International Journal of Trend in Scientific Research and Development (IJTSRD)
Volume 5 Issue 4, May-June 2021 Available Online: www.ijtsrd.com e-ISSN: 2456 – 6470

# A Review: Hypertension

**Shweta Pawar[1], Sujit Kakde[2], Ashok Bhosale[3]**

[1]Student, [2]Giude, [3]Principal,
[1,2,3]PDEA'S Shankarrao Ursal College of Pharmaceutical Science and Research Centre, Pune, Maharashtra, India

**ABSTRACT**

Hypertension is a major public health problem and important area of research due to its high prevalence and being major risk factor for cardiovascular diseases and other complications.

**Objectives**

1. To assess the prevalence of hypertension and its associated factors and
2. to estimate awareness, treatment, and adequacy of control of hypertension among study subjects.

According to the Joint National Committee 7 (JNC7), normal **blood pressure** is a systolic BP < 120 mmHg and diastolic BP < 80 mm Hg. **Hypertension** is defined as systolic BP level of ≥140 mmHg and/or diastolic BP level ≥ 90 mmHg.

A number of factors increase BP, including
(1) obesity, (2) insulin resistance, (3) high alcohol intake, (4) high salt intake (in salt-sensitive patients), (5) aging and perhaps (6) sedentary lifestyle, (7) stress, (8) low potassium intake, and (9) low calcium intake.

***KEYWORDS:*** *health problem, cardiovascular disease, systolic and diastolic, factors*

***How to cite this paper:*** Shweta Pawar | Sujit Kakde | Ashok Bhosale "A Review: Hypertension" Published in International Journal of Trend in Scientific Research and Development (ijtsrd), ISSN: 2456-6470, Volume-5 | Issue-4, June 2021, pp.1320-1326, URL: www.ijtsrd.com/papers/ijtsrd42416.pdf

IJTSRD42416



## INTRODUCTION

Hypertension is a major public health problem due to its high prevalence all around the globe. Around 7.5 million deaths or 12.8% of the total of all annual deaths worldwide occur due to high blood pressure. It is predicted to be increased to 1.56 billion adults with hypertension in 2025.

Raised blood pressure causes many complications like chronic heart disease, stroke, and coronary heart disease. Elevated BP is positively match up to the risk of stroke and coronary heart disease. Other than coronary heart disease and stroke, its complications include heart failure, peripheral vascular disease, renal impairment, retinal haemorrhage, and visual impairment.

There are several factors make liable to hypertension and those factors vary from country to country even from some place or region like urban or rural. By perceiving the effect of urbanization on our collective health, World Health Organization has chosen "Urbanization and Health" as the theme for World Health Day 2010. Urbanization is considered a determinant of health and one of the key drivers of non communicable diseases (NCDs), especially in low- and middle-income countries (LMICs). Urban people are more at risk of these diseases as compared to their rural counterparts. As per the findings of National Family Health Survey (NFHS-4), the prevalence of hypertension, obesity, and blood glucose in urban area of Uttar Pradesh was 10.5%, 23.9, and 9.9%, respectively. However, the prevalence of the same phenomenon was 8.3%, 10.8%, and 8.2%, respectively in rural area. Thus comparatively it is seen that all the parameters are having higher prevalence in urban area as compared to rural area. Rapid **Urbanization** is the process through which cities grow, and higher and higher percentages of the population comes to live in the city, mechanization, sedentary life, and dietary changes act together as a web of risk factors which entangles people in it and leads to several chronic diseases. In order to take effective prevention measures, identification of the risk factors is an essential prerequisite.

## DEFINITION OF HYPERTENSION:

The relationship Between BP and cardiovascular and renal events is continuous, making the distinction between normotension and hypertension, based on cut-off BP values, somewhat arbitrary. However, 'hypertension' is defined as the level of BP at which the benefits of treatment (either with lifestyle interventions or drugs) unequivocally outweigh the risks of treatment.

