SUMMARY

Hypertension is a progressive cardiovascular syndrome syndrome arising from complex and interrelated etiologies. Early markers of the syndrome are often present before blood pressure elevation is sustained . Progression is strongly associated with functional and structural cardiac and vascular abnormalities that damage the heart, kidneys, brain, vasculature, and other organs and lead to premature morbidity and death.(Giles TD et al.,2005)

More than a quarter of the adult population of the world – totalling nearly one billion – had hypertension in the year 2000, and this proportion is predicted to increase by about 60 % to a total of 1.56 billion by the year 2025.(Kearney PM et al.,2005)

Prehypertension is defined as a systolic BP level of 120–139 mmHg and/or diastolic BP level of 80–89 mmHg. Several studies showed that "prehypertension" is common and is associated with the metabolic syndrome and other CV risk factors, such as obesity, elevated triglycerides, elevated LDL cholesterol, and low levels of HDL cholesterol. Furthermore, during follow-up, subjects with prehypertension are more susceptible to developing true hypertension and coronary atherosclerosis. (Grossman A., et al 2006)

The objectives of defining this classification of blood pressure were to draw the clinical and public healthy attention on the prevention of people in this range. Prehypertension is a precursor of clinical hypertension and is closely related with the increased incidence of cardiovascular disease. (Vasan RS et al., 2001)

The relationship between uric acid and microalbuminuria in healthy adults without other cardiovascular risk factors may help to clarify the role of uric acid in cardiovascular disease. Elevated serum uric acid level associated with microalbuminuria among nondiabetic and nonhypertensive subjects without a history of cardiovascular disease or renal dysfunction, particularly subjects with prehypertension. (Knight EL et al., 2005)

Obesity is a risk factor for many borderline conditions such as prehypertension. The relationship between obesity and elevation of blood presssure is well established both in adults and children. Obese individuals exhibit higher levels of blood pressure from childhood to old age. Obese subjects display higher BP levels than non-obese individuals even in the normotensive range. (Stabouli S et al, 2005)

With each cigarette, the blood pressure rises transiently and the pressor effect may be missed if the blood pressure is measured 30 minutes after the last smoke. The transient rise in blood pressure may be most prominent with the first cigarette of the day even in habitual smokers. In one study of normotensive smokers, there was an average elevation in systolic pressure of 20 mmHg after the first cigarette. (Groppelli A et al., 1992)

In prehypertensive patients there is one or more cardiovascular risk factors like higher levels of total cholesterol (TC), low density lipoprotein cholesterol (LDL-C), triglyceride (TG), glucose, insulin, body mass index (BMI), a decreased high density lipoprotein cholesterol (HDL-C) and a less favorable body fat distribution, and the presence of one or more of these factors have been found to increase the progression of prehypertension to hypertension. (Haffner SM et al., 1992)

Excessive alcohol intake is a more frequent contributor to elevated blood pressure. In studies in which mean blood pressure levels were reported according to alcohol use categories, blood pressure elevations were 1. 6 to 10. 9 mmHg higher in the higher alcohol use groups than in the low or no use groups. When dose response relationship was evaluated, the subjects with the highest alcohol intake had the highest blood pressures. The magnitude of the increase in blood pressure in heavy drinkers averages about 5 to 10 mmHg, with systolic increases nearly always greater than diastolic increases. (Milton H et al., 1982)

Having a family history of high blood pressure places people in a higher risk category than others with no family history of high blood pressure. It is clear that family history plays an important role in determining risk, but there are probably more important factors, and they are under people control. (Winnicki M ., 2006)

Sedentary normotensive have 20–50% higher risk of developing hypertension than individuals who are undertaken physical exercise regularly.(Paffenbargar R.S.,1991)

Exposure to stress increases sympathetic outflow, and repeated stress-induced vasoconstriction may result in vascular hypertrophy, leading to progressive increases in peripheral resistance and blood pressure.(Oparil S et al.,2003)

It is important to measure blood pressure accurately. Many people have blood pressures in the prehypertensive range (ie, 120–139 mm Hg systolic; 80–89 mm Hg diastolic). Many people in this group can expect to develop hypertension in time, as the prevalence of hypertension increases steadily with age unless effective preventive measures are implemented,

such as losing weight, exercising regularly, and avoiding excessive consumption of sodium and alcohol. (Wenger NK 1988)

The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure indicates that people with a systolic blood pressure of under 130 mm Hg and a diastolic blood pressure of under 85 mm Hg should be screened every 2 years, while people with elevated blood pressure (130/85 mm Hg or above) should be screened more frequently. (Chobanian AV et al 2003)

Individuals with prehypertensive levels of blood pressure have an increased risk of developing cardiovascular disease relative to those with optimal levels. The association pronounced among individuals with diabetes mellitus, and among those with high BMI. (Kshirsagar A V et al., 2006)

Prehypertension accelerates the development of left ventricular (LV) hypertrophy and diastolic dysfunction. prehypertensive individuals have significantly greater age-related increase in LV wall thickness and LV mass and an increased incidence of LV concentric remodeling and LV hypertrophy, compared with individuals with normal blood pressure. (Markus M R ., 2008)

The proportion of cardiovascular disease is attributable to high blood pressure . This burden applies to different economic regions, age-groups, and blood pressure levels and is not limited to subjects with hypertension but who are not classified as hypertensive. Worldwide, 7. 6 million premature deaths (13. 5% of the global total) and 92 million disability-adjusted life-years (6. 0% of the global total) were attributed to high BP. About 50% of this affliction occurred in individuals with

hypertension, but the remainder was in individuals with lesser degrees of high BP.(Lawes CM et al.,2008)

Both lifestyle changes and drug therapy have been shown at least temporarily to slow the progression of prehypertension into hypertension. The trials, up til now, have likely been too late to stop the progress, because, at least in the spontaneously hypertensive rate, antihypertensive therapy must be given much earlier in the life span to prevent the future development of hypertension.(Bavikati VV et al., 2008)

JNC-7 recommends 5 nonpharmacological treatments for prehypertension (DASH dietary pattern rich in potassium and calcium and reduced in total and saturated fat, weight loss, reduced sodium intake, regular physical activity, and moderation of alcohol intake). Each has been proven in clinical trials to significantly lower BP, and most have been shown to prevent the development of hypertension. (Chobanian AV et al., 2003)

The JNC-7 report recommends drug treatment if a trial of lifestyle modification fails to reduce BP to 130/80 mm Hg or less in patients with either DM or CKD, implying that prehypertension in these clinical subgroups should be treated pharmacologically. These recommendations are based on evidence from clinical trials that suggest that the goal of BP treatment should be lower in this population once hypertension develops. (Hansson L et al., 1998)