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Review Article

Hypertensive Patients and Their Management in Dentistry

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Hypertension is a common disease encountered in dental setting. Its wide spreading, terrible consequences, and life-long treatment require an attentive approach by dentists. Hypertension management in dental office includes disease recognition and correct measurement, knowledge of its treatment and oral adverse effects, and risk assessment for dental treatment. Dentist role in screening undiagnosed and undertreated hypertension is very important since this may lead to improved monitoring and treatment.

1. Introduction

Hypertension is defined as values >140 mmHg SBP and/or >90 mmHg DBP, based on the evidence from RCTs that in patients with these BP values treatment-induced BP reductions are beneficial (Table 1) [1]. The same classification is used in young, middle-aged, and elderly subjects, whereas different criteria, based on percentiles, are adopted in children and teenagers for whom data from interventional trials are not available [1].

JNC 7 introduced in 2003 the category of prehypertension, which is defined as SBP of 120 to 139 mmHg and DBP of 80 to 89 mmHg (Table 2) [2]. Patients with prehypertension are at increased risk of developing hypertension, those with blood pressure values 130–139/80–89 mmHg have a two times greater risk of developing hypertension than those with lower values [3].

Hypertension is a highly prevalent cardiovascular disease, which affects over 1 billion people worldwide [2]. Although more than 70% of hypertensive patients are aware of the disease, only 23–49% are treated, and fewer (20%) achieving control [2, 4, 5]. Hypertension prevalence varies by age, race, education, and so forth.

According to ESC-ESH guidelines in 2013, there are limited comparable data available on the prevalence of hypertension and the temporal trends of BP values in different European countries [6]. Overall the prevalence of hypertension appears to be around 30–45% of the general population, with a steep increase with ageing. There also appear to be noticeable differences in the average BP levels across countries, with no systematic trends towards BP changes in the past decade [7–29].

A permanent high blood pressure (BP) affects blood vessels in the kidneys, heart, and brain, increasing the incidence of renal and cardiac coronary heart disease and stroke. Hypertension was called the "silent killer" because it often affects target organs (kidney, heart, brain, eyes) before the appearance of clinical symptoms.

2. Etiology and Classification of Hypertension

Hypertension is classified as primary or essential hypertension (without an organic cause) and secondary hypertension (it has a well-established organic cause).

2.1. Primary or Essential Hypertension (without an Organic Cause). Primary hypertension is the term used for medium

Table 1: Definitions and classification of office blood pressure levels (mmHg)[a] [1].

Category	Systolic mmHg		Diastolic mmHg
Optimal	<120	and	<80
Normal	120–129	and/or	80–84
High normal	130–139	and/or	85–89
Grade 1 hypertension	**140–159**	*and/or*	**90–99**
Grade 2 hypertension	**160–179**	*and/or*	**100–109**
Grade 3 hypertension	**≥180**	*and/or*	**≥110**
Isolated systolic hypertension	**≥140**	*and*	**<90**

[a]The blood pressure (BP) category is defined by the highest level of BP, whether systolic or diastolic. Isolated systolic hypertension should be graded 1, 2, or 3 according to systolic BP values in the ranges indicated.

Table 2: JNC 7 classification of hypertension [2].

Classification	SBP (mmHg)	DBP (mmHg)
Normal	<120	and <80
Prehypertension	120–139	or 80–89
Stage I hypertension	140–159	or 90–99
Stage II hypertension	≥160	≥100

to high BP for a long time (chronic) without a known cause, which is a very common form of hypertension, comprising about 90–95% of all patients with hypertension [30].

2.2. Secondary Hypertension. Hypertension with an organic cause, well established the following:

(i) renal (parenchyma or renal vascular) chronic pyelonephritis, acute and chronic glomerulonephritis, polycystic kidney disease, renal vascular stenosis or renal infarction, other severe kidney disease (arteriolar nephrosclerosis), renin-secreting tumors;

(ii) endocrine: oral contraceptives, adrenal hyper function (Cushing's syndrome, primary aldosteronism, congenital or hereditary adrenogenital syndrome), pheochromocytoma, myxedema, acromegaly, thyroid and parathyroid hyper function;

(iii) neurological: psychogenic "diencephalic syndrome," familiar dysautonomia (Riley-Day), polyneuritis (acute porphyria, lead poisoning), increased intracranial pressure;

(iv) others: coarctation of the aorta, increased intravascular volume (transfusion excessive polycythemia vera), polyarteritis, hypercalcemia, drugs (corticosteroids, cyclosporine), sleep apnea, pregnancy toxemia, acute intermittent porphyria.

