

## ASSOCIATION BETWEEN ABNORMAL BODY MASS INDEX AND BLOOD PRESSURE IN DIABETIC PATIENTS IN THE AREA OF KANPUR (NORTH INDIA)

ANANT SACHAN<sup>a</sup>, ANIL KUMAR<sup>b1</sup>, R. K. SRIVASTAVA<sup>c</sup>, SHIRIN JAHAN<sup>d</sup>, PRANJAL PANKAJ<sup>e</sup> AND YATINDRA KATIYAR<sup>f</sup>

<sup>a,c,d</sup>Department of Anatomy, Rama Medical College Hospital and Research Centre Kanpur, Uttar Pradesh, India

<sup>b</sup>Central Research Laboratory, Rama Medical College Hospital and Research Centre, Mandhana, Kanpur, (UP) India

<sup>e</sup>Department of Medicine, Rama Medical College Hospital and Research Centre Kanpur, Uttar Pradesh, India

<sup>f</sup>Department of Pharmacy, GSVM Medical College, Kanpur, India

### ABSTRACT

The entire study was carried at Rama Medical College, Hospital and Research Centre, Kanpur jointly in the department of Anatomy and Central Research Laboratory. The main focus was to establishment an association between abnormal body mass index and blood pressure in diabetic patients of this region. The mean age of the participants at the time of registration was around 45.33±9.1years. The mean HbA1c values for hypertension were found significantly different between patients and controls. The mean blood pressure values were mainly systolic calculated at onset of hypertension point and found statistically significant. It was also noted that the systolic blood pressures were recorded to increase gradually with the increase in BMI in the patients. There was a statistically significant increase in the systolic blood pressure between diabetics and non diabetics group (p<0.0001).

**KEYWORDS:** Systole and Diasystole, Body Mass Index, Hypertension, Statistically Significant

Obesity has been described as a public health problem of epidemic proportions (WHO, 1998). This chapter defines obesity and provides an overview of the literature regarding the prevalence and etiology of obesity, the risk factors and physiological complications associated with obesity and the estimated economic costs of obesity.

#### Definition & Classification of Obesity

Obesity is characterized by excess adipose tissue and it is normally defined using Quetelet's index, more commonly known as the body mass index (BMI). BMI has been used for many large-scale epidemiological studies and only individuals who are unusually muscular

could be misclassified as overweight or obese (Prentice, 1998). BMI is calculated by dividing body weight (kg) by height squared (m<sup>2</sup>). The World Health Organization (WHO) has published guidelines based on the associations between BMI and all cause mortality and a BMI of 30kg/m<sup>2</sup> is widely recognized as the cut off point for adult obesity (see table 1.1). The classification for obesity in children is complicated and is not a perfect measure because during childhood there are sporadic changes in height and body composition. However, internationally based cut-off points have been published (Edmunds, Waters & Elliott, 2001; Cole, Bellizzi, Flegal & Dietz, 2000).

**Table 1: WHO Classification of under and overweight in adults according to BMI**

Classification	BMI (kg/M <sup>2</sup> )	Risk of Comorbidities
Underweight	<18.5	Low
Healthy Weight	18.5-24.9	Average
Overweight	25.0-29.9	Increased
Obese Class I	30.0-34.9	Moderate
Obese Class II	35.0-39.9	Severe
Obese Class III (Morbid)	>40.0	Very Severe

Although BMI is a widely used method for defining obesity it must be acknowledged that the cut-off point is arbitrary and the main function of the categories is to enable comparison with other countries (James, Leach, Kalamara & Shayeghi, 2001). The BMI cut-off point indicates the increased risk of health complications. It does not mean that everyone with a BMI greater than 30kg/m<sup>2</sup> will develop obesity-related health problems:

#### Diabetes

Diabetes is a chronic disease that occurs either when the pancreas does not produce enough insulin or when the body cannot effectively use the insulin it produces (1). There are mainly two types of diabetes; Type 1 diabetes is immune-mediated and requires daily administration of insulin. The other common type is type