## HYPERTENSION AND CARDIOVASCULAR RISK

Several calculation modalities are used today for cardiovascular risk assessment. Cardiovascular risk assessment should be performed in all hypertensive patients. Risk assessment methods being based on the population in which the patient lives and the inclusion of factors such as ethnicity variations, socioeconomic status, and medication use will contribute to improvements in risk assessments. The results should be shared with the patient, and modifiable risk factors must be effectively treated. Hypertension rarely occurs in isolation, and often clusters with other cardiovascular risk factors such as dyslipidaemia and glucose intolerance. This metabolic risk factor clustering has a multiplicative effect on cardiovascular risk. Consequently, quantification of total cardiovascular risk (i.e.,

the likelihood of a person developing a cardiovascular event over a defined period) is an important part of the risk stratification process for patients with hypertension.

Since 2003, the European Guidelines on CVD prevention have recommended use of the Systematic Coronary Risk Evaluation (SCORE) system because it is based on large, representative European cohort data sets. The SCORE system estimates the 10-year risk of a first fatal atherosclerotic event, in relation to age, sex, smoking habits, total cholesterol level.

TABLE 01: Factors influencing cardiovascular risk in patients with hypertension

**Source**

| Demographic characteristics and laboratory parameters |
|---|
| Sex[a] (men >women) |
| Age[a] |
| Smoking (current or past history)[a] |
| Total cholesterol[a] and HDL-C |
| Uric acid |
| Diabetes[a] |
| Overweight or obesity |
| Family history of premature CVD (men aged <55 years and women aged <65 years) |
| Family or parental history of early-onset hypertension |
| Early-onset menopause |
| Sedentary lifestyle |
| Psychosocial and socioeconomic factors |
| Heart rate (resting values >80 beats/min) |
| **Asymptomatic HMOD** |
| Arterial stiffening:<br>Pulse pressure (in older people) ≥60 mmHg<br>Carotid–femoral PWV >10 m/s |
| ECG LVH (Sokolow–Lyon index >35 mm, or R in aVL ≥11 mm; Cornell voltage duration product >2440 mm*ms, or Cornell voltage >28 mm in men or >20 mm in women) |
| Echocardiographic LVH [left ventricular mass index: men >50 g/m$^{2.7}$; women >47 g/m$^{2.7}$ (height in m$^{2.7}$); indexation for BSA may be used in normal-weight patients; left ventricular mass/BSA g/m$^2$ >115 (men) and >95 (women)] |
| Microalbuminuria (30–300 mg/24 h), or elevated albumin–creatinine ratio (30–300 mg/g; 3.4–34 mg/mmol) (preferentially on morning spot urine)[b] |
| Moderate CKD with eGFR 30–59 ml/min/1.73 m$^2$ (BSA)[b] |
| Ankle-brachial index <0.9 |
| Advanced retinopathy: haemorrhages or exudates, papilloedema |
| **Established cardiovascular or renal disease** |
| Cerebrovascular disease: ischaemic stroke, cerebral haemorrhage, TIA |
| CAD: myocardial infarction, angina, myocardial revascularization |
| Presence of atheromatous plaque on imaging |
| Heart failure, including HFpEF |
| Peripheral artery disease |
| Atrial fibrillation |
| Severe CKD with eGFR <30 ml/min/1.73 m$^2$ |

BSA, body surface area; CAD, coronary artery disease; CKD, chronic kidney disease; CVD, cardiovascular disease; eGFR, estimated glomerular filtration rate; HDL-C, HDL cholesterol; HFpEF, heart failure with preserved ejection fraction; HMOD, hypertension-mediated organ damage; LVH, left ventricular hypertrophy; PWV, pulse wave velocity; SCORE, Systematic COronary Risk Evaluation; TIA, transient ischaemic attack.
[a]CV risk factors included in the SCORE system.
[b]Proteinuria and reduced eGFR are independent risk factors. See Table 6 for cardiovascular risk modifiers.

There is also emerging evidence that an increase in serum uric acid to levels lower than those typically associated with gout is independently associated with increased cardiovascular risk in both the general population and in hypertensive patients. Measurement of serum uric acid is recommended as part of the screening of hypertensive patients.

