

UNDERWRITER RESOURCE

SERVING THE LIFE INSURANCE INDUSTRY

Volume 2, Issue 1
January 2008

The *Underwriter Resource* is published six times per year to inform, instruct and inspire the would-be, beginning and professional underwriter.

Hypertension: An Overview

Hypertension affects 65 million adults in the United States and is a major risk factor for myocardial infarction, stroke, heart failure and renal failure. Data from observational studies involving more than 1 million individuals have indicated that death from both ischemic heart disease and stroke increases progressively and linearly from levels as low as 115 mmHg systolic blood pressure (SBP) and 75 mmHg diastolic blood pressure (DBP). This increased risk is present in individuals ranging from ages 40 to 89; for every 20 mmHg increase of SBP or 10 mmHg increase of DBP mortality from ischemic heart disease and stroke doubles. Randomized controlled clinical trials have convincingly demonstrated that the treatment of hypertension reduces the risk of death from cardiovascular and renal disease (NIH Publication No. 04-5230).

"Mortality from hypertension is determined more by the overall risk burden than the level of blood pressure."

The prevalence of hypertension is higher at the older ages (table 1) and in those with increasing body mass index (BMI), non-Hispanic black ethnicity and those with less education.

Data from the National Health and Nutrition Examination Survey (NHANES) 1999-2004 indicate that only two thirds of those with hypertension are aware of their diagnosis and only half are receiving treatment for their hypertension. Of those receiving therapy, a third do not have adequate control of the blood pressure (Hypertension. 2007; 49:69-75).

The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC 7) defines hypertension as SBP greater than or equal to 140 mmHg or DBP greater than or equal to 90. Those with a diagnosis of hypertension are further classified as Stage 1 or Stage 2 hypertension based on the magnitude of the blood pressure elevation (table 2). This definition is based on office blood pressure measurements (Ambulatory Blood Pressure Monitoring, Underwriter Resource. September 2007; volume 1, issue 1).

Table 1. Prevalence of Hypertension in the US Population 2003-2004

Age	Prevalence as a Percentage of Population
18-39	7.3
40-59	28.1
>60	66.3

Source: Hypertension. 2007; 49:69-75

Table 2. Classification of Hypertension

	SBP mmHg	DBP mmHg
Normal	<120	<80
Prehypertension	120-139	Or 80-89
Stage 1 Hypertension	140-159	Or 90-99
Stage 2 Hypertension	≥160	≥100

Source: The JNC 7 report (NIH Publication No. 04-5230)

Table 3. Four-Year Rates of Progression to Hypertension (95% CI)

Baseline BP category mmHg	Age 35 to 64	Age 65 to 94
<120/80	5.3 (4.4-6.3)	16.0 (12.0-20.9)
120-129/80-84	17.6 (15.2-20.3)	25.5 (20.4-31.4)
130-139/85-89	37.3 (33.3-41.5)	49.5 (42.6-56.4)

Source: Lancet 2001; 358:1862-86

Data from a Framingham cohort study (Lancet 2001; 358:1862-86) indicates that those with blood pressure which is higher than normal but not high enough to qualify for the diagnosis of hypertension are at a significant risk for developing hypertension in four years when compared to those who have blood pressure in the normal range. Therefore this group is referred to as having prehypertension (table 3).

Inside this issue...

- Hypertension: An Overview
- Hypertension Related Mortality: A Cost Effective Approach to Risk Selection

Dr. RK Illango, President
RK Illango Consulting, Inc.
21021 Goshen Road
Gaithersburg, MD 20882

www.rkillango.com
301.963.4650 tel
301.963.4651 fax
rk@rkillango.com



Hypertension is most often the result of a complex interaction of genetic and environmental factors, and in ninety five percent of cases, no cause can be identified. These cases are referred to as primary or essential hypertension; the others are referred to as secondary hypertension. The frequently encountered causes of secondary hypertension are summarized in table 4.

Evaluation of a Hypertensive Patient

Evaluation of a patient with hypertension involves the following:

Causes	Comments
Chronic kidney disease	Most common cause of secondary hypertension.
Renal artery stenosis	Constitutes one to two percent of hypertensive patients. Fibromuscular hyperplasia is the cause in those under age 50 especially women; the rest are due to atherosclerotic stenosis of proximal renal arteries.
Primary hyperaldosteronism	Most common, potentially curable cause, usually due to adrenal adenoma or bilateral hyperplasia.
Cushing's syndrome other glucocorticoid excess	Includes those on steroid therapy.
Pheochromocytoma	Uncommon, found in less than 0.1% of all cases of hypertension.
Coarctation of the aorta	Uncommon cause.
Hypertension associated with pregnancy	One of the common causes of maternal and fetal morbidity and mortality.