<sup>1</sup>Corresponding Author

2 diabetes and characterized by insulin resistance or relative insulin deficiency (1, 2). Type 2 diabetes is the most common form and comprises of 90% of people with diabetes around the world (1). The prevalence of type 2 diabetes rates continue to increase with increasing number of patients at risk of serious diabetes-related complications. Having type 2 diabetes is increase the risk of a myocardial infarction two times more and the risk of suffering a stroke two to four times more. It is also a leading cause of blindness, limb amputation and kidney failure (1, 3-5). Although trials of secondary prevention after myocardial infarction show as good or better short term effect of interventions in patients with diabetes as in patients without, patients with diabetes have not had a similar reduction in longer-term case fatality rates of cardiovascular disease (CVD) (6). Population based studies of CVD risk factor trends among subjects with and without diabetes show differing trend in disfavor of those with diabetes (7). Studies of adherence to guidelines for CVD prevention targets in patients with diabetes in general practice have shown that only 13% reach all the targets (8). Previous studies have found appropriate lifestyle intervention and/or drug treatment is effective in delaying or preventing both diabetes and its complications (9-12). Accordingly, simple, sensitive and acceptable tools for identification of subjects at risk are warranted.

### **Epidemiology of type 2 diabetes**

The world prevalence of diabetes in 2010 among adults aged 20-79 years is estimated to 6.4%, affecting 285 million adults (13). Between 2010 and 2030, there is an expected 70% increase in numbers of adults with diabetes in developing countries and a 20% increase in developed countries (13). In several studies that a clustering of features, such as high plasma glucose, obesity, dyslipidemia (high triglyceride and total cholesterol levels low high density lipoprotein (HDL) cholesterol levels and hypertension, referred to as insulin resistance or the metabolic syndrome, is a marker of increased risk for the development of type 2 diabetes as well as for CVD (16,17). Environmental and lifestyle factors are the main causes of the dramatic increase in type 2 diabetes prevalence (18-20). Genetic factors probably identify those most vulnerable to these changes. Furthermore, studies have shown certain ethnic groups to be more susceptible to developing diabetes than others (21, 22).

### **Risk factors for type 2 diabetes:**

Many studies have elaborated the associations between several risk factors and the risk of type 2 diabetes. Body mass index (BMI), lipids, hypertension,

smoking; physical inactivity, low education, dietary patterns, family history, and recently also specific genes are the most frequently documented risk factors for type 2 diabetes (26-32).

### **Hypertension**

Hypertension is one of the most important causes of the total disease burden in the world (1). According to large observational studies, hypertension is thus associated with high incidence of cardiovascular disease, such as stroke, ischemic heart disease, and other vascular diseases (2-5). An increased incidence of cardiovascular disease has in fact been seen in relation to blood pressure levels across the entire blood pressure distribution (6-8), also within the normal blood pressure range (9-11). For half a century, treatment and awareness of high blood pressure has been insufficient, as described by the rule of halves (12-14), meaning that only half of those identified with hypertension were aware of their condition, and only half of those aware were treated, and of those treated only half achieved treatment goals. Even though treatment has contributed to a reduction of cardiovascular events, the control of high blood pressure and hypertension can still be improved (15).