3. Pathogenesis of Essential Hypertension

From family and epidemiological studies it is clear that hypertension results from a complex interaction between genetic factors and the environment [31]. There are at least 50 known factors which increase blood pressure, among which the most important are [32, 33]:

(i) age (over 55 years for men, over 65 years for women);

(ii) a family history of premature cardiovascular disease;

(iii) smoking;

(iv) increased consumption of alcohol;

(v) sedentariness;

(vi) cholesterol rich diet;

(vii) coexistence of other diseases (diabetes, obesity, dyslipidemia).

4. Treatment of Hypertension

Adopting a healthy lifestyle is critical in preventing high blood pressure. The major changes in lifestyle that could lead to lower blood pressure include reduction of body weight in overweight or obese patients [34], adopting a low-salt diet [35] rich in potassium and calcium [36], increasing physical activity [37], moderate alcohol consumption [37], and smoking cessation [38].

Hypertension drug treatment depends on the stage of hypertension, associated diseases, and risk factors present. Recommendations are based on the definition and classification of hypertension adopted by The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure in USA in 2003 [2] and the conclusions of the European Society of Hypertension-European Society of Cardiology (ESH-ESC) in 2013 [1]. They determine the maximum physiological BP 130 mmHg systolic and 85 mmHg for diastolic, BP values of 139 mmHg systolic, and 89 mmHg diastolic standing at the upper limit of normal. The recommendations emphasize that the decision to drop blood pressure in a particular patient should not rely solely on the values of BP but also on total cardiovascular risk assessment of that patient (Table 3). From the meta-analysis by Staessen et al. [39] results show that all classes of antihypertensive agents provide similar cardiovascular protection. To prevent stroke and congestive heart failure, feared complications of hypertension, the results of recently published trials suggest that some classes can achieve selective benefits.

4.1. Total Cardiovascular Risk Assessment [1]. ESC-ESH report from June 2013 presents SCORE model of assessment

Table 3: Total cardiovascular risk assessment [1].

Recommendations	Class[a]	Level[b]	Ref[c]
In asymptomatic subjects with hypertension but free of CVD, CKD, and diabetes, total CV risk stratification using the SCORE model is recommended as a minimal requirement	I	B	[66]
As there is evidence that OD predicts CV death independently of SCORE, a search for OD should be considered, particularly in individuals at moderate risk	IIa	B	[67, 68]
It is recommended that decisions on treatment strategies depend on the initial level of total CV risk	I	B	[69–71]

CKD: chronic kidney disease; CV: cardiovascular; CVD: cardiovascular disease; OD: organ damage; SCORE: Systematic Coronary Risk Evaluation.
[a]Class of recommendation.
[b]Level of evidence.
[c]Reference(s) supporting levels of evidence.

of total cardiovascular risk using charts and interactive site http://www.heartscore.org. The charts must be interpreted considering physician's knowledge and experience [1]. Risk may be higher than indicated in the charts in the following:

(1) sedentary subjects and those with central obesity; the increased relative risk associated with overweight is greater in younger subjects than in older subjects;

(2) socially deprived individuals and those from ethnic minorities;

(3) subjects with elevated fasting glucose and/or an abnormal glucose tolerance test, who do not meet the diagnostic criteria for diabetes;

(4) individuals with increased triglycerides, fibrinogen, apolipoprotein B, lipoprotein levels, and high-sensitivity C-reactive protein;

(5) individuals with a family history of premature CVD (before the age of 55 years in men and 65 years in women).

Three important causes of primary hypertension are salt/volume overload, activation of the reninangiotensin-aldosterone system (RAAS), and activation of the sympathetic nervous system (Table 4) [4].

Salt (sodium chloride) overload/volume overload is one of the common causes of hypertension. Essential hypertension has been associated with high sodium intake in a variety of scientific models, clinical studies and trials, and it is certified that decreasing the sodium intake ameliorates this effect [40, 41]. High sodium intake increases blood pressure by expanding intravascular volume and may have direct neurohormonal effects on the cardiovascular system [4, 41]. Thiazide diuretics are indicated by JNC 7 [2] as initial therapy for most patients with hypertension, either alone or in combination with another class of antihypertensive agents.

