

**I A F F**

# **Recognition and Prevention of Occupational Heart Diseases**



**International Association  
of Fire Fighters**





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# A Guide to the Recognition and Prevention of Occupational Heart Disease for the Fire and Emergency Medical Services



**Department of Occupational Health and Safety  
International Association of Fire Fighters, AFL-CIO, CLC**

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# Foreword

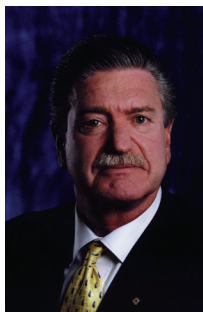
Heart disease continues to be the leading cause of job related deaths among IAFF members in both Canada and the United States. In 1996, over 56% of line of duty deaths were attributable to heart disease and a related disorder, stroke. This IAFF manual, *A Guide to the Recognition and Prevention of Occupational Heart Disease for the Fire and Emergency Medical Services*, is designed as a guide to assist the fire fighter, EMT, and paramedic in understanding heart disease and its prevention. In addition, the IAFF continues to pursue expansion of presumptive heart disease legislation and further protection from the myriad of hazards to which our members are exposed.

This manual is intended to broaden the fire service's approach to implementing a successful heart disease prevention program. It includes chapters on the anatomy and normal function of the heart, risk factors for heart disease, and the prevention of heart disease. In addition, specific chapters address the unique risk of cardiovascular disease among fire fighters and existing heart disease legislation as it relates to our membership. The manual was developed by the IAFF Department of Occupational Health and Safety with the assistance of our Medical Residency Program. The IAFF Medical Residency Program, now entering its twelfth year, has provided numerous services to the IAFF and our affiliates. The manual highlights, once again, the importance of this program.

We encourage all locals to support heart disease prevention programs for all members. Such a program should be the objective of every fire and emergency department in cooperation with its local IAFF affiliate.

We all realize the inherent hazards of fire fighting and emergency medical response. It is important for us to be recognized as a unique population with special needs. We need for our fire departments and health care providers to approach our daily health and fitness needs with comprehensive knowledge about our occupation and its attendant risks. In meeting this goal, this manual synthesizes the resources of occupational medicine, the NFPA and government standards, and relevant documents to create a comprehensive guidebook for our membership.

We have dedicated this manual to Brother James E. Thornton who served as President of the Virginia Professional Fire Fighters and Norfolk Fire Fighters Local 68. Jim was an outspoken advocate for fire fighters and pressed for the development of this manual during the 41st IAFF Convention in 1992. He died of cancer on November 3, 1992. He was 48 years old.



**Harold A. Schaitberger**  
General President



**Thomas H. Miller**  
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# Acknowledgments

The IAFF Department of Occupational Health and Safety would like to lend its appreciation and gratitude to the physicians who have rotated through the IAFF Medical Residency Program. Initiated through convention action in 1986, the IAFF entered into a cooperative agreement with the Johns Hopkins University School of Hygiene and Public Health to host physicians for 60-day rotations in their second year of post-graduate study specializing in occupational medicine. This publication represents another noted achievement of this program.

We would like to especially thank those IAFF Medical Residents who helped in developing and updating this manual -- Jane Barlow, MD, MPH, MBA; Jim Martin, MD, MPH; Mike McGrail, MD, MPH; Ron Joines, MD, MPH, Ann Kuhnen, MD, MPH, and John D. Piacentino, MD, MPH. Also, Andrew Levinson, MPH, IAFF Occupational Safety and Health Assistant and Lisa Aaron for their efforts in completing this manual.

We also wish to recognize Jim Melius, MD, DrPH, Chairman of the IAFF Medical Advisory Board for his input during the development of this work effort and for his technical review of the information provided herein.

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# Chapter 1

## INTRODUCTION

From before birth until death, the heart performs as a pump, forcing blood through the circulatory system without interruption. Without this function, serious brain damage can occur within 4 to 5 minutes and certain death in 10 minutes. As shown in Figures 1.1 and 1.2, heart disease is the major killer of both men and women in the United States and in Canada. Clearly, the importance of a healthy heart can not be overstated.

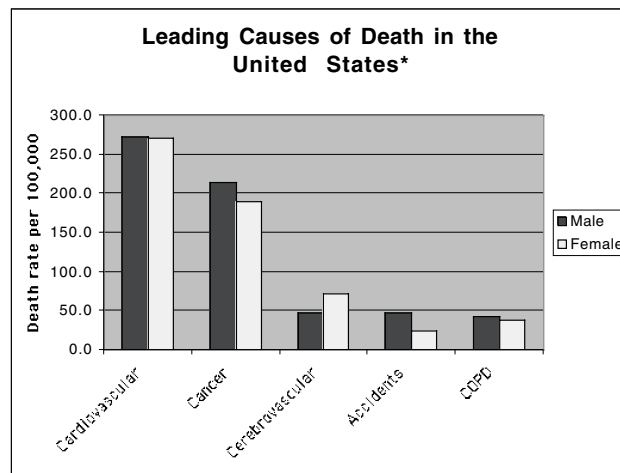


Figure 1.1

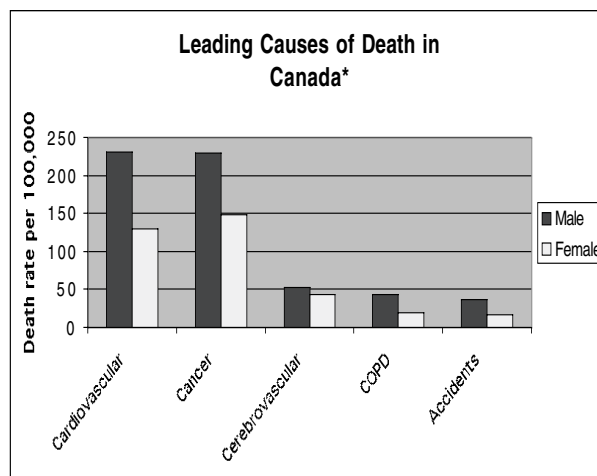


Figure 1.2

Heart disease can be prevented and treated. Increased social awareness of the adverse health effects of a high fat diet, smoking, obesity, and sedentary lifestyle has changed the way many people care for themselves. The result of "healthier" lifestyles, as well as improving treatments for heart disease, has been a decrease in the death rate from heart attacks over the past two decades. Representative mortality data from the United States and Canada is presented in Figures 1.3 and 1.4.

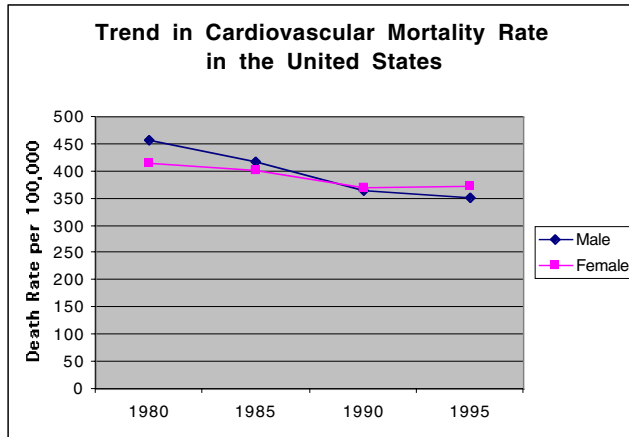


Figure 1.3

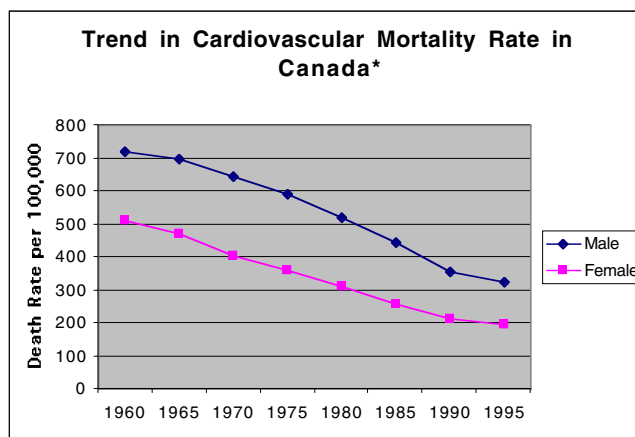


Figure 1.3

This manual is written for fire fighters and emergency medical responders, a group of individuals who face special risks of cardiac disease. The manual contains a review and an analysis of several topics, including the evidence that fire fighters and emergency medical responders are at increased risk for developing heart disease, the normal anatomy and function of the heart, the types of diseases that afflict the heart, the factors that increase fire fighters' risk of developing heart disease, and what fire fighters and emergency medical responders can do to prevent heart disease. Information is also included that addresses the Public Safety Officers Benefits Program in the United States and a review of provincial and state heart legislation.

Throughout this manual, information and statistics are presented from both Canada and the United States. While specific numbers differ between the two countries, the trends are nearly identical and could be readily interchanged. The manual also has several diagrams that will help the reader understand the text of each chapter.

The International Association of Fire Fighters knows you will find this manual informative and useful.

## END NOTES

- Figure 1.1 National Vital Statistics Reports, Vol 47, No. 19, June 30, 1999  
 Figure 1.2 Statistics Canada, Health Statistics Division.  
<http://www.statcan.ca/english/Pgdb/People/Health/health36.html>  
 Figure 1.3 National Center for Health Statistics, Age-adjusted death rates for 72 selected causes, United States, 1979-98. Unpublished data, GMWK293.  
 Figure 1.4 Health Canada. Cardiovascular Disease Surveillance On-Line.  
[http://cythera.ic.gc.ca/spansweb/cvd/index\\_e.html](http://cythera.ic.gc.ca/spansweb/cvd/index_e.html)

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# Chapter 2

## REVIEW OF FIRE FIGHTER MORTALITY STUDIES

Fire fighters have voiced concern that they have unusually high rates of heart disease. One of the few ways to assess the validity of this concern is to analyze heart disease rates in fire fighter mortality studies.

Mortality studies look at the number of deaths in a group, such as fire fighters, to determine if a particular disease is causing more deaths than expected. Many mortality studies of fire fighters have been performed, looking for an association between fire fighting and various diseases. The results have been mixed. This chapter will review several key studies and their strengths and limitations.

## PROBLEMS WITH MORTALITY STUDIES

Most mortality studies gather information using death certificates of former fire fighters. The number of deaths in fire fighters due to various causes is compared to another group, the reference population, to look for differences. A typical comparison group is the overall United States white male population. However, this is not the most appropriate comparison group. There are important differences between fire fighters and the general population of the United States, other than the experience of fire fighting. In general, qualified fire service personnel are healthier than the U.S. population. Ideally, the study and comparison groups would have similar race, gender, age, health index and life-style profiles and the profession of fire fighting would be the only significant difference. In reality, some differences will exist, leaving the results of such studies open to interpretation and debate.

Mortality studies also rely on accurate coding of death certificates. However, the diagnostic categories used are general and often overlap, adding uncertainty to the studies. In comparison with cancer, cardiovascular disease is not very accurately diagnosed and recorded on death certificates. For example, one doctor may classify a death as a “heart attack” while a different doctor may classify the same death as “sudden death due to arrhythmia.” Both doctors may have made accurate (and related) diagnoses, but the current death certificate system may not reconcile the differences and similarities of these diagnoses. As a result, one diagnosis may be counted as a death due to atherosclerotic coronary vascular disease, while the other might not be.

Another source of uncertainty arises if a fire fighter leaves the job because of cardiovascular disease, or if a fire fighter develops heart disease but dies from another cause, such as cancer or an accident. Many fire fighters can no longer work as fire fighters after they develop cardiovascular disease. These cases would not be counted in mortality studies, therefore, mortality studies may tend to underestimate the risk of cardiovascular disease. Remember, these studies actually measure the risk of *death* from cardiovascular disease, rather than the risk of *developing* cardiovascular disease.

Studies of fire fighters also suffer a limitation known as the “healthy worker effect.” In part, fire fighters are selected for employment on the basis of their physical fitness. They are in fact healthier than the general population because less healthy individuals are less likely to be hired as fire fighters. Because of this selection bias, fire fighters would be expected to have a decreased incidence of cardiovascular disease mortality when compared to the general population. Therefore, it would take a very large increase in fire fighter cardiovascular mortality to result in a significant measurable increase in a mortality study.

## REVIEW OF MORTALITY STUDIES

Between individual fire fighters, the types and amounts of toxic exposure vary from fire to fire and job to job. In addition, life time exposure can vary considerably with the use, or lack of use, of personal protective equipment. Because accurate data about hazardous exposures is unknown, researchers commonly lump all fire fighters together for study and duration of employment is generally used as a substitute for dose. These study techniques may dilute a real effect from a particular exposure that affects only a portion of fire fighters.

Despite the problems associated with mortality studies and the tendency for any real effects to be obscured, several studies have reported an increased risk of cardiovascular disease in fire fighters. Additionally, in a recent reanalysis of 23 mortality studies, Choi<sup>1</sup> adjusted for the healthy worker effect and showed that four studies which previously showed no increased association between fire fighting and cardiovascular mortality underestimated the occupational risk of fire fighting and cardiovascular mortality. He concluded that overall, there is strong evidence of an increased risk between work as a fire fighter and death from cardiovascular disease.

In 1959, Mastromatteo<sup>2</sup> reported a significant increased risk of death in the city of Toronto fire fighters from all causes and specifically from cardiovascular disease. He compared Toronto fire fighters with males in the city of Toronto and the province of Ontario over the time frame 1921 to 1953.

Coronary artery disease deaths were also elevated in the study by Bates,<sup>2</sup> who assessed city of Toronto fire fighters hired between Jan. 1, 1949, and Dec. 31 1959. These fire fighters were followed through 1984 and compared to city of Toronto males. There were 21 deaths from coronary artery disease, which was 1.73 times (73% increase) more heart disease deaths than in the comparison group. The highest death rate was for those 45 to 49 years of age in the 1970s. Heart disease death rates decreased as the fire fighters grew older, which would not be expected. (Heart disease death rates should increase with age, not decrease.) Bates explains the falling rates in the older fire fighters by several factors. First, the death rate from heart disease in the population had generally been decreasing, and so one would expect a similar trend in fire fighters. Fire fighters that had already died of a heart attack in an earlier age group left a lower risk for the remaining population. Finally, in the 1970s, policy changes encouraged fire fighters with evidence of coronary artery disease to retire or transfer to non-fire fighting duties, also leaving a lower risk population.

Sardinas et al also found some evidence of increased risk of coronary artery disease in his study of Connecticut fire fighters and police officers, studied for the years 1960 to 1978<sup>3</sup>. When fire fighters were compared with other workers, 1.52 times more heart disease deaths were identified. However, when compared to police officers, the mortality of fire fighters was somewhat lower.

Feuer conducted a mortality study of police and fire fighters in New Jersey<sup>4</sup>. The 901 deaths reported to the Police and Firemen Retirement System between 1974 and 1980 were compared to the U.S. general population, the New Jersey general population and the New Jersey police. A statistically significant increased mortality rate (1.2 times more heart disease deaths) was reported for atherosclerotic heart disease in working fire fighters as compared to New Jersey males. When compared with police officers, fire fighters had a slightly elevated risk of dying from a heart attack. When divided into groups by duration of employment, less than or equal to 22 years, 22 to 27 years, and greater than 27 years, elevated heart attack rates of 1.43, 1.11, and 0.91 were found respectively. This inverse relationship suggests that individuals who are more susceptible to heart disease are affected early in their career. Since only 3 of the 115 fire fighters died during working hours, a chronic rather than an acute process was suggested.

In a recent study L'Abbe and Tomlinson looked at 5,414 male fire fighters from six fire departments within metropolitan Toronto<sup>5</sup>. Each individual's employment record was linked

to Canada's National Mortality Data Base, and the number and cause of death was determined for the period January 1, 1950 through December 31, 1989. Mortality analysis of this group was specific for age and calendar period. The causes of death were compared to those of the Ontario male population for the same time period. L'Abbe reported a very slight increase in risk of dying from an acute myocardial infarction. Although not statistically significant, the levels reported are higher than expected in a population screened before entering employment and whose work required a higher level of physical fitness than that of the general population (i.e. the healthy worker effect). Also of interest is the finding that the majority of deaths attributed to arteriosclerosis were due to aortic aneurysm.

Another large Canadian study of Edmonton and Calgary fire fighters was reported by Guidotti<sup>6</sup>. In comparison to Alberta males of the same time period (1927-1987), a non-significant, slight elevation of coronary artery disease deaths (1.1 times more deaths than expected) was noted.

In contrast to these studies, several other major studies have found little or no excess in risk for cardiac disease. Eliopoulos<sup>7</sup> studied 990 Western Australian Fire Brigade employees between 1939 and 1978 using death certificate data. Western Australian males of the appropriate age and time group were used as the comparison group. No evidence of increased mortality from circulatory disease was found. However, analysis did reveal evidence of the healthy worker effect.

Mortality experience among 5655 Boston fire fighters with three or more years of service between 1915 and 1975 was reported by Musk<sup>8</sup>. Deaths from all causes were 91% of expected for Massachusetts males and 94% of expected compared to U.S. white males. In separate studies, Demers<sup>9</sup> and Beaumont<sup>10</sup> also reported a less than expected mortality due to disease of the heart and other circulatory disease compared to U.S. rates for white males. Interpretation of these studies is limited by several of the factors mentioned earlier including: the healthy worker effect, changes in fire fighter exposure since the studies were done, and inaccuracies in death certificate coding.

## CONCLUSION

Two studies have shown an increasing risk of circulatory disease with increasing duration of employment. Heyer<sup>11</sup> reported a trend of increasing death rates from circulatory disease with increasing duration of exposure. Vena and Fiedler<sup>12</sup> reported similar findings. Heyer demonstrated persistence of this trend at least 30 years or more after selection to the fire fighter work force. In fact, fire fighters with 30 years or more of fire fighting duty had a relative risk of 1.84 compared to those with less than 15 years of service.

In summary, mortality studies are an imprecise method of studying the relationship between fire fighting and cardiovascular disease. Selection of fire fighters based on their physical fitness would be expected to decrease their cardiovascular disease risk in comparison to the general population. Although they may be more fit than the general population, fire fighters are exposed to carbon monoxide, cyanide and other toxic gases as well as strenuous physical demands during the course of employment that increase cardiovascular disease risks. Despite these limitations, some studies have shown an increased risk of cardiovascular disease among fire fighters. Moreover, when adjusting for the healthy worker effect, several studies which previously detected no risk, also show an increased risk between fire fighting and cardiovascular mortality. The plausibility of this finding is supported by the exposure of fire fighters to substances and physical stresses which increase their risk of cardiovascular disease.

## END NOTES

- <sup>1</sup> Choi B. A Technique to Re-Assess Epidemiologic Evidence in Light of the Healthy Worker Effect: The Case of Firefighting and Heart Disease. *J Occup Med.* 2000; 42: 1021-1034.
- <sup>2</sup> Mastromatteo E. Mortality in City Firemen, II: A Study of Mortality in Firemen of a City Fire Department. *AMA Arch Ind Health.* 1959; 20: 227-33.
- <sup>3</sup> Bates JT. Coronary Artery Disease Deaths in the Toronto Fire Department. *Journal of Occupational Medicine.* 1987; 29(2): 132-135.
- <sup>4</sup> Sardinias A, Miller JW, Hansen H. Ischemic Heart Disease Mortality of Firemen and Policemen. *American Journal of Public Health.* 1986; 76(9): 1140-1141.
- <sup>5</sup> Feuer E, Rosenman K. Mortality in Police and Firefighters in New Jersey. *American Journal of Industrial Medicine.* 1986; 9: 517-527.
- <sup>6</sup> L'Abbe KA, Tomlinson GA. Scientific Report: Mortality Study of Fire Fighters in Metropolitan Toronto. Industrial Disease Standards Panel. Toronto, Ontario. March 1992.
- <sup>7</sup> Guidotti TL. Mortality of Urban Firefighters in Alberta, 1927-1987. *American Journal of Industrial Medicine.* 1993; 23: 921-940.
- <sup>8</sup> Eliopoulos E, Armstrong BK, Spickett JT, Heyworth F. Mortality of Fire Fighters in Western Australia. *British Journal of Industrial Medicine.* 1984; 41: 183-187.
- <sup>9</sup> Musk AW, Monson RR, Peters JM, Peters RK. Mortality among Boston Firefighters, 1915-1975. *British Journal of Industrial Medicine.* 1978; 35: 104-108.
- <sup>10</sup> Demers PA, Heyer NJ, Rosenstock L. Mortality among Firefighters from Three Northwestern United States Cities. *British Journal of Industrial Medicine.* 1992; 49: 664-670.
- <sup>11</sup> Beaumont JJ, Chu GST, Jones JR, Schenker MB, Singleton JA, Piantanida LG, Reiterman M. An Epidemiologic Study of Cancer and other Causes of Mortality in San Francisco Firefighters. *American Journal of Industrial Medicine.* 1991; 19: 357-372.
- <sup>12</sup> Heyer N, Weiss NS, Demers P, Rosenstock L. Cohort Mortality Study of Seattle Fire Fighters: 1945-1988.
- <sup>13</sup> Vena JE, Fiedler RC. Mortality of a Municipal-worker Cohort: IV. Firefighters. *American Journal of Industrial Medicine.* 1987; 11: 671-684.

# Chapter 3

## THE NORMAL HEART

The heart efficiently pumps blood through a simple yet ingenious design. To comprehend what can go wrong with the heart, a basic understanding of normal heart anatomy and function is required.

## OVERVIEW

The heart has four chambers: two ventricles and two atria. Each chamber moves blood by contraction of its muscular walls, making the chamber smaller and forcing blood out of the heart into the blood vessels.

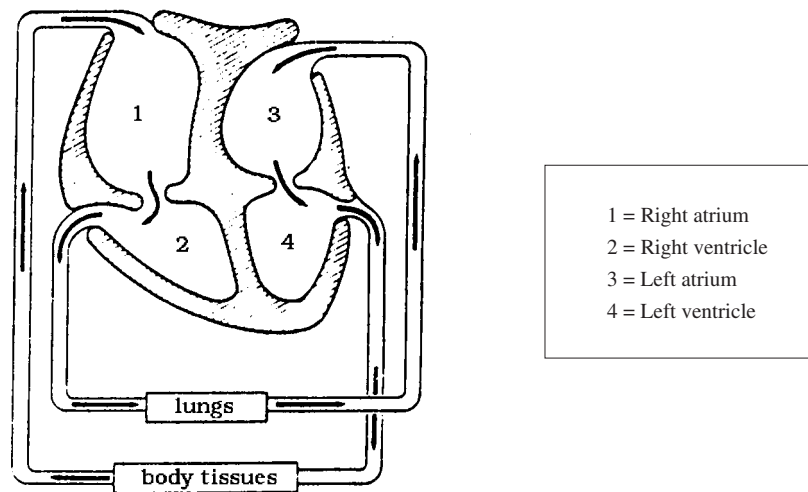


Figure 3.1 Diagram of the heart's chambers.

The two ventricles are located on the bottom of the heart and are the main pumping chambers. The atria are the upper two chambers and help ensure complete filling of the ventricles. The ventricles will actually fill to 70 percent of capacity without the help of the atria, which fill the remaining 30 percent of the ventricles as they contract. The additional 30 percent boost is not needed at rest. However, with exercise, this extra volume of blood is very important. Without it, the heart “fails” to maintain adequate blood flow, resulting in symptoms such as shortness of breath.

Functionally, the heart can be divided into right and left sides. As seen in Figure 3.1, the right side of the heart pumps blood through the pulmonary arteries to the lungs. The left side of the heart pumps blood out the aorta to the rest of the body. The blood pressure is significantly higher in the left heart circulation (e.g. 120/80 mmHg) as compared to the right heart circulation (e.g. 22/0 mmHg). Because of this pressure difference, the left ventricle has significantly thicker muscular walls than the right ventricle. Given the left ventricle’s important task of pumping blood to all the organs of the body, it is apparent why a healthy left ventricle is critical to a person’s well-being. The higher pressures and increased muscle thickness of the left ventricle also predispose it to most of the diseases that affect the heart.

## HEART VALVES

The heart valves function as one-way gates for blood and are of vital importance in maintaining normal heart output. Their function ensures that blood flows in only one direction in the heart. The location of the four heart valves is seen in Figure 3.2.