**RESULT**: CVR assessment should be performed in all hypertensive patients, the results shared with the patient, and modifiable risk factors effectively treated. Risk assessment methods being based on the population in which the patient lives and the inclusion of factors such as ethnicity variations, socioeconomic status, and medication use will contribute to the improvement of risk assessments.

**BLOOD PRESSURE MEASUREMENT:**

Auscultatory or oscillometric semiautomatic or automatic sphygmomanometers are the preferred method for measuring BP in the doctor's office. These devices should be validated according to standardized conditions and protocols. BP should initially be measured in both upper arms, using an appropriate cuff size for the arm circumference. A consistent and significant SBP difference between arms (i.e., >15 mmHg) is associated with an increased cardiovascular risk, most likely due to atheromatous vascular disease. Where there is a difference in BP between arms, ideally established by simultaneous measurement, the arm with the higher BP values should be used for all subsequent measurements.

In older people, people with diabetes, or people with other causes of orthostatic hypotension, BP should also be measured 1 and 3 min after standing. Orthostatic hypotension is defined as a reduction in SBP of at least 20 mmHg or in DBP of at least 10 mmHg within 3 min of standing, and is associated with an increased risk of mortality and cardiovascular events. Heart rate should also be recorded at the time of BP measurements because resting heart rate is an independent predictor of cardiovascular morbid or fatal events, although heart rate is not included in any cardiovascular risk algorithm.

**HOME BLOOD PRESSURE MONITORING**

Home BP is the average of all BP readings performed with a semiautomatic, validated BP monitor, for at least 3 days and preferably for 6–7 consecutive days before each clinic visit, with readings in the morning and the evening, taken in a quiet room after 5 min of rest, with the patient seated with their back and arm supported. Two measurements should be taken at each measurement session, performed 1–2 min apart.

Blood pressure was measured two times on the right arm of the selected subject using automatic electronic device (OMRON HEM-7261). The average of two readings was used.

It's recommended to use an upper arm blood pressure monitor for the most accurate blood pressure reading results.

**HOW TO USE HOMEBLOOD PRESSURE MONITOR:**

- **Be still.** Don't smoke, drink caffeinated beverages or exercise within 30 minutes before measuring your blood pressure. Empty your bladder and ensure at least 5 minutes of quiet rest before measurements.
- **Sit correctly.** Sit with your back straight and supported (on a dining chair, rather than a sofa). Your feet should be flat on the floor and your legs should not be crossed. Your arm should be supported on a flat surface (such as a table) with the upper arm at heart level. Make sure the bottom of the cuff is placed directly above the bend of the elbow. Check your monitor's instructions for an illustration or have your healthcare provider show you how.
- **Measure at the same time every day.** It's important to take the readings at the same time each day, such as morning and evening. It is best to take the readings daily however ideally beginning 2 weeks after a change in treatment and during the week before your next appointment.
- **Take multiple readings and record the results**. Each time you measure, take two or three readings one minute apart and record the results. If your monitor has built-in memory to store your readings, take it with you to your appointments. Some monitors may also allow you to upload your readings to a secure website after you register your profile.
- **Don't take the measurement over clothes.** Several studies have been done to determine what is a normal variation between right and left arm. In general, any difference of 10 mm Hg or less is considered normal and is not a cause for concern.

**WHITE-COAT HYPERTENSION:**

**White coat hypertension** (WHT), more commonly known as **white coat syndrome**, is a form of labile **hypertension** in which people exhibit a **blood pressure** level above the normal range, in a clinical setting, although they do not exhibit it in other settings. Although the prevalence varies between studies, white-coat hypertension can account for up to 30–40% of people (and >50% in the very old) with an elevated office BP. It is more common with increasing age, in women, and in non-smokers. Its prevalence is lower in patients with HMOD, when office BP is based on repeated measurements, or when a doctor is not involved in the BP measurement. A significant white-coat effect can be seen at all grades of hypertension (including resistant hypertension), but the prevalence of white-coat hypertension is greatest in grade 1 hypertension.

