the JNC-7 also stressed the importance of systolic hypertension. They found systolic BP >140 in people over 50 was a more important risk factor than elevated diastolic pressure. Also sobering was their discovery that for each systolic/diastolic BP rise of 20/10 mm Hg, the risk doubles for development of cardiovascular disease [1].

Detecting hypertension enables dentists to improve the overall health of his or her patients. A corollary to this public health mission is to emphasize the fact that as many as 50% of patients fail to take their medicines as prescribed [2]. Having an elevated BP in a dental office often reminds already diagnosed patients of the importance of taking their medicines.

Taking BP readings also allows the dentist to document vital signs before giving a local anesthetic. Several organizations, including the American Association of Oral Maxillofacial Surgeons, in its Parameters of Care Documents, and the American College of Prosthodontists, in its Local Anesthesia Parameter, recommend this practice [8,9]. The tacit rationale is that some patients, because they have more pressing health care needs, should not receive the injection and subsequent invasive dental care if their BP is too high. From a risk-management perspective, a dentist may increase his or her malpractice exposure if no preoperative vital signs are recorded and a patient suffers a medical complication following injection of the local anesthetic. The argument could be made: ''Had the dentist taken the BP before the procedure, he or she would have discovered the patient's readings were elevated, would therefore not have given the anesthesia, and the heart attack, stroke, or seizure could have been prevented.'' Several articles report serious medical complications in hypertensive patients following dental care, but no direct cause-and-effect relationship has ever been proven linking the administration of local anesthetics in dental care [10,11] and the development of the medical problems listed above. Dentists, however, can practice defensively by simply documenting BP values on all patients before administering any drug. Of interest is Astra Zeneca, in its product insert for Xylocaine, recommends taking vital signs after each local anesthetic injection [12].

Finally, should a medical emergency occur, having pretreatment ''baseline'' vital signs is important. A patient whose BP is significantly lower or

higher during an emergency than his or her baseline value is of great concern [13].

Treatment

It is estimated that up to 50 million Americans have hypertension. Some epidemiologists feel that it is the number one public health problem in developed countries [5]. Unfortunately, around 90% to 95% of all cases of hypertension have no known etiology and, therefore, treatment involves medicines, usually for life. These patients are said to have primary or essential hypertension. The remaining 5% to 10% have known identifiable causes, such as faulty heart valves, catacholamine secreting tumors, increased thyroid hormones, oral contraceptives, and, most commonly, renal-vascular disease. These patients have secondary hypertension and sometimes the cause can be removed, resulting in a cure. The use of oral contraceptives is the number one cause of secondary hypertension in women and chronic use of nonsteroidal anti-inflammatory agents raises BP values an average of 5 mm [2].

For cases of hypertension with no known etiology, genetics certainly play a predisposing role. African Americans have the highest incidence of hypertension of all people in the entire world, while recent data suggest that Puerto Rican Americans have the highest death rate from hypertension-related disease (154 per 100,000) [14]. Those with genetically predisposed hypertension respond unfavorably to obesity, stress, and a high sodium diet, while other patients can handle these without any negative BP effects. Nonetheless, the initial treatment of hypertension involves diet restrictions, regular exercise, weight control, and low limits on the use of alcohol. The Dietary Approaches to Stop Hypertension plan has been well studied and recommends the intake of fruits, vegetables, and low-fat dairy products, with restriction of sodium to less than 2.4 g a day. These lifestyle modifications often cost nothing; rather, they typically save money and possess few contraindications or side effects. Still many fail to achieve desirable BP with these modifications and therefore are prescribed medicines to bring the BP within the therapeutic range.

The goals of medical management of hypertension are to reduce pressure into at least the prehypertension range ($<140/90$), but what difference does this really make? Research indicates that proper management reduces the incidence of myocardial infarctions by 20% to 25%, stroke by 35% to 40%, and heart failure by around 50%. Achieving and maintaining ideal body weight lowers systolic BP reading by 5 to 20 points with exercise lowering it an additional 5 to 9 mm of Hg. The BP goal for patients with existing end-organ disease or diabetes is 130/80 mm Hg or below [1]. Contemporary data indicates that ideal therapy should strive to not only lower 24-hour mean blood pressure values, but to also reduce dramatic BP swings. Having

stable 24-hour BP values seems to reduce the rate of heart attacks and strokes [15,16].