The "Renin Angiotensin Aldosterone System" (RAAS) hormonal axis also contributes to hypertension in many patients [4]. Renin, a hormone synthesized and released by the kidney in response to intravascular volume depletion and hyperkalemia, promotes the conversion of angiotensinogen (produced by the liver) to angiotensin I, which is converted to angiotensin II by the angiotensin-converting enzyme (ACE) in the lung. One mechanism of increasing blood pressure by angiotensin II is increasing renal sodium reabsorption, producing vasoconstriction, and activating the sympathetic nervous system [4]. But angiotensin II also increases the production and secretion of aldosterone from the adrenal cortex, and aldosterone increases renal sodium reabsorption [4]. Thus, the RAAS system increases blood pressure through increasing renal sodium reabsorption (which leads to intravascular volume expansion) and vasoconstriction.

There are several classes of medications used to block various components of the RAAS pathway, like β-Blockers such as propranolol, carvedilol, and metoprolol (decrease renal renin release), direct renin inhibitor aliskiren (binds to renin and thus prevents the conversion of angiotensinogen to angiotensin I), ACE inhibitors (block ACE and prevent the conversion of angiotensin I to angiotensin II), angiotensin II receptor blockers (prevent angiotensin II from binding to its receptor, decreasing vasoconstriction and renal sodium reabsorption), aldosterone-receptor blockers (such as spironolactone and eplerenone), and other medications such as amiloride (decrease the effects of aldosterone-mediated renal sodium reabsorption) [4].

Activation of the sympathetic nervous system (SNS) also contributes to the development, maintenance, and progression of hypertension. Therapies have been developed to target the central, peripheral, and renal SNS to improve the control of blood pressure: peripheral α1-receptor blockers (such as terazosin and tamsulosin), central α2-agonist clonidine, and β-blockers, vasodilators such as minoxidil, nitrates, and hydralazine [2, 42].

5. Oral Manifestations Caused by the Adverse Effects of Antihypertensive Drugs

5.1. Xerostomia. Many antihypertensives medications like ACEIs, thiazide diuretics, loop diuretics, and clonidine are associated with xerostomia [43–46]. Its likelihood increases with the number of concomitant medications. Xerostomia has many consequences, like decay, difficulty in chewing, swallowing, and speaking, candidiasis, and oral burning syndrome. Sometimes the feeling is transient and salivary function is adjusted by the patient itself. There are situations

Table 4: Mechanisms implicated in essential hypertension and antihypertensive medication classes targeting these mechanisms [4].

Mechanism	Medication targeting the mechanism	Examples
Volume overload	Diuretics	Hydrochlorothiazide, Chlorthalidone, Metolazone, Furosemide, Torsemide
	Dihydropyridine CCBs	Amlodipine, Nifedipine
Renin-angiotensin-aldosterone system	ACEIs	Lisinopril, Captopril
	ARBs	Losartan, Valsartan
	β-blockers	Metoprolol, Carvedilol
	Direct renin inhibitors	Aliskiren
	Aldosterone receptor blockers	Spironolactone, Eplerenone
Sympathetic nervous system	Central α-blockers	Clonidine
	Peripheral α-blockers	Tamsulosin, Terazosin
	β-blockers	Metoprolol, Carvedilol
	Nondihydropyridine CCBs	Verapamil, Diltiazem
	Vasodilators	Minoxidil, Hydralazine, Nitrates

ACEI: angiotensin-converting enzyme inhibitor; ARB: angiotensin II receptor blocker; CCB: calcium-channel blocker.
Data from [2].

when is required to change the antihypertensive medication. It is often necessary to treat xerostomia directly with parasympathomimetic agents such as pilocarpine or cevimeline. Other recommendations include frequent sipping of water, sugarless candies, coffee consumption reduction, and avoiding alcohol containing mouthwashes. To reduce the risk of caries topical applications of fluoride, particularly in the form of gels with high concentrations applied by brush or trays [47], are recommended.

5.2. Gingival Hyperplasia. It can be caused by calcium channel blockers, with an incidence ranging from 6 to 83% [48–52]. The majority of cases are associated with nifedipine. The effect could be dose related. Gingival hyperplasia is manifested by pain, gingival bleeding, and difficulty in mastication. A good oral hygiene greatly reduces its incidence. By changing antihypertensive medication hyperplasia can be reversed [53].

5.3. Lichenoid Reaction. Many antihypertensives (thiazide diuretics, methyldopa, propranolol, captopril, furosemide, spironolactone, and labetalol) are associated with oral lichenoid reactions [54, 55]. Clinical forms differ greatly from lichen planus itself. The easiest way to treat it is to change antihypertensive medication, and lichenoid reactions are resolving after discontinuation of the responsible drug. If medication could not be changed, lichenoid reactions are treated with topical corticosteroids [47].