HMOD is less prevalent in white-coat hypertension than in sustained hypertension, and recent studies show that the risk of cardiovascular events associated with white-coat hypertension is also lower than that in sustained hypertension. Conversely, compared with true normotensives, patients with white-coat hypertension have increased adrenergic activity, a greater prevalence of metabolic risk factors, more frequent asymptomatic cardiac and vascular damage, and a greater long-term risk of new-onset diabetes and progression to sustained hypertension and LVH.

White-coat hypertension has also been shown to have a greater cardiovascular risk in isolated systolic hypertension and older patients [91], and does not appear to be clinically innocent.

**MASKED HYPERTENSION:**

**Masked hypertension** is defined as a normal **blood pressure** (BP) in the clinic or office (<140/90 mmHg), but an elevated BP out of the clinic (ambulatory daytime BP or home BP>135/85 mmHg).

**What causes masked hypertension**? **Masked hypertension** can occur if your home or work environment is more stressful than at your doctor's office. Use of alcohol, caffeine or cigarettes at home can also **cause** increased **blood pressure**.

**Masked hypertension** is associated with increased cardiovascular risk in both untreated and treated subjects. In contrast, **white-coat hypertension** is a cardiovascular risk factor in untreated but not in treated subjects.

Masked hypertension can be found in approximately 15% of patients with a normal office BP. The prevalence is greater in younger people, men, smokers, and those with higher levels of physical activity, alcohol consumption, anxiety and job

stress. Obesity, diabetes, CKD, family history of hypertension, and high–normal office BP are also associated with an increased prevalence of masked hypertension. Masked hypertension is associated with dyslipidaemia and dysglycaemia, HMOD, adrenergic activation, and increased risk of developing diabetes and sustained hypertension. Meta-analyses and recent studies have shown that the risk of cardiovascular events is substantially greater in masked hypertension compared with normotension, and close to or greater than that of sustained hypertension. Masked hypertension has also been found to increase the risk of cardiovascular and renal events in diabetes, especially when the BP elevation occurs during the night.

**BLOOD PRESSURE DURING EXERCISE:**

It is important to recognize that BP increases during dynamic and static exercise, and that the increase is more pronounced for SBP than for DBP, although only SBP can be measured reliably with non-invasive methods. There is currently no consensus on normal BP response during exercise. The increase in SBP during exercise is related to pre-exercise resting BP, age, arterial stiffness and abdominal obesity, and is somewhat greater in women than in men and in unfit individuals. There is some evidence that an excessive rise in BP during exercise predicts the development of hypertension, independently from BP at rest.

Normally **during exercise**, **blood pressure** increases to push the flow of oxygen-rich **blood** throughout the body. However, in some individuals, the response to **exercise** is exaggerated. Instead of reaching a systolic (upper number) **blood pressure** of around 200 mmHg at maximal **exercise**, they spike at 250 mmHg or higher.

Nevertheless, exercise testing is not recommended as part of the routine evaluation of hypertension because of various limitations, including a lack of standardization of methodology and definitions. Importantly, except in the presence of very high BP values (grade 3 hypertension), patients or athletes, with treated or untreated hypertension should not be discouraged from regular exercise, especially aerobic exercise, which is considered beneficial as part of lifestyle changes to reduce BP.

Becoming more active can lower both your top and bottom **blood pressure** numbers. How much lower isn't entirely clear, but studies show reductions from 4 to 12 mm Hg diastolic and 3 to 6 mm Hg systolic. Regular **exercise** also helps you maintain a healthy weight — another important way to control **blood pressure**.