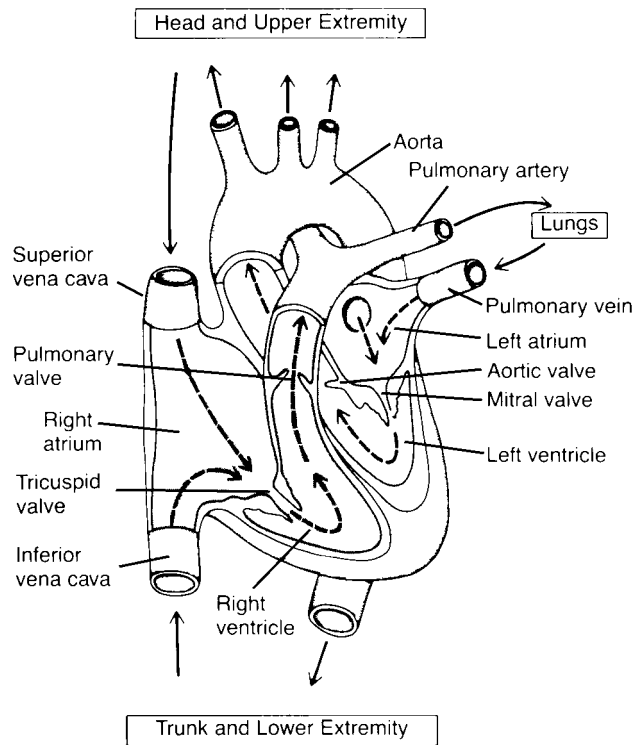


Figure 3.2 The circulatory system

The mitral valve is located between the left atrium and left ventricle, allowing blood to flow from atria to ventricle only. The aortic valve prevents blood from flowing back from the high pressure aorta into the left ventricle, when it relaxes to refill. Like the mitral valve, the tricuspid valve is the one-way valve that separates the right atrium and right ventricle. And, the right side of the heart has the pulmonic valve, like the aortic valve, to prevent blood from flowing backward into the right ventricle from the pulmonary arteries. The snapping shut of these heart valves generate the heart sounds that are heard with a stethoscope. Close-up pictures of the structure of the aortic and mitral valves are presented in Figure 3.3.

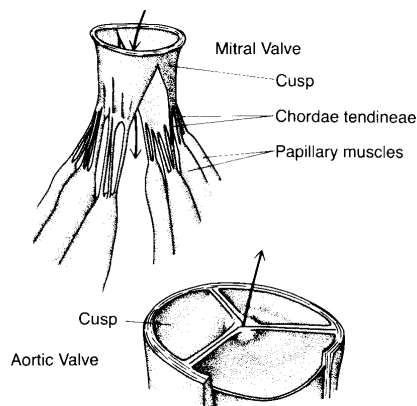


Figure 3.3 The structure of the mitral and aortic valves.

## THE CIRCULATORY SYSTEM

One of the principle functions of the heart is to drive the circulation to deliver oxygen to the body. The basic oxygen carrying unit is an iron-containing molecule called hemoglobin, which is packaged inside red blood cells (RBCs). By the billions, RBCs travel through the circulatory system, to deliver oxygen to all the cells of the body. We can follow a single RBC as it makes one complete journey through the body's circulatory system. (You can follow the course of the RBC described below in Figure 3.2.) Starting in the right atrium, the RBC passes through the tricuspid valve into the right ventricle. When the ventricle contracts, the RBC is pumped out of the heart through the pulmonic valve into the pulmonary artery. It then travels through progressively smaller artery branches until it is in the smallest blood vessels within the lungs, called capillaries. The capillaries are so small that RBCs must pass through them single file. While in the lung capillaries, the RBC picks up its supply of oxygen. It then returns to the heart through the pulmonary veins and enters the left atrium. The RBC leaves the left atrium through the mitral valve into the left ventricle. The left ventricle pumps the RBC through the aortic valve, into the high pressure circulation of the aorta. The RBC travels through successive branching arteries, to arrive in the small capillaries located in most body tissues. Here the RBC delivers its oxygen supply to the tissues, after which it begins its return journey through progressively larger veins. The RBC completes its "round trip" journey by returning to the right atrium via the major veins called the inferior and superior vena cava.

## BLOOD VESSELS

The body has two general blood vessel types, arteries and veins. Arteries carry blood away from the heart and veins bring blood back to the heart. Arteries have relatively thick walls which include a muscular layer to help withstand and control the high pressures generated by the heart pump. On the other hand, veins have relatively thin walls which actually stretch and distend quite easily (as in the extreme case of varicose veins). The venous circulation is under low pressure and has no dedicated pump, like the heart, to move blood. In fact, venous blood flow depends on the low pressures generated by gravity and by the pumping action of the skeletal muscles of the arms and legs pressing on vein walls. The pressure difference between the arterial and venous circulation is apparent to those who have witnessed the spurting blood from a severed artery, as compared to the steady oozing blood flow from a cut vein.

Because veins and the atria work at such low pressures, the heart does not need valves as blood enters the heart (See Figure 3.3). Some backward flow into the veins does occur when the atria contract (which a trained eye can see in the neck veins of most people) but the amount is small enough to be functionally insignificant.

## MYOCARDIUM

The majority of the heart, as shown in Figure 3.4, consists of a special type of muscle. When examined under the microscope, the heart muscle cells appear to connect end-to-end, forming long fibers which are interwoven. Individual cells are connected by intercalated discs.

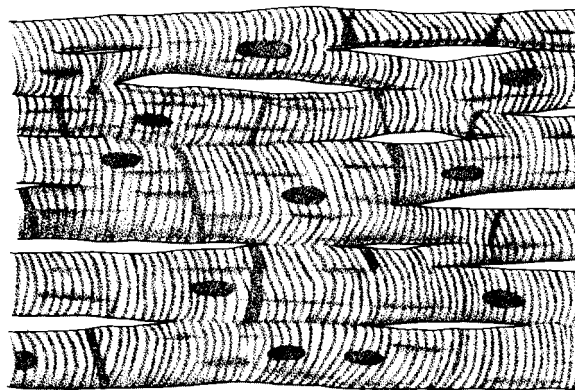


Figure 3.4 The microscopic appearance of heart muscle.

## CORONARY ARTERIES

These fibers form an intricate lattice work. The muscle cells communicate with each other by passing electric current, which can be measured on an electrocardiogram (ECG or EKG). The electric current releases calcium inside the muscle cell, causing the cell to contract and shortening the cell length.

Many connected cells in the same muscle fiber contract simultaneously to shorten the fiber length. Many fibers shortening simultaneously results in the contraction of the heart (heart chambers become smaller) which forces or “pumps” blood out. Fibers lengthen when the myocardium relaxes, allowing the heart to refill with blood.

Like any muscle, the heart requires oxygen to produce the energy it uses while pumping blood. Because the heart never stops to rest, the heart muscle has a well developed arterial system to deliver oxygenated (oxygen rich) blood to the muscle.

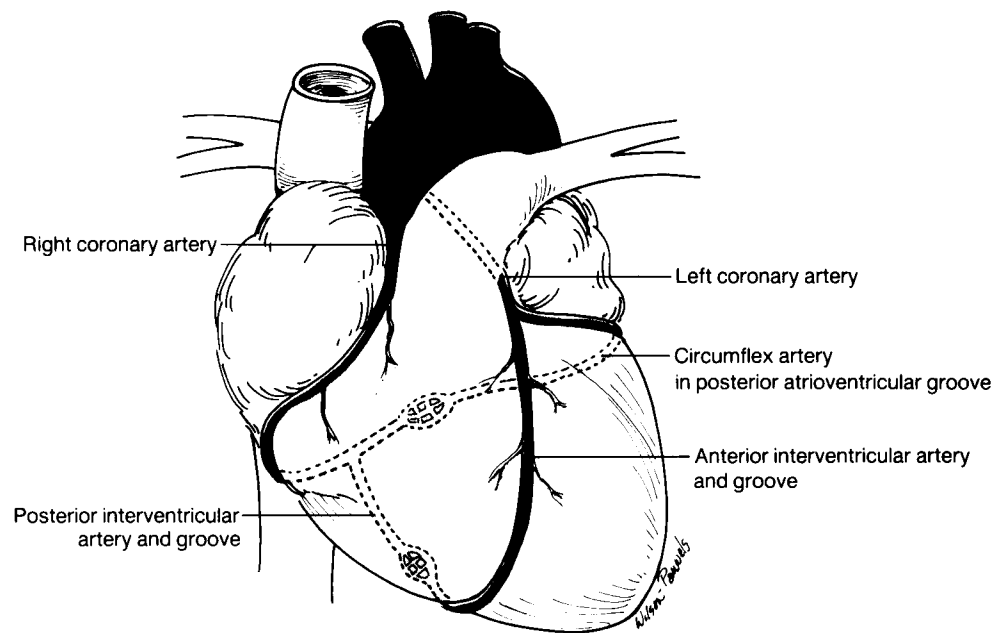


Figure 3.5 The major coronary arteries.

The major arteries of this system include the right coronary artery and the left coronary artery, which has two major branches, the left anterior descending (called the anterior interventricular branch in Figure 3.5) and circumflex arteries. Most often it is blockage in one or more of these arteries that leads to ischemic (lack of oxygen) heart disease and its major clinical manifestation, a heart attack.

## CONDUCTION SYSTEM

As mentioned earlier, electric current is what triggers heart muscle cells to contract. For the heart to function properly, the electric current must flow through the heart muscle cells in a coordinated fashion, so the muscle cells contract together and at an appropriate rate. The heart has specialized cells that form a “wiring” system, called the cardiac conduction system, that coordinates the cooperative contractions.

As depicted in Figure 3.6, the sinoatrial node (SA node) located in the atria, starts the heart beat. It also controls how fast the heart beats. From the SA node, a wave of current crosses both atria, causing them to contract and push blood into the ventricles. To get from the atria into the ventricles, the electric current must pass through the atrioventricular (AV node), which delays the current momentarily before sending it into the ventricles. Smooth, even contraction

of the ventricles is ensured by a “wiring” system called the AV bundle and Purkinje fibers, which lead away from the AV node and instantaneously deliver current to many contiguous areas of the ventricles.

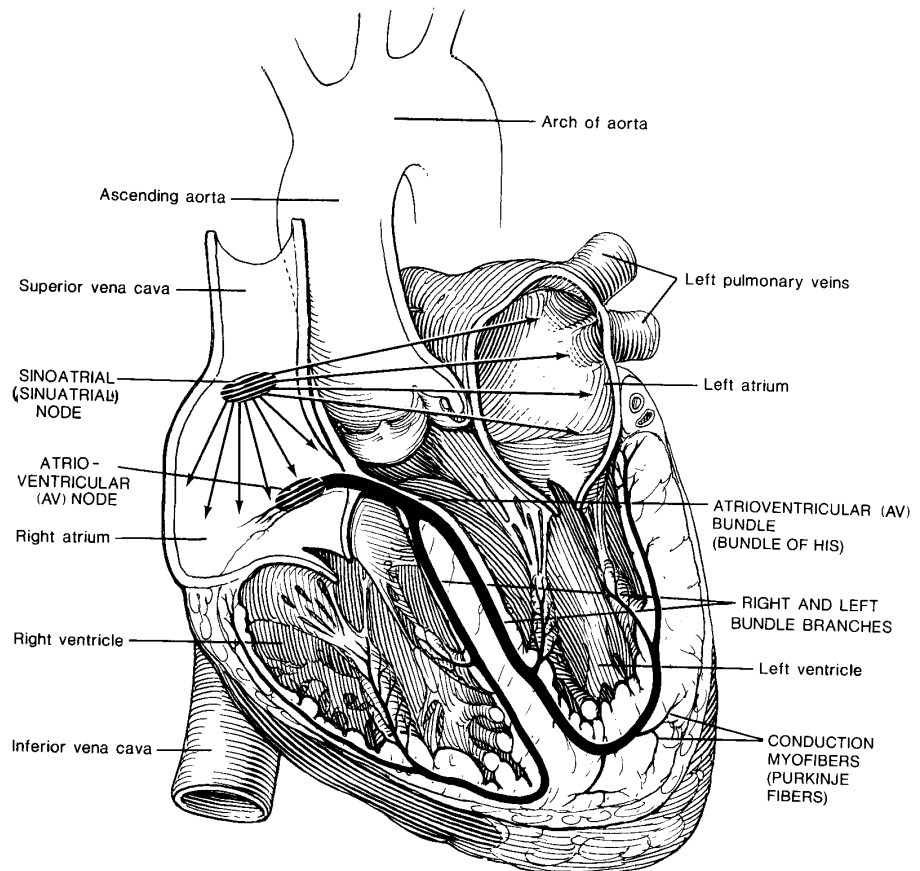


Figure 3.6 The conduction system of the heart.

## BLOOD PRESSURE

Blood pressure refers to the pressure inside the vascular system. This pressure causes blood to flow, delivering life sustaining oxygen and nutrients to all the body’s organs. If too high, blood pressure can also have profound adverse effects on a person’s health. Therefore, blood pressure is commonly checked at every health care visit. The instrument used to measure blood pressure is called a sphygmomanometer.

Blood pressure is reported as two numbers, like 120 over 80 (often written as 120/80) and is measured in millimeters of mercury (mmHg). A blood pressure of 120 mmHg means that this pressure level would elevate a column of the liquid metal mercury 120 millimeters against gravity. The upper number (in this case 120) is the *systolic* pressure and represents the highest or peak pressure in the arterial circulatory system. This peak pressure coincides with the contraction of the heart ventricles. The lower number is the *diastolic* pressure and is the lowest pressure level obtained in the arterial circulation. This pressure level occurs when the myocardium is relaxed and the heart is refilling with blood.

The level of blood pressure is determined by a number of things, including the volume of fluid in the blood, the diameter of the blood vessels (remember, blood vessels have a muscular layer in their walls that can control the vessels’ diameter), and the force of the heart contractions. The circulatory system can be thought of as a simple fluid system, much like a

garden hose filled with water, where fluid is added through the digestive tract and removed by the kidneys. If the amount of fluid in the system increases (add water into the garden hose), as occurs with excessive dietary intake of salt, the pressure goes up. If the size of the system shrinks by blood vessels contracting and getting smaller (put the same amount of water in a narrower garden hose), the pressure goes up. In addition, a heart beating more energetically (opening the spigot connected to the garden hose) will contribute to an increase in pressure.

Obviously, the mechanisms of blood pressure control are far more complex, but this simplified analogy gives a basic understanding of what controls blood pressure and how it can be manipulated. For instance, decreasing salt intake decreases the volume of fluid in the blood, so blood pressure decreases. Certain medications dilate the blood vessels and decrease the vigor of myocardial contraction, so blood pressure decreases. Certain medications cause increased urine formation, resulting in lower blood fluid levels, and decreased blood pressure. Conversely, kidney disease prevents fluid from leaving the system, so blood pressure increases.

## **A DYNAMIC SYSTEM**

The heart is a dynamic pump, it can change the speed and force with which it operates. When you exert yourself, the muscles of your body use more energy, which requires more oxygen. The body sends a notice of this increased demand to the heart through special nerves and chemical messengers like epinephrine (also called adrenaline). These factors cause the heart to speed up its rate and increase the vigor and force with which the myocardium contracts. Blood is pushed more energetically into the circulation, making the blood pressure go up (especially the systolic or “peak” pressure).

As noted earlier, the arteries can also affect blood pressure. The small muscle layer in the artery walls also can respond to these messengers. If the artery contracts down to a smaller diameter, the pressure goes up. Conversely, if the artery dilates to a larger diameter, the pressure goes down. At higher pressures, blood flow speed increases, resulting in oxygen delivery to where it is needed more quickly and in greater amounts.

It is important to realize that other things besides physical exertion can affect heart rate and blood pressure through nervous system and chemical messengers. Strong emotions (e.g. fear, anxiety) and pain can cause the release of epinephrine and other hormones that increase heart rate and blood pressure. Likewise, many medications can interact with this system to alter heart rate and blood pressure.

## **CONCLUSION**

Each of the systems reviewed, the coronary arteries, the heart muscle, the heart valves, and the conduction system can develop abnormalities resulting in clinical heart disease. These will be explained in the following chapters.

## **CREDITS**

Figure 3.1 The Heart, Figure 2, p. 2. Canadian Centre for Occupational Health and Safety, Hamilton, Ontario, Canada.

Figure 3.2 Reproduced with permission. Guyton, A.C.: Human Physiology and Mechanisms of Disease. Fifth edition. Philadelphia, W.B Saunders Co., 1992, Figure 8-1, p. 81.

Figure 3.3 Reproduced with permission. Guyton, A.C.: Human Physiology and Mechanisms of Disease. Fifth edition. Philadelphia, W.B Saunders Co., 1992, Figure 8-5, p. 84.

Figure 3.4 Reproduced with permission. Guyton, A.C.: Human Physiology and Mechanisms of Disease. Fifth edition. Philadelphia, W.B Saunders Co., 1992, Figure 8-2, p. 81.

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Figure 3.5 Reproduced with permission. Akesson, E.J., J.A. Loeb, and L. Wilson-Pauwels: Thompson's Core Textbook of Anatomy. Second Edition. Philadelphia, J.B. Lippincott Company, 1990, Figure 5-12, p. 67.

Figure 3.6 Reproduced with permission. Tortora, Gerard J.: Principles of Human Anatomy. Fifth edition. New York, Harper & Row Publishers, Inc., 1989, Figure 13-6 (a), p. 355.



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# Chapter 4

## HEART DISEASE

As was discussed in the chapter entitled “The Normal Heart”, the heart can be divided into several systems that all act in concert to ensure normal function. Each of these systems is prone to specific problems which can disrupt the overall function of the heart. Furthermore, abnormal function (dysfunction) in any one system may result in abnormal function in a different system. The complexities of the heart and heart disease have filled many medical textbooks. The purpose of this chapter is to review the more common diseases that afflict the heart. Each section of this chapter will discuss the diseases that primarily affect a specific heart system.

## DISEASES OF THE CORONARY ARTERIES

The coronary arteries provide the myocardium with the oxygen and nutrients needed to maintain normal function. A number of disease processes can interrupt the flow of blood through these arteries, resulting in different kinds of health problems. The most common problem is blockage of the coronary arteries by cholesterol plaques, a disease process called atherosclerosis.

## ATHEROSCLEROSIS

The terminology health care professionals use when talking about atherosclerosis and heart attacks can be confusing. A basic understanding of several terms helps to clear this confusion. Because of the hard nature of atherosclerosis, areas of build up which result in a narrowing of the artery are referred to as atherosclerotic *plaques*, or cholesterol *plaques*. Athero is the Greek term for “gruel” and refers to the cholesterol deposits that build up in the arteries. *Sclerosis* is the Greek term for “hardness” and refers to the hard nature of the cholesterol plaques in arteries.

Atherosclerosis is a disease process causing thickening and hardening of the medium and large sized arteries. It is the underlying cause of the vast majority of the heart attacks and strokes that occur in the United States and Canada.

When atherosclerotic plaques build up and sufficiently block the flow of blood in the coronary arteries and cause symptoms, all the different resulting diseases are collectively called atherosclerotic coronary vascular disease (ASCVD) or atherosclerotic heart disease (ASHD). Many authors simply refer to this process by the less precise terms coronary artery disease (CAD) or coronary vascular disease (CVD). All four terms can be used interchangeably, however we will predominately use ASCVD. A number of factors, such as high cholesterol and smoking, have been shown to be associated with increased risk of developing ASCVD. Because this class of heart disease is the single largest cause of death in the United States and Canada, a separate chapter in this manual addresses these “risk factors” and how to lower your risk of developing ASCVD.

## PATHOGENESIS

The artery wall has several layers, much like an onion. Atherosclerotic plaques form primarily in the innermost lining of the artery, called the endothelium.

Most of the layers are strong, fibrous tissue and muscle, meant to withstand the high pressures inside the arterial system. However, the inner most lining of the artery is only a single, delicate cell layer thick and is called the endothelium. The endothelium has several tasks. Perhaps the most important is to prevent blood from forming clots inside the blood vessels. In fact, tearing of the endothelium (as occurs when an artery is cut) is what triggers platelets and other blood proteins to form a blood clot.

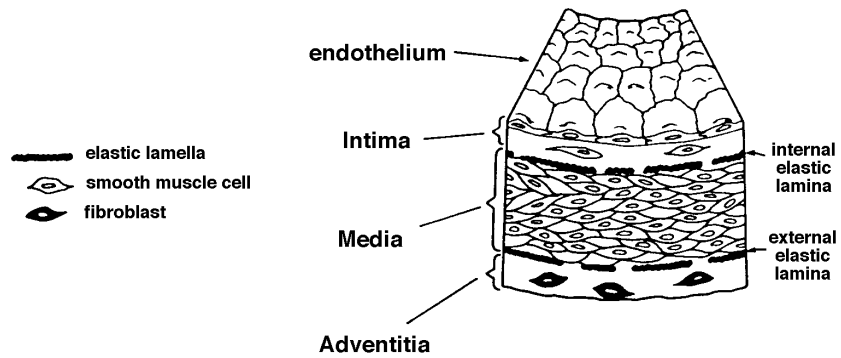


Figure 4.1 Normal Artery Structure

Atherosclerosis begins with damage to the endothelium, such as mechanical trauma, immunologic or chemical injury. A source of mechanical trauma is turbulent blood flow near areas where blood vessels branch off. The exact process by which damage occurs in humans has yet to be fully explained. As seen in Figure 4.2, the local trauma results in an area inside the artery where the endothelial lining is missing.

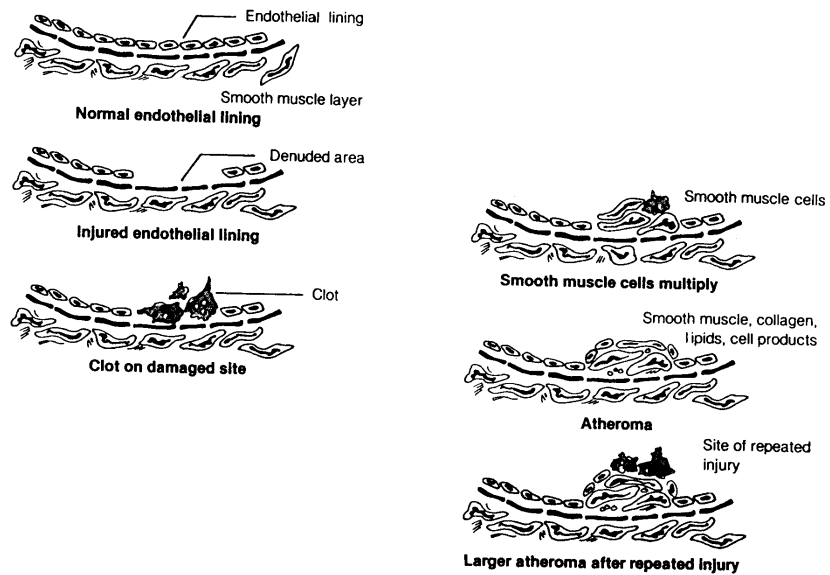


Figure 4.2 The Evolution of Atheroma

As a result, the platelets and clot forming proteins in the blood form a small blood clot. This is how the blood clotting mechanism should normally react in the body. After the clot has formed, over time, the body removes the blood clot and repairs the damage to the artery lining. The area of damage is infiltrated by smooth muscle and other types of cells, and it is this area of cellular buildup where lipids (like cholesterol) and collagen are deposited to form an atherosclerotic plaque, or *atheroma*, for short. The atheroma is gradually covered over by the endothelial layer. As seen in Figure 4.2, this section of endothelium is prone to recurrent injury. With each injury, the atheroma grows thicker, blocking more of the artery.

The formation of atherosclerosis is a very gradual process, taking many years to progress to the point of causing symptoms. The process often begins when a person is in their 20s and 30s. The worsening blockage of an artery depicted in Figure 4.3 takes 20 or more years to develop.

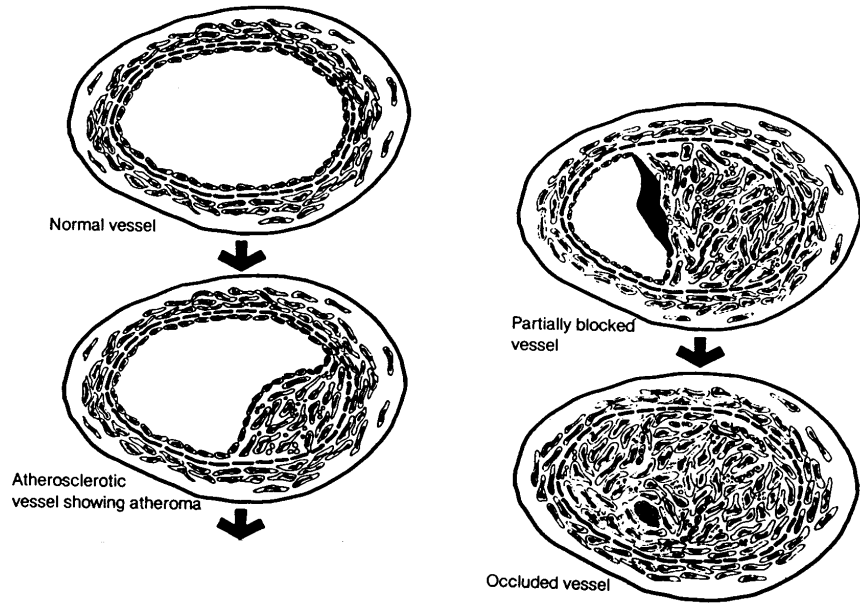


Figure 4.3 Evolution of Blockage in an Artery  
(as seen on cross section of the artery at the point of blockage)

The degree and location of coronary artery blockages are currently best assessed by a heart catheterization, also called a cardiac catheterization. In this procedure, a cardiologist runs a small tube (the catheter) into the coronary arteries, typically through a large needle inserted into an arm or groin artery. Once the catheter is in the coronary artery, contrast material (some people call it “dye”) is injected through it. The contrast fills the arteries, revealing any blockages, their severity and their location on x-rays taken during the procedure.