Source: Current Medical Diagnosis and Treatment 2008

Major Risk Factors	Target Organ Damage and Associated Disorders
Smoking Dyslipidemia Age older than 60 years Sex (men and postmenopausal women) Family history of cardiovascular disease: women under age 65 or men under age 55	Heart diseases • Left ventricular hypertrophy • Angina/prior myocardial infarction • Prior coronary revascularization • Heart failure Stroke or transient ischemic attack Peripheral arterial disease Retinopathy Renal dysfunction Diabetes mellitus

Source: JNC 6 report (NIH Publication No. 98-4080)

Blood Pressure Stages (mmHg)	Risk Group A	Risk Group B	Risk Group C
Prehypertension (120–139/80–89)	Lifestyle modification	Lifestyle modification	Drug therapy
Stage 1 (140–159/90–99)	Lifestyle modification (up to 12 months)	Lifestyle modification (up to 6 months)	Drug therapy
Stage 2 (≥160 / ≥100)	Drug therapy	Drug therapy	Drug therapy

Source: JNC 6 and 7 reports

- Identifying lifestyle and other cardiovascular risk factors.
- Looking for the presence of concomitant diseases such as diabetes, chronic kidney disease or cardiovascular disorders.
- Screening for evidence of target organ damage such as left ventricular hypertrophy, albuminuria and measuring carotid intima-media thickness (IMT) using carotid ultrasound.

As the vast majority of hypertension cases are primary, additional diagnostic procedures to identify the cause of hypertension is only required when signs and symptoms or routine testing indicates a secondary cause for the hypertension or when onset of hypertension is sudden or is difficult to control.

Risk Stratification

The goal of therapy in hypertension is reduction in cardiovascular and renal morbidity and mortality. This is achieved by reducing global risk burden (table 5) and reducing blood pressure, according to The Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC 6).

It recommends that both global risk burden and severity of hypertension be used to categorize hypertensives into risk groups when devising treatment strategies for hypertension (table 6). The risk groups are as follows:

- Risk group A (low risk) would include those who have no cardiovascular risk factors and those with no evidence of target organ damage or associated disorders such as cardiovascular disorders, renal disorders or diabetes.
- Risk group B (intermediate risk) would include those with one or more cardiovascular risk factors but no evidence of target organ damage or associated cardiovascular disorders, renal disorders or diabetes.
- Risk group C (high risk) would include those with evidence of target organ damage or those who have associated cardiovascular disorders, renal disorders or diabetes.

Obesity and physical inactivity are also predictors of cardiovascular risk and interact with other risk factors, but they are of less significance in the selection of antihypertensive treatment.

The effectiveness of this therapeutic strategy has been evaluated by investigators using data from the NHANES Epidemiologic Follow-up Study (Hypertension. 2000; 35:539-543). In this study (table 7), participants were grouped into risk categories as recommended in the JNC 6 report and the effect of therapy was measured. The results were expressed as the number of patients who required treatment with a reduction of 12 mmHg SBP over ten years to prevent one death.

Specifically, the number who would require treatment to prevent a death from all causes in patients with prehypertension, Stage 1 hypertension, or Stage 2 hypertension was, respectively, 81, 60, and 23 for those in risk group A; 19, 16, and 9 for those in risk group B; and 14, 12,

and 9 for those in risk group C. This analysis confirms that the mortality from hypertension is determined more by the overall risk burden than the level of blood pressure (table 8).

Therapy

When treating hypertensives, the reduction of global risk burden is accomplished by adoption of healthy lifestyles and by modifying cardiovascular risk factors. The impact of life style modifications on blood pressure reduction is quite significant. For instance, a 22 pound weight loss results in 5 to 20 mmHg reduction in systolic pressure, a low fat diet rich in fruits and vegetables reduces blood pressure by 8 to 14 mmHg and regular aerobic physical activity reduces blood pressure by 4 to 9 mmHg. The results are even much larger when changes are made in more than one factor.

The primary focus of drug therapy is reduction of SBP as most will reach the DBP goal once the SBP is reduced. Treating SBP and DBP to targets that are less than or equal to 140/90 mmHg is associated with a decrease in cardiovascular complications. In clinical trials, antihypertensive therapy has been associated with reductions in stroke incidence by 35 to 40 percent; myocardial infarction by 20 to 25 percent and heart failure by greater than 50 percent. In patients with hypertension and diabetes or renal disease, the goal is reduction of blood pressure to less than or equal to 130/80 mmHg (JNC 7).