5.4. Other Undesirable Effects. ACE inhibitors are associated with cough and loss of taste (ageusia) or taste alteration (dysgeusia). Dysgeusia has also been reported with other antihypertensives use, like β-blockers, acetazolamide, and diltiazem. It has been postulated that dysgeusia may result through a mechanism affecting salivary handling of metal ions such as magnesium [56, 57].

6. Drug Interactions between Antihypertensives and Drugs Used in Dentistry

Most antihypertensive drugs have drug interactions with LA (local anesthetic) and analgesics.

(i) Interaction of LA with nonselective beta-blockers may increase LA toxicity [58].

(ii) The cardiovascular effects of epinephrine used during dental procedures may be potentiated by the use of medications such as nonselective b-blockers (propranolol and nadolol). Guidelines recommend decreasing the dose and increasing the time interval between epinephrine injections [59].

(iii) Long-term use of NSAIDs may antagonize the antihypertensive effect of diuretics, beta-blockers, alpha blockers, vasodilators, ACE inhibitors [4]. Short-term administration has, however, a clinically meaningful effect. Other pain relievers such as paracetamol can be used to avoid this side effect.

Dental treatment in hypertensive patients necessitates special attention, because any stressful procedure may increase blood pressure and trigger acute complications such as cardiac arrest or stroke.

Control of pain and anxiety is very important in patients with high medical risk. Patients with cardiovascular disease have a high risk of complications due to endogenous catecholamines (adrenaline and noradrenaline) released from pain and stress. These catecholamines may increase dramatically BP and cardiac output. This effect is reduced by controlling dental pain. Local anesthetics with epinephrine produce a longer and more effective anesthesia than simple LA, thus avoiding an exaggerated response to stress [60]. LA with vasoconstrictor should be avoided or used in low doses in patients taking nonselective beta-blockers or in patients with uncontrolled hypertension. The maximum

Table 5: Office blood pressure measurement [1].

When measuring BP in the office, care should be taken
(i) to allow the patients to sit for 3–5 minutes before beginning BP measurements;
(ii) to take at least two BP measurements, in the sitting position, spaced 1-2 min apart, and additional measurements if the first two are quite different. Consider the average BP if deemed appropriate;
(iii) to take repeated measurements of BP to improve accuracy in patients with arrhythmias, such as atrial fibrillation;
(iv) to use a standard bladder (12-13 cm wide and 35 cm long), but have a larger and a smaller bladder available for large (arm circumference >32 cm) and thin arms, respectively;
(v) to have the cuff at the heart level, whatever the position of the patient;
(vi) when adopting the auscultatory method, use phases I and V (disappearance) Korotkoff sounds to identify systolic and diastolic BP, respectively;
(vii) to measure BP in both arms at first visit to detect possible differences. In this instance, take the arm with the higher value as the reference;
(viii) to measure at first visit BP 1 and 3 min after assumption of the standing position in elderly subjects, diabetic patients, and other conditions in which orthostatic hypotension may be frequent or suspected;
(ix) to measure, in case of conventional BP measurement, heart rate by pulse palpation (at least 30 s) after the second measurement in the sitting position.

recommended dose of epinephrine in a patient with cardiac risk is 0.04 mg, which is equal to that containing about two cartridges of LA with 1 : 100000 epinephrine or 4 cartridges with 1 : 200000 epinephrine [60]. In patients with severe disease it may be useful to measure BP and heart rate after anesthetic injection. Slow administration and aspiration can prevent undesirable reactions.

Other contraindications to vasoconstrictor AL include severe uncontrolled hypertension, refractory arrhythmias, myocardial infarction or stroke by age less than 6 months, unstable angina, coronary artery bypass graft under 3 months, congestive heart failure, and untreated hyperthyroidism [61].

Due to higher concentrations of epinephrine (almost 12 standard cartridges) in gingival retraction cords used for prosthetics impressions and its rapid uptake in circulation, the use of epinephrine for gingival eviction in patients with cardiovascular disease is contraindicated [4, 62].

7. Hypertensive Patient Management in the Dental Office

Initial evaluation of each patient with hypertension should include detailed family history of cardiovascular disease and other related diseases, history of hypertension, medications, duration and antihypertensive treatment history, severity of disease, and its complications [61]. Before starting dental treatment, dentist has to assess the presence of hypertension, to determine the presence of associated organ disease and determine dental treatment changes needed [63].