Remember, atherosclerosis is the process that blocks the coronary arteries. The blockages can have several different effects on people, most commonly causing angina, a heart attack or sudden death. Although these three problems are related because atherosclerosis of the coronary arteries is their underlying cause, the problems affect a person’s health in different ways.

## ANGINA

The term angina (short for angina pectoris) refers to the chest pain that occurs when the heart is not receiving enough blood, usually because of partial blockages (as seen in Figure 4.3) of one or more of the coronary arteries. Discomfort from heart injury feels different from other kinds of pain. While the heart has sensory nerves that can detect pain, these nerves are rudimentary and differ from the pain fibers found in our skin. As a result, the chest pain caused by ASCVD is typically described as a general chest “pressure,” often radiating into the neck and arms. This poorly localized, vague pain is in direct contrast to the more sharp, well localized pain that occurs when the skin is injured.

The pain fibers in the heart are stimulated when the amount of oxygen getting to the heart is not adequate to meet the myocardium’s need for oxygen. A typical scenario involves a coronary artery blockage that prevents an increase in blood flow to deliver more oxygen to the

## HEART ATTACK

heart when it is working hard. For example, a man feels well at rest. However, when walking up a flight of stairs, he develops chest pressure. When he stops and rests, the “angina” gradually resolves. Mechanistically speaking, what has happened in this scenario is that our patient feels well at rest because the heart has adequate coronary artery blood flow. However, when he starts to exert his leg muscles by going up the stairs, the heart speeds up and the blood pressure goes up to supply the leg muscles with more oxygen-rich blood. The increased heart rate and blood pressure means that the heart muscle is also working harder, and that it too needs more oxygen. Unfortunately, the blockages in his coronary arteries limit the increase in blood flow to the heart muscle to deliver more oxygen. The heart muscle “downstream” from the blockages becomes *ischemic*, meaning that it has an inadequate amount of oxygen. The pain fibers in the ischemic myocardium are activated, causing chest pain. When he rests, the heart rate slows and the blood pressure decreases. As a result, the heart muscle is able to get an adequate oxygen supply, and the chest pain gradually resolves.

Despite the chest pain, the heart muscle suffers no permanent damage if the ischemic condition does not last longer than a few minutes. The poor oxygen levels may “stun” the myocardium so that it does not contract, however, as the ischemia resolves, the heart muscle contraction returns to normal. When angina can be predictably brought on and relieved, as in the above scenario, it is called *stable* angina.

Unfortunately, angina is a poor warning signal (albeit an important one) that the coronary arteries have significant blockages. This is due to the poor degree to which pain fibers in the heart are developed. For unexplained reasons, roughly 25% of persons with heart disease do not feel the pain of angina. This is particularly common among diabetics. Even in those who feel pain, recent evidence suggests that people with ASCVD can have episodes of myocardial ischemia and have no chest discomfort what-so-ever. A few people have actually had heart attacks and never even knew it! This condition is aptly called “silent ischemia” and medical science is wrestling with what this condition means for your health, and how to deal with it.

A heart attack is one of the most common and most serious manifestations of ASCVD. As depicted in Figure 4.4, most heart attacks occur when a coronary artery is completely (or almost completely) occluded very quickly by a blood clot.

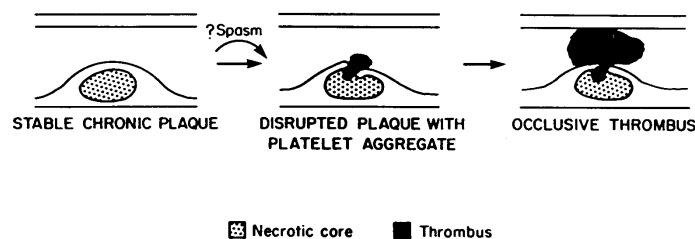


Figure 4.4 Coronary Artery Occlusion in a Heart Attack

As was noted earlier, the process begins with a small blood clot forming on a recently damaged plaque. However, in this instance, the blood clot does not stay small. Rather, it quickly grows into a large clot that completely blocks the coronary artery. This blockage can be further aggravated by the coronary artery narrowing in response to the blockage formation. This coronary artery “spasm” happens to different degrees in different people.

The heart muscle supplied by the occluded artery becomes ischemic, regardless of how hard the heart is working, *causing chest pain or angina*. If blood flow is not reestablished in 10 to 15 minutes, heart muscle cells begin to die, resulting in a *myocardial infarction*.

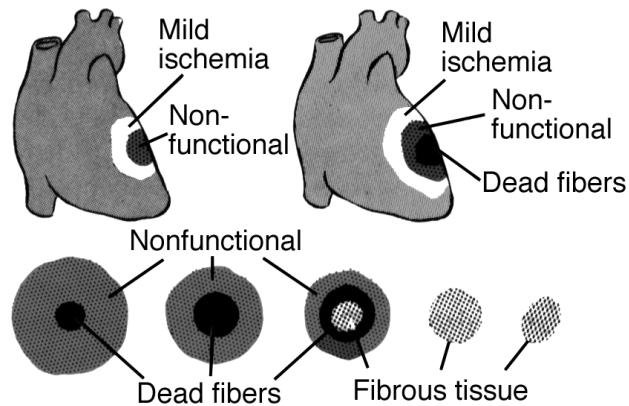


Figure 4.5 Depiction of a Heart Attack

The amount of heart muscle that dies depends on which coronary artery is blocked and where along the length of the artery the blockage has occurred. The left anterior descending coronary artery supplies most of the blood to the critical left ventricle. A blockage far upstream of this (or any) coronary artery will result in a much “larger” myocardial infarction than an occlusion toward the “downstream” end of the artery. The amount of damage to the heart can also be influenced by how well conditioned the heart is. Finally, the size of the damage can be decreased by reopening the occluded artery with “clot-dissolving” drugs or angioplasty at a hospital.

Angina, or chest pain, is a sign that the heart muscle is not receiving enough oxygen but does not necessarily mean a heart attack. During a heart attack the pain is typically more severe, lasts longer (more than 10 to 15 minutes) and does not improve with rest.

Normal functions of the heart are impaired when a portion of the myocardium dies. First, the efficiency of the heart decreases, as the dead heart muscle is replaced by scar tissue that can not contract. As a result of this decreased efficiency, the heart may not be able to pump an adequate supply of blood to the rest of the body. This condition is called *heart failure*. Many symptoms develop in heart failure, including shortness of breath, fatigue and swelling of the lower legs. Until the scar tissue forms and strengthens (takes about 6 weeks) the heart wall is very weak in the area of the dead heart muscle cells. This area is prone to break open, or rupture, usually resulting in rapid death. Heart attacks can also disrupt the specialized conduction system, resulting in abnormal heart rhythm. These problems are some of the more common from a long list of potential complications that occur as a result of a heart attack.

## SUDDEN DEATH

A “sudden death” may be the first sign that someone has severe heart disease. The person suddenly (and literally) drops dead. The exact mechanism of death is not known for sure. Perhaps a blood clot forms (as described in the “heart attack” section above) in an area that results in massive damage. If the left main coronary artery becomes occluded, much of the heart’s blood supply is blocked, and the heart quickly stops working. Sudden death can also result from serious abnormal heart beats (arrhythmia) reviewed later in this chapter. In both cases, the cessation of blood flow in the circulatory system results in no blood flow to the brain, causing unconsciousness within seconds and death within minutes.

Several other less common manifestations of ASCVD include passing out, abnormal shortness of breath, chronic fatigue, acute episodes of exhaustion, abnormal EKG and abnormal heart beats. Each of these can occur by themselves or in a variety of combinations.

**OTHER DISEASES  
OF THE CORONARY  
ARTERIES**

A number of other conditions can eventually lead to blockage of the coronary arteries. Inflammation of the arteries due to a number of rare diseases, called vasculitis, can cause angina and heart attacks. Large doses of ionizing radiation can occasionally damage the coronary arteries. Rarely, blunt trauma to the chest damages the coronary arteries, resulting in blockage formation. All these events are rare and are not associated with any exposures typically associated with fire fighting.

Developmental abnormalities of the coronary arteries can also cause artery blockage. This problem is relatively rare and not associated with fire fighting.

**DISEASES OF THE  
HEART MUSCLE  
(CARDIOMYOPATHY)**

A number of conditions directly affect the heart muscle cells. These diseases of the myocardial cells are collectively called cardiomyopathies. Three major groups of cardiomyopathy exist. Figure 4.6 depicts how the ventricle's myocardium appears for each of the three cardiomyopathies and shows how well the muscle contracts (systole) and relaxes (diastole).

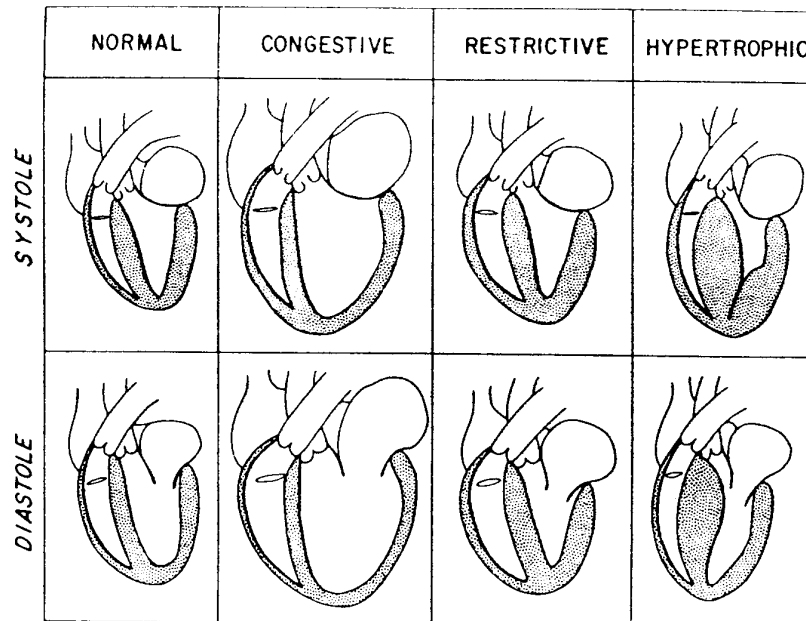


Figure 4.6 The Three Types of Cardiomyopathy Compared to a Normal Heart

**DILATED (OR  
CONGESTIVE)  
CARDIOMYOPATHY**

Several diseases and chemicals can damage but not kill the heart muscle cells so that they do not contract normally. The myocardium becomes weak and "flabby." The result is a heart that enlarges (becomes dilated) and is much less efficient at pumping blood. Many times, the exact cause of an individual's cardiomyopathy goes undetected.

Viral infections, alcohol abuse, and chronic uncontrolled hypertension are the most common causes of dilated cardiomyopathy. Occasionally, cases have been associated with either chronic or high level exposure to several elements, including arsenic, cobalt, lead and antimony. Although fire fighters are not routinely exposed to these elements, certain groups of fire fighters may be exposed if working in industrial areas which use these elements. Chronic exposure to low levels of organic solvents (such as toluene and trichloroethylene) can also cause cardiomyopathy. For fire fighters, exposure to these chemicals is also situational.

**HYPERTROPHIC  
CARDIOMYOPATHY**

Cardiomyopathy has not been shown in any study to affect the health of fire fighters more than the rest of the population. Still, it is important to realize certain exposures can cause myocardial disease and that fire fighters may encounter those exposures.

This group of diseases results from heart muscle cells actually growing. When this occurs, the muscle walls grow so thick that they actually block the heart valves, so blood can't flow through the heart. The cause of this condition is unknown and has not been associated with any occupational exposures. There is a tendency for this problem to occur within families.

**RESTRICTIVE  
CARDIOMYOPATHY**

This group of diseases is called "restrictive" because the heart muscle has difficulty relaxing, so that the heart has trouble refilling with blood. Unable to refill with blood, heart efficiency decreases. The heart can't easily "relax" because various materials, such as a protein called amyloid, infiltrates and intermingles with the otherwise normal heart muscle cells, making the heart walls stiff. Most of the restrictive cardiomyopathies occur because of many different diseases like sarcoidosis, endocrine disorders, and certain cancers. Like hypertrophic cardiomyopathy, no clear occupational exposures are associated with the development of this disorder.

**LEFT VENTRICULAR  
HYPERTROPHY**

Left ventricular hypertrophy (LVH) must be distinguished from hypertrophic cardiomyopathies (see above). Hypertrophy refers to the growth and thickening of the heart muscle cells, but in this case it is in response to the heart having to work harder than normal. LVH most often develops in people with chronic, untreated high blood pressure. It occurs because the heart muscle, like any muscle in the body, bulks up (hypertrophies) when under heavy work loads. In this way, the heart muscle is no different than a muscle in the arm or leg of a weight lifter. Unfortunately, LVH predisposes the heart to many of the other diseases reviewed in this chapter.

**DISEASES OF THE  
HEART VALVES**

In most cases, valvular heart disease affects only one or two of the heart valves. Generally speaking, the left side of the heart (mitral and/or aortic valves) are more frequently affected. Most heart valve disease can be attributed to damage of the valve by infection (often by rheumatic fever) or congenital abnormalities. Years of constant opening and shutting causes small amounts of damage to progress into significant abnormalities.

The two most frequent valve problems are called stenosis and insufficiency. Stenosis means that the valve can't open all the way, restricting blood flow through the heart. The heart must work harder to pump blood through these "narrowed" valves. Insufficiency means the valve leaks, so that blood flows the wrong way across the valve. This "backward" flow of blood results in the heart having to work harder, because it has to constantly "re-pump" a portion of the blood it just finished pumping. These valve conditions have characteristic heart "murmurs" that can be heard with a stethoscope. When either of these problems becomes severe enough to cause symptoms, open heart surgery is typically done to replace the diseased valve.

Increased risk of developing heart valve disease has not been associated with fire fighting, nor with any of the exposures fire fighters are likely to encounter.

**MITRAL VALVE  
PROLAPSE**

Mitral valve prolapse (MVP) refers to a largely genetic condition of the mitral valve that affects up to 10% (primarily women) of the population of Canada and the United States. In MVP, the mitral valve leaflets are too long. The result is that the valve does not close properly. Instead of stopping shut in a normal position, the valves swing too far back, and "prolapse" into the left atrium. Although this condition can be associated with a heart murmur, atypical chest pains, and abnormal heart beats, the vast majority of people with MVP have a normal life.

**INFECTIOUS  
ENDOCARDITIS**

Infectious endocarditis is the diagnosis given when a heart valve is infected by certain micro-organisms like bacteria. In addition to damaging the valve, infected valves frequently become covered with "blood clot-like" growths that can break off and travel through the

## DISEASES OF THE CONDUCTION SYSTEM

### HEART BLOCK

arteries, eventually blocking an artery somewhere else in the body. Should the newly blocked artery be one that leads to the brain, a stroke may occur. Heart valves with previous damage from any cause are at increased likelihood to become infected. Although this is a relatively common diagnosis, no information suggests that fire fighters are at increased risk for this disease.

As described earlier in this manual, the conduction system consists of specialized heart cells that rapidly carry electric current through the heart, delivering the current to most all areas of the heart so that they contract together. This conduction system can have a large number of problems and abnormalities. The more serious problems are reviewed here.

The term “heart block” refers to electric signals not passing down the conduction system in a normal fashion. Blockages can be of varying degrees of severity, from a simple delay of the current to complete blockage. Complete blockage is called “complete heart block” or “third degree heart block.” This problem is analogous to cutting an electrical wire.

Third degree heart block is the most dangerous type of heart block and usually results from damage to the conduction system during a heart attack. Lack of oxygen causes cells that make up the conduction system to die. Unfortunately, the cells most commonly affected are those in the AV node. As was noted in the chapter reviewing normal heart anatomy and function, the electric signal that triggers a heart beat enters the ventricles through the AV node. Thus, when this critical area is damaged, no current enters the ventricles. Since the current is what triggers the heart muscle to contract, the ventricles stop working.

Fortunately, the heart has special areas in the ventricles that can generate an electric current. This will trigger the ventricles to contract, however, this “escape” heart beat is very slow, typically 20 to 30 beats per minute. This slow heart rate may or may not be adequate to keep a person alive. To treat this condition, doctors can implant artificial pacemakers. Pacemakers are small devices with a battery that deliver electric shocks to the myocardium in the ventricles, causing them to contract at more normal heart rates, above 60 beats per minute.

### ARRHYTHMIAS

The term arrhythmia refers to abnormal heart beats. Arrhythmia occur because the normal, orderly delivery, and movement of the electric current in the heart is disrupted. Many things can disrupt the current. A common problem is an area of irritation in the heart that causes the heart muscle to generate electric currents that normally do not develop. This irritation can result from ischemia, scar tissue, cardiomyopathies and valve problems.

Because the atria and ventricles are electrically separate (connected only by the AV node), arrhythmia can occur in one or the other. When electric current is completely erratic in the atria, small areas of the atria contract haphazardly. This condition is called *atrial fibrillation*. If you were to look at the atria (top half of the heart) during atrial fibrillation, they would look like a bag of worms. This type of contraction does not generate enough force to pump any blood. However, as the atria are not essential for the ventricles to fill, most people do well with atrial fibrillation. Frequently, the erratic current in the atria arriving at the AV node at rapid and varying intervals enters the ventricles at an unpredictable, quickly changing rate. This causes the ventricles to have a very irregular heart beat in the range of 150 beats per minute. This can usually be controlled with medication.

The same erratic current can occur in the ventricles, and is called *ventricular fibrillation*. Again, like the atria, the ventricles’ uncoordinated contractions make it look like a bag of worms. Because the ventricles do not pump any blood during fibrillation, there is rapid loss of consciousness and death. The treatment is to “cardiovert” the heart by delivering a large electric shock to the heart. This shock can cancel the erratic current in the heart, allowing the normal heart beat mechanism to take over.

Ventricular tachycardia is another dangerous arrhythmia. The ventricles develop a rapid heart beat (150 to 300 beats per minute) in which electric currents bypass the normal heart conduction system. In addition to the rapid heart beat, the heart muscle contraction is less coordinated than normal. The resulting rapid, inefficient pumping of the heart can result in a low blood pressure that requires emergency medical treatment.

A number of chemicals have been shown to “sensitize” the myocardium, making the development of arrhythmias from irritations of the myocardium more likely. This is particularly worrisome in people who already have underlying heart disease. These include halogenated hydrocarbons, organophosphates, antimony, arsenic and arsine. Although exposure to these chemicals will be highly situational for fire fighters, the increasing use of synthetic polymers and chemicals increases the potential for exposure, especially to halogenated hydrocarbons.

## CONCLUSION

The different systems of the heart can suffer from a variety of diseases. Atherosclerotic coronary vascular disease is clearly the most common health problem facing fire fighters. This disease process takes many years to develop and is preventable in many cases. Other diseases of the heart do not appear to affect fire fighters at unusually high rates. Still, the probability of developing certain conditions like arrhythmias and cardiomyopathy can be increased by certain chemical exposures. It is important that fire fighters be aware of the potential danger of such exposures and avoid them as much as possible.

## CREDITS

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# Chapter 5

## **RISK FACTORS FOR ATHEROSCLEROTIC CORONARY VASCULAR DISEASE**

Atherosclerotic coronary vascular disease (ASCVD) is one of the leading causes of death in the United States and Canada. Many research scientists have sought to define the factors that increase a person's risk of developing ASCVD. In order to discover these individual characteristics, it is necessary to link vital statistics (such as mortality studies) with genetic factors, occupational risks, and lifestyle choices in individual people. Thousands of people have been studied in hundreds of studies. One of the first and most important studies was the Framingham Heart Study. Since 1950, most all of the adults in the town of Framingham, Massachusetts have been followed very closely for the development of heart disease. This ongoing study has discovered several characteristics that are clearly associated with an increased chance of developing ASCVD.

This chapter will review the known and suspected risk factors for developing ASCVD. For purposes of organization, this chapter has been divided into four sections:

- Major risk factors we are unable to control;
- Major risk factors we can control;
- “Minor” risk factors; and
- “Special” risk factors faced by fire fighters.

All four groups are important, but those factors that we can control deserve particular attention.

## **MAJOR RISK FACTORS BEYOND OUR CONTROL**

This section reviews those factors associated with an increased risk of developing ASCVD that we can not reasonably control.

### **AGE**

The mortality of ASCVD shows a striking relationship to age. Although rare in younger women, the disease is already a major cause of death in men 35 to 44 years of age. This death rate increases so rapidly that for those people over age 55, 35 percent of all deaths are due to ASCVD. This increase in heart disease mortality with age is logical, because cholesterol plaques build up over time. The older you get, the more time plaques have to develop. Figure 5.1 depicts how heart disease rates increase in both men and women as they grow older.

### **GENDER**

As can also be seen in Figure 5.1, the age specific mortality incidence rates for heart disease are much higher in men than in women. However, as men and women age, the difference in death rates from ASCVD becomes smaller. Unfortunately, at all ages, the survival of women after myocardial infarction (MI) is uniformly poorer than among men; this is largely unexplained but may be related to the smaller heart size of women. The apparent protective effects of female hormones, called estrogens, have been implicated as the reason for the lower risk of heart disease among premenopausal women. But, after menopause, female estrogen levels diminish, resulting in less protection from the development of atherosclerotic plaques. Post-menopausal estrogen replacement therapy has been in common practice for several years, and recent studies have determined that women who take hormone replacement gain some level of protection against heart disease, as compared to women who do not.

## Percentage of Deaths due to Cardiovascular Disease by Age and Sex Canada

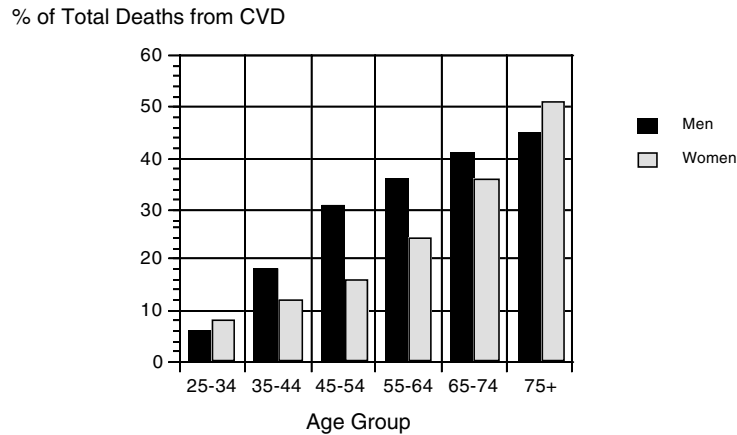


Figure 5.1

### FAMILY HISTORY

Family and genetic factors appear to play an important role in determining the relative importance of the risk factors reviewed in the next section (in particular for high blood pressure, diabetes, and high cholesterol). However, there appears to be a genetic predisposition to ASCVD in some families that is independent of these other known risk factors. This “genetic risk” portends that the risk of family members developing ASCVD increases as the age of blood relatives who have developed coronary heart disease decreases. For example, having a close relative (father, mother, brother, sister) have a heart attack before age 55, dramatically increases a person’s risk of developing coronary artery disease.

### MAJOR RISK FACTORS WE CAN CONTROL

This section reviews those risk factors which we can control with reasonable effort, thereby reducing the risk of developing ASCVD. Information and strategies for controlling these factors are examined in Chapter 6.

### CHOLESTEROL

Cholesterol belongs to a larger family of biological chemicals called lipids (fats). Cholesterol is an essential ingredient in our bodies. Many parts of the cells that make up our bodies are made of cholesterol. In addition, cholesterol is transformed into many other chemicals that are important for good health, such as Vitamin D and certain hormones.