Many classes of drugs are available to reduce blood pressure. Of these, diuretics, beta blockers, ace inhibitors, calcium channel blockers and angiotensin II receptor blockers are the ones utilized most frequently. Most cases of hypertension would require a combination of two or more antihypertensive agents selected from different drug classes for effective control of blood pressure. ♦

Hypertension Related Mortality: A Cost Effective Approach to Risk Selection

Hypertension is a frequently encountered impairment in underwriting as it affects one in three adult Americans. Its role in increasing cardiovascular and renal morbidity and mortality in diabetes is well known. Even in non-diabetics, it is a major risk factor for death from coronary artery disease, stroke, heart failure and end-stage renal disease.

The following statistics from the American Heart Association (Circulation. 2007; 115: e111-e115) illustrate the magnitude of the mortality risk caused by hypertension. The overall death rate from hypertension in 2004 was 17.9/100,000 population. In 2002 hypertension was indicated as one of the causes of death in 277,000 cases. The age adjusted death rate for hypertension increased from 1994 to 2004 by 25.2 percent and the actual number of deaths rose 54.6 percent. About 69 percent of people who have a first heart attack, 77 percent who have a first stroke and 74 percent who have congestive heart failure, have blood pressure (BP) higher than 140/90 mmHg. Total life expectancy was 5.1 years longer for normotensive men and 4.9 years longer for normotensive women compared with hypertensives of the same sex at age 50.

Blood Pressure and Mortality Risk

BP level alone is an unreliable indicator of risk in an individual patient even in the absence of cardiovascular disorders, renal disorders or diabetes. In fact, the cardiovascular risk profile and the presence and degree of target organ damage are better predictors of mortality and should be used to select the treatment and determining BP goals and prognosis (Hypertension: Overview in this issue).

The Framingham Heart Study and other important, prospectively designed studies have clearly demonstrated that left ventricular hypertrophy (LVH) is a major cardiovascular risk factor that is independent of degree of elevation of both systolic and diastolic pressures. Many other studies have reported that hypertensive therapy results in reduced left ventricular mass and wall thicknesses, even though it is uncertain if there is a proportionate reduction in risk. The major underlying mechanism associated with risk from LVH appears to be progressive impairment of coronary flow reserve, due to deposition of collagen in the ventricular wall and around the coronary vessels. This is so, even in the absence of occlusive atherosclerotic disease. The results of these hemodynamic and structural alterations affecting the left ventricle are usually recognized as electrocardiographic and echocardiographic LVH, which may be manifested clinically by angina pectoris, cardiac dysrhythmias, systolic or diastolic dysfunction and cardiac failure, as well as by silent ischemia, myocardial infarction and sudden cardiac death (Hypertension. 1999; 34:782-789).

Carotid intima-media thickness assessed by ultrasonography, has been regarded as a valid indicator of generalized atherosclerosis. Consistently elevated SBP but not elevated DBP has been associated with increased prevalence of early atherosclerotic manifestations, including carotid intima-media

Table 7. Distribution of NHANES I Epidemiologic Follow-up Study Participants According to BP Level and Risk Categorization

Risk Group	Prehypertension 130-139/85-89	Stage 1 140-159/90-99	Stage 2 ≥160/≥100
	Number of (%) patients		
A (low risk)	276 (3.9)	257 (3.6)	107 (1.5)
B (intermediate risk)	1371 (19.3)	2208 (31.1)	1505 (21.2)
C (high risk)	300 (4.2)	603 (8.5)	463 (6.5)

Source: Hypertension 2000; 35:539-543

Table 8. Estimated Impact of 12 mmHg Reduction in SBP on the Number Needed To Treat to Prevent One All-Cause Death over 10 years

Risk Group	130-139/85-89	140-159/90-99	≥160/≥100
A (low risk)	81	60	23
B (intermediate risk)	19	16	9
C (high risk)	14	12	9

Source: Hypertension 2000; 35:539-543

thickening, in cross-sectional studies. Further, treatment of elevated SBP slowed progression of carotid stenosis and reduced cardiovascular risk in older persons with isolated systolic hypertension (Hypertension. 1999; 34:51-5).

Renal Function and Cardiovascular Mortality

The association between mild renal dysfunction and cardiovascular risk has been confirmed in multiple studies. Mild renal dysfunction, defined as a glomerular filtration rate less than 60 mL per minute or the presence of albuminuria of greater than equal to 30 mg per day, is relatively common in patients with long-standing primary hypertension, varying from 10 to 40 percent in various studies. Although mild renal dysfunction does not necessarily imply progression toward end-stage renal disease, it contributes heavily to the burden of cardiovascular risk. In fact, it has recently been reported that its presence results in a three-time higher incidence of fatal events, regardless of the presence of other common risk factors (J Am Soc Nephrol. 2001; 12:218-225).

