HEALTH & DIVING REFERENCE SERIES

THE HEART & DIVING
DAN’s Health & Diving Resource Series is a comprehensive collection of online and printed resources developed from years of DAN-supported research and insights gained from assisting thousands of members through dive and medical emergencies. These materials provide valuable information on topics critical to diver health and safety, as well as common issues encountered by new and experienced divers. As your dive safety association, it is our duty to provide the diving community with these vital education and reference tools. The series offers greater insight into topics such as ears and equalization, cardiovascular health, decompression sickness, hazardous marine injuries, and much more. Through information and education, we hope to enhance diver safety and incident prevention.

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Scuba diving is an appealing recreational activity for people of all ages. Indeed, diving in favorable conditions requires little exertion, making it easy for the uninitiated to assume that diving is a safe and effortless pastime. But it is essential to keep in mind that during any dive, perilous conditions and circumstances can arise that may call for vigorous exercise on a moment’s notice. Immersion alone is a stressor on the body, especially the heart and circulatory system. People who have limited exercise capacity may be pushed to their limit by diving—to the point of serious injury and even death. This chapter explains some basic information about the heart in relation to diving to help keep you safe and healthy as you dive.
Scuba diving exposes you to many effects, including immersion, cold, hyperbaric gases, elevated breathing pressure, exercise and stress, as well as a positive risk of gas bubbles circulating in your blood. Your heart’s capacity to support an elevated blood output decreases with age and with disease. Having a healthy heart is of the utmost importance to your safety while scuba diving, as well as to your ability to exercise generally and your life span. The information in this booklet is devoted to helping you understand how heart disease can affect you while you’re diving and how you can promote optimal heart health.

EFFECTS OF IMMERSION

Immersion in water near the temperature of the human body exposes your body to a pressure gradient, which shifts blood from the vessels in your legs to those in your chest cavity. This increases the volume of blood within your chest by up to 24 ounces (700 milliliters). Your heart thus takes in an additional 6 to 8 ounces (180 to 240 milliliters) of blood, resulting in an enlargement of all four chambers, an increase in pressure in your right atrium, a more than 30-percent increase in cardiac output and a slight increase in your overall blood pressure. Baroreceptors (sensors that perceive a change in blood pressure) within your body’s major vessels react to all these changes by decreasing the activity of your sympathetic nervous system, which governs what’s popularly called the “fight-or-flight” response. As a result, your heart rate declines and the concentration in your plasma of norepinephrine, a hormone of the sympathetic nervous system drops; in response to the drop in norepinephrine, your kidneys excrete more sodium, and your urine production increases.

EFFECTS OF COLD

Water has high thermal conductivity—that is, your body loses more heat when you’re immersed in water than when you’re in dry air. You’ll feel more comfortable at a given air temperature than when you’re immersed in water of the same temperature. And when your body loses heat, that intensifies the narrowing of your peripheral blood vessels (a condition known as “peripheral vasoconstriction”). This in turn sends more blood to your heart, which increases the filling pressure on the right side of your heart and makes it pump more blood. Constriction of the body’s small arteries also increases the resistance to blood flowing through the periphery of your body, which raises your blood pressure, meaning your heart has to exert itself more to maintain an adequate flow of blood throughout your body.

EFFECTS OF PRESSURE

Breathing air under increased pressure, as you do when scuba diving, also affects your heart and circulatory system. Increased levels of oxygen cause vasoconstriction, increase your blood pressure and reduce your heart rate and heart output. And increased levels of carbon dioxide—which may accumulate in the body when you exercise during a dive, due to reduced pulmonary ventilation caused by dense gases—can increase the flow of blood through your brain, which can speed up oxygen toxicity if you’re breathing a hyperoxic gas mix (one with an elevated level of oxygen).

EFFECTS OF EXERCISE

Diving can be very physically demanding, but recreational divers have the option of choosing diving conditions and activities that typically do not require a lot of exertion. Nevertheless, any dive places some metabolic energy demands on your body. For example, slow, leisurely swimming on the surface represents a moderate-intensity activity (see Table