Because it is such a critically important substance, a complex “carrier” system has developed to move cholesterol through the entire body. This system consists of a number of proteins that bind to cholesterol and transport it to where it is needed. Cholesterol, a *lipid*, when bound together with one of these carrier *proteins*, is called a *lipoprotein*. Both cholesterol and carrier proteins can be measured in blood samples.

Although there are many different types of lipoproteins, two are of particular importance when looking at the risk of having a heart attack. Low Density Lipoprotein (LDL cholesterol) is 45 per cent cholesterol by weight and is the major carrier of cholesterol to the body’s tissues. Because this lipoprotein can deliver too much cholesterol to the wrong places (like the heart arteries) resulting in cholesterol plaque build up, people often refer to this as “bad” cholesterol.

High Density Lipoprotein (HDL cholesterol) is 30 per cent cholesterol by weight and is involved in the reverse transport of cholesterol away from body tissues and out of the body. HDL cholesterol removes excess cholesterol from the arteries, helping to prevent cholesterol

plaque build up. Because this lipoprotein appears to remove excess cholesterol, it is often referred to as “good” cholesterol.

Among the numerous risk factors for developing ASCVD, one of the best documented is the association between blood cholesterol levels and heart attack rates.<sup>2</sup> When looking at total serum cholesterol levels, the risk of developing ASCVD increases as the cholesterol level increases.<sup>3</sup> Figure 5.2 shows how heart attack death rates go progressively higher as cholesterol levels increase.

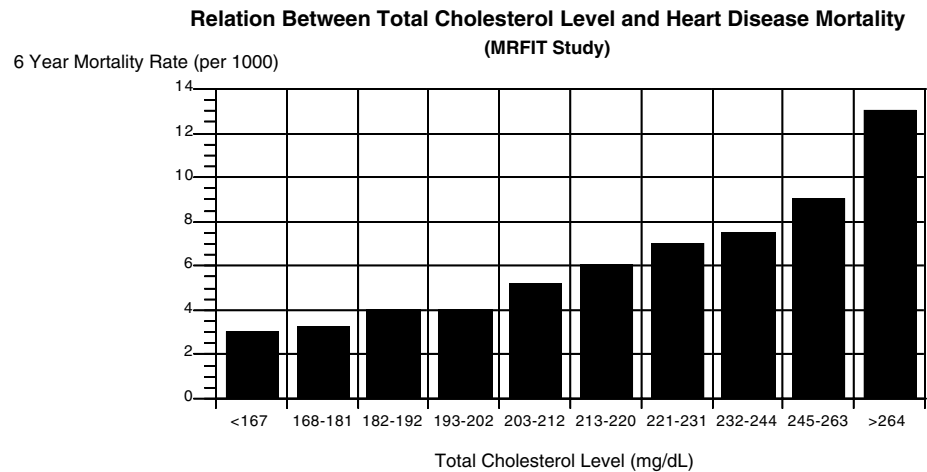


Figure 5.2

A slightly different way of looking at this relationship is shown in Table 5.1. This table shows the results of a large study in which the people studied were divided into 5 groups of equal size. The first group is those people (Percentile 0-20) with the lowest total cholesterol levels. The last group (Percentile 80-100) is those with the highest total cholesterol levels. When compared to the lowest level group, the highest level group has a 3.4 times higher risk of developing ASCVD. As seen in Table 5.1, risk increases as cholesterol levels rise.

<b>The Risk of ASCVD Increasing with Cholesterol Levels</b>		
<b>Percentile</b>	<b>Serum Cholesterol (mg/dL)</b>	<b>Relative Risk of ASCVD</b>
<b>0-20</b>	<b>&lt;181</b>	<b>1.0</b>
<b>20-40</b>	<b>182-202</b>	<b>1.3</b>
<b>40-60</b>	<b>203-221</b>	<b>1.7</b>
<b>60-80</b>	<b>222-245</b>	<b>2.2</b>
<b>80-100</b>	<b>&gt;246</b>	<b>3.4</b>

Table 5.1

Although not shown here, LDL cholesterol levels have the same relationship; that is, an increasing LDL cholesterol level increases the risk of developing heart disease.<sup>4</sup>

From these kinds of data, the National Cholesterol Education Program Expert Panel from the American Medical Association developed these guidelines for assessing cholesterol levels, shown in Table 5.2.

Recommended Cholesterol Levels.		
Interpretation Level	Total Cholesterol Level	LDL Cholesterol Level
Desirable level	<200	<130
Borderline level	200 to 239	130 to 159
High-risk range	>240	>160

The recommended levels for total and LDL cholesterol are even lower if one has additional risk factors, such as diabetes or hypertension.

Table 5.2

In addition, desirable HDL cholesterol levels should be over 35 mg/dL. As seen in Figure 5.3, as HDL cholesterol levels increase, the risk of ASCVD decreases.

**Relation Between HDL Cholesterol Levels and Heart Disease Rates (Framingham Heart Study)**

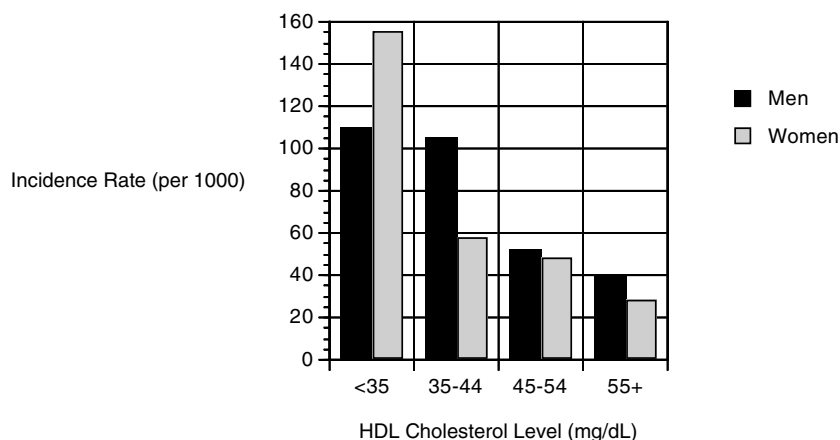


Figure 5.3

Many times, genetic factors inherited from parents play a large role in determining cholesterol levels. Several “familial dyslipoproteinemias”, which can cause extremely high cholesterol and LDL levels, are well described and are quite common. Regardless of why cholesterol levels are elevated, diet and exercise (as well as certain medications) can significantly improve cholesterol levels.

## TRIGLYCERIDE

Triglycerides are a different type of lipid which (like cholesterol) can be measured in the blood. This type of lipid makes up the fat stored by our bodies and makes up most of the fat in the animal meats we eat. When in the blood, triglycerides can actually form microscopic fat globules.

Triglycerides have been weakly associated with increased risk of ASCVD in a number of studies, although this evidence is still debated by some scientists.<sup>6</sup> Regardless, the National Institutes of Health have classified blood (serum) triglyceride levels under 250 mg/dl as normal, 250 to 500 mg/dl as borderline risk, and above 500 mg/dl as high risk.

## TOBACCO USE

Tobacco is a major risk factor for the development of ASCVD. According to the Surgeon General, in 1987, 23% of the total ASCVD deaths in the United States (about 200,000) were due to cigarette smoking.<sup>7</sup> Many studies have demonstrated that smokers have a 70% higher risk of dying from ASCVD than do non-smokers.<sup>8</sup> Several mechanisms may account for this increase in risk. First, cigarette smoke has been demonstrated to have adverse effects on cholesterol levels, lowering HDL levels and increasing LDL levels. Tobacco use has also been correlated with alterations in blood pressure and increases in blood carbon monoxide levels. Finally, tobacco smoke exerts many effects on blood clotting factors, platelet function and other hematological parameters that probably play a role in the formation of atherosclerotic plaques.<sup>8,9</sup>

In addition to helping cholesterol plaques form in the arteries, smoking appears to increase the likelihood of having a heart attack once plaques are present. Nicotine, a stimulant present in tobacco smoke, interacts with the body's nervous system to cause the release of epinephrine (adrenaline). Epinephrine increases the tone of the muscular layer present in arteries, resulting in enhanced constriction and narrowing of the arteries. Carbon monoxide also decreases the ability of the blood to transport oxygen by tightly binding to hemoglobin. These effects combine to worsen any partial blockage that may already be present from cholesterol plaques. Tobacco smoke also increases the ability of blood to form blood clots on the atherosclerotic plaque, causing sudden narrowing (if not complete blockage) of the arteries affected by ASCVD.<sup>10</sup>

As shown in Figure 5.4, the risk of developing a manifestation of heart disease is much higher in smokers as compared to non-smokers.

**Risk of Selected Heart Disease Manifestations by Smoking Status  
(Framingham Heart Study)**



Figure 5.4

## HYPERTENSION

Hypertension (high blood pressure) is a modifiable risk factor that frequently coexists with other risk factors. Because blood pressure can change in response to a variety of stimuli, the resting blood pressure (blood pressure when you are resting and relaxed) is what is measured to determine if someone has hypertension. The most frequently used numbers to define hypertension are a systolic blood pressure over 140 or a diastolic blood pressure over 90. A large number of medical conditions can cause hypertension, but most people with hypertension

have no apparent “underlying” disease causing the elevated pressures. This is called “essential” hypertension. Hypertension is often referred to as a “silent killer” as people typically live 20 years or more without obvious symptoms related to hypertension. The only way to detect this problem early, before it can cause damage, is to have blood pressure measurements taken regularly.

Hypertension is associated with an increased risk of heart disease. As seen in Figures 5.5 and 5.6, the risk increases as both diastolic and systolic blood pressures increase.

### Relation of Diastolic Blood Pressure and Heart Disease (Framingham Heart Study)

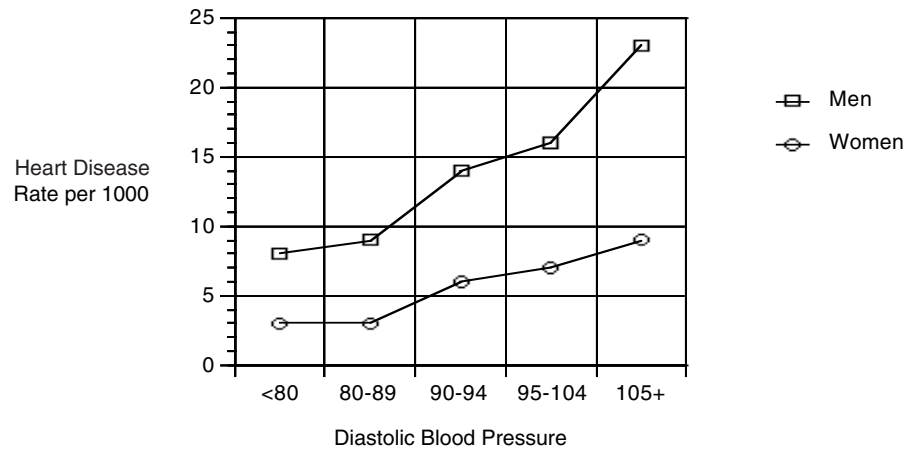


Figure 5.5

### Relation of Systolic Blood Pressure and Heart Disease (Framingham Heart Study)

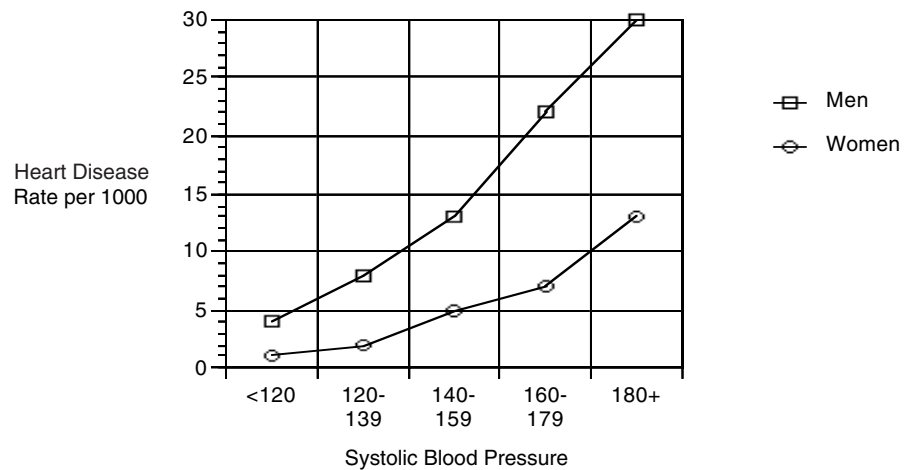


Figure 5.6

Hypertension may accelerate the formation of atherosclerotic plaques by damaging the walls of blood vessels. These damaged areas are prone to the formation of cholesterol plaques.

## DIABETES MELLITUS

High blood pressures also force the heart to work harder, since the heart must pump blood out against a high pressure. This higher work load puts the heart at increased risk of a heart attack if a coronary artery has a blockage. (Remember, elevated blood pressures are based on resting blood pressures, not when someone is under physical or psychological stress.)

Diabetes is a disease of glucose (sugar) metabolism due to deficiencies or resistance to insulin, a hormone which induces cells to take up glucose from the blood. Type I diabetes is due to a lack of insulin, whereas type II results from an inability of insulin to properly induce cells to take up blood sugar. The result of both is high blood sugar levels, often accompanied by high cholesterol levels and hypertension. These high levels of sugar interact in complex ways with proteins in the body to accelerate the formation of cholesterol plaques in the arteries. Persons who are afflicted with diabetes have a clear increase in both risk and severity of coronary artery disease and fatal MI.<sup>11</sup> Current research is trying to determine whether good control of blood sugar levels decreases a diabetic person's risk of developing heart disease.

## OBESITY

The precise role of obesity as an independent cardiac risk factor remains unclear. It is known that the risk of ASCVD is increased in obese persons, but this risk does not increase with increasing weight. Overweight and obesity do raise the risk of developing other risk factors, including hypertension, elevated cholesterol, and diabetes. It is postulated that these associated risk factors are responsible for most of the increase in ASCVD seen in obese persons.

Interestingly, new studies have also demonstrated a relationship between the distribution of fat in the body and cardiac risk. Persons who have a higher proportion of fat deposition around the abdomen as opposed to the hips and thighs have a greater risk of developing ASCVD.<sup>12</sup> These findings have raised new hypotheses about separate metabolic entities and about the causes of heart disease.

## SEDENTARY LIFESTYLE

Most epidemiologic studies show an inverse relationship between physical activity and risk from ASCVD and all other causes. The more that you exercise, the lower your risk of heart disease.<sup>13</sup> In fact, populations with the highest heart disease mortality rates are from sedentary, highly industrialized societies. Physical activity may have a favorable effect on the heart via several mechanisms: increasing HDL levels, increasing coronary artery size, decreasing myocardial oxygen demand as the "training effect" improves heart muscle conditioning, and decreasing tendencies for "over zealous" blood clot formation. Physical fitness also has a positive impact on other cardiac risk factors, such as obesity and diabetes. Regardless of how it works, as seen in Figure 5.7, exercise reduces the risk of having a heart attack.

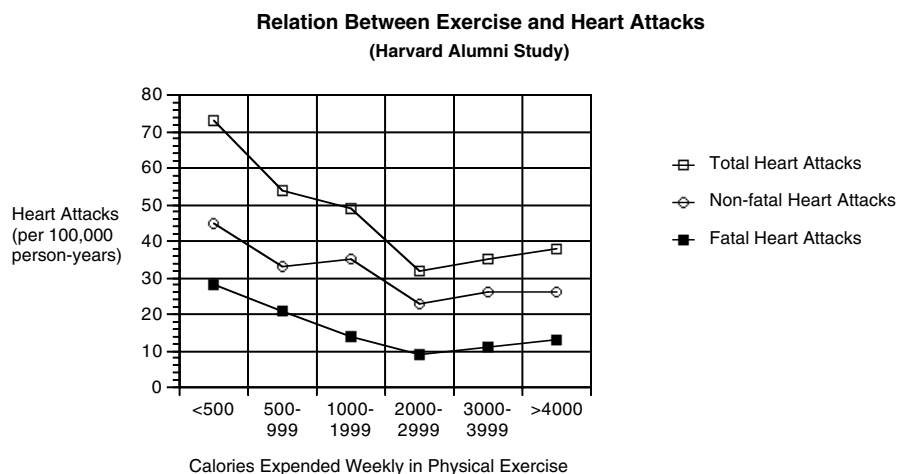


Figure 5.7

**MULTIPLE RISK FACTORS**

It is important to understand that the more risk factors that you have, the higher the risk of developing ASCVD. Figure 5.8 shows results from the Framingham study. This figure indicates that 50 year old men with two risk factors have a three times greater risk of developing ASCVD than men with no risk factors. The risk continues to increase in persons with additional risk factors.

**Relation of Number of Risk Factors and Coronary Artery Disease**

Framingham Heart Study

Rate of Developing Heart Disease (per 1000)

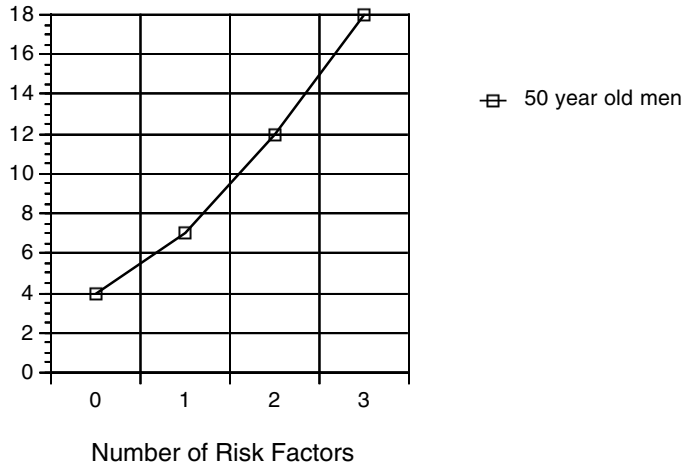


Figure 5.8

**MINOR RISK FACTORS**

This section briefly reviews several potential risk factors for developing ASCVD. Many of these factors are still under active investigation to better define and quantify any increased risk they confer.

**TYPE A PERSONALITY**

Several large studies have evaluated the correlation of type A personalities to the risk of developing ASCVD. Type A people are characterized as being highly competitive, ambitious and in a constant struggle with their environment. On the other hand, Type B people are defined as being more passive (“laid-back”) and struggle less with their environment. According to earlier studies, type A personality appeared to have about twice the risk of developing angina as Type B persons, but Type A persons do not have an increase in death rates from heart attacks! Newer studies and a follow up to the Western Collaborative Trial have failed to duplicate these findings. Thus, at present no clear evidence for a link between Type A behavior and heart disease has been established.<sup>14,15,16</sup>

**WATER HARDNESS**

Some large studies have found that cardiovascular disease is inversely associated with water hardness. That is to say, the harder the water in a person’s water supply, the less likely that person will develop ASCVD. Unfortunately, no study has clearly linked the specific components in the water to increased risk. Selenium and zinc have been described as protective, lead and calcium have been described as conferring increased risk, and magnesium has been put in both categories. The inconsistency of the findings precludes any definite conclusions on whether the trace mineral composition of water represents an ASCVD risk factor.<sup>17</sup>

**HYPERCOAGULABILITY**

The term “Hypercoagulability” specifically describes people whose blood forms a clot faster than expected. In many cases, this condition can be linked to elevated levels of a clotting protein called fibrinogen. Fibrinogen levels increase with smoking, high cholesterol levels, obesity and increased stress. Yet, even when accounting for these associated risk factors, high fibrinogen levels are independently associated with increased ASCVD risk. The exact mechanism by which fibrinogen contributes to atherosclerotic plaque formation is not known. However, it is well known that regular aspirin use and exercise lower fibrinogen levels and lower the risk of heart attack in persons over 50.<sup>18</sup>

**COFFEE CONSUMPTION**

Studies have been indeterminate about whether either coffee or caffeine consumption is a risk factor for ASCVD. If coffee does influence risk, the effect is probably small.

**ALCOHOL**

The epidemiologic evidence about the relationship between alcohol consumption and heart disease is conflicting.<sup>19</sup> There is evidence to suggest that the moderate consumption of alcohol (1 to 2 ounces per day) may increase HDL and result in a slight reduction of heart disease risk. However, consumption of alcohol is known to increase blood pressure and has been demonstrated to increase the risk of heart disease when consumed in immoderate amounts. Very large amounts of alcohol are directly toxic to the myocardium and can lead to heart failure. Thus, given the propensity of alcohol to cause other serious health problems, there is serious concern about recommendations to consume alcohol for its beneficial effects.

**FIRE FIGHTERS  
RISK FACTORS**

This section reviews occupational exposures faced by fire fighters that may increase the risk of developing atherosclerotic plaques or may precipitate a heart attack in fire fighters with underlying ASCVD.

**CARBON  
MONOXIDE**

Carbon monoxide (CO) is formed by the incomplete combustion of carbon containing materials. Personal monitors worn by fire fighters have recorded very high concentrations of CO at fires.<sup>20,21</sup> Carbon monoxide decreases the oxygen carrying ability of the blood and poses acute and chronic health effects.

Carbon monoxide acts as a chemical asphyxiant and starves the heart of its normal oxygen supply by binding to the oxygen carrying molecule, hemoglobin. Carbon monoxide binds to hemoglobin 200 times more effectively than oxygen, so it takes only a small percentage of CO in the inspired air for hemoglobin molecules to become filled with CO. Once bound to hemoglobin, it is difficult to remove CO from the oxygen binding site. The net effect is that even low concentrations of CO in the air can have significant negative effects on the body’s ability to transport oxygen.

At high CO exposure levels, the oxygen carrying capacity of blood becomes compromised and the death of myocardium (similar to a heart attack) can occur, even without any blockages in the coronary arteries. The physical demands of fire fighting increase the body’s demand for oxygen and can worsen symptoms. High level of exposure can also result in loss of consciousness due to lack of oxygen sent to the brain. In persons with known ASCVD accompanied by angina, CO exposure has been shown to reduce exercise capacity and result in vulnerability to cardiac rhythm disturbances.<sup>22</sup> The reduced capability of the blood to carry oxygen following CO exposure functionally worsens existing heart artery blockages, resulting in decreased oxygen delivery to heart muscle without the blockages actually becoming larger. Reduced exercise capacity and ischemic responses have also been shown in healthy young adults exposed to CO.<sup>23</sup>

Long-term exposure to CO is suspected to cause the formation of atherosclerotic plaques. Since several studies demonstrated atherosclerosis developing at accelerated rates in animals chronically exposed to CO.<sup>24</sup> This hypothesis is reinforced by the observation that, in animals,

chronic exposure to high concentrations of CO in cigarette smoke increases the risk of developing heart disease. The exact mechanism by which this occurs is unknown.

The evidence from epidemiologic studies is also suggestive that CO has the same effect in people. In northern Japan, chronic high-level exposures to CO have been reported to produce a seven-fold increase in ASCVD occurrence in tatami mat makers.<sup>25</sup> These workers heat their tightly sealed buildings with charcoal braziers. Likewise, New York City tunnel officers (chronically exposed to CO from auto exhaust) were found to have a 35% increase in risk of dying from ASCVD. This excess risk declined after tunnel ventilation improvements decreased tunnel CO levels.<sup>26</sup>

Since the only significant entryway for CO into the body is through the lungs, fire fighters can protect themselves from excessive exposure by proper use of personal protective equipment, such as the self-contained breathing apparatus.

**POLYCYCLIC  
AROMATIC  
HYDROCARBONS**

Polycyclic aromatic hydrocarbons (PAHs) are a group of carbon compounds that are formed in most fires involving carbon containing materials, including wood, fuels and many man-made materials. Like carbon monoxide, studies of long term exposure to PAHs have shown an accelerated formation of atherosclerotic plaques in animals.<sup>27</sup> However, this association has never been clearly demonstrated in people. Unlike CO, PAHs are not asphyxiants. However, they are considered carcinogens in the lung and colon.

**OTHER CHEMICALS**

A number of other chemicals that can be present in combustion products can have adverse effects on the heart. Cyanide and hydrogen sulfide act as chemical asphyxiants, potentially resulting in myocardial ischemia. Arsenic and carbon disulfide exposure may contribute to atherosclerotic plaque formation. Lead, cadmium, and organic solvent exposure may contribute to the development of high blood pressure, thus indirectly affecting the heart.<sup>28</sup> The potential for exposure to these dangerous chemicals increases as the normal industrial mechanisms used to control exposure fail during fire and disaster situations.

**OCCUPATIONAL  
STRESS**

Many, but not all studies have demonstrated an association between job stressors and the risk of ASCVD. Job-related factors that appear to influence the induction or exacerbation of ASCVD included perceived job stress, role ambiguity, job autonomy, and job change. However, such causality is controversial. The presumed mechanisms of stress on ASCVD include the activation of nervous and hormonal systems that increase blood pressure and induce cholesterol level abnormalities, as well as changes in personal habits of smoking and diet.