It is critical that health care providers follow the standards for accurate BP measurement. BP should be categorized as normal, elevated, or stages 1 or 2 hypertension to prevent and treat high BP. Normal BP is defined as  $<120/<80$  mm Hg; elevated BP  $120-129/<80$  mm Hg; hypertension stage 1 is  $130-139$  or  $80-89$  mm Hg, and hypertension stage 2 is  $\geq 140$  or  $\geq 90$  mm Hg. Prior to labeling a person with hypertension, it is important to use an average based on  $\geq 2$  readings obtained on  $\geq 2$  occasions to estimate the individual's level of BP. Out-of-office and self-monitoring of BP measurements are recommended to confirm the diagnosis of hypertension and for titration of BP-lowering medication, in conjunction with clinical interventions and tele-health counseling. Corresponding BPs based on site/methods are: office/clinic  $140/90$ , HBPM  $135/85$ , daytime ABPM  $135/85$ , night-time ABPM  $120/70$ , and 24-hour ABPM  $130/80$  mm Hg. In adults with an untreated systolic BP (SBP)  $>130$  but  $<160$  mm Hg or diastolic BP (DBP)  $>80$  but  $<100$  mm Hg, it is reasonable to screen for the presence of white coat hypertension using either daytime ABPM or HBPM prior to diagnosis of hypertension. In adults with elevated office BP ( $120-129/<80$ ) but not meeting the criteria for hypertension, screening for masked hypertension with daytime ABPM or HBPM is reasonable.

Antihypertensive drug treatment is a cornerstone in lowering blood pressure (BP), where the majority of

patients often require 2 or more antihypertensive drugs to achieve BP goals (BP<140/90 mmHg in general; BP<130/80 mmHg in patients with diabetes). 2, 5, 8, 9 Factors other than pharmacological treatment affect a patient's ability to achieve BP control, e.g. lifestyle modifications, 10-12 doctor's inertia 13-19 and a patient's socioeconomic status. 20-22

## MATERIALS AND METHODS

Body Mass Index-

The formula for calculating BMI is:

$$\text{BMI (kg / m}^2\text{)} = \frac{\text{Weight (kg)}}{\text{Height (m)}^2}$$

**Table 2: Estimation of body mass index**

BMI (kg/m <sup>2</sup> )	Weight Status
Below 18.5	Underweight
18.5 to 24.9	Healthy weight
25.0 and above	Overweight
30.0 and above	Obese

### HbA1c

#### Materials Required:

**Collection of blood sample:** About 2ml of patient's blood will be collected by vein puncture into a tube, and the tube containing a mixture (anticoagulant mixture) of ethylene diammine tetra acetic acid (EDTA) and sodium fluoride in the ratio of 1:2 (W/W). 5 mg of the mixture is enough for 2ml of blood sample. The tube should be thoroughly shaken for complete mixing.

#### Study design and population

This is a cross-sectional study. The source population is Type 2 Diabetes Mellitus patients attending diabetic clinic in HUSM, Kubang Kerian Kelantan in February 2010. The inclusion criteria include Type 2 Diabetes Mellitus, Malay and adults aged 18 years old. Newly diagnosed Diabetes Mellitus type 2 patients of less than one year, patients diagnosed as having Diabetic Type 1, gestational diabetes and/or chronic kidney disease were excluded. Systematic random sampling was applied.

#### Operational definitions

In this study HbA1c values below 6.5% was taken as glycaemic control as recommended by The International Diabetes Federation 13. The HbA1c test is a way to assess blood glucose control during the previous

three to four months. It is measured by cation exchanger high performance liquid chromatography (HPLC).

#### Method of data collection

The methods of data collection were via: questionnaire, physical and anthropometric measurements and review of medical records. Short questionnaire was answered by the patients with guidance from single research assistant in Bahasa Melayu. Socio-demographic characteristics include age, sex, and household income per month, educational level and type of occupation. Type of occupation was classified into sedentary jobs which require prolonged sitting or no physical activity and active jobs which require physical activity. The clinical histories of the patients include the duration of diabetes, family history of diabetes, and family history of obesity, smoking habit, dietary fiber intake habit and physical activity status. The patient was considered smoker if he was smoking at the time of the study.

#### Data analyses

Data was analyzed using Statistical Packages for Social Sciences (SPSS) version 12.0.1 15. The distributions and frequencies were examined. The continuous variables were expressed as mean and standard deviation or median and inter quartile range. The frequencies and percentages for categorical variables were calculated. The correlation between anthropometric parameters and HbA1c was tested using Pearson product moment correlation and the correlation coefficient *r* was used to test the strength and direction of correlation. Assumptions and outliers were checked using scatter plot.