**FIGURE**

| Recommendations | Class[a] | Level[b] |
|---|---|---|
| Screening programmes for hypertension are recommended. All adults (18 years or older) should have their office BP measured and recorded in their medical file, and be aware of their BP [12,98]. | I | B |
| • Further BP recording is indicated, at least every 5 years if BP remains optimal. | I | C |
| • Further BP recording is indicated, at least every 3 years if BP remains normal. | I | C |
| • If BP remains high–normal, further BP recording, at least annually, is recommended. | I | C |
| • In older patients (> 50 years), more frequent screening of office BP should be considered for each BP category because of the steeper rise in SBP with ageing. | IIa | C |
| It is recommended that office BP should be measured in both arms at least at the first visit because a between-arm SBP difference of > 15 mmHg is suggestive of atheromatous disease and is associated with an increased CV risk [45]. | I | A |
| If a between-arm difference in BP is recorded, then it is recommended that all subsequent BP readings use the arm with the higher BP reading. | I | C |
| It is recommended that the diagnosis of hypertension should be based on:<br>• Repeated office BP measurements on more than one visit, except when hypertension is severe (e.g. grade 3 and especially in high-risk patients). At each visit, three BP measurements should be recorded, 1–2 min apart, and additional measurements should be performed if the first two readings differ by > 10 mmHg. The patient's BP is the average of the last two BP readings. | I | C |

| | | |
|---|---|---|
| Or<br>• Out-of-office BP measurement with ABPM and/or HBPM, provided that these measurements are logistically and economically feasible. | I | C |
| Out-of-office BP (i.e. ABPM or HBPM) is specifically recommended for a number of clinical indications, such as identifying white-coat and masked hypertension, quantifying the effects of treatment, and identifying possible causes of side effects [17,54,62,68,72] (e.g. symptomatic hypotension). | I | A |
| It is recommended that all hypertensive patients undergo pulse palpation at rest to determine heart rate and search for arrhythmias such as AF [20,47]. | I | C |
| Other BP measures and indices (pulse pressure, BP variability, exercise BP, and central BP) may be considered but are not often used for routine clinical use at present. They may provide useful additional information in some circumstances and are valuable tools for research. | IIb | C |

ABPM, ambulatory blood pressure monitoring; AF, atrial fibrillation; BP, blood pressure; HBPM, home blood pressure monitoring.
[a]Class of recommendation.
[b]Level of evidence.

**THE KIDNEY IN HYPERTENSION:**

Hypertension is the second most important cause of CKD after diabetes. Hypertension may also be the presenting feature of asymptomatic primary renal disease. An alteration of renal function is most commonly detected by an increase in serum creatinine. This is an insensitive marker of renal impairment because a major reduction in renal function is needed before serum creatinine rises. Furthermore, BP reduction by antihypertensive treatment often leads to an acute increase in serum creatinine by as much as 20–30%, especially with renin–angiotensin system (RAS) blockers, which has a functional basis and does not usually reflect manifest renal injury, but the long-term clinical significance is unclear. The diagnosis of hypertension-induced renal damage is based on the finding of reduced renal function and/or the detection of albuminuria. CKD is classified according to estimated glomerular filtration rate (eGFR), calculated by the 2009 CKD-Epidemiology Collaboration formula.

The albumin: creatinine ratio (ACR) is measured from a spot urine sample (preferably early morning urine), and is the preferred method to quantify urinary albumin excretion. A progressive reduction in eGFR and increased albuminuria indicate progressive loss of renal function, and are both independent and additive predictors of increased cardiovascular risk and progression of renal disease.

**Hypertensive kidney disease** is a medical condition referring to **damage** to the **kidney** due to **chronic** high blood pressure. It manifests as **hypertensive** nephrosclerosis (sclerosis referring to the stiffening of **renal** components).

RENAL HYPERTENSION, also called renovascular hypertension, is elevated blood pressure caused by kidney disease. It can usually be controlled by blood pressure drugs. Some people with renal hypertension can be helped by angioplasty, stenting, or surgery on the blood vessels of the kidney.

**THE BRAIN IN HYPERTENSION:**

Hypertension increases the prevalence of brain damage, of which transient ischaemic attack (TIA) and stroke are the most dramatic acute clinical manifestations. In the asymptomatic phase, brain damage can be detected by MRI as white matter hyperintensities, silent microinfarcts, (most of which are small and deep, i.e., lacunar infarctions), microbleeds and brain atrophy. White matter hyperintensities and silent infarcts are associated with an increased risk of stroke and cognitive decline due to degenerative and vascular dementia. Availability and cost do not permit the widespread use of brain MRI for the evaluation of hypertensive patients, but white matter hyperintensity and silent brain infarcts should be sought in all hypertensive patients with neurological disturbances, cognitive decline, and, particularly, memory loss.