Drugs used to treat hypertension

As the following list reveals, the medicines used in the treatment of hypertension are diverse and ever increasing. Many patients are prescribed more than one drug, and publishing a complete list of all agents is beyond the scope of this article. The dentist is encouraged to check with current references when questions arise about medicines, their side effects, and drug-to-drug interactions.

Diuretics (eg, hydrochlorothiazide, triamterene, furosemide) are the most researched class of drugs and work to reduce BP by both decreasing vascular resistance and by reducing blood volume. For most patients, the first drug given for the treatment of high BP is a diuretic.

Beta-blockers (eg, propranolol, sotolol) are also often frequently prescribed and decrease BP by reducing the rate and force of contractions. They are often used on patients with coexisting cardiac issues, such as angina and histories of myocardial infarctions. Selective beta-blockers (eg, atenolol, metoprolol) preferentially target and block the beta-1 receptors on the heart, avoiding the beta-2 receptors of the bronchioles. These bronchiole receptors react to sympathetic stimulation by relaxing smooth muscles, yielding bronchodilation. Nonselective beta-blockers are therefore contraindicated in patients with asthma, as their inhalers (beta agonists) are "blocked" by their antihypertensive medicine.

ACE inhibitors (eg, captopril, enalapril) work by retarding the renin-angiotensin system. They produce vasodilatation by interfering with the conversion of angiotensin 1 into angiotensin 2. With a reduction in angiotensin 2, vasoconstriction decreases, lowering BP.

Calcium channel blockers (eg, amlodipine, nifedipine, diltiazem) typically reduce all the variables in BP by minimizing calcium influx into smooth and cardiac muscle. They decrease total peripheral resistance and often slow the heart rate and decrease the force of contraction.

Alpha blocking agents (eg, prazosin, terazosin) impede the sympathetic transmitter norepinephrine from binding to receptors in the arterioles, leading to vasodilatation.

Direct acting vasodilators (eg, nitroglycerin, minoxidil) work independent of the ANS to relax vascular smooth muscle.

Some agents (eg, methyldopa, clonidine) act in the central nervous system to decrease sympathetic nervous system output.

A relatively new class of drugs is the class of angiotensin 2 receptor blockers (eg, losartin, telmisartan), which work by preventing this vasoconstrictor from binding on smooth muscle sites in the arterials, thus promoting vasodilatation.

Certainly, each class of antihypertensive has its own side effects. Table 2 lists systemic side effects of interest to dentists and Table 3 lists oral side effects if any. Finally, Table 4 lists some common drug interactions between these hypertensive agents and medicines dentists may use or prescribe [17–19].

Some side effects deserve special attention. Gingival overgrowth can be seen with most calcium channel blockers, with an incidence from 1.7% to 38% [20]. Nifedipine is the most notorious, and while surgery may temporarily reduce the painful bleeding gums, drug cessation is usually necessary for cure. Here the dentist must communicate with the physician to switch to another class of drug or another channel blocker.

Xerostomia is another side effect common to virtually all antihypertensives, with patients taking more than one drug the most severely affected. Switching drugs rarely helps; they all produce dry mouth. Class V caries, burning tongue, decreased removable prosthesis retention, and difficulty chewing and swallowing are all known complications of xerostomia [20]. Treatment involves topical fluoride and, possibly, systemic medicines, such as pilocarpine or cevimeline. Many patients simply carry bottled water and chew sugarless gum. These are simple but effective remedies. Dentists should also advise their patients to avoid alcohol-containing mouthwashes, as these exacerbate dry mouth.

Lichenoid drug reactions, a condition clinically indistinguishable from lichen planus, occur from many antihypertensives. Switching medicines may help, but biopsy is warranted if the lesions fail to regress. Treatment for the lichenoid lesions is necessary only if they become symptomatic. The high-potency steroid clobetasol or the antimetabolite cyclosporine are both generally effective [21].