#### Blood Pressure Measurement

To reduce misclassification of hypertension, blood pressure should be measured in a standardized fashion (81, 82). Intra-individual variation in blood pressure, rather than a so-called white coat effect (83), is known to cause an overestimation of hypertension, and accordingly repeated readings are recommended (84). The recent ESH/ESC guidelines further recommend (28):

- To allow the patients to sit for 3-5 minutes before beginning blood pressure measurements.
- To take at least two blood pressure measurements in the sitting position spaced 1-2 min apart.
- Consider the mean of the two blood pressure readings if deemed appropriate.
- 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 (arm circumference 17-22 cm), respectively.

- To have the cuff at the heart level, whatever the position of the patient.

Furthermore, a quiet, comfortable location at normal room temperature is optimal. Ideally, the patient should not recently have eaten, smoked, exercised, or taken caffeine (29, 82). When adopting the auscultator method, the phase I (appearance) and V (disappearance) of Korotkoff sounds should be used to identify systolic and diastolic blood pressure, respectively. Provided that the arm is supported at the heart level, sitting and supine blood pressure measurements are considered comparable (82). The observer should be well trained in the techniques of blood pressure measurement and use an accurate and properly maintained device (28, 85).

The study was approved by the Institutional Ethics Committee. This study was conducted in Rama Medical College Hospital and Research center, Kanpur. Data were recorded from outpatients visited Rama Hospital. In this study total 160 subjects were observed for BMI, type 2 diabetes and hypertension. All 160 participants were diagnosed for hypertension and diabetes. Measurements such as BMI, glycated

hemoglobin (HbA1c) levels, and blood pressure readings were made as per international guidelines.

BMI classifications approved by the WHO were used to categories patients: normal weight (BMI=18.5-24.9 kg/m<sup>2</sup>), overweight (25.0-29.9), class I obesity (30.0-34.9), class II obesity (35.0-39.9) and class III obesity ( $\geq 40$ ). We evaluated associations between BMI and age at onset of hypertension.

## RESULTS

Descriptive statistics on the two data sets are summarized in Table 1 and Table 2. The mean age of the participants at the time of registration is around 45.33 $\pm$ 9.1 years.

The mean HbA1c values for hypertension are significantly different between patients with and without diabetes. The mean blood pressure values mainly systolic measured at onset of hypertension point and found statistically significant. The systolic blood pressures were found to increase progressively with the increase in BMI in both groups of patients. There was a statistically significant increase in the systolic blood pressure between diabetes and non diabetes group ( $p < 0.0001$ ).

**Table 3: Calculation of hypertension in diabetic and non diabetic cases and controls**

S.N.	Characteristics	Hypertension in patients with T2DM (n=55)	Hypertension in patients without diabetes (n=105)
1.	Sex distribution		
	Male	44.3% (n=25)	35.2% (n=37)
	Female	55.3% (n=30)	64.7% (n=68)
2.	BMI distribution		
	Overweight	71.4% (n=40)	67.6% (n=71)
	obesity	28.5% (n=15)	32.4% (n=34)

**Table 4: Calculation of hypertension parameter in diabetic and non diabetic cases and controls**

S.N.	Characteristics	Hypertension in patients with T2DM (n=55)	Hypertension in patients without diabetes (n=105)	P value
1.	Mean age	45.33 $\pm$ 9.1	43.27 $\pm$ 9.6	0.1887
2.	Mean blood pressure Systolic (mmHg)	128.5 $\pm$ 18.7	120.38 $\pm$ 17.6	0.0069
3.	Mean BMI (kg/m <sup>2</sup> )	28.7 $\pm$ 2.9	38.7 $\pm$ 2.7	< 0.0001
4.	Mean HbA1C (%)	7.8 $\pm$ 1.0	5.2 $\pm$ 0.7	<0.0001

## DISCUSSION

This study found similar results as mentioned in HUSM, Kubang Kerian Kelantan in February 2010. The obtained data are also co-related with several co-workers such as stated by Reaven and Banting in 1988.