A family history of cerebral haemorrhage at middle age and early-onset dementia should prompt MRI. Cognitive impairment in older patients is, at least in part, hypertension-related, and cognitive evaluation tests should be considered in the clinical assessment of hypertensive patients with a history suggestive of early cognitive impairment. The Mini-Mental State Examination has been the most widely used method in clinical trials, but is now being superseded by more sophisticated cognitive tests that are more suitable for routine clinic visits.

**High blood** pressure (or "**hypertension**") has been shown to damage the tiny blood vessels in the parts of **your brain** responsible for cognition and memory, greatly increasing your risk of developing Alzheimer's disease or another dementia.

Neurological regulation of **blood pressure** and flow depends on the cardiovascular centers located in the

medulla oblongata. This cluster of neurons responds to changes in **blood pressure** as well as blood concentrations of oxygen, carbon dioxide, and other factors such as pH.

**GENETICS AND HYPERTENSION:**

A positive family history is a frequent feature in hypertensive patients, with the heritability estimated to vary between 35 and 50% in most studies. However, hypertension is a highly heterogeneous disorder with a multifactorial aetiology. Several genome-wide association studies and their meta-analyses have identified 120 loci that are associated with BP regulation, but together these only explain about 3.5% of the trait variance. Several rare, monogenic forms of hypertension have been described such as glucocorticoid-remediable aldosteronism, Liddle's syndrome, and others, where a single gene mutation fully explains the pathogenesis of hypertension and dictates the best treatment modality. There are also inherited forms of phaeochromocytoma and paraganglioma, which are also rare causes of hypertension. Outside of specialist clinics evaluating patients for these rare causes of secondary hypertension, there is no role for genetic testing in hypertension in routine clinical care.

*Hypertension* tends to run in families. Individuals whose parents have *hypertension* have an elevated risk of developing the condition, particularly if both parents are affected. However, the inheritance pattern is unknown. Rare, *genetic* forms of *hypertension* follow the inheritance pattern of the individual condition.

Angiotensinogen (AGT) AGT was the first **gene** to show linkage with human essential or primary **hypertension**. In addition to linkage to the AGT locus, **hypertension** and plasma angiotensinogen levels were both found to be associated with the 235T and 174M variants of AGT.

**HYPERTENSION AND PREGNANCY:**

Hypertensive disorders in pregnancy affect 5–10% of pregnancies worldwide and remain a major cause of maternal, foetal, and neonatal morbidity and mortality. Maternal risks include placental abruption, stroke, multiple organ failure, and disseminated intravascular coagulation. The fetus is at high risk of intrauterine growth retardation (25% of cases of preeclampsia), prematurity (27% of cases of preeclampsia), and intrauterine death (4% of cases of preeclampsia).

The definition of hypertension in pregnancy is based on office BP values, SBP at least 140 mmHg and/or DBP at least 90 mmHg, and is classified as mild (140–159/90–109 mmHg) or severe (≥160/110 mmHg), in contrast to the conventional hypertension grading.

1. **Pre-existing hypertension:** precedes pregnancy or develops before 20 weeks of gestation, and usually persists for more than 6 weeks postpartum and may be associated with proteinuria.
2. **Gestational hypertension:** develops after 20 weeks of gestation and usually resolves within 6 weeks postpartum.
3. **Pre-existing hypertension plus superimposed gestational hypertension with proteinuria**.
4. **Preeclampsia:** gestational hypertension with significant proteinuria (>0.3 g/24 h or ≥30 mg/mmol ACR). It occurs more frequently during the first pregnancy, in multiple pregnancy, in hydatidiform mole, in antiphospholipid syndrome, or with pre-existing hypertension, renal disease, or diabetes. It is often associated with foetal growth restriction due to placental insufficiency and is a common cause of prematurity. The only cure for preeclampsia is delivery. As proteinuria may be a late manifestation of preeclampsia, it should be suspected when de-novo hypertension is accompanied by headache, visual disturbances, abdominal pain, or abnormal laboratory tests, specifically low platelets and/or abnormal liver function.