Orthostatic hypotension occurs to varying degrees in all patients taking antihypertensive medicines. Dentists are encouraged to slowly return patients to an upright position following dental care, and to have them sit on the edge of the chair for 30 to 60 seconds before standing. Another precaution, noted in JNC7, involves the interaction between antihypertensive drugs and general anesthetic agents. Although this ‘‘problem’’ will

Table 2
Systemic side effects of hypertensive drugs

Drug	Systemic side effect
Diuretic	Orthostatic hypotension, blood dyscrasia
Beta blockers	Orthostatic hypotension, blood dyscrasia
ACE inhibitors	Orthostatic hypotension, renal failure, neutropenia
Calcium channel blockers	Orthostatic hypotension, renal failure
Alpha blockers	Orthostatic hypotension
Direct-acting vasodilator	Blood dyscrasia, orthostatic hypotension
Central-acting agents	Rebound hypertension if agent stopped abruptly, dizziness
Angiotensin 2 receptor blocker	Cough, muscle cramping, orthostatic hypotension

Data from Refs. [17–19].

be the responsibility of the anesthesiologist, dentists should be aware that intra- and postoperative BP control is challenging on medically treated hypertensive patients who must receive their dentistry under general anesthesia [22].

Finally, the drug interaction between nonsteroidal anti-inflammatory drugs and most antihypertensive agents only begins to occur after 2 to 3 weeks of daily use of nonsteroidal anti-inflammatory drugs. Indomethacin seems to be the nonsteroidal anti-inflammatory drug most likely to reduce BP medication effectiveness.

Hypertension, sleep apnea, and dentistry

Sleep apnea is now a frequently diagnosed condition characterized by partial or complete obstruction of the upper airway while sleeping. This leads to less restful sleep and daytime somnolence, and contributes to the development of hypertension. Oral appliances that advance the mandible during sleep help some patients with obstructive sleep apnea [23] and a well-designed study recently revealed that such appliances could actually help lower BP [24]. A 4-week trial had patients wear a device that on average advanced the mandible 7 mm. A modest but significant reduction in diastolic BP (1.8 mm) versus the control was discovered but no change in the systolic BP was found. It will be of great interest to see what effect these appliances have on the future treatment of apnea-associated hypertension.

Blood pressure values and recommendations for dental care

Above what BP values should we not provide dental treatment? Should the number differ depending on whether we are giving emergency care for a swollen symptomatic patient versus elective care, such as in-office bleaching?

Table 3
Oral side effects of antihypertensive medicines

Drug	Oral adverse side effects
Diuretics	Dry mouth, lichenoid reaction
Beta blockers	Dry mouth, taste changes, lichenoid reaction
ACE inhibitors	Loss of taste, dry mouth, ulceration, angioedema
Calcium channel blockers	Gingival enlargement, dry mouth, altered taste
Alpha blockers	Dry mouth
Direct-acting vasodilators	Facial flushing, possible increased risk of gingival bleeding and infection
Central-acting agents	Dry mouth, taste changes, parotid pain
Angiotensin 2 antagonists	Dry mouth, angioedema, sinusitis, taste loss

Data from Refs. [17–19].

Table 4
Drug interactions with antihyperintensive agents

Drug	Interactive drug	Effect
Diuretics	NSAIDs	Decreased antihypertensive effect
Diuretics	Barbiturates	Orthostatic hypertension
Diuretics	Fluconazole	Elevated fluconazole levels
Beta blockers	NSAIDs	Decreased antihypertensive effect
Beta blockers (nonselective)	Epinephrine	Transient BP elevations
Beta blockers	Local anesthetics	Decreased rate of amide metabolism
Beta blockers (nonselective)	Bronchodilators	Decreased response to inhaled bronchodilator
ACE inhibitors	NSAIDs	Decreased antihypertensive effect
Calcium channel blockers	Benzodiazepines	Increased sedation
Calcium channel blockers	Parenteral anesthetic agents	Intraoperative hypertension
Calcium channel blockers	Aspirin	Increased antihypertensive effect
Calcium channel blockers	NSAIDs	Decreased antihypertensive effect
Alpha blockers	NSAIDs	Decreased antihypertensive effect
Alpha blockers	CNS depressant	Increased antihypertensive effect
Direct-acting vasodilators	NSAIDs	Decreased antihypertensive effect
Direct-acting vasodilators	Opioids	Increased antihypertensive effect
Centrally-acting agents	Epinephrine	Transient elevation in BP
Centrally-acting agents	NSAIDs	Decreased antihypertensive effect
Centrally-acting agents	Sedatives	Increased sedation
Centrally-acting agents	Opioids	Increased antihypertensive effect
Angiotensin 2 receptor blocker	Systemic antifungal	Increased antihypertensive effect
Angiotensin 2 receptor blocker	Sedatives	Increased antihypertensive effect

Abbreviations: CNS, central nervous system; NSAIDs, nonsteroidal anti-inflammatory drugs.