## ACKNOWLEDGEMENT

The Authors are grateful to the principal of Rama Medical College, Hospital and Research Centre, Kanpur for his kind consideration and permission of the

original research work at this centre and for the financial assessment.

## REFERENCES

- Prentice A.M., 1998. Body mass index standards for children. *BMJ*, **317**: 1401-1402.
- Edmunds L., Waters E. and Elliott E.J., 2001. Evidence based management of childhood obesity. *BMJ*, **323**: 916-919.
- Cole T.J., Bellizzi M.C., Flegal K.M. and Dietz W.H., 2000. Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ*, **320**: 1240-1243.
- James P.T., Leach R., Kalamara E. and Shayeghi M., 2001. The Worldwide Obesity Epidemic. *Obesity Research*, **9**: S228-S233.
- World Health Organization. Fact Sheet No.312: What is Diabetes? September 5, 2009.
- International Diabetes Federation. Diabetes Atlas. 4th edn. Brussels: International Diabetes Federation, 2009.
- International Diabetes Federation. Diabetes Atlas. 3rd edn. Brussels: International Diabetes Federation, 2006.
- Centers for Disease Control. National Diabetes Fact Sheet-2007. February 18, 2009.
- Agency for healthcare research and quality. Screening for type 2 diabetes mellitus in adults. Accessed on: February 19, 2009.
- Cubbon R.M., Wheatcroft S.B., Grant P.J., Gale C.P., Barth J.H. and Sapsford R.J., 2007, Temporal trends in mortality of patients with diabetes mellitus suffering acute myocardial infarction: a comparison of over 3000 patients between 1995 and 2003. *Eur. Heart J.*, **28**(5):540-5.
- Preis S.R., Pencina M.J., Hwang S.J., D'Agostino R.B., Sr. Savage P.J. and Levy D., 2009. Trends in cardiovascular disease risk factors in individuals with and without diabetes mellitus in the Framingham Heart Study. *Circulation*, **120**(3):212-20.
- Jenssen T.G., Tonstad S., Claudi T., Midthjell K. and Cooper J., 2008. The gap between guidelines and practice in the treatment of type 2 diabetes A nationwide survey in Norway. *Diabetes Res Clin Pract*, **80** (2):314-20.
- Tuomilehto J., Lindstrom J., Eriksson J.G., Valle T.T., Hamalainen H. and Ilanne-Parikka P., 2001. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N. Engl. J. Med.*, **344** (18):1343-50.
- Knowler W.C., Barrett-Connor E., Fowler S.E., Hamman R.F., Lachin J.M. and Walker E.A., 2002. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N. Engl. J. Med.*, **346**(6):393-403.
- Ramachandran A., Snehalatha C., Mary S., Mukesh B., Bhaskar A.D. and Vijay V., 2006. "The Indian Diabetes Prevention Programme shows that lifestyle modification and metformin prevent type 2 diabetes in Asian Indian subjects with impaired glucose tolerance (IDPP-1)." *Diabetologia*, **49**(2):289-97.
- Effect of intensive blood-glucose control with metformin on complications in overweight patients with type 2 diabetes (UKPDS 34). *Lancet* 1998; **352**: 854-65.
- Shaw J.E., Sicree R.A. and Zimmet P.Z., 2010 Global estimates of the prevalence of diabetes for 2010 and 2030. *Diabetes Res. Clin. Pract.*, **87**(1):4-14.
- Taskinen M.R., 2003. Diabetic dyslipidaemia: from basic research to clinical practice. *Diabetologia*, **46**(6):733-49.
- Reaven G.M. Banting lecture 1988. Role of insulin resistance in human disease. *Diabetes* 1988; **37**(12):1595-607.
- Diamond J. The double puzzle of diabetes. *Nature* 2003; **423**(6940):599-602.
- Hu F.B., Manson J.E., Stampfer M.J., Colditz G., Liu S. and Solomon C.G., 2001. Diet, lifestyle, and the risk of type 2 diabetes mellitus in women. *N. Engl. J. Med.*, **345**(11):790-7.
- Zimmet P., Alberti K.G.M.M. and Shaw J., 2001. Global and societal implications of the diabetes epidemic. *Nature*, **414** (6865):782-7.
- Shai I., Jiang R., Manson J.E., Stampfer M.J., Willett W.C. and Colditz G.A., 2006. Ethnicity, obesity, and risk of type 2 diabetes in women. *Diabetes Care*, **29**(7):1585-90.
- McBean A.M., Li S., Gilbertson D.T. and Collins A.J., 2004. Differences in diabetes prevalence, incidence, and mortality among the elderly of