**Antenatally unclassifiable hypertension:** this term is used when BP is first recorded after 20 weeks of gestation and it is unclear if hypertension was pre-existing. Reassessment 6 weeks postpartum will help distinguish pre-existing from gestational hypertension.

**CLINICAL MANAGEMENT OF HYPERTENSION IN PREGANANCY:**

**Mild hypertension of pregnancy (BP 140–159/90–109** mmHg**).** The goal of drug treatment of hypertension in pregnancy is to reduce maternal risk; however, the agents selected must be safe for the fetus. The benefits of drug treatment for mother and fetus in hypertension in pregnancy have not been extensively studied, with the best data from a single trial using alpha-methyldopa, performed 40 years ago. A further study suggested that tighter vs. less tight control of BP in pregnancy showed no difference in the risk of adverse perinatal outcomes and overall serious maternal complications. However, secondary analysis suggested that tighter control of BP may reduce the risk of developing more severe hypertension and preeclampsia.

**High blood pressure** during **pregnancy** poses various risks, including: Decreased blood flow to the placenta. If the placenta doesn't get enough blood, your baby might receive less oxygen and fewer nutrients. This can lead to slow growth (intrauterine growth restriction), low birth weight or premature birth.

In the United States, **high blood pressure** happens in 1 in every 12 to 17 **pregnancies** among women ages 20 to 44. **High blood pressure** in **pregnancy** has become more common. However, with good **blood pressure** control, **you** and your baby are more likely to stay **healthy**.

Most women with pre-existing hypertension and normal renal function will not have severe hypertension and are a low risk for developing complications during pregnancy. Indeed, some of these women may be able to withdraw their medication in the first half of pregnancy because of the physiological fall in BP. Despite the paucity of evidence, European Guidelines have recommended initiating drug treatment:

1. In all women with persistent elevation of BP at least 150/95 mmHg;
2. In women with gestational hypertension (with or without proteinuria), pre-existing hypertension with the superimposition of gestational hypertension, or hypertension with subclinical HMOD, when BP is more than 140/90 mmHg.

**CONCLUSION:**

**Hypertension** is a very important disorder in aged people and is associated with higher risk of cardiovascular morbidity and mortality. The fact of reducing **blood**

**pressure** values decreases the risk for cardiac death as well as neurological, metabolic, and musculoskeletal system sequelae in aged people.

**High blood pressure** (**hypertension**) is a common condition in which the long-term force of the blood against your artery walls is high enough that it may eventually cause health problems, such as heart disease.

**High blood pressure** can cause many complications. **High blood pressure** (**hypertension**) can quietly damage your body for years before symptoms develop. Uncontrolled **high blood pressure** can lead to disability, a poor quality of life, or even a fatal heart attack or stroke.

Common factors that can lead to **high blood pressure** include: A diet **high** in salt, fat, and/or cholesterol . **Chronic** conditions such as kidney and hormone problems, diabetes, and **high** cholesterol. Family history, especially if your parents or other close relatives have **high blood pressure**.

**REFERENCES**:


[1] Ahmed A., Rahman M., Hasan R., et al. Hypertension and associated risk factors in some selected rural areas of Bangladesh. *International Journal of Research in Medical Sciences.* 2014;2(3):p. 925. doi: 10.5455/2320-6012.ijrms20140816. [CrossRef] [Google Scholar]

[2] Forouzanfar MH, Liu P, Roth GA, Ng M, Biryukov S, Marczak L, et al. Global burden of hypertension and systolic blood pressure of at least 110 to 115 mm Hg, 1990–2015. *JAMA* 2017; 317:165–182. Cited Here

[3] Tsai WC, Wu HY, Peng YS, Yang JY, Chen HY, Chiu YL, et al. Association of intensive blood pressure control and kidney disease progression in nondiabetic patients with chronic kidney disease: a systematic review and meta-analysis. *JAMA Intern Med* 2017; 177:792–799. Cited Here.