Data from Refs. [17–19].

Should we limit the amount of epinephrine for hypertensive patients? These reasonable questions defy simple answers. Many of us have seen patients with no prior history of hypertension present to our offices with a toothache, swelling, and BP in the 190/110 range. They are sick, anxious, desperate, and difficult to turn away just because of their elevated BP. As mentioned before, many such patients have their pressure respond favorably by simple relaxation techniques. Sounds, smells, lighting, colors, and the perception that the doctor is competent all affect anxiety and BP.

A recent article by Mishima and colleagues [25] noted how pleasant sounds of water decreased the patient's BP while, conversely, the sounds of a dental turbine increased readings. This study also noninvasively investigated cerebral blood flow and metabolism. Both changed solely in response to auditory stimulation. Certainly, playing pastoral, relaxing music and having patients go to their ''happy place'' affects BP favorably for some but not all. Others may respond to pharmacologic anxiolytic

measures, such as the use of nitrous oxide or orally administered sedatives, techniques available to most general dentists. A 2005 study by Grossman [26] looked at treating hypertensive patients with 5 mg of diazepam versus the ACE inhibitor, captopril. Patients in this study presented to the emergency room with BP readings greater than 190/100 and responded equally well to both treatments, reducing, on average, their systolic BP by an impressive 30 mm Hg, and diastolic values by 25 mm. If, however, pharmacologic anxiolytic interventions are not available or if they fail to bring the patient's BP down, what is a dentist to do? A phone consultation can be made to the patient's physician, who may have some constructive advice. Malamed [27] states, though, that many physicians are not familiar with the doses of epinephrine used by dentists, and often inappropriately give the "go ahead and treat but use no epinephrine" advice. He goes on to state that "the primary responsibility for the care of the patient rests solely in the hands of the person who performs the treatment, not the one who gives the advice." In addition, many times "emergency" patients do not have a regular physician with whom to consult, leaving the dentist to take full responsibility.

A condition termed symptomatic or malignant hypertension takes precedence over any dental care, even emergency care. Patients with this condition often present with headaches, changes in mental status, alterations in their retina and fatigue. Left untreated, blindness, acute renal failure, myocardial infarctions, and strokes may result [2]. Values typically found with symptomatic hypertension are those in the 225/125 range and immediate referral to the emergency room is indicated. These symptomatic hypertensive patients rarely seek dental care first, leading back to our question: Above which BP values should the dentist not treat? Many well-respected authors have published 180/110 for the absolute cutoff for any dental treatment [28–30] but this value may, in fact, be too high for patients who have had previous hypertensive-related organ damage, such as myocardial infarctions, strokes, or labile angina. Conversely, an otherwise healthy patient with a negative medical history with values around 200/110 may often be treated without any perioperative complications.

The concept of "how healthy is the patient," otherwise termed "risk assessment," is key in determining the likelihood of complications. The physical classification system of the American Society of Anesthesiologists (ASA) has been in use since 1941. The higher the ASA class, the more at-risk the patient is both from a surgical and anesthetic perspective [31].

ASA Class I. A normal healthy patient
ASA Class II. A patient with mild systemic disease
ASA Class III. A patient with severe systemic disease
ASA Class IV. A moribund patient who is not expected to survive without the operation

The prudent dentist might elect not to perform elective care on an ASA Class III patient whose BP is 175/105. Classifying a patient into the ASA I to IV scheme, however, is subjective even for physicians, prompting many to seek out a better risk-assessment strategy.