Particular attention should be given to accurate measurement of BP in pregnant women, since pregnancy may alter the patient BP values, more than 10% of pregnant women having clinically relevant hypertension [64]. BP monitoring is also necessary in diabetic patients, patients with autonomous dysfunction, and elderly patients for which orthostatic hypotension is a big problem [2]. The dentist must be familiar with other diseases treated with antihypertensive drugs (such as atenolol, amlodipine, and carteolol) as headaches, regional pain, renal failure, glaucoma, and congestive heart failure.

8. BP Measurement in the Dental Office

Patients with hypertension are at increased risk of developing adverse effects in a dental office. Therefore, measuring BP will be done in the dental office to every new patient, for each visit. In patients with chronic systemic diseases, BP measurement will be carried out during more complicated dental interventions as oral surgery, restorative treatment complicated with longer sessions, placing dental implants, and periodontal surgery.

Routine measurement of BP may reduce the risk of cardiovascular events and acute complications during dental treatment, especially when conscious sedation or general anesthesia is required. BP monitoring is vital for emergency treatment of patients who have side effects. Routine monitoring of patients with known hypertension allows the dentist to determine if BP is adequately controlled.

Best BP measurements were obtained with mercury sphygmomanometers, no longer available now. Aneroid sphygmomanometers used should be checked every 6 months. Electronics BP units are simple to use but not as accurate as the aneroid.

ESC-ESH guidelines in 2013 and JNC 7 in 2003 described the method that health care professionals should use to obtain office blood pressure measurements (Table 5) [1, 2].

One must use a properly calibrated and validated blood pressure instrument. Patients should be seated in a chair with their feet on the floor for 5 minutes in a quiet room. Their arm should be supported at the level of the heart and an appropriately sized blood pressure cuff (cuff bladder encircling at least 80% of the arm) must be used. Accurate measurement of blood pressure is important to avoid overdiagnosis and underdiagnosis, as well as overtreatment and undertreatment, of hypertension [4].

Table 6: White-coat hypertension, the white-coat effect, and masked hypertension [4].

Diagnosis	Office blood pressure	Blood pressure outside office	Associated with adverse outcomes
WCH	Elevated	Normal	Controversial
WCE	Elevated	Normal or high	Controversial
Masked hypertension	Normal	Elevated	Yes

9. White-Coat Hypertension, the White-Coat Effect, and Masked Hypertension

Office BP is usually higher than BP measured out of the office, which has been ascribed to the alerting response, anxiety, and/or a conditional response to the unusual situation [1]. *White-coat hypertension* (WCH) refers to a persistently elevated office blood pressure in the presence of a normal blood pressure outside of the office [4]. WCH is different from the white-coat effect (WCE), which refers to a high office blood pressure but whereby hypertension may or may not be present outside the office setting. *Masked hypertension* refers to when a patient has a normal office blood pressure but has hypertension outside of the office (Table 6). WCH, the WCE, and masked hypertension can be diagnosed through various methods including home blood pressure monitoring and 24-hour ambulatory blood pressure monitoring. WCH and masked hypertension are important for clinicians to recognize. It is controversial as to whether WCH is associated with increased cardiovascular risk, but patients with masked hypertension are at increased cardiovascular risk. The prevalence of WHC during physician visits is approximately 20% [4, 65]. The prevalence of WCH in the setting of visits to the dentist's office has not been established. ESC-ESH guidelines recommend that the terms "white-coat hypertension" and "masked hypertension" be reserved to define untreated individuals [1].

Routine measurement of blood pressure values in the dental office [63] is as follows:

(i) measuring and recording the TA at the first visit,

(ii) measuring and recording BP at recheck:

(a) every two years for patients with BP < 120/80 mmHg;

(b) every year for patients with BP 120–139/80–89 mmHg;

(c) every visit for patients with BP > 140/90 mmHg;

(d) every visit for patients with coronary artery disease, diabetes mellitus, or kidney disease with BP > 135/85 mmHg;

(e) every visit for patients with established hypertension.

10. Summary

Hypertension is the most commonly diagnosed disease worldwide and is associated with increased cardiovascular risk and mortality. Many patients with hypertension have uncontrolled disease. The dentist has an important role in screening undiagnosed and undertreated hypertension, which may lead to improved monitoring and treatment. It is generally recommended that emergency dental procedures be avoided in patients with a blood pressure of greater than 180/110 mmHg. Because of the high prevalence of disease and medication use for hypertension, dentists should be aware of the oral side effects of antihypertensive medications. Also, dentists should consider management of drug-drug interactions of antihypertensives with medications commonly used during dental visits.

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