A number of studies have demonstrated that fire fighters suffer from increased psychological stress.<sup>29</sup> The psychological stress accompanying alarm response is frequently accompanied by physical changes. For example, the heart rate of fire fighters has been found to markedly increase (an average of 47 to 61 beats per minute) when a fire alarm sounds, and the heart rate will remain elevated until arrival at the fire scene, despite minimal physical exertion.<sup>30, 31</sup>

The level to which stress increases heart disease risk has yet to be accurately determined, but may be small relative to other risk factors.

**NOISE**

Noise is a physical stressor that is well known to cause the release of adrenaline stimulating the sympathetic nervous system and resulting in increased blood pressure. These responses have been found in both human and animal studies. The characteristics of the noise that have been associated with heart disease include unpredictability, a lack of meaningfulness, high volume, and of an intermittent nature.<sup>32</sup>

Studies of fire fighters' reaction to alarm signals have confirmed that noise can induce measurable biologic and psychological effects. Although the physical activity necessary to get

into a truck following an alarm signal should not increase the heart rate to more than 100 beats per minute, studies have demonstrated even greater response. Heart rates, particularly among younger fire fighters, increase to as much as 130-150 beats per minute after exposure to the station fire alarm.<sup>33</sup> Several additional studies have demonstrated 47-61 beat per minute increases after exposure to alarms. It has been theorized that these responses result from release of catecholamines, like adrenaline. In animals, adrenaline has been demonstrated to disrupt the endothelium. This endothelial damage can result in premature atherosclerosis and could be a contributing factor to the increased risk of heart disease among fire fighters.

Noise exposure has been shown in some, but not all, studies to increase the risk of ASCVD. One study performed in Germany found that steelworkers in noisy jobs had a higher incidence of circulatory and neurologic irregularities than their coworkers in less noisy areas of the plant. Further evidence is provided by a NIOSH study performed in conjunction with Raytheon. The medical records of factory workers regularly exposed to noise levels of 95 dB or greater were compared to workers exposed at 80 dB or less. The findings showed a statistically significant increase in the number of cardiovascular diseases in the high exposure group. Thus, loud noise exposure appears to result in a small, but measurable, increase in heart disease risk.

One possible solution to address this situation is to modify existing alarm systems. Skolnick has suggested that alarms should sound only in stations that are required to respond.<sup>34</sup> To minimize the impact on hearing and the cardiovascular system, they should ring in gradually increasing volume rather than at full volume from the outset.

## HEAT AND COLD

Both excessive heat and cold have been linked to an increased risk of heart attack, especially in those persons with existing coronary artery blockages. Cold causes blood vessels to constrict, elevating blood pressure, thereby increasing how hard the heart must work. Heat increases the heart's work load as it must pump more blood to the skin as the body tries to cool itself.<sup>35</sup> Either stress can presumably induce a heart attack if demands on the heart are excessive.

## CONCLUSIONS

Atherosclerotic heart disease is the major killer of adult men and women in North America. Analysis of the characteristics and habits of populations have delineated many factors which affect an individual's risk of developing ASCVD. Elevated blood cholesterol, smoking, high blood pressure, obesity, and sedentary lifestyle are the major "classic" risk factors that can be controlled by an individual's actions. These issues are important as studies in Los Angeles County safety personnel and Dallas fire fighters have revealed comparable rates of high blood cholesterol, tobacco abuse, hypertension, obesity, and poor physical conditioning in fire fighters when compared to the general population. In addition, fire fighters face important and unique occupational hazards, such as carbon monoxide exposure, extreme temperatures, noise, and psychological stress in the daily performance of their duties. By controlling both personal and professional risk factors, fire fighters can promote their long term cardiovascular health.

## END NOTES

<sup>1</sup> Meade TW, Berra A. Hormone Replacement Therapy and Cardiovascular Disease. *Br Med Bull* 1992; 48:276-308.

<sup>2</sup> Keys A. *Seven Countries: Death and Coronary Heart Disease in Ten Years*. Cambridge: Harvard University Press, 1979.

<sup>3</sup> Stamler J, Wentworth D, Neaton JD. Is the Relationship Between Serum Cholesterol and Risk of Premature Death from Coronary Heart Disease Continuous and Graded? Findings in 356,222 primary Screenings of the Multiple Risk Factor Intervention Trial. *JAMA* 1986, 256:2823-2828.

<sup>4</sup> Wallace RB, Anderson RA. Blood Lipids, Lipid Related Measures, and the Risk of Atherosclerotic Cardiovascular Disease. *Epidemiol Rev*. 1987; 9:95-119.

<sup>5</sup> Multiple Risk Factor Intervention Trial Research Group. Multiple Risk Factor Intervention Trial: Risk Factor Changes and Mortality Results. *JAMA* 1982; 248:1465-1477.

- <sup>6</sup> Wallace RB, Anderson RA. Blood Lipids, Lipid Related Measures, and the Risk of Atherosclerotic Cardiovascular Disease. *Epi Rev* 1987; 9:95-119.
- <sup>7</sup> Reducing the Health Consequences of Smoking: 25 years of Progress. A Report of the Surgeon General. Washington D.C.: US Department of Health and Human Services, Public Health Service, Office on Smoking and Health, DHHS Pub No. (CDC) 89-8411, 1989.
- <sup>8</sup> Report of the Surgeon General. The Health Consequences of Smoking: Cardiovascular Disease. Rockville, MD: U.S. Dept. of Health and Human Services, Public Health Service, Office on Smoking and Health, 1983.
- <sup>9</sup> McGill HC. Potential Mechanisms for the Augmentation of Atherosclerosis and Atherosclerotic Disease by Cigarette Smoking. *Prev Med* 1979; 8:390-403.
- <sup>10</sup> Blackburn H, Luepker R. Heart Disease. In Maxcy-Rosenau-Last, Public Health and Preventive Medicine, 13th ed. Eds J Last, R Wallace. Norwalk, CT: Appleton and Lange, 1992, p.827-847.
- <sup>11</sup> Pyorala K, Laakso M, Uusitupa M. Diabetes and Atherosclerosis: An Epidemiologic View. *Diabetes Metab Rev* 1987; 3:463-524.
- <sup>12</sup> Bjorntorp P. The Associations Between Obesity, Adipose Tissue Distribution, and Disease. *Acta Med Scand* 1988; 723s:121-134.
- <sup>13</sup> Powell KE, et al. Physical Activity and the Incidence of Coronary Heart Disease and Death. *Ann Rev Pub Health* 1987; 8:253-287.
- <sup>14</sup> Rosenman RH, Brand RJ, Jenkins CD, et al. Coronary Heart Disease in the Western Collaborative Group Study: Final Follow-up Experience of 8 1/2 years. *JAMA* 1975; 233:872-877.
- <sup>15</sup> Shekelle RB, Hulley SB, Neaton JD, et al. The MRFIT Behavior Pattern Study. Type A Behavior and the Incidence of Coronary Heart Disease. *Am J Epidemiol* 1985; 122:559-570.
- <sup>16</sup> Ragland DR, Brand RJ. Type A Behavior and Mortality from Coronary Heart Disease. *N Eng J Med*. 1988; 318:65-69.
- <sup>17</sup> Ragland DR, Brand RJ. Coronary Heart Disease Mortality in the Western Collaborative Group Study: Follow up Experience of 22 years. *Am J Epidemiol* 1988; 127: 462-475.
- <sup>18</sup> Sharrett AR, Feinleib M. Water Constituents and Trace Elements in Relation to Cardiovascular Diseases. *Prev Med* 1975; 4:20-36.
- <sup>19</sup> Mead TW. Clotting Factors and Ischemic Heart Disease. In Meade TW (ed): *The Epidemiologic Evidence from Anticoagulants in Myocardial Infarction: A Reappraisal*. New York: John Wiley and Sons, 1984.
- <sup>20</sup> Wilhelmsen L, et al. Fibrinogen as a Risk Factor for Stroke and Myocardial Infarction. *N Eng J Med* 1984; 311:501-505.
- <sup>21</sup> Steering Committee of the Physicians' Health Study Research Group HMS. Preliminary Report: Finds from the Aspirin Component of the Ongoing Physicians' Health Study. *NEJM* 1988; 318:262-264.
- <sup>22</sup> Astrup P. Enhancing Influence of Carbon Monoxide on the Development of Atheromatosis in Cholesterol Fed Rabbits. *J Atheroscler Res* 1967; 7:343-54.
- <sup>23</sup> Goldsmith JR. Carbon Monoxide Research. Recent and Remote. *Arch Environ Health* 1970; 21:118-120.
- <sup>24</sup> Kaelber CT, Barboriak J. Symposium on Alcohol and Cardiovascular Diseases. *Circulation* 1981; 64.
- <sup>25</sup> Loke J, et al. Acute and Chronic Effects of Fire Fighting on Pulmonary Function. *Chest* 1980; 77:369-373.
- <sup>26</sup> Loke J, et al. Carboxyhemoglobin Levels in Firefighters. *Lung* 1976; 154:35-39.
- <sup>27</sup> Decoufle P, Lloyd JW, Salvin LG. Mortality by Cause Among Stationary Engineers and Stationary Firemen. *JOM* 1977; 19:679-82.
- <sup>28</sup> Petronio L. Chemical and Physical Agents of Work-related Cardiovascular Diseases. *Eur Heart J* 1988; 9:26-34.
- <sup>29</sup> Barnard RJ, Gardner GW, Diaco NV. "Ischemic" Heart Disease in Firefighters with Normal Coronary Arteries. *JOM* 1976; 18:818-820.
- <sup>30</sup> Benowitz NL. Cardiotoxicity in the Workplace. *Occ Med: State of the Art Rev*, 1992; 7:465-478.

<sup>31</sup> Goldhaber SZ. Cardiovascular Effects of Potential Occupational Hazards. *JACC* 1983; 2:1210-15.

<sup>32</sup> Guidotti, TL. Human Factors in Fire Fighting: Ergonomic, Cardiopulmonary, and Psychogenic Stress Related Issues. *Int Arch Occ Environ Health* 1992; 64:1-12.

<sup>33</sup> Kuorinka I, Korhonen O. Firefighters' Reaction to Alarm, An ECG and Heart Rate Study. *JOM* 1981; 23:762-766.

<sup>34</sup> Skolnick MH. Heart Disease Among Fire Fighters: Part 2. *Int Fire Fighter*. 1964; 47:8-9.

<sup>35</sup> Duncan HW, Gardner GW, Barnard RJ. Physiological Responses of Men Working in Firefighting Equipment in the Heat. *Ergonomics* 1979; 22:521-527.

## CREDITS

Figure 5.1 Reproduced with permission. Heart and Stroke Foundation of Canada: Cardiovascular Disease in Canada. Ottawa, Canada, 1993. Figure 6, page 10.

Figure 5.2 Stamler J, Wentworth D, Neaton JD. Is the relationship between serum cholesterol and risk of premature death from coronary heart disease continuous and graded? Findings in 356,222 primary screenees of the Multiple Risk Factor Intervention Trial. *Journal of the American Heart Association* 256:2823-2828, 1986.

Figure 5.3 The Framingham Heart Study, National Heart Institute, United States Department of Health and Human Services.

Figure 5.4 The Framingham Heart Study, National Heart Institute, United States Department of Health and Human Services.

Figure 5.5 The Framingham Heart Study, National Heart Institute, United States Department of Health and Human Services.

Figure 5.6 The Framingham Heart Study, National Heart Institute, United States Department of Health and Human Services.

Figure 5.7 Powell KE, et al. Physical activity and the incidence of coronary heart disease and death. *Annual Review of Public Health* 1987; 8:253-287.

Figure 5.8 The Framingham Heart Study, National Heart Institute, United States Department of Health and Human Services.

Table 5.1 Stamler J, Wentworth D, Neaton JD. Is the Relationship Between Serum Cholesterol and Risk of Premature Death from Coronary Heart Disease Continuous and Graded? Findings in 356,222 Primary Screenees of the Multiple Risk Factor Intervention Trial. *JAMA* 1986, 256:2823-2828.

Table 5.2 National Cholesterol Education Program Expert Panel, American Medical Association.



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# Chapter 6

## PREVENTING HEART DISEASE

As was discussed in Chapter 2, multiple studies have demonstrated that fire fighters have an increased risk of death due to heart disease. A wide variety of occupational and non occupational factors contribute to this high incidence. From an occupational perspective, the fire fighting environment, particularly visible smoke plumes, oxygen deficiency, and chemical off gassing from fires, constitutes a significant health hazard. Smoke plumes contain a number of chemicals believed or demonstrated to promote or exacerbate heart disease. These substances include carbon monoxide, organic solvents, heavy metals, and other chemicals that may have significant adverse effects on the heart. Recognized standards for respirator use mandate the use of SCBA and other personal protective equipment when the environment is hazardous, is suspected of being hazardous, or may rapidly become hazardous. Thus, SCBA should be used at these times to reduce the harmful impact of fire fighting on the heart. The effectiveness of SCBA in preventing these exposures is well documented. In persons who have an adequate face seal and properly functioning equipment, the positive pressure environment of an SCBA effectively reduces exposure to zero. The second essential component of identifying and preventing occupation related heart disease is documentation. Fire departments must document member's specific chemical exposures when known or site specific chemical hazards when available.

There are a number of important lifestyle and health related factors that are of great importance in determining your risk of heart disease. These modifiable risk factors include: smoking, hypertension, high blood cholesterol (hypercholesterolemia), diabetes, and obesity. These risk factors become more important as we age, and heart disease becomes more common. While aging is not reversible, the lifestyle choices that we make are and can dramatically reduce the risk of atherosclerotic heart disease (ASCVD). If you have already been diagnosed with hypertension, diabetes, or elevated cholesterol, the treatment plan recommended by your physician, coupled with the choices that you make, provide a comprehensive strategy to reduce your risk of heart disease. These important choices include: tobacco use, exercise, weight control, and stress management. In this chapter we shall discuss ideas which will assist you in making and maintaining heart healthy choices.

## TOBACCO AND HEART DISEASE

Tobacco use is one of the most important risk factors for developing coronary artery disease. According to American Heart Association estimates, approximately 200,000 Americans die every year from coronary heart disease due to smoking.<sup>1</sup> While the harmful effects of smoking have been known for decades, recent evidence also indicates that the nicotine in chewing tobacco can affect blood pressure and other cardiovascular disease risk factors.<sup>2</sup>

Tobacco use, whether from smoking or chewing, has a harmful effect on the heart's performance. Acutely, nicotine prompts the release of the hormone adrenaline, increasing blood pressure, narrowing coronary arteries, and "whipping" the heart to beat faster and more forcefully. Furthermore, burning tobacco releases carbon monoxide which limits the oxygen supply to the heart. Studies have demonstrated that heart pain, called angina, and adverse electrocardiogram changes can be brought on in persons with pre-existing heart disease by the carbon monoxide released from burning cigarettes.<sup>3</sup> Carbon monoxide starves the heart muscle of vital oxygen. Chronically, tobacco can also cause elevated blood pressure and worsen hypercholesterolemia, artery damage, and peripheral vascular disease.

## SMOKING CESSATION

The good news is that a person who quits or reduces smoking can quickly reduce their chances of heart disease. Long term survival studies have clearly demonstrated this fact. Immediately after quitting, your risk of having a heart attack begins to decrease. It is reduced by 50% within one year of cessation, and approaches the risk of nonsmokers after two years.<sup>4</sup> This benefit occurs even in persons who are over 55 and have been smoking for many years.<sup>5</sup>

**CIGARETTES AND OTHER FORMS OF TOBACCO ARE ADDICTING.** Physically, nicotine's addictive nature is due to its stimulation of the brain and heart. Psychologically, cigarette use is reinforced by specific activities and social interaction. We may use cigarettes as a reward after persevering through stressful situations, or as a means to strike up a conversation. Given these facts, it is no surprise that so many people have been unsuccessful in the attempt to "kick the habit". In fact, most persons who quit for good are able to do so only after multiple attempts. Fortunately, there are millions of former smokers who have successfully beaten the habit, and can attest to the fact that their lives are just as happy and fulfilling, or even more so, without smoking.

In consideration of the ill health effects associated with tobacco use, the IAFF has proposed three main goals in its Wellness-Fitness Initiative Tobacco Cessation Policy:<sup>6</sup>

- All new fire department candidates shall be tobacco free upon appointment and throughout their length of service to the department.
- Current fire department uniformed personnel shall not use tobacco products inside the work-site, within or on fire department apparatus, or inside training facilities.
- A fire department sanctioned tobacco cessation program shall be made available to incumbent tobacco users. Tobacco cessation programs must be non-punitive and must include short and long term goals.

The Wellness-Fitness Initiative also describes examples of tobacco cessation programs.

The IAFF has also published "The IAFF Smoking Cessation Manual: Issues, Policies, and Programs" to assist you in your smoking cessation efforts. It outlines the most commonly used techniques, including aversive therapy, self-help and group programs, nicotine replacement (with "patches" or "gum"), acupuncture, and other methods. No single method is right for everyone. But, regardless of which method you choose, several decisions will increase your success rate.

### STEPS YOU CAN TAKE TODAY

- **Contact the IAFF for a copy of the smoking cessation manual.**
- **Consider using nicotine replacement therapy, such as "the patch" or "nicotine gum," regardless of which group or self-help method you choose. When used in a comprehensive program, nicotine replacement roughly doubles the success rate.**

If you desire additional information on individual programs or your department wishes to begin a smoking cessation program, contact the IAFF Department of Occupational Health and Safety.

As we have discussed, both diabetes and hypertension can greatly affect our risk of developing ASCVD. As blood pressure rises, the work load of the heart increases proportionately. This stress could place the heart at risk, especially if its blood supply is already compromised by ASCVD. Hypertension, if uncorrected, may also eventually damage the walls of the blood

## HIGH BLOOD PRESSURE AND DIABETES

vessels, and cause damage to the brain, kidneys, eyes, and heart. In persons with diabetes, high blood glucose levels can cause disease in small blood vessels and accelerate atherosclerosis, further damaging the cardiovascular system. Although genetic factors play a role in high blood pressure and diabetes, both conditions can be greatly affected by our choice of lifestyle.

The treatment of high blood pressure is often a two step process, involving both non-drug and drug therapy. Non drug therapy focuses on exercise, weight loss, reduced sodium intake, and moderation of alcohol use. In many cases, mild or borderline hypertension can be controlled by these interventions alone.<sup>7,8</sup> These steps are also effective in preventing high blood pressure from developing in the first place. Exercise independently lowers your risk of developing hypertension. Low to moderate intensity exercise, such as gardening, walking, or cycling, is effective in reducing blood pressure. It is most effective when done daily, but three times per week also offers a benefit. The weight loss that often accompanies increased activity is also very beneficial; for every kilogram (2.2 pounds) that you lose, your blood pressure declines by 1-2 points (millimeters of mercury).<sup>9</sup> Guidelines for healthy salt use will be discussed along with other nutritional factors later on in this chapter. Excessive alcohol consumption is probably the most common cause of reversible, curable hypertension. While studies have consistently demonstrated a small beneficial effect of moderate alcohol consumption on the risk of developing heart disease, drinking over 2 ounces a day can actually increase your blood pressure.<sup>10,11</sup> Generally, these simple steps should be tried for three to six months in persons with mild to moderate hypertension before drug therapy is begun.

Drug therapy is effective in reducing blood pressure 10% or more in most people.<sup>12</sup> Studies have repeatedly shown that a reduction of this magnitude will substantially reduce your risk of coronary artery disease. A wide variety of single drugs and combinations are available to individualize the treatment of hypertension; the side effect profiles of these drugs vary considerably, making it possible to find an acceptable and effective option for virtually everyone. While in many cases, drug therapy for hypertension is for a lifetime, by strictly adhering to the non-drug alternatives discussed above, you can lessen the amount of drug necessary to control your blood pressure and, in some cases, even eliminate your need for medication.

Like, hypertension, both the prevention and treatment of diabetes also include weight loss, changes in diet, and exercise. The effectiveness of these efforts has not been rigorously studied among diabetics. Should these efforts to control diabetes be insufficient, your doctor may recommend medication. It is important to take replacement insulin and other medications as prescribed and to immediately report any side effects to your physician. This medication prevents the life threatening complications of diabetes and may also reduce the risk of heart disease.

#### **STEPS YOU CAN TAKE TODAY**

- **Moderate alcohol use (<2 drinks per day)**
- **Daily, low to moderate intensity exercise (walking, cycling etc.)**
- **Eat less than 6 grams of salt per day (see nutrition section for advice)**
- **Maintain a stable, healthy body weight (see obesity section)**

## OBSESITY

About 32 million American adults are obese.<sup>13</sup> Excess body weight increases the work load on a heart that may be unprepared for the additional stress. Obesity also contributes to other ASCVD risk factors, such as high cholesterol, hypertension, and adult onset diabetes. For instance, the prevalence of hypertension is three times higher in obese persons than in those of normal weight.<sup>14</sup> The risk of developing arthritis, gall bladder disease, and many cancers is also increased in obese persons. The end result can be a shorter and less productive life.

How do you know if your current weight is unhealthy? Many organizations have attempted to answer this question and several widely used measures of obesity are currently in use. An older, but still widely used, definition is 20% or more above your ideal body weight range, which is established from charts such as the Metropolitan Life Insurance Company Table.<sup>15,16</sup> These tables define obesity based solely upon the weight where your health risks begin to increase. An increasingly common method uses the body mass index, or BMI. The BMI is calculated by dividing your weight in kilograms by the square of your height (in meters); above 27.8 in men or 27.3 in women is considered overweight.<sup>17</sup> The BMI is not an accurate predictor of obesity in very muscular people.

An ideal body weight can be achieved with an appropriate balance of caloric intake and expenditures. Unfortunately, many of us tend to overestimate the amount of calories we expend with physical activity. The best tip for weight loss is to avoid starvation or fad diets. Skipping meals leads to gorging and can create a vicious cycle. Drink 8 glasses of water per day and eat from all of the basic food groups: grains, vegetables, dairy, and meat. If a sensible diet is complemented by a moderate amount of physical activity, it will, in most cases, result in gradual weight loss without a rebound effect. Consult your physician for any questions or concerns that you may have about beginning a new diet.

## DIET

Hypercholesterolemia, obesity, hypertension, diabetes and gout can all be greatly influenced by our choice of diets. A healthy balance of caloric intake, fat, carbohydrate and salt consumption are crucial elements in our avoidance of heart disease. The *IAFF/IAFC Fire Service Joint Labor Management Wellness-Fitness Initiative* encourages continuous education of company officers and supervisors regarding healthier eating habits. Use of a nutritional counselor can help with analyzing individual diets, customizing diet prescriptions and developing specialized meals for nutritional replenishment after incidents.

## FAT

Fats are an essential part of our diet, providing the building blocks of many body components. However, according to the American Heart Association, the average American needs to reduce their fat consumption by about one half. On average, no more than 30% of our calories should come from any fat, saturated fats should be about 10% of calories, and polyunsaturated fats about 10% of total calories.<sup>18,19</sup> The reason is simple: the primary effect of high fat consumption is to raise blood cholesterol levels. By maintaining an "ideal" blood cholesterol level (less than 200 mg/dL), you may reduce your risk of heart attack by up to two thirds. And, if you already have hypercholesterolemia, studies have convincingly demonstrated that you can reduce your risk of having a heart attack by 2% for every 1% reduction in cholesterol levels.<sup>20</sup>

Fat itself is not unhealthy, but our choice of the quantity and kind of fats that we consume can affect our health. There are three main types of fats. Saturated fats come primarily from animals and tend to be the worst offenders in increasing blood cholesterol levels. Hydrogenated fats, fats which are thickened for use in products like cookies or margarine, probably act the same way. In contrast, the polyunsaturated fats, derived mostly from vegetable oils, tend to decrease cholesterol levels. Monounsaturated fats from peanut, canola, and olive oils tend not to greatly influence blood cholesterol levels. Exceptions include the coconut and palm oils, which tend to resemble the saturated animal fats, and the fish fats which the body treats more like vegetable oils. The fat composition of common spreads and cooking oils are presented in Figure 6.1 to help you make an educated choice.

The American Heart Association has also published guidelines for cholesterol consumption, recommending an upper limit of 300 mg/day from our diets.<sup>21</sup> Consumed cholesterol also raises our blood cholesterol levels, but not as substantially as the amount of fat that we eat. Figure 6.2 illustrates the cholesterol content of many common foods. How does your diet measure up?