- four racial/ethnic groups: Whites, Blacks, Hispanics, and Asians. *Diabetes Care*, **27**(10):2317-24.
- Valdes S., Botas P., Delgado E., Alvarez F. and Cadorniga F.D., 2007. Population-based incidence of type 2 diabetes in northern Spain: the Asturias Study. *Diabetes Care*, **30**(9):2258-63.
- Meisinger C., Thorand B., Schneider A., Stieber J., Doring A. and Lowel H., 2002. Sex differences in risk factors for incident type 2 diabetes mellitus: the MONICA Augsburg cohort study. *Arch Intern Med.*, **162**(1):82-9.
- Haffner S.M., Miettinen H. and Stern M.P., 1997. Relatively more atherogenic coronary heart disease risk factors in prediabetic women than in prediabetic men. *Diabetologia*, **40**(6):711-738.
- Bassuk S.S. and Manson J.E., 2008. Lifestyle and risk of cardiovascular disease and type 2 diabetes in women: A review of the epidemiologic evidence. *Am J Lifestyle Med.*, **2**(3):191-213.
- Gadsby R., 2002. Epidemiology of diabetes *Adv Drug Deliv Rev.*, **54**(9):1165-72.
- Chan J.C., Malik V., Jia W., Kadowaki T., Yajnik C.S. and Yoon K.H., 2009. Diabetes in Asia: epidemiology, risk factors, and pathophysiology. *JAMA*, **301**:2129-40.
- Lyssenko V., Jonsson A., Almgren P., Pulizzi N., Isomaa B. and Tuomi T., 2008. Clinical risk factors, DNA variants, and the development of type 2 diabetes. *N. Engl. J. Med.*, **359**(21):2220-32.
- Lim S.S., Vos T., Flaxman A.D., Danaei G., Shibuya K. and Adair-Rohani H., A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 2012; **380**: 2224-60.
- Stokes J., Kannel W.B., Wolf P.A., D'Agostino R.B. and Cupples L.A., 1989. Blood pressure as a risk for cardiovascular disease. *Hypertension*, **13**(5 suppl):113-8.
- Kannel W.B., Wolf P.A., Verter J. and McNamara P.M., 1970. Epidemiologic assessment of the role of blood pressure in stroke. The Framingham study. *JAMA*, **214**: 301-10.
- Kannel W.B., Castelli W.P., McNamara P.M., McKee P.A. and Feinleib M., 1972. Role of blood pressure in the development of congestive heart failure. The Framingham study. *NEJM*, **287**:781-7.
- Whelton P.K., Perneger T.V., Brancati F.L. and Klag M.J., 1992. Epidemiology and prevention of blood pressure-related renal disease. *J. Hypertens Suppl.*, **10**:S77-84.
- MacMahon S., Peto R., Cutler J., Collins R., Sorlie P. and Neaton J., 1990. Blood pressure, stroke, and coronary heart disease. Part 1. Prolonged differences in blood pressure: prospective observational studies corrected for the regression dilution bias. *Lancet*, **335**:765-74.
- Lewington S., Clarke R., Qizilbash N., Peto R. and Collins R., 2002. Prospective Studies Collaboration. Age-specific relevance of usual blood pressure and vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. *Lancet*, **360**:1903-13.
- Staessen J.A., Li Y., Thijs L. and Wang J.G., 2005. Blood pressure reduction and cardiovascular prevention: an update including the 2003-2004 secondary prevention trials. *Hypertens Res.*, **28**: 385-407.
- Vasan R.S., Larson M.G., Leip E.P., Evans J.C., O'Donnell C.J. and Kannel W.B., 2001. Impact of high-normal blood pressure on the risk of cardiovascular disease. *NEJM*, **345**:1291-7.
- Liszka H.A., Mainous A.R., King D.E., Everett C.J. and Egan B.M., 2005. Prehypertension and cardiovascular morbidity. *Ann. Fam. Med.*, **3**:294-9.
- Zhang Y., Lee E.T., Devereux R.B., Yeh J., Best L.G., Fabsitz R.R. and Howard B.V., 2006. Prehypertension, diabetes, and cardiovascular disease risk in a 47 population based sample: the Strong Heart Study. *Hypertension*, **47**: 410-4.
- Wilber J.A. and Barrow J.G., 1972. Hypertension: A community problem. *Am. J. Med.*, **52**: 653-63.
- Hart J.T., 1970. Semicontinuous screening of a whole community for hypertension. *Lancet*, **2**: 223-6.
- Smith W.C., Lee A.J., Crombie I.K., Tunstall-Pedoe H., 1990. Control of blood pressure in Scotland: The rule of halves. *BMJ*, **300**: 981-3.