[4] Banegas JR, Lopez-Garcia E, Dallongeville J, Guallar E, Halcox JP, Borghi C, et al. Achievement of treatment goals for primary prevention of cardiovascular disease in clinical practice across Europe: the EURIKA study. *Eur Heart J* 2011; 32:2143–2152. Cited Here.

[5] Chow CK, Teo KK, Rangarajan S, Islam S, Gupta R, Avezum A, et al. PURE Study InvestigatorsPrevalence, awareness, treatment, and control of hypertension in rural and urban communities in high-, middle-, and low-income countries. *JAMA* 2013; 310:959–968. Cited Here.

[6] Kearney PM, Whelton M, Reynolds K, Muntner P, Whelton PK, He J. Global burden of hypertension: analysis of worldwide data. *Lancet* 2005; 365:217–223. Cited Here.

[7] Vishram JK, Borglykke A, Andreasen AH, Jeppesen J, Ibsen H, Jorgensen T, et al. MORGAM ProjectImpact of age on the importance of systolic and diastolic blood pressures for stroke risk: the MOnica, Risk, Genetics, Archiving, and Monograph (MORGAM) project. *Hypertension* 2012; 60:1117–1123. Cited Here.

[8] Berry JD, Dyer A, Cai X, Garside DB, Ning H, Thomas A, et al. Lifetime risks of cardiovascular disease. *N Engl J Med* 2012; 366:321–329. Cited Here.

[9] Aktas MK, Ozduran V, Pothier CE, Lang R, Lauer MS. Global risk scores and exercise testing for predicting all-cause mortality in a preventive medicine program. *JAMA* 2004; 292:1462–1468. Cited Here.

[10] Cooney MT, Dudina AL, Graham IM. Value and limitations of existing scores for the assessment of cardiovascular risk: a review for clinicians. *J Am Coll Cardiol* 2009; 54:1209–1227. Cited Here.

[11] Borghi C, Agabiti Rosei E, Bardin T, Dawson J, Dominiczak A, Kielstein JT, et al. Serum uric acid and the risk of cardiovascular and renal disease. *J Hypertens* 2015; 33:1729–1741. discussion 1741.Cited Here.

[12] Parati G, Pomidossi G, Casadei R, Mancia G. Lack of alerting reactions to intermittent cuff inflations during noninvasive blood pressure monitoring. *Hypertension* 1985; 7:597–601. Cited Here.

[13] Bobrie G, Clerson P, Menard J, Postel-Vinay N, Chatellier G, Plouin PF. Masked hypertension: a systematic review. *J Hypertens* 2008; 26:1715–1725. Cited Here.

[14] Mancia G, Bombelli M, Cuspidi C, Facchetti R, Grassi G. Cardiovascular risk associated with white-coat hypertension: pro side of the argument. *Hypertension* 2017; 70:668–675. Cited Here.

[15] Huang Y, Huang W, Mai W, Cai X, An D, Liu Z, et al. White-coat hypertension is a risk factor for cardiovascular diseases and total mortality. *J Hypertens* 2017; 35:677–688. Cited Here.

[16] Briasoulis A, Androulakis E, Palla M, Papageorgiou N, Tousoulis D. White-coat hypertension and cardiovascular events: a meta-analysis. *J Hypertens* 2016; 34:593–599. Cited Here.

[17] Hodgkinson J, Mant J, Martin U, Guo B, Hobbs FD, Deeks JJ, et al. Relative effectiveness of clinic and home blood pressure monitoring compared with ambulatory blood pressure monitoring in diagnosis of hypertension: systematic review. *BMJ* 2011; 342:d3621. Cited Here.

[18] Chappell LC, Shennan AH. Assessment of proteinuria in pregnancy. *BMJ* 2008; 336:968–969. Cited Here.

[19] Redman CW. Fetal outcome in trial of antihypertensive treatment in pregnancy. *Lancet* 1976; 2:753–756. Cited Here.