**STEPS YOU CAN TAKE TODAY**

- **Consume no more than 30% of your daily calories from fat.**
- **Minimize cholesterol consumption to no more than 300 mg/day.**
- **Try to eat mostly vegetable oils, olive and peanut oil.**
- **Avoid coconut and palm oil.**

## Fat Composition of Oils

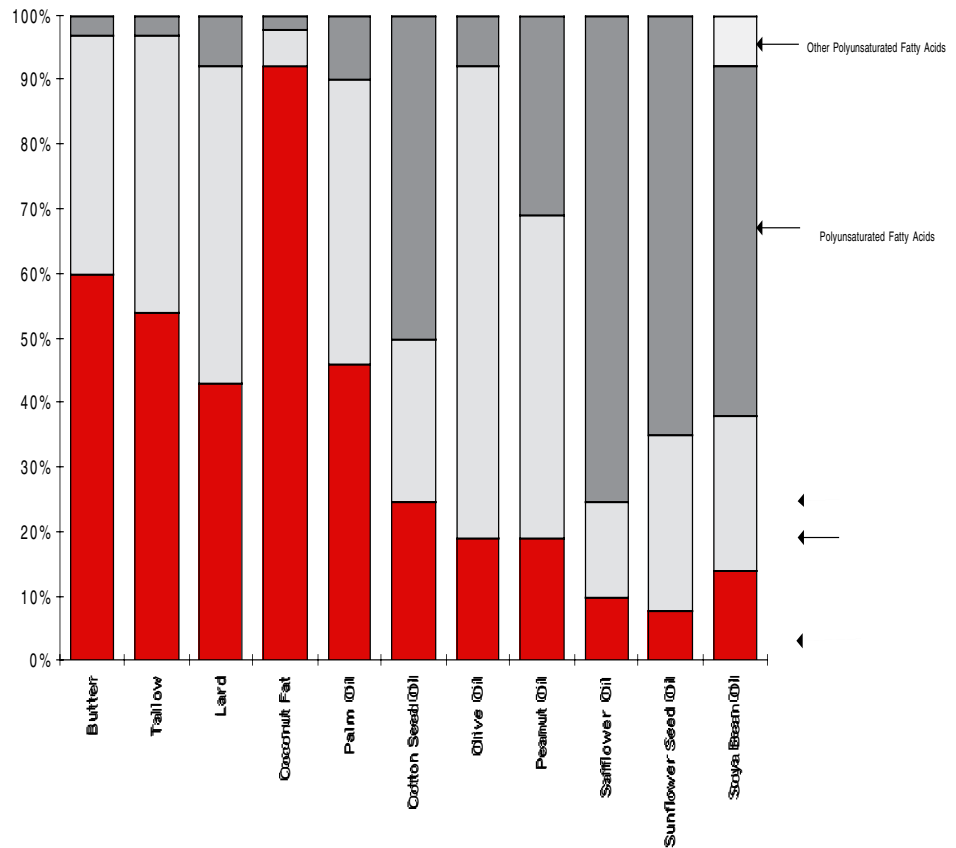


Figure 6.1 Fat composition of common cooking oils and spreads

### Cholesterol Composition of Common Foods (mg/100g)

<b>MEAT</b>		<b>FISH</b>		<b>MILK &amp; MILK PRODUCTS</b>	
Pork (extra lean)	70	Caviar	300	Whole Milk (3.5% fat)	10
Beef (extra lean)	70	Lobster	150	Skim Milk	3
Veal (extra lean)	90	Oysters	150	Cream Cheese	105
Lamb (extra lean)	70	Crab	150	Fat Cheese (45%)	90
Venison	110	Sole	50	Fat Cheese (30%)	57
		Red Perch	40	Low Fat Cheese (20%)	30
<b>INNER ORGANS</b>		Mackerel	35	Low Fat Cheese (10%)	12
Pork Liver	360	Cold Fish	30	Ice Cream	45
Beef Kidney	350				
Calf Liver	420	<b>FAT</b>		<b>EGGS</b>	
Brain	3,100	Butter	280	1 Egg	280
		Pork/Animal Fat	100	Egg White	0
<b>CHICKEN</b>	75	Vegetable Margarine	0	Egg Yolk (100 g)	1400

Figure 6.2 Cholesterol Composition of Common Foods

## SODIUM CHLORIDE

Sodium chloride, commonly known as table salt, is used extensively in processed, canned, and fast food. Excess salt contributes to high blood pressure, water retention, and weight gain. The average daily consumption of most Americans greatly exceeds requirements. In developed countries like the U.S., the average daily intake of salt is about 12.5 grams per day, but even a profusely sweating athlete needs only about 5 grams of salt each day to replace losses.<sup>22</sup> The American Heart Association recommends that the average person limit their salt intake to 3 grams, or about 1 to 1 1/2 teaspoons per day.<sup>23</sup> This goal can easily be exceeded by minimizing the amount of processed, pre-prepared and fast foods we eat, and avoiding added salt during cooking. Finally, our taste for salt depends upon the amount we consume. If we reduce our intake, within one to two months the body loses its craving for salt and we prefer less of it in our food

### STEPS YOU CAN TAKE TODAY

**Keep salt intake to 3 grams per day.**

## SUGAR

The American Heart Association recommends that foods high in complex carbohydrates, such as bread, pasta, vegetables, and fruits, should provide 45-55% of our daily calories. Simple sugars, like table sugar, corn sweeteners, and honey, should be a smaller part of the diet. Complex carbohydrates are slowly released into our blood stream providing uniform levels of blood sugar and have a lower caloric density (lower number of calories per ounce of food) than other nutritional sources. Thus, the rapid swings in blood sugar, which can promote binge eating, can be avoided. All of these factors combine to make a diet high in complex carbohydrates an essential component of the lifetime goal of a healthful body weight. Reduced intake of refined sugars may also lower the risk of dental caries.

## A SENSIBLE DIET

Keeping in mind the dietary facts outlined above, the question remains how do we incorporate this into creating a healthy and tasty diet? Fortunately there are a number of good books available in bookstores that provide specific advice on appropriate meals and also provide delicious, low fat recipes. A good start, however, is the American Heart Association Diet, "An Eating Plan for Healthy Americans" (See Figure 6.3). Details from this plan are outlined below.

## **GENERAL TIPS FOR A HEALTHY DIET**

To provide well balanced nutrition and control the amount and kind of fat, saturated fatty acids, and dietary cholesterol you should:

- Eat no more than 6 ounces (cooked) per day of lean meat, fish, and skinless poultry.
- Try main dishes featuring pasta, rice, beans and/or vegetables or create “low meat” dishes by mixing these foods with small amounts of lean meat or fish.
- Limit fat and oil consumption to 5 to 8 teaspoon servings in cooking and baking and in salad dressings and spreads.
- Use cooking methods that require little or no fat - boil, broil, bake, roast, poach, steam, sauté, stir-fry or microwave.
- Trim off the fat you can see before eating meat and poultry. Drain off all fat after browning. Chill soups and stews after cooking so you can remove the hardened fat from the top.
- The 3 to 4 egg yolks per week included in your eating plan may be used alone or in cooking and baking (including store bought products).
- Limit your use of organ meats such as liver, brains, chitterlings, kidney, heart, gizzard, sweetbreads, and pork maws.
- Choose skim or 1% fat milk and non-fat or low-fat yogurt and cheeses.
- Eat 5 or more servings of fruits or vegetables per day.
- Eat 6 or more servings of breads, cereals, or grains per day.

## **PHYSICAL ACTIVITY**

As a fire fighter, it is essential that you maintain a high level of physical fitness in order to perform your job well. Regular aerobic exercise also acts as the “glue” that ties together the elements that optimize our chance of avoiding heart disease.<sup>25</sup> As mentioned earlier, a program of regular aerobic exercise can help maintain normal body weight, blood pressure, cholesterol, and blood sugar, and improve cardiovascular fitness. In fact, a recent analysis demonstrated that inactive persons have a 90% higher risk of heart attack than physically active persons.<sup>26</sup>

Cardiovascular fitness can be achieved with the initiation of a regular exercise program. Your choice of exercise will depend on your current level of fitness, whether you like to work out alone or prefer group activities, and whether you prefer to exercise indoors or outside. Age also plays a role. Consult your physician before beginning your exercise program if:

- You are over 40 years old and have not been physically active;
- You experience any unpleasant symptoms during exertion such as chest pain, dizziness, or marked breathlessness with mild exertion;
- You have any chronic illness or take any daily medications.

Taking all factors into account, however, the benefits of regular exercise far exceed the risks. This is particularly true for a profession such as fire fighting, where job performance and safety may be dramatically affected by the physical conditioning of the fire fighter.

<b>Do Condition Heart &amp; Lungs</b>	<b>Can Condition Heart &amp; Lungs</b>	<b>Do Not Condition Heart &amp; Lungs Much</b>
Aerobic Dancing	Downhill Skiing	Badminton
Bicycling	Basketball	Baseball
Cross-Country Skiing	Field Hockey	Bowling
Hiking (uphill)	Calisthenics	Croquet
Ice Hockey	Handball	Football
Jogging	Racquetball	Gardening
Jumping Rope	Soccer	Golf
Rowing	Squash	Housework
Running in Place	Tennis	Ping-pong
Stair-climbing	Volleyball	Shuffleboard
Stationary Cycling	Walking Moderately	Social Dancing
Swimming		Softball
Walking Briskly		Walking Leisurely

© American Heart Association

Figure 6.3 Exercise Choices for a Healthy Heart

# Diet Planning

## Meat, Poultry and Fish

High in Protein, B Vitamins and Iron and Other Minerals

Servings per day: No more than 6 oz. cooked lean meat, poultry and fish.

Serving size: 3 oz. cooked (4 oz. raw) lean meat, poultry or fish.

Here are some good examples to help you judge serving sizes of meat, poultry and fish. A 3 ounce portion equals:

- the size of a deck of playing cards
- 2 thin slices of lean roast beef (each slice 3" x 3" x 1/4")
- 1/2 of a chicken breast or a chicken leg with thigh (without skin)
- 3/4 cup of flaked fish

Choose from:

- Fish; fresh, frozen, canned in water (or rinsed)
- Shellfish
- Chicken (without skin)
- Cornish hen (without skin)
- Turkey (without skin)
- Turkey, ground
- Lean beef (from the round, sirloin, loin)
- Lean or extra lean ground beef
- Lean ham
- Lean pork (tenderloin, loin chop)
- Lamb (except rib)
- Veal (except commercially ground)
- Wild game; rabbit, venison pheasant, duck (without skin)

Tips:

- Shrimp and crayfish are higher in cholesterol than most other types of fish, but lower in fat and saturated fatty acids than most meats and poultry.
- Buy "choice" or "select" grades of beef rather than "prime."
- Ham and Canadian bacon are higher in sodium than other meats.
- Domesticated versions of game (duck and goose) are not as lean as wild game
- Organ meats are very high in cholesterol. However, liver is rich in iron and vitamins and a small serving (3 ounces) is okay about once a month.
- Trim off all the fat before cooking meat.
- Drain or skim off fat from cooked meats before using juices in stews, soups, gravies etc.
- Remove the skin and fat under the skin from poultry pieces before cooking. If you're roasting a whole chicken or turkey, leave the skin on to keep the bird from getting too dry while roasting. Then remove the skin before carving and serving the meat.
- Select whole turkeys that have not been injected with fats or broths.
- Frozen dinners and entrees may also fit into the plan. Look for those that are made specially for low-fat, low-cholesterol, low-sodium diets.
- One cup serving of cooked beans peas or lentils or 3 ounces of soybean curd (tofu) can replace a 3 ounce serving of meat poultry or fish.



## Eggs

High in Protein, B Vitamins, and Iron and Other Minerals

Servings per week: 3 to 4 egg yolks a week may be eaten (egg whites are not limited).

Tips:

- Because of their cholesterol content (213 mg per yolk), limit your whole eggs and egg yolks to no more than 3 to 4 per week. Be sure to count any egg yolks used in cooking and in store-bought foods in your total for the week.
- Use two egg whites, or one egg white plus 2 teaspoons of unsaturated oil, in place of one whole egg in cooking. You can also use cholesterol-free commercial egg substitutes.
- Eat only cooked eggs and egg whites—not raw.



## Vegetables and Fruits

High in Vitamins, Minerals and Fiber, Low in Fat, Calories and Sodium. Contain no Cholesterol

Servings per day: 5 or more fruits or vegetables.

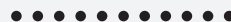
Serving size 1 medium size piece of fruit or 1/2 cup fruit juice. 1/2 - 1 cup cooked or raw vegetables.

Choose from:

- All vegetables and fruits except coconut. Olives and avocados should be counted as fats (see Fats and Oils section). Starchy vegetables are listed with Breads, Cereals, Pasta and Starchy Vegetables because they are similar in calories per serving to the other foods in that group.

Tips:

- Enjoy plenty of fruits and vegetables. If you are watching your weight, these foods will give you vitamins, minerals and fiber with few calories. Be sure to include sources rich in vitamin C and vitamin A.
- Check the labels for sodium content of canned vegetables.



## Milk Products

High in Protein, Calcium, Phosphorus, Niacin, Riboflavin, Vitamins A and D

Servings per day: 2 or more for adults over 24 years and children 2 - 10 years  
3 - 4 for ages 11 - 24 and women who are pregnant or breast feeding.

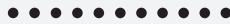
Serving size: 1 cup skim, 1/2% or 1% fat milk  
1 cup nonfat or low-fat yogurt  
1 oz. low-fat cheese or 1/2 cup low-fat cottage cheese

Choose from:

- Milk products with 0-1% fat, including: skim milk; 1/2-1% fat milk; nonfat or low-fat dry milk powder; evaporated skim milk; buttermilk made from skim or 1% fat milk; nonfat or low-fat yogurt drinks made with skim or 1% fat milk and cocoa (or other low-fat drink powders)
- Low-fat cheeses, including: dry-curd; skim or low-fat cottage cheese; natural or processed cheeses with no more than 5 grams of fat per ounce

Tips:

- Skim, 1/2% fat and 1% fat milk all provide the same nutrients as whole milk and 2% fat milk. But they are much lower in fat, saturated fatty acids, cholesterol and calories.
- If you're used to whole milk products, you may find it easier to make the change slowly to lower fat foods. Try 2% fat milk first. Then when you're used to that, move to 1% fat milk. That will make it much easier if you decide to change to skim milk.



## Snacks

Choose from:

- *Choose snacks from other food groups:* Fruits and juices; Raw vegetables and low-fat dips; Low-fat cookies; Low-fat crackers; Plain unsalted popcorn; Unsalted pretzels; Hard candy or gum drops; Sugar, syrup, honey, jam, jelly, or marmalade (as spreads).



## Beverages

Choose from:

- First Choices: Fruit or vegetable juice; coffee; tea; plain or flavored mineral water; low-sodium broth; and low-sodium bouillon.
- Other Choices: Fruit punches; carbonated soft drinks;
- Alcoholic beverages—If you drink them, do so in moderation. Have no more than two drinks per day of wine, beer or liquor, and only when caloric limits allow. The amounts to count as *one* drink (1/2 ounce pure alcohol) are 12 oz. Beer; 1 1/2 oz. 80 proof spirits (bourbon, gin, rum, Scotch, tequila, vodka, whiskey); 1 oz. 100 proof spirits; 4 oz. Wine (red, white, rose)



## Fats and Oils

Some of these foods are high in Vitamins A or E, but all are high in fat and calories.

Servings per day: No more than a total of 5 - 8 depending on your caloric needs

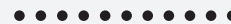
Serving size: 1 tsp. vegetable oil or regular margarine  
2 tsp. diet margarine  
1 Tbsp. salad dressing  
2 tsp. mayonnaise or peanut butter  
3 tsp. seeds or nuts  
1/8 of medium avocado  
10 small or 5 large olives

Choose from:

- Vegetable oils and margarines with no more than 2 grams of saturated fatty acids per tablespoon including, canola; corn; olive; safflower; sesame; soybean; sunflower.
- Salad dressings and mayonnaise with no more than 1 gram of saturated fatty acids per tablespoon.

Tips:

- Use fats and oils sparingly—and use the ones lowest in saturated fatty acids and cholesterol.
- Use hydrogenated shortenings sparingly and choose those made from vegetable fat. They are lower in saturated fatty acids than those made from animal/vegetable fat blends.
- Use cooking styles that add little or no fat to food, and ask for them when eating out.
- Remember to count the “hidden fat” in bakery and snack foods as well as the fats used in cooking and on vegetables and breads.
- Remember that although coconut oil, palm oil and palm kernel oil are vegetable oils and have no cholesterol, they are high in saturated fatty acids. *Read food labels carefully.*



## Desserts

Choose from:

- Desserts low in saturated fatty acids, cholesterol and calories. For a special treat, share a dessert portion with someone.
- *First Choices* (low in fat and saturated fatty acids): Fruit—fresh, frozen, canned or dried Low-fat yogurt with fruit Crackers and cookies (as listed in the Breads section); Angel food cake; Frozen low-fat or nonfat yogurt; Sherbet or ice milk; Flavored gelatin; Water ices or sorbets.
- *Special Occasions Only* (higher in fat and calories): Homemade desserts (cakes, pies, cookies, puddings)—made with margarine or oils low in saturated fatty acids; skim or 1% fat milk, and egg whites or egg substitutes (or egg yolks within limits). Store-bought desserts are now made with unsaturated oils and are either low-fat or nonfat. *Be sure to read ingredient lists.*



## Breads, Cereals, Pasta and Starchy Vegetables

Low in Fat and Cholesterol; High in B Vitamins, Iron and Fiber

Servings per day: 6 or more.

Serving size: 1 slice of bread  
 1/4 cup nugget or bud-type cereal  
 1/2 cup hot cereal  
 1 cup flaked cereal  
 1 cup cooked rice or pasta  
 1/4 - 1/2 cup starchy vegetables  
 1 cup low-fat soup

Choose from:

- Breads and rolls, including wheat, rye, raisin or white bread; English muffins; frankfurter and hamburger buns; water (not egg) bagels; pita bread; tortillas (not fried)
- Crackers and snacks, including animal, graham, rye, soda, saltine, oyster crackers; matzo; fig bar; ginger snap; molasses cookies; bread sticks; melba toast; rusks and flat bread; pretzels (unsalted); popcorn (see "Fats and Oils" for preparation)
- Quick breads, including homemade using margarine or oils low in saturated fatty acids; skim or 1% fat milk, and egg whites or egg substitutes (or egg yolks within limits); biscuits; muffins; cornbread; fruit breads; soft rolls; pancakes; French toast; waffles
- Hot or cold cereals, all kinds (granola-type may be high in fat or saturated fatty acids)
- Rice and pasta, all kinds (pasta made without egg yolk)
- Starchy vegetables, including potatoes; corn; lima beans; green peas; winter squash; yams; sweet potatoes
- Soups, including chicken noodle; tomato-based seafood; chowders; minestrone; onion; split pea

Tips:

- Many kinds of crackers and snacks are now available with no added salt or unsalted tops. Some are high in saturated fatty acids, so read the labels.
- If you use any egg yolks in cooking quick breads, be sure to count them in your daily allowance.
- Cereals, pasta and rice cooked without salt are lower in sodium than instant or ready-to-eat types of these foods.
- Most soups are high in sodium and some are high in fat. When buying soups, read labels and choose those low in sodium and fat. You can also make your own soups and control both sodium and fat.



### For More Information

For help in changing your recipes to fit this plan, you may find the *American Heart Association Cookbook, Fifth Edition* useful. More tips for **dining out and preparing food** are in the *American Heart Association Low-Fat, Low-Cholesterol Cookbook*. You'll find low-fat recipes that are also low in sodium in the *American Heart Association Low-Salt Cookbook*. All of these are available in your local bookstores.

The *American Heart Association Fat and Cholesterol Counter* lists the fat, saturated fatty acid, cholesterol, sodium and calorie content of more than 450 common foods. You can find it in bookstores and grocery stores. For more information about nutrition, diet and heart disease, contact your nearest American Heart Association. Or call toll-free 800-AHA-USA1 [1-800- 242-8721].

© American Heart Association

Figure 6.4 Planning your Diet

The exact frequency and intensity of exercise necessary to be “protective” against heart disease is not known, however, in the past it has been recommended that aerobic exercise be done for 30 minutes at least three times weekly. To exercise aerobically, you have to train within a narrow band called the target heart rate, which is defined as 60-80% of your maximum heart rate. Your maximum heart rate can be calculated by subtracting your age from 220. For example, if you are 40 years old, then your maximum heart rate is 180 and your target heart rate is between 98 and 144 beats per minute. After you have been exercising regularly and feel physically fit, advancing to 85% of your maximum heart rate is appropriate.

**PSYCHOLOGICAL  
STRESS**

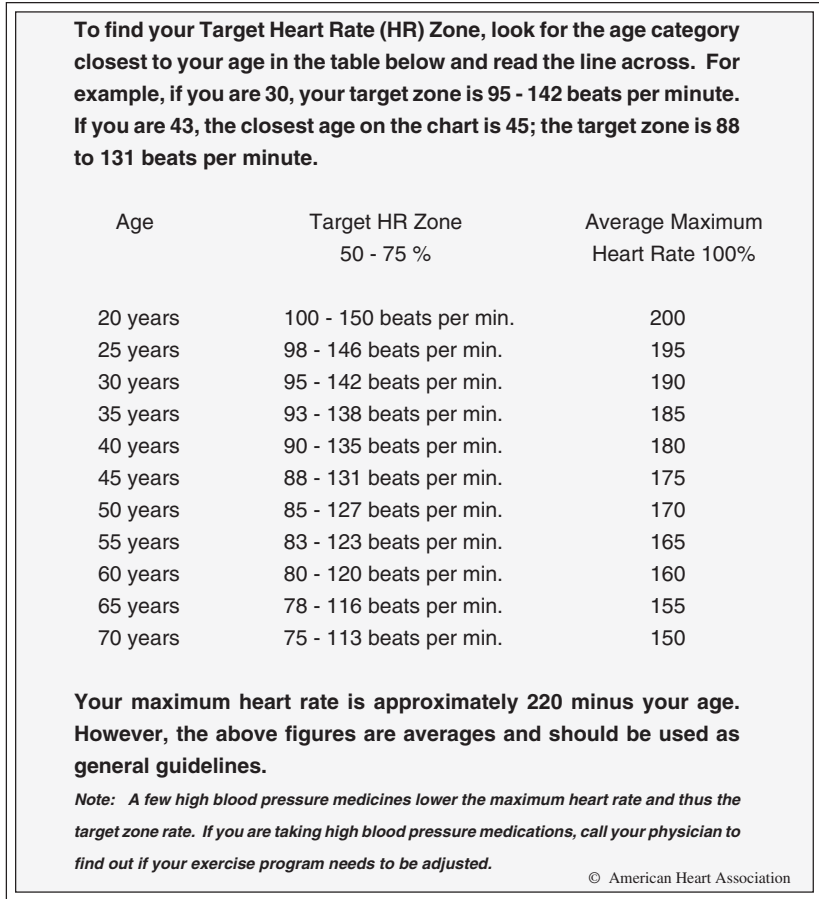


Figure 6.5 Target Heart Rate

Recent evidence also suggests that even less intense exercise can reduce the risk of heart disease. Daily activities that expend as little as 150-300 kcal, such as walking or gardening, are also associated with a significantly lower risk of ASCVD. The bottom line is: get some exercise every day.

Stress is unavoidable in daily life. It motivates us to excel and to survive. For example, the stress from responding to a fire may help enhance our performance at the scene. But, too much stress can overwhelm us, lead to marital strife, and can leave us feeling helpless or inadequate. Personal or occupational stressors may also lead to behavior which is unhealthy, such as physical inactivity, over eating, smoking, or excessive alcohol intake. Although not considered to be a major risk factor, psychological stress has been linked to an increased risk of developing ASCVD by some studies.<sup>27</sup> Classically, this increased risk is associated with the “Type A” personality. These individuals have a strong sense of time urgency (trying to accomplish more and more in less and less time) and are aggressive, ambitious, competitive, impatient, and frequently frustrated.

There are a number of strategies that can help us cope effectively with stress. Recognition of our internal stress level is the first step in the maintenance of a healthy “stress equilibrium”. The demands of contemporary society may lead us to shoulder more burdens than is healthy. At times we are tempted to try and meet our perceived needs, or those of our family, by shouldering additional responsibilities such as a second job. Relaxation is relegated to the status of a luxury that we cannot afford, rather than an essential component of a healthy and productive life. Appropriate help seeking and information gathering is another important

## WELLNESS AT WORK

technique. Obtain information or help with issues which worry you, such as a troublesome tax question or a new physical ailment. Share these problems and possible solutions with friends and family. Seeking appropriate professional advice can also help to solve the problem and help to avoid much unnecessary anxiety.<sup>28</sup> Identify which stress factors in your environment can be eliminated, changed, or rearranged. Learn constructive and positive self statements to help you recognize and utilize your strengths.<sup>29</sup> Overall, although we do not currently understand the role that stress plays in the development of heart disease, we believe that the common ways to reduce stress, such as a sensible exercise program, can help reduce heart disease risk.