It appears that mild renal dysfunction is also associated with the presence LVH and atherosclerosis. This link was explored in a study of 358 previously treated hypertensive patients by a group of investigators from University of Genoa, Genoa, Italy (Hypertension. 2003; 42:14-18). They found that the prevalence of mild renal dysfunction, LVH, and carotid plaque in this group was 18 percent, 48 percent, and 28 percent, respectively. Mild renal dysfunction was related to the presence of several risk factors, such as older age, higher blood pressure levels, abnormal lipids and smoking habits. Patients with the highest left ventricular mass and carotid IMT (upper quartiles) showed a higher prevalence of mild renal dysfunction. After adjusting for duration of hypertension, mean blood pressure, smoking habits and age, they found that the risk of LVH and/or carotid atherosclerosis increased by 43 percent with each standard deviation reduction in creatinine clearance, and by 89 percent with each standard deviation increase in albuminuria. Similar findings were documented in many other studies. Thus, decreased creatinine clearance and increased urinary albumin excretion predict the presence of LVH and carotid atherosclerosis with a high degree of certainty and could be used to screen for them.

Uric Acid and Cardiovascular Disease

Hyperuricemia is frequently encountered in hypertensive patients. Several large epidemiological studies have identified the association of cardiovascular risk and increased serum uric acid in the general population and among patients with hypertension. It remains to be determined whether the relationship between uric acid and cardiovascular risks is circumstantial or causal. A study of 619 subjects at the National Cardiovascular Center in Osaka, Japan determined that serum uric acid level was independently associated with left ventricular mass index (LVMI) in males and females. Further, the combination hyperuricemia and LVMI was an independent predictor of CV events (Hypertension. 2006; 47:195-202).

Brain Natriuretic Peptide and LV Dysfunction

Brain natriuretic peptide (BNP) is a cardiac biomarker that has been extensively used in screening for the presence of symptomatic and asymptomatic systolic and diastolic left ventricular function in the general population. It is well

documented that any degree of left ventricular dysfunction leads to substantial increased mortality (Utilizing NT-ProBNP in the Selection of Risks for Life Insurance at www.rkillango.com).

Screening Insurance Applicants

The high incidence of hypertension and the significant mortality risk it poses requires an efficient and cost effective risk selection strategy. Currently, attending physician records are the main source of information for risk selection in hypertension; electrocardiograms are obtained at larger amounts and older ages. As this approach is expensive and time consuming, alternate strategies need to be explored. The fact that the global risk burden is much more predictive of mortality than the level of BP requires that the focus be more on the risk burden than the actual blood pressure values. Besides, the unreliability of office blood pressure measurements (Ambulatory Blood Pressure Monitoring, Underwriter Resource. September 2007, volume 1 issue 1) and the fact that a third of those with hypertension have never been diagnosed makes excessive emphasis on recorded office blood pressure risky.

The optimal method for screening insurance applicants for global risk burden based on current medical knowledge is as follows:

- Screen for the traditional cardiovascular risk factors.
- Obtain an echocardiogram or electrocardiogram to evaluate for LVH.
- Obtain a B mode ultrasonogram of the carotid to determine IMT.
- Screen for kidney dysfunction utilizing the calculated glomerular filtration rate (GFR) and urine for microalbuminuria.

Screening for traditional risk factors is a routine practice in the insurance industry. But, echocardiography, electrocardiography and B mode ultrasound of the carotid on all hypertensives insurance applicants is not viable option because of cost and inconvenience to applicants. Based on clinical studies, this may not be necessary as calculated GFR, microalbuminuria and serum uric acid would detect most cases of LVH and early atherosclerosis and is inexpensive.

Therefore, it is logical and practical to use a combination of traditional cardiovascular risk factors, calculated GFR, urine for microalbumin and serum uric acid as screening tests for cardiovascular risk on all applicants. This would ensure that the third of those with undiagnosed hypertension with increased global risk burden are not missed. Attending physician statements, BNP and other cardiovascular tests only need to be requested for applicants with abnormal screening tests. This would cut costs and decrease turnaround time since these more costly evaluation tools would be ordered only when further evaluation is warranted for those at higher risk. ♦

©2008 - RK Illango Consulting, Inc. has made all reasonable efforts to ensure that information provided through its publication is accurate at the time of inclusion and accepts no liability for any inaccuracies or omissions. All rights reserved. No part of this publication may be reproduced in any form without the prior permission of the publisher.