The concept of metabolic equivalent or METS is in vogue. One MET is defined as 3.5 mL of 0_2/Kg/min [29,32]. It essentially is a test of the patient's ability to perform physical work. Some examples are:

1 to 4 METS: eating, dressing, walking around house, dishwashing
4 to 10 METS: climbing at least one flight of stairs, walking level ground 6.4 km/hr, running short distance, game of golf
$\geq$10 METS: swimming, singles tennis, football

People with capacities of 4 METS or less are at high risk for medical complications. Those who can perform 10 METS or more are at very low risk. A person who is anxious with a BP 200/115 but can perform 10 METS of work would likely have no problems with a simple extraction.

Certainly the dentist should document BP values and refer patients with elevated numbers for prompt medical attention following the rendering of any emergency dental care.

One other variable to consider: How long will the procedure last? If the invasive procedure is to extract a mobile abscessed tooth with a predicted surgical time of 3 minutes, many doctors will proceed. Alternatively, if the plan is to extract a lone standing mandibular first molar that appears ankylosed and is suffering from acute pulpitis (making profound local anesthesia challenging), the wise provider may opt to write a prescription for antibiotics and analgesics, and reappoint or refer to a hospital dental clinic or oral-maxillofacial surgeon. Fig. 1 presents an algorithm for treating the hypertensive dental patient. The algorithm assumes no other medical contraindications, such as a recent stroke, unstable dysrhythmias, myocardial infarction, or pregnancy.

To recapitulate: Questions for the dentist to consider when deciding on dental care are:

- What is the actual BP number?
- Is therapy elective or emergent?
- Will the procedure be long or invasive?
- What is the health of the patient?
- Is there any advice from the patient's physician?

No absolute black-or-white cut-off numbers exist. The dentist must decide if the benefits of proceeding with a procedure outweigh any systemic risks.

The role that the epinephrine in the dental anesthetic has on exacerbating hypertension is also controversial. Several articles conclude that little if any cardiovascular change occurs from slow (with aspiration) administration of two to three cartridges of local anesthetic with epinephrine 1:100,000 (total

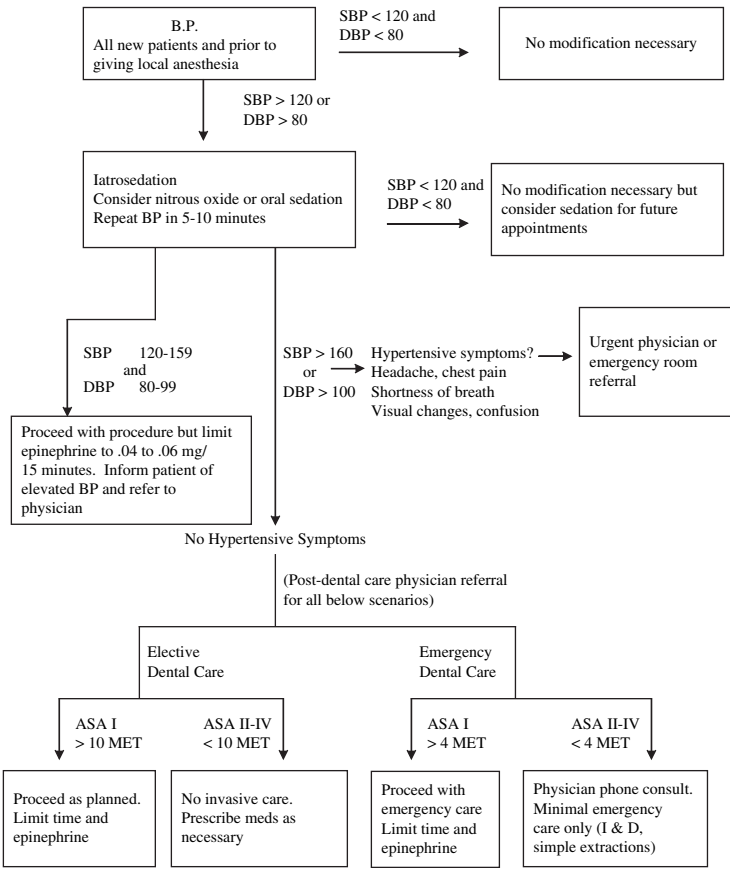

Fig. 1. Algorithm for treating the hypertensive dental patient. SBP, systolic blood pressure; DBP, diastolic blood pressure.