The maintenance of good health is essential to the job of fire fighting. As such, there has been a growth of programs sponsored by fire departments to promote cardiovascular fitness. However, programs that are limited to episodic fitness tests may actually cause more harm by requiring fire fighters to abruptly meet test demands without adequate preparation. The IAFF, through formal policy, recommends that wellness and fitness programs emphasize the general health benefits to the fire fighter as well as benefits to the fire department.

“The IAFF will provide the necessary guidance and assistance in implementing physical fitness programs for any local and affiliate which requests such assistance. Such programs may be mandatory; however, agreement to initiate it must be mutual between the administration and its members represented by the local union. Any program of physical fitness must be at a minimum positive and not punitive in design; allow for age and position in the department; allow for on-duty time participation utilizing facilities provided or arranged by the department; provide for rehabilitation and remedial support for those in need; and be reasonable and equitable to all participants.”

The *IAFF/IAFC Fire Service Joint Labor Management Wellness/Fitness Initiative* is a comprehensive program that addresses the needs of the total individual in a program to build and maintain fit fire fighters and EMS personnel. Fitness—physical, mental, and emotional—requires an effective wellness program available to recruits, incumbents, and retirees. Components of the Wellness/Fitness Initiative include medical evaluation, fitness, injury/fitness/medical rehabilitation, behavioral health, and data collection.

The *IAFF/IAFC Fire Service Joint Labor Management Wellness/Fitness Initiative*, once implemented, should realize significant cost savings in lost work time, workers compensation, and disability. In addition, through data collection and analysis, participating departments will create an invaluable database suitable for comparisons throughout the fire service. The *IAFF/IAFC Fire Service Joint Labor Management Wellness/Fitness Initiative* is a complete physical fitness and wellness program package that seeks to maintain fit, healthy, and capable fire fighters and EMS responders throughout their career. The developed program includes a manual, video, and data collection protocol. If you or your department has an interest in beginning a program at your work place, or improving an already existing one, further information is available from the IAFF.

## SUMMARY

In conclusion, there is much you can do to reduce your risk of developing coronary artery disease. The common denominator is prevention. Eat sensibly, avoid high fat, high salt, and processed foods; avoid smoking and excessive alcohol intake; and maintain a moderate exercise program. Most importantly, wear proper personal protective gear to minimize the potential for harmful exposures. These steps, along with periodic health maintenance visits with your physician, may add years to your life and prevent years of disability. The mortality from heart disease has finally begun to decline due to many factors including the growing awareness in our population of how to stay healthy. By following these simple recommendations, thousands of additional deaths can be prevented each year. You too, can be in the group with lower risk for developing heart disease. If you have any questions, or would like to start a heart disease prevention program at your local, contact the IAFF Department of Occupational Health and Safety for advice and additional educational material.

## END NOTES

- <sup>1</sup> Orleans CS, Shipley RH. Take Heart, There Are Ways to Quit Smoking. The Heart Book, Delair Publishing Company, 1981.
- <sup>2</sup> Siegel D, Benowitz N, Ernster VL, et. al. Smokeless Tobacco, Cardiovascular Risk Factors, and Nicotine and Cotinine Levels in Professional Baseball Players. *Am J Pub Health* 1992; 82:417-421.
- <sup>3</sup> Heyden S, Stucky B. Cholesterol, in *The Heart Book*, Delair Publishing Company New York, New York, 1981.
- <sup>4</sup> Report of the Surgeon General: The Health Consequences of Smoking: Cardiovascular Disease. Rockville, MD : US. Department of Health and Human Services, Public Health Service, Office on Smoking and Health, 1983.
- <sup>5</sup> Hermanson B, Omenn GS, Kronmal RA, et al. Beneficial Six Year Outcome of Smoking Cessation in Older Men and Women with Coronary Artery Disease: Results From the CASS Registry. *N Eng J Med* 1988; 319:1365-9.
- <sup>6</sup> The Fire Service Joint Labor Management Wellness-Fitness Initiative, 2nd Ed. 2000, International Association of Fire Fighters.
- <sup>7</sup> Ibid, #5.
- <sup>8</sup> Stamler J, Stamler R. Intervention for the Prevention and Control of Hypertension and Atherosclerotic Diseases: United States and international experience. *Am J Med.* 1984; 76:13-36.
- <sup>9</sup> Kaplan NM. Arterial Hypertension. In: *Internal Medicine*. Ed: JH Stein. Boston, MA: Little, Brown, and Company, 1990, p. 246.
- <sup>10</sup> Ibid, #8.
- <sup>11</sup> Nonpharmacologic Approaches to the Control of High Blood Pressure. Final Report of the Subcommittee on Nonpharmacologic Therapy of the 1984 Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension* 1986; 8:444-67.
- <sup>12</sup> Ibid, #8.
- <sup>13</sup> Department of Health and Human Services. Nutrition Monitoring in the United States. Washington, D.C.: Government Printing Office, 1986: 2,54, 59-62.
- <sup>14</sup> Van Itallie TB. Health Implications of Overweight and Obesity in the United States. *Ann Int Med* 1985; 103: 983-988.
- <sup>15</sup> Foster WR, Burton BT, eds. National Institutes of Health Consensus Conference: Health Implications of Obesity. *Ann Intern Med* 1985; 103:977-1077.
- <sup>16</sup> 1983 Metropolitan Height and Weight Tables. *Statistical Bulletin, Metropolitan Insurance Co* 1983; 64:3.
- <sup>17</sup> Ibid, #14.
- <sup>18</sup> American Heart Association. Controlling Your Risk Factors for Heart Attack, page 22.
- <sup>19</sup> Nestle M: Hypertension, in *Nutrition in Clinical Practice*, Jones Medical Publications, Greenbrae, California, p. 209, 1985.
- <sup>20</sup> The Lipid Research Clinics Coronary Primary Prevention Trial Results II. The Relationship of Reduction in Incidence of Coronary Heart Disease to Cholesterol Lowering. *JAMA* 1984; 251:365-74.
- <sup>21</sup> American Heart Association: The American Heart Association Diet: An Eating Plan for Healthy Americans.
- <sup>22</sup> Ibid, #18.
- <sup>23</sup> Ibid, #17.
- <sup>24</sup> Ibid, #17
- <sup>25</sup> Williams S: Regular Exercise, Cardiovascular Fitness, and the Prevention of Coronary Heart Disease, in *The Heart Book*. Siegfied Heyden, Editor. Delair Publishing Company, New York, New York, 1981.
- <sup>26</sup> Powell KE, Thompson PD, Caspersen CJ, Kendrick JS. Physical Activity and the Incidence of Coronary Heart Disease. *Ann Rev Pub Health* 1987; 8:253-287.
- <sup>27</sup> Haynes SG, Feinleib M, Kannel WB. The Relationship of Psychosocial Factors to Coronary Heart Disease in the Framingham Study. Eight year Incidence of Coronary Heart

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Disease. Am J Epidemiol. 1980; III: 37.

<sup>28</sup> J. Blumenthal. Stress, in The Heart Book Siegfied Heyden, Editor, Delair Publishing Company New York, New York, 1981.

<sup>29</sup> Ibid, #27.

## CREDITS

Figure 6.1 Reproduced with permission. Heyden, S.: The Heart Book. New York, NY: Delair Publishing Company, Inc., 1981.

Figure 6.2 Reproduced with permission. Heyden, S.: The Heart Book. New York, NY: Delair Publishing Company, Inc., 1981.

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# Chapter 7

## HEART DISEASE AND FIRE FIGHTER BENEFITS

## FIRE FIGHTER DEATH AND INJURY STATISTICS

For more than forty years, the International Association of Fire Fighters has been addressing the issues of fire fighters and cardiovascular disease. The IAFF has protected its members by pursuing enactment of legislation that provides protection and compensation for those fire fighters whose health has deteriorated through the performance of their fire fighting occupation. Such IAFF sponsored benefit laws have ranged from federal legislation, to provide compensation for the families of those fire fighters who die or are severely disabled in the line-of-duty, to state and provincial legislation extending retirement and/or worker compensation benefits to those who become disabled from occupationally contracted diseases.

The profession of fire fighting is and has always been a hazardous occupation. Each year the IAFF publishes an annual *Death and Injury Survey*, and each year the hazards of fire fighting continue to exist and display ever varied forms. During the ten year period, 1987-1996, the D & I survey has found that professional fire fighters experienced 347 line-of-duty deaths, 537 occupational disease deaths, 336,678 injuries and 6,170 forced retirements due to occupationally induced diseases or injuries. Fire fighter line-of-duty fatalities have ranked fire fighting among other publicized hazardous occupations in the private sector, such as mining and construction.

Of the injuries reported, approximately 80 per cent occur while at the emergency scene. Sprains and strains are the leading cause of on-duty injury followed by lacerations and contusions, burns, inhalation of hazardous materials, and eye injuries. The data showed that more than 40% of all fire fighters can be expected to be injured at least once during the course of the year. Occupational diseases such as heart disease and cancer constitute more than 90 % of all reported fire fighter deaths when their occurrences are combined. As shown in Table 7.1, fire fighter fatalities from heart attacks are significantly above those experienced by other occupations.

<b>Occupational Fatalities Due to Heart Attack</b>	
<b>Occupation</b>	<b>Deaths per 100,000 Workers</b>
<b>All Private Industry</b>	<b>0.43</b>
<b>Construction</b>	<b>2.06</b>
<b>Transportation</b>	<b>1.20</b>
<b>Agriculture</b>	<b>.48</b>
<b>Wholesale/Retail Trade</b>	<b>.27</b>
<b>Services</b>	<b>.25</b>
<b>Manufacturing</b>	<b>.20</b>
<b>Fire Fighting</b>	<b>5.11</b>

Table 7.1

## FIRE FIGHTING ENVIRONMENT

These figures only roughly scratch the surface when it comes to detailing the hazards of fire fighting. The nature of the job is so varied and extensive, often people do not truly understand what a fire fighter's job is all about. Each day, fire fighters are physically and psychologically challenged on the job. Challenges exist as building fires, vehicle accidents, hazardous materials incidents, rescues, wildland fires, explosions, chemical exposures, extreme temperature environments, infectious disease exposures, occupational disease and cancer, fire ground accidents, environmental exposures, psychological stress, noise, fitness for duty, and personal time management.

Fire fighters are constantly making transitions from the calm, peaceful environment of the fire house to the hostility presented by fire. These constant transformations from quiet to raging infernos have numerous psychological and physiological side-effects. Within 15-30 seconds after the fire alarm sounds, research studies have found that a fire fighter's heart rate can increase by as much as 117 beats per minute. In addition, a fire fighter's heart can beat at twice its normal rate throughout the entire fire fighting operation. These extreme physiological stresses obviously lead to severe coronary problems, which have been documented by numerous authorities.

The working environment can also mean a transition from below freezing temperatures to temperatures between 100 degrees and 500 degrees Fahrenheit at the fire itself. These temperature extremes can lead to frostbite along with numerous cardiovascular and pulmonary disorders such as acute circulatory collapse, hypertension, pneumonia, and bronchitis.

Fire fighting involves strenuous physical activity that is made more burdensome by the protective clothing and breathing apparatus worn by the fire fighter. The equipment adds 45 to 65 pounds to his or her weight. Nevertheless, the fire fighter performs such vital activities as carrying heavy hose up flights of stairs, fighting water pressure to keep the hose directed at the flames, climbing onto rooftops carrying tools to ventilate the burning structure and so forth.

When a fire destroys a residential home or an industrial factory, it represents a work environment that closely resembles, at first glance, what fire fighters faced several hundred years ago. However, technology has created a distinct difference in the modern fire environment: benzene, polyvinyl chloride, asbestos, and polychlorinated biphenyls, to name a select few. These chemical compounds are commonplace ingredients in our environment as components of household furniture, plastic pipes, wall coverings, automobiles, buses, airplanes, and coverings for electrical and other insulation materials.

The potential of these chemical substances to cause bodily harm to the fire fighter is dependent upon many factors at the fire scene: the level of concentration, respiratory rate and volume, time of exposure, clothing worn and type of respiratory equipment used. Unlike even human skin, which can resist certain materials, the lungs lack any protection to resist these toxic substances. In addition, these toxic materials can be absorbed by a surface area in the lungs that is forty (40) times greater than the body's outside surface area.

The health effects can be short-term or even initially nonexistent. Such exposures can and do result in long-term illnesses involving the cardiovascular system, the respiratory system, the central nervous system and other body organs.

Our modern day society has contributed to many of the hazards fire fighters face. During the last two decades, the rapid proliferation of synthetic products entering the marketplace has added a new dimension to fire fighting. The proliferation of these products in our society has caught the scientific community off-guard. There are simply too many new chemicals and too few funds to research all of their potential hazards. Since the beginning of World War II, the production of synthetic chemicals has increased 350-fold. With the addition of thousands of new chemicals annually, it becomes impossible to study the carcinogenic properties of each and every one of them. The National Institute for Occupational Safety and Health (NIOSH)

## HEART DISEASE STUDIES

Registry of Toxic Effects of Chemical Substances contains more than 65,000 substances, which have been shown to produce some type of toxic effect on animals and/or humans. NIOSH estimates that there are approximately 100,000 substances that may have some type of toxic effect.

Fire fighters, like most workers, have little idea about the identity of many of the materials they are potentially exposed to or the hazards of such exposures. Nevertheless, fire fighters continue to respond to the scene and work immediately to save lives and reduce property damage without regard to the potential hazards that may exist. A fire emergency has no controls or occupational safety and health standards to reduce the effect of toxic chemicals. It is an uncontrollable environment that is fought by fire fighters using heavy, bulky and often times inadequate personal protective equipment and clothing. The effect is not only physically demanding, but involves exposure to air contaminants, some of which are known to cause diseases of the cardiovascular system.

The fire fighting profession illustrates the selective impact of past safety and health initiatives. Despite the advances made in safety and health areas, fire fighters are still being killed, injured and diseased at an alarming rate. Professional fire fighters have inordinate numbers of line-of-duty deaths, deaths due to occupational diseases, forced retirements, and line-of-duty injuries.

To review, exposures to hazards occur during fire fighting activities; victim rescue and body removal from structural and non-structural fires; during rescue and extrication from vehicles; while performing emergency medical duties as first responders, emergency medical technicians and paramedics; during hazardous material events; and during day-to-day activities involving fire fighters' peers and the general public. Fire fighters face the possibility of death or injury every time they respond to an alarm where they provide emergency assistance to the citizens of this country. While risk may be part of the profession, fire fighter deaths and injuries should not be part of the job. Life threatening and health hazards are numerous for fire fighters. The greatest effort should be made to eliminate such hazards.

Studies that link fire fighting with heart disease fall into three main groups--laboratory studies, field studies and epidemiological studies. The first, animal laboratory experiments have identified exposure to noise and certain chemicals (such as the common solvent carbon disulfide; carbon monoxide; arsenic; the common combustion by-products; polycyclic aromatic hydrocarbons; and elevated levels of the stress hormone, adrenalin) to contribute to the atherosclerotic process.

The second group, field studies, documents the exposure of fire fighters to these agents through industrial hygiene, biological, and physiological monitoring. Industrial hygiene data indicates that the fire environment contains a number of potentially dangerous toxins. Most frequent exposures, affecting the cardiovascular system, include carbon monoxide, poly aromatic hydrocarbons, cyanide, benzene, and hydrochloric acid. Arsenic as well as other toxic metals, organic solvents such as carbon disulfide, and many other toxins may also be present depending upon the products of combustion and conditions at the scene. Due to the highly unpredictable nature of the fire environment, it is almost impossible to predict with any certainty all of the exposures that could be encountered at any given fire. Blood testing of fire fighters has demonstrated elevated levels of carboxyhemoglobin, a biological marker for carbon monoxide exposure that exceed levels found in both the smoking and non-smoking population. Increased levels of urinary catecholamines (a metabolite of adrenalin) in fire fighters following fire operations, have demonstrated increased adrenalin levels. Electrocardiographic monitoring of fire fighters performing maximal exercise without the benefit of warm up time, a situation which mimics real conditions, suggests diminished oxygen supply to the heart during the initial stages of activity under these circumstances.

The third group, as addressed in Chapter 2, are epidemiologic studies of fire fighters and other occupational groups. This group of studies are performed to determine if exposures actually result in elevated rates of heart disease.

For example, the three epidemiologic studies of fire fighters in New Jersey, Connecticut, and Toronto, have demonstrated increased mortality rates from heart disease in comparison to the general population. However, there have also been a number of other epidemiologic studies that have not found an increased risk. This is due to a number of factors:

- Due to statistical constraints the number of individuals studied may not be sufficient to detect a difference;
- The studies rely on mortality, and measure only deaths from heart disease. Differences in survivorship between an occupational group and the general population resulting from disparities in the quality and accessibility of medical care or other factors, may result in misleading conclusions about disease prevalence;
- As mortality studies, the investigations rely upon death certificates which are frequently inaccurate and may erode the ability of the study to detect real differences;
- Due to the selection forces at the work place, occupational groups tend to be healthier than the general population with disease incidence significantly less than the general population. An increase in the prevalence of a medical condition arising from work place exposures may therefore-be missed with comparison to the general population. This “healthy worker effect” is accentuated with fire fighters who are extremely healthy, and has been termed the “super healthy worker effect”. This problem may be controlled by using another, similar occupational group as a control. This has been accomplished in a number of studies of fire fighters using policemen as a comparison group. This may not be appropriate for the evaluation of heart disease, however, since a number of studies have also demonstrated an elevated rate of heart disease in policemen as well as fire fighters;
- When studying an occupational group, certain sub-populations may be at greater risk for a disease due to differences in exposures, administrative policies, or other reasons. The ability of a study to identify and establish the increased rates in these subgroups may be limited due to statistical and study design constraints.

Any of these factors could result in an otherwise well designed epidemiologic study failing to find an increase in the prevalence of an illness even if one existed (i.e. a “false negative” result).

Due to the difficulty in conducting reliable industrial hygiene monitoring in the dangerous and unpredictable fire environment, epidemiologic studies linking carbon monoxide exposure to heart disease in fire fighters have not been practical. However, studies with New York City tunnel workers and motor vehicle examiners have demonstrated chronic exposure to carbon monoxide to be associated with increased rates of heart disease. Recent epidemiologic studies have also demonstrated an increased rate of heart disease in shift workers. Although not conclusive, the epidemiologic data presented above does demonstrate an association with the exposures encountered during fire fighting and an increased risk of atherosclerotic heart disease.

**IAFF POSITION ON  
HEART DISEASE**

**STATE PRESUMPTIVE  
LAWS**

Accordingly, it is the position of the International Association of Fire Fighters that cardiovascular disease is exacerbated by fire fighting duties and that fire fighting increases the incidence of cardiovascular disease in fire fighters. It has been well demonstrated that occupational heart disease is occupationally related to fire fighting. Fire fighters and emergency medial personnel face the possibility of death or injury every time they respond to an alarm where they provide emergency assistance. While risk may be part of the profession, fire fighter and emergency medial personnel deaths, injuries, and occupational diseases should not be part of the job.

In recognition of the causal relationship of the fire fighting occupation and heart disease, thirty-six states have adopted some type of presumptive heart law to afford protection to fire fighters with these conditions. These heart laws presume, in the case of fire fighters, that heart disease is occupationally related. Consequently, their provisions place the burden of proof to deny worker compensation and/or retirements benefits on the fire fighter's employer. Additionally, many workers' compensation boards in Canada and the United States have established a history of identifying heart and lung disease and cancer in fire fighters as employment-related. The states that have heart disease presumptive laws are identified in Table 7.2. While all these state laws recognize heart disease as occupationally related, some have exclusions and prerequisites for obtaining benefits. The IAFF maintains full copies of all state laws or regulations and a number of worker compensation awards from the United States and Canada that address heart disease. The IAFF also provides assistance and information to obtain or maintain heart legislation and regulations.

<b>STATES WITH PRESUMPTIVE HEART LAWS</b>	
Alabama	Nevada
California	New Hampshire
Connecticut	New Jersey
Florida	New York
Georgia	North Dakota
Hawaii	Ohio
Idaho	Oklahoma
Illinois	Oregon
Iowa	Pennsylvania
Kentucky	Rhode Island
Louisiana	South Carolina
Maine	South Dakota
Maryland	Tennessee
Massachusetts	Texas
Michigan	Utah
Minnesota	Vermont
Missouri	Virginia
Nebraska	Wisconsin

Table 7.2

**PUBLIC SAFETY  
OFFICERS BENEFITS**

The Public Safety Officers Benefits Act (Public Law 94-430) became law on September 29, 1976. The legislation provided for a \$50,000 death benefit for fire fighters (paid and volunteer) and law enforcement officers that died in the line-of-duty (emergency or non-emergency) from a traumatic injury. The Act does not cover deaths resulting from occupational illness or pulmonary or heart disease unless a traumatic injury is a substantial factor to the death.

The benefits are paid if toxicology reports demonstrate a carbon monoxide level to be 10 percent or greater (15 percent or greater for the smoker) at the onset of a heart attack. Additionally, the Public Safety Officers Benefits Program has developed a formula that addresses oxygen therapy provided to the victim prior to the death.

The Act did exclude federal fire fighters, however on October 12, 1984 the Act was amended to correct this exclusion. Likewise, on October 15, 1986 public sector EMS personnel were also amended into the coverage of the Act.

On November 11, 1988, the benefit was increased from \$50,000.00 to \$100,000.00 and made retroactive to June 1, 1988. The dependency test for parent(s) was eliminated. Additionally, it provided that on October 1, 1988 and every year thereafter, the benefit would be increased to reflect any increase in the consumer price index.

On November 29, 1990, Congress again amended the PSOB benefits program to include permanent and total disability. The amendment was tightly drawn, sharply limited in scope, and intended to cover only those public safety officers permanently unable to perform any gainful employment. The PSOB disability amendment recognizes that state, local and agency benefit programs are primarily responsible for the hundreds, perhaps thousands, of public safety officer disability pensions awarded each year. The PSOB Act is clear that benefits awarded are supplementary in nature and by law are not to offset any worker compensation payment or disability pension benefit.

Even where an officer is disabled by a severe, catastrophic injury received in the line of duty, PSOB's benefit does not come into play unless the injuries are so disabling as to permanently prevent any gainful employment. This standard recognizes that in all but rare cases a disabled public safety officer will have the capacity to supplement a state or local disability pension with employment earnings of varying degrees. It is clear that Congress intended the PSOB disability for the rare occasion where a public safety officer miraculously survives line of duty injuries that, except for modern medical technology, would have resulted in death. Such a survivor, however, is so severely handicapped with permanent disabilities that any type of gainful employment is simply not possible.

A detailed data sheet on the PSOB Program is provided in the Appendix. The following summarizes the legislative history and amendments to this legislation:

- ***Public Safety Officers' Benefits Act of 1976, Part J.*** Original act detailing the provision of death benefits. Introduced to the 94th Congress, September 29, 1976.
- ***Federal Register, May 6, 1977, Part II.*** The rules adopted by the Law Enforcement Assistance Administration for implementation of the PSOB Act are written.
- ***Federal Register, Vol. 43, No. 180 - September 15, 1978.*** Results of meeting detailing the contribution of carbon monoxide and heart disease in the deaths of public safety officers.
- ***Federal Register, Vol. 50, No. 128, July 3, 1985.*** An amendment to the PSOB Act which transfers the administration of the Act from the LEAA to the Bureau of Justice Assistance. In addition, federal public safety officers are now covered under the act and "gross negligence" and "intoxication" standards are defined within this amendment.

- ***Federal Register, Vol. 53, No. 50, March 15, 1988.*** Amendment to the PSOB Act which includes provision of death benefit coverage to members of public rescue squads or ambulance crews. Also an explanation of EMS coverage in correspondence from the U.S. Department of Justice.
- ***Federal Register, Vol. 57, No. 113, June 11, 1992.*** Amendment to the PSOB Act to include coverage for disability benefits. Such disability is defined as permanent and total as a direct result of a catastrophic personal injury sustained in the line of duty which will prevent an individual from performing any gainful work.