- Lindblad U., Ek J., Eckner J., Larsson C.A., Guangliang S. and Råstam L., 2011. Prevalence, awareness, treatment and control of hypertension - rule of thirds in The Skaraborg Project. *SJPHC*, **30**: 88-94.
- IDF-WPR. International Diabetes Federation Western Pacific Region (IDF-WPR) Type 2 Diabetes Practical Targets and Treatment, Fourth Edition. 2005.
- SPSS Inc. SPSS 12.0.1 for Windows.) Chicago: SPSS Inc., 2003.
- Mancia G., Fagard R., Narkiewicz K., Redon J., Zanchetti A. and Böhm M., 2013. Practice guidelines for the management of arterial hypertension of the European Society of Hypertension (ESH) and the European Society of Cardiology (ESC): ESH/ESC Task Force for the Management of Arterial Hypertension. *J. Hypertens.*, **31**:1925-38.
- National Clinical Guidance Centre, Hypertension (NICE CG 127). National Institute for Health and Clinical Excellence, 2011.
- Coope J., 1974. A screening clinic for hypertension in general practice. *J Royal College of General Practitioners*, **24**:161-6.
- Pickering T.G., Hall J.E., Appel L.J., Falkner B.E., Graves J. and Hill M.N., 2005. Recommendations for blood Pressure Measurement in humans and experimental animals: Part 1: Blood pressure measurement in humans: 53 a statement for professionals from the Subcommittee of Professional and Public Education of the American Heart Association Council on High Blood Pressure Research. *Hypertension*, **45**: 142-61.
- Mancia G., Parati G., Pomidossi G., Grassi G., Casadei R. and Zanchetti A., 1987. Alerting reaction and rise in blood pressure during measurement by physician and nurse. *Hypertension*, **9**: 209-15.
- Rosner B. and Polk B.F., 1983. Predictive values of routine blood pressure measurements in screening for hypertension. *Am. J. Epidemiol.*, **117**: 429-42.
- Godwin M., Delva D., Seguin R., Casson I., MacDonald S. and Birtwhistle R., 2004. Relationship between blood pressure measurements recorded on patients' charts in family physicians' offices and subsequent 24 hour ambulatory blood pressure monitoring. *BMC Cardiovasc Disord*, **4**: 2.