epinephrine dose .036–.054 mg) [33,34]. Certainly patients release their own epinephrine and other vasoactive mediators in amounts in excess of this if they are feeling pain during a procedure. A stressed patient can release up to 40 times his or her baseline catecholamine level [35]. We also know that the half-life of epinephrine is only 2 to 5 minutes [36]. It is rapidly inactivated by catechol-0-methyltransferase. Many therefore feel vasoconstrictors are not contraindicated for treating hypertensive patients, especially for painful procedures where vasoconstrictor-free local anesthetics often fail to produce profound or lasting anesthesia. This conclusion is supported in the JNC 7. Dentists should strive to limit the total quantity of circulating epinephrine, which includes that administered by the dentist in the local anesthesia and that released by the patient's adrenal medulla. Avoiding any "extra" vasoconstrictor, however, seems prudent on all compromised patients. For such patients, the dentist is thus wise to choose

a nonepinephrine-containing gingival retraction cord. Caution should be taken to avoid direct intravascular injections and the use of the periodontal ligament syringe with 1:50,000 epinephrine is ill-advised due to potential rapid epinephrine absorption [37]. Also recommended is giving local anesthetic injections for hypertensive patients one quadrant at a time, especially those with existing end-organ damage. For long procedures where multiple injections are contemplated, the dentist should take BP readings every 10 to 15 minutes throughout the procedure.

Drug interactions with epinephrine

The ability of epinephrine to react with various antihypertensive agents (and other classes of drugs) to potentially yield cardiovascular complications is the subject of much research and debate [13,17,19,20,27]. A summary of epinephrine and drug interactions follows:

Epinephrine and nonselective beta-blockers: Hypertension and a reflex bradycardia are potential consequences of this drug combination.

Epinephrine and tricyclic antidepressants: This mix also may yield acute hypertensive changes, but is more of a problem with the vasoconstrictors levonordefrin and norepinephrine, neither currently available in the United States.

Epinephrine and diuretics: Diuretics often produce hypokalemia, which is exacerbated by epinephrine use. Low blood potassium levels increase the risk for dysrhythmias.

Epinephrine and cocaine: Although sometimes difficult to obtain from the patient history, any suspicion of cocaine use should prompt the dentist to use epinephrine with extreme caution. Those two drugs together often result in BP spikes and fatal dysrhythmias. Avoiding any dental care for 24 hours following suspected cocaine use is rational.

Other than perhaps the ASA Class IV patient with a MET capacity <4, or a person who recently used cocaine, no absolute contraindication for using epinephrine in the 0.04- to 0.06-mg range exists. Rarely does a dentist ever have to give more than two cartridges of local with epinephrine at one time. If multiple quadrants of dentistry are planned, take vital signs after quadrant one is completed and, if close to baseline, proceed with the next quadrant.

The dentist's BP

Performing dentistry is often stressful, with the administration of local anesthetic being one of the most challenging times. Studies have demonstrated elevations in the dentist's BP while giving local anesthesia (systolic

BP up 24%; diastolic BP up 28%) [38]. Dentists should be mindful of their own health, and have their BP monitored frequently.

Hypertension and intraoperative bleeding

Common sense dictates that elevated BP during surgery leads to increased intraoperative bleeding. While bleeding may not be of importance during restorative dentistry, it is during oral surgery. In fact, oral-maxillofacial surgeons sometimes ask anesthesiologists to lower pressure for procedures that typically cause significant hemorrhage, such as a Le Fort osteotomy [39]. This ''hypotensive anesthesia'' has been shown to reduce overall blood loss. In the context of dentists operating in their office under local anesthesia, this purposeful reduction in BP is not feasible, but appropriate precautions should be made if aggressive oral surgery is planned (eg, full mouth extractions with alveoloplasty) and the patient's BP is elevated. This is especially so if the patient is taking anticoagulants, such as aspirin or warfarin. The current trend is to not take patients off warfarin for oral surgery [40,41]. A rational approach on such patients, especially if BP is elevated, is to perform one or two extractions and verify good coagulation before continuing.

Summary

Most dentists will treat patients with hypertension daily as one in four Americans has this disease. This article reviews contemporary diagnosis and treatment, and makes suggestions for providing dental care to this large and diverse group of people.

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