The following summarizes precedent court cases that affected the law:

- ***Beverly Morrow, et al. v. The United States.*** U.S. Court of Claims case in which a fire fighter's widow sought benefits following the death of her husband of a heart attack six weeks after an initial attack associated with a smoke inhalation incident. Benefits were denied as death was attributed to heart disease.
- ***Hubert Smykowski, et al. v. The United States.*** U.S. Court of Claims case in which a police officer died after a struggle with a suspect. Court upheld the Administrators ruling that the stress and strain of a struggle was not a traumatic injury as defined by the PSOB Act.
- ***Carrie Rose, etc. v. Arkansas State Police.*** The U.S. Supreme Court ruled that any law that authorizes a state to offset state worker compensation benefits against benefits paid by the Federal PSOB program is in conflict with the supplementary nature of the PSOB Act and is therefore invalid under the supremacy clause of the U.S. Constitution.

Finally, since 1991 the IAFF has been attempting to secure public safety officer benefits for Canadian fire fighters. The IAFF supports the creation of a federally funded PSOB in Canada which at this time has not been accepted at the federal level by any ministry. However, the establishment of such a fund is well within the purview of the federal government, and the IAFF continues to work towards implementation.

## CREDITS

Table 7.1 Annual Death and Injury Survey, International Association of Fire Fighters; and Occupational Injuries and Illnesses in the United States by Industry, US Bureau of Labor Statistics, US Department of Labor.

Table 7.2 International Association of Fire Fighters, Department of Occupational Health and Safety, 1994.



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# Glossary

**Angina (Angina Pectoris)** Pain originating in the heart muscle in response to a shortage of oxygen which is usually due to coronary artery disease. Angina can be brought on by exercise, excitement, or other occasions of increased heart activity and oxygen demand.

**Aorta** The major artery that is an outlet from the left ventricle through which oxygenated blood is pumped to the rest of the body.

**Aortic Valve** The three leaflet valve which regulates the blood flow from the left ventricle to the aorta. Closure of the aortic valve prevents back flow into the left ventricle from the aorta.

**Arrhythmia** An abnormal rate or rhythm of heart contraction. Fatal arrhythmias, such as ventricular fibrillation, are associated with sudden death and are more common in persons with coronary artery disease.

**Arteries** Muscular walled blood vessels that carry blood away from the heart.

**Atherosclerosis** A degenerative disease of the arteries caused by the deposition of cholesterol plaques on the innermost lining of the arteries, or endothelium.

**Atherosclerotic Coronary Vascular Disease (ASCVD)** Atherosclerotic disease of the arteries that supply blood to the heart muscle, or myocardium.

**Atrium** One of two thin walled heart chambers which pump blood to an associated ventricle.

**Atrioventricular Node (AV node)** A collection of specialized muscle cells located at the junction of the atria and ventricles which is responsible for relaying the electrical impulses from the atrial to the ventricular conduction system.

**AV Bundle** A specialized bundle of muscle cells that function as electrical pathways in the ventricles, coordinating electrical stimulation and contraction of the muscle.

**Blood Pressure** The pressure within the arteries generated by the heart's pumping; usually described with two numbers, the highest being the pressure that occurs during systole, and the lower number, the pressure during diastole.

**Capillary** The smallest diameter blood vessels which supply most of the body tissues. Red blood cells pass through in single file, which allows for oxygen exchange to occur through the thin walls of these blood vessels.

**Cardiac Catheterization** A heart imaging study for the diagnosis of coronary artery and other cardiac diseases. A small tube is inserted into an artery in the groin and advanced into the heart. Then a type of dye is injected, allowing visualization of the heart chambers and coronary arteries by x-ray.

**Cardiomyopathy** A disease of the heart muscle.

**Cardioversion** Electrical shock applied to the chest wall in an attempt to correct an abnormal heart rhythm.

**Cholesterol** A lipid complex formed within the liver from fats and protein. Cholesterol is used as the building block of beneficial substances, such as bile and Vitamin D, but it also plays a crucial role in the development of atherosclerosis.

**Coagulation** The clotting of blood. Abnormalities, such as an atherosclerotic plaque in the lining of the arteries (the endothelium), may generate favorable conditions for coagulation, leading to the formation of a blood clot within the artery.

**Conduction System** The network of specialized tissues within the heart responsible for the generation and transmission of the electrical impulses that allow for sequential stimulation and coordinated contraction of heart muscle. Components include the sinoatrial and atrioventricular nodes, the AV bundle, and Purkinje fibers.

**Coronary Arteries** The arteries that supply oxygenated blood to the heart muscle.

**Coronary Artery Disease (CAD)** Atherosclerotic disease of the coronary arteries.

**Chronic Obstructive Pulmonary Disease (COPD)** A chronic and permanent lung disease, usually caused by tobacco smoking. The two most common forms, chronic bronchitis and emphysema, result in a decreased ability to move air through the lungs. The increased work of breathing and lower concentration of blood oxygen that results from these conditions can cause the heart to work harder. Many persons with COPD also suffer from ASCVD as a result of smoking. Severe COPD may lead to enlargement of the right side of the heart and pulmonary hypertension.

**Diabetes Mellitus** A metabolic disease that results in abnormal elevation of blood glucose. Diabetes may be caused by deficiency in insulin production, or insensitivity of the body tissues to insulin. Diabetes is categorized as Type I (insulin dependent diabetes or IDDM), and Type II (non insulin dependent diabetes NIDDM).

**Diastole** The phase of heart activity characterized by atrial contraction, relaxation and filling of the ventricles and lower arterial blood pressure.

**Electrocardiogram** A painless study of the electrical activity of the heart which allows for the diagnosis of arrhythmias, ischemia, heart attacks, abnormalities in heart size and other conditions due to the characteristic changes in electrical patterns which occur in these circumstances.

**Endocarditis** Inflammation of the lining of the heart, or endocardium, usually caused by an infection.

**Endocardium** The innermost lining of the chambers of the heart.

**Endothelium** The layer of cells that line the interior of blood vessels.

**Epidemiology** The study of disease within populations.

**Epinephrine** Also called adrenaline. It is a potent hormone that results in numerous excitatory effects, including arterial constriction, increased blood pressure, and an increase in the work and oxygen requirements of the heart.

**Escape Beats** Heart beats that originate from a location other than the Sinoatrial node (SA node).

**Fibrillation** Rapid, uncoordinated, and ineffective electrical activity in a muscle making it incapable of well coordinated contraction.

**Gout** A metabolic condition characterized by elevation of uric acid, a by-product of cell degradation.

**Healthy Worker Effect** A phenomenon frequently observed in occupational epidemiologic studies. Due to the usual good health of workers, the baseline incidence of illness tends to be less than the general population. An elevated incidence of an illness created by work place conditions may not be sufficient to exceed the baseline rate in the general population, against which the work force is frequently compared. This may result in an underestimate of an occupational disease in the study population.

**Heart Attack** Permanent injury to heart muscle from oxygen deprivation; usually a result of coronary artery disease. See also myocardial infarction.

**Heart Block** Delays in the propagation of cardiac electrical impulses.

**Heart Failure** Deficient pumping of the heart due to heart injury, fluid overload, kidney malfunction or a combination of all of these.

**Heart Murmur** Heart sounds due to the turbulence from blood coursing through the heart, lungs, or blood vessels. In some cases, heart murmurs arise from the increased turbulence caused by pathological conditions, such as a stenotic or insufficient valve, or heart wall defect.

**Hemoglobin** An iron-based molecule which serves as the oxygen carrier for red blood cells.

**High Density Lipoprotein (HDL)** A form of cholesterol with a high fraction of protein, that is associated with lower incidence of coronary artery disease.

**Hypercholesterolemia** The presence of an abnormally elevated blood cholesterol.

**Hypertension** Elevated blood pressure, usually defined as a systolic blood pressure greater than 140 mm Hg , or diastolic blood pressure greater than 90 mm Hg.

**Hypertrophy** An increase in the size or bulk of an organ due to enlargement of cell size; frequently associated with an increased work burden of the affected body part.

**Incidence** The rate of new disease within a specified time period.

**Insufficiency** Incompetence. Frequently refers to a malfunctioning heart valve which allows back flow of blood into a chamber against the preferred direction of flow.

**Ischemia** Inadequate oxygen supply to an area of the heart.

**Left Heart Circulation** The flow of blood generated by the left atrium and ventricle which results in the transport of oxygenated blood to the remainder of the body (systemic circulation).

**Left Atrium** The thin walled chamber of the left side of the heart that receives blood from the lungs, and with its contraction, pumps blood through the mitral valve to the left ventricle.

**Left Ventricle** The thick walled chamber of the left side of the heart that receives blood from the left atrium, and with its contraction, pumps oxygenated blood to the systemic circulation.

**Lipid** A fat soluble substance; frequently refers to the fatty substances within the human body.

**Low Density Lipoprotein** A form of cholesterol with a low fraction of protein associated with a higher incidence of coronary artery disease.

**Mitral Valve** The valve that regulates blood flow between the left atrium and ventricle.

**Mitral Valve Prolapse** A condition characterized by an abnormal length of the mitral valve leaflets, which allows for a sinking back or prolapse of the valve into the left atrium during systole. This condition, which is more common in females, may be associated with a heart murmur and atypical chest pains, but rarely affects life expectancy.

**Mortality Studies** Epidemiologic studies used to assess the prevalence of specific diseases by comparing the causes of death in one population with those in another. The comparison population is usually obtained from the vital statistics of the general population. This approach is only helpful in studying those conditions which cause or contribute to death.

**Myocardial Infarction (MI)** Permanent injury to heart muscle from oxygen deprivation; usually a result of coronary artery disease.

**Myocardium** The muscular layer of the heart between the pericardium and endocardium. It is responsible for the pumping action of the heart.

**Nicotine** One of the many active ingredients in tobacco smoke. Nicotine is a physically addictive drug with stimulant properties. Nicotine stimulates the release of epinephrine, a powerful hormone which constricts blood vessels, raises blood pressure, and causes the heart to work harder, increasing cardiac oxygen requirements.

**Pacemaker** Any biologic rhythmic center that establishes a pace of activity. An artificial pacemaker is sometimes inserted for the treatment of rhythm disturbances that arise due to a malfunction of the heart's usual pacemaker, the sinoatrial node (SA node).

**Parasympathetic Nervous System** A division of the autonomic nervous system which exerts a modulating effect on the cardiovascular system.

**Pathogenesis** The origin or development of a disease or disease process.

**Pericardium** The outermost lining of the heart.

**Platelets** Irregularly shaped small cells in blood which aid in clotting. Platelets may also be responsible for the development of thrombi within the circulatory system.

**Prevalence** The rate of disease within a population. Prevalence includes both pre-existing and new cases of a disease.

**Pulmonary** Pertaining to the lungs.

**Pulmonary Artery** The artery arising from the right ventricle through which deoxygenated blood is pumped to the lungs.

**Pulmonic Valve** The three leaflet valve through which blood passes from the right ventricle to the pulmonary artery. Closure of the pulmonic valve prevents back flow from the artery to the right ventricle.

**Purkinje Fibers** Modified cardiac muscle cells responsible for the transmission of electrical impulses which allow for coordinated contraction of heart muscle.

**Red Blood Cell** A cell that carries hemoglobin and allows for oxygen exchange with body tissues.

**Right Heart Circulation** The circulation of the right side of the heart, comprised of the right atrium, right ventricle, and pulmonary arteries. Responsible for transporting de-oxygenated blood from the vascular system to the lungs for oxygenation.

**Right Atrium** The chamber of the right side of the heart that receives blood from the venous system and pumps it through the tricuspid valve to the right ventricle.

**Right Ventricle** The thick walled chamber of the right side of the heart that receives deoxygenated blood from the right atrium and pumps it through the pulmonic valve and pulmonary artery and ultimately to the lungs.

**Risk Factor** An identifiable trait or behavior pattern associated with a specific disease outcome.

**Sinoatrial Node (SA node)** Specialized heart tissue located at the entrance of the superior vena cava and right atrium which possess an intrinsic automaticity. The SA node serves as the origin of the normal cardiac impulse, and is the pacemaker for the electrical activity of the heart.

**Statistically Significant** A term used in biostatistics and epidemiology to denote that a certain set of findings is meaningful, and is unlikely to represent a chance or random event. This is usually accomplished by statistical tests which conventionally assign a value of 0.05 as the upper limit of probability that a study finding should achieve to be considered as significant. If the statistical test demonstrates that the probability of the finding is less than 1 in 20, say 1 in 200 (or 0.005), then the finding is presumed to be “statistically significant”, and probably not attributable to a random or chance event.

**Stenosis** A closing or narrowing of a passageway or channel sufficient to impede flow. This is a frequently used term in relation to heart valves such as the aortic valve, which may develop stenosis as a complication from a prior infection or congenital abnormality. The increase resistance to blood flow can overwork the heart, progressing to heart failure. The term may also apply to arteries which have become narrowed from atherosclerosis.

**Sudden Death** The sudden, unexpected death of an individual, usually from cardiovascular causes. The causative event may be sudden occlusion of a major coronary artery from a large thrombus, or an abnormal heart rhythm.

**Sympathetic Nervous System** A division of the autonomic nervous system which exerts an excitatory effect on the cardiovascular system.

**Systole** The portion of the heart activity cycle described by the contraction of the ventricles, which results in flow of blood to the pulmonic and systemic circulation. The atria are at rest during systole, and fill with blood from the venous and pulmonary circulation.

**Tachycardia** An unusually fast beating of the heart, defined as a rate of over 100 beats per minute.

**Thrombolysis** The dissolution of a blood clot. This is a common medical therapy for treatment of a myocardial infarction resulting from a blood clot, or thrombus.

**Thrombus** A clot formed from blood elements within the cardiovascular system. A thrombus can be attached to the walls of a blood vessel or the heart, and can detach and travel downstream to occlude a blood vessel.

**Tricuspid valve** The three leaflet valve that regulates blood flow between the right atrium and ventricle.

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**Triglycerides** Fatty components in the blood stream from our diet. Triglycerides are transported to the liver where they are used as the building blocks for other lipids, such as cholesterol.

**Veins** Thin walled, distensible blood vessels responsible for carrying deoxygenated blood from the tissues to the right side of the heart.

**Vena Cava** The largest common veins of the body. The superior and inferior vena cava return deoxygenated blood to the right atrium.

**Ventricles** The thick walled, muscular chambers of the heart responsible for pumping blood to the pulmonary and systemic circulation.

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# Appendix

## Public Safety Officers Benefit (PSOB) Information







# BJA Bureau of Justice Assistance Fact Sheet

## HISTORY

### Public Safety Officers' Benefits Program

The Public Safety Officers' Benefits (PSOB) Act (42 U.S.C. 3796, et seq.) was enacted in 1976 to assist in the recruitment and retention of law enforcement officers and fire fighters. Specifically, Congress was concerned that the hazards inherent in law enforcement and fire suppression and the low level of State and local death benefits might discourage qualified individuals from seeking careers in these fields, thus hampering the ability of communities to provide for public safety. The PSOB Act was designed to offer peace of mind to men and women seeking careers in public safety and to make a strong statement about the value American society places on the contributions of those who serve their communities in potentially dangerous circumstances. The resultant PSOB Program, which is administered by the Bureau of Justice Assistance (BJA), presents a unique opportunity for the U.S. Department of Justice; Federal, State, and local public safety agencies; and national public safety organizations to become involved in promoting the protection of public safety officers before tragedies occur. Each year, the PSOB Program receives substantial information about line of duty deaths that is used to enhance public safety officer training. The PSOB Program also encourages public safety agencies to adopt model policies that can help guide an agency through the tragic event of a line of duty death.

### PSOB Service Standard Commitment

The mission of the PSOB staff is to assist public safety officers, their agencies, and their families before, during, and after a tragedy occurs. Three core values guide our daily operations and measure our performance. They are:

- We will respond rapidly and accurately to PSOB death and disability benefits claims.
- We will be humane in our support of public safety officers, their agencies, and their families.
- We will seek and pursue opportunities to expand our assistance to the public safety field.

To improve our response time, we continuously assess our allocation of staff and organizational processes. We will respond to the public safety field within two (2) weeks once an eligible death benefits case is complete; within four (4) weeks once an ineligible death benefits case is complete; and within six (6) weeks once a

disability case is complete. To ensure accuracy, we will use medicolegal experts and in-depth legal analyses from outside the PSOB Program.

To provide our services in the most sensitive and professional manner, PSOB staff receive training on key issues associated with grief, critical incident stress, and post-traumatic stress disorder. We also solicit and use information provided to us on the tone and impact of our verbal and written communication with the public safety field. One example of the PSOB Program giving more to the field is a series of regional training sessions conducted to help law enforcement agencies prepare for the loss of an officer. It is essential that all public safety agencies be prepared to effectively assist the family, fellow officers, and the community to move forward in the aftermath of a tragedy. Our commitment to support the public safety community has never been stronger, and it will continue to grow.

**PSOB PROGRAM  
BENEFITS**

The PSOB Program provides a onetime financial benefit to the eligible survivors of public safety officers whose deaths are the direct and proximate result of a traumatic injury sustained in the line of duty. The benefit was increased from \$50,000 to \$100,000 for deaths occurring on or after June 1, 1988. Since October 15, 1988, the benefit has been adjusted each year on October 1 to reflect the percentage of change in the Consumer Price Index. For fiscal year 1998, the benefit is \$141,556. The PSOB Program provides the same benefit to public safety officers who have been permanently and totally disabled by a catastrophic personal injury sustained in the line of duty if that injury permanently prevents the officer from performing **any** gainful work. Approximately 15 disability claims have been submitted annually since the PSOB Act was amended in 1990 to include permanent and total disability. Medical retirement for a line of duty disability does not, in and of itself, establish eligibility for PSOB benefits. Since 1977, on average, the PSOB Program has received 275 benefit claims each year for line of duty deaths of public safety officers. PSOB Program staff respond rapidly and with sensitivity to requests for assistance from claimants and public safety agencies. They also provide moral support and, when necessary, referrals to organizations such as Concerns of Police Survivors (COPS), which can provide long-term support for surviving family members and coworkers of deceased public safety officers.

**PSOB PROGRAM  
EFFECTIVE DATES**

The effective dates for PSOB Program benefits are as follows:

**Death Benefits**

- State and local law enforcement officers and fire fighters are covered for line of duty deaths occurring on or after September 29, 1976.
- Federal law enforcement officers and fire fighters are covered for line of duty deaths occurring on or after October 12, 1984.
- Members of Federal, State, and local public rescue squads and ambulance crews are covered for line of duty deaths occurring on or after October 15, 1986.

**Disability Benefits**

Federal, State, and local law enforcement officers, fire fighters, and members of public rescue squads and ambulance crews are covered for catastrophic injuries sustained on or after November 29, 1990. The public safety officer must be separated from his or her employing agency for medical reasons, and must be receiving the maximum allowable disability compensation from his or her jurisdiction, in order to initiate a claim for PSOB disability benefits. Eligible officers may include persons who are comatose, in a persistent vegetative state, or quadriplegic.

**PUBLIC SAFETY  
OFFICERS ELIGIBLE FOR  
PSOB PROGRAM  
BENEFITS**

Under the PSOB Program, a *public safety officer* is a person serving a *public agency* in an official capacity, with or without compensation, as a law enforcement officer, fire fighter, or member of a public rescue squad or ambulance crew. *Law enforcement officers* include, but are not limited to, police, corrections, probation, parole, and judicial officers. *Volunteer fire fighters and members of volunteer rescue squads and ambulance crews* are covered under the program if they are officially recognized or designated members of legally organized volunteer fire departments, rescue squads, or ambulance crews.

A *public agency* is defined as the United States; any U.S. State; the District of Columbia; the Commonwealth of Puerto Rico; any U.S. territory or possession; any unit of local government; any combination of such States or units; and any department, agency, or instrumentality of the foregoing. To be eligible for benefits, a public safety officer's death or total and permanent disability must result from injuries sustained in the

**SURVIVORS ELIGIBLE FOR PROGRAM DEATH BENEFITS**

line of duty. *Line of duty* is defined in the PSOB regulations (28 CFR 32) as any action that the public safety officer whose primary function is crime control or reduction, enforcement of the criminal law, or suppression of fires is authorized or obligated by law, rule, regulation, or condition of employment or service to perform. Other public safety officers— whose primary function is not law enforcement or fire suppression—must be engaged in their *authorized* law enforcement, fire suppression, rescue squad, or ambulance duties when the fatal or disabling injury is sustained.

Once BJA approves a claim for death benefits, the benefit will be paid to eligible survivors in a lump sum, as follows:

- *If there are no surviving children of the deceased officer*, to the surviving spouse.
- *If there is a surviving child or children and a surviving spouse*, one-half to the child or children in equal shares and one-half to the surviving spouse.
- *If there is no surviving spouse*, in equal shares to the child or children.
- *If none of the above apply*, in equal shares to the parent or parents.

Under the PSOB Act, *child* is defined as any natural child who was born before or after the death of the public safety officer, or who is an adopted child or stepchild of the deceased public safety officer. At the time of death, the *child* must be 18 years of age or younger; or 19 through 22 years of age and pursuing a full-time course of study or training, if the child has not already completed 4 years of education beyond high school; or 19 years or older and incapable of self-support due to a physical or mental disability. For PSOB Program benefits to be paid, a public safety officer must be survived by an eligible survivor; public safety officers cannot predesignate their beneficiaries.

**PSOB PROGRAM LIMITATIONS AND**

No PSOB Program benefit can be paid:

- If the death or permanent and total disability was caused by the intentional misconduct of the public safety officer or if the officer intended to bring about his or her own death or permanent and total disability.
- If the public safety officer was voluntarily intoxicated at the time of death or permanent and total disability.
- If the public safety officer was performing his or her duties in a grossly negligent manner at the time of death or permanent and total disability.
- To a claimant whose actions were a substantial contributing factor to the death of the public safety officer.
- To members of the military serving as law enforcement officers, fire fighters, or rescue squad or ambulance crew members, or to any of their survivors.

PSOB benefits do not cover death or permanent and total disability resulting from stress; strain; occupational illness; or a chronic, progressive, or congenital disease (such as heart or pulmonary disease), unless there is a traumatic injury that is a substantial contributing factor in the death or permanent and total disability. Medical proof of the traumatic injury (such as a blood test for carbon monoxide poisoning) may be necessary for coverage in these cases.

**REDUCTION OF BENEFITS**

The PSOB Program benefit is reduced by certain payments made under the District of Columbia Code and may itself reduce benefits under Section 8191 of the Federal Employees' Compensation Act. However, State and local benefits must not be reduced by benefits received under the PSOB Act, and the PSOB benefit is not reduced by any benefit received at the State or local level.

**INTERIM PAYMENT**

If BJA determines an urgent claimant need before the final action of paying a death benefit, an interim benefit payment not exceeding \$3,000 may be made to the eligible survivor(s) if it is probable that the death is compensable.

**ATTACHMENT AND TAX EXEMPTION**

PSOB death and disability benefits are not subject to execution or attachment by creditors. The Internal Revenue Service (IRS) has ruled that the benefit is not subject to Federal income tax (IRS Ruling No. 77-235, IRS 1977-28) or to Federal estate tax (IRS Ruling No. 79-397).

**ATTORNEYS' FEES**

The PSOB Act authorizes BJA to establish the maximum fee that may be charged for services rendered to the claimant by another party in connection with any PSOB claim filed with BJA. Contracts for stipulated fee and contingent fee arrangements are prohibited by PSOB regulations (28 CFR 32.22(b)). BJA assumes no responsibility for payment of claimant attorney fees (28 CFR 32.22(d)).

**FILING A CLAIM**

Eligible survivors or disability claimants may file claims directly with BJA or through the public safety agency, organization, or unit in which the public safety officer served. In most cases, the public safety agency provides BJA with sufficient information to determine whether the circumstances of the death or permanent and total disability support a benefit payment. The public safety agency prepares a Report of Public Safety Officer's Death or Permanent and Total Disability Claim Form to accompany the claim for death benefits completed by the eligible survivor(s) or, in the case of disability claims, the injured officer. BJA will determine whether and to whom a benefit should be paid.

**FOR FURTHER INFORMATION**

For more information about the Public Safety Officers' Benefits Program or to share your observations and recommendations, please contact:

U.S. Department of Justice Response Center  
Tel: 800-421-6770 or 202-307-1480

Bureau of Justice Assistance Public Safety Officers' Benefits Program 810 Seventh Street, NW. Washington, DC 20531 202-307-0635; Fax: 202-307-3373 Toll Free: 888-744-6513 Internet: <a href="http://www.ojp.usdoj.gov/BJA/">http://www.ojp.usdoj.gov/BJA/</a>	Bureau of Justice Assistance Clearinghouse P.O. Box 6000 Rockville, MD 20849-6000 800-688-4252 Fax: 301-519-5212 Bulletin Board System: 301-738-8895 Internet: <a href="mailto:look@ncjrs.aspensys.com">look@ncjrs.aspensys.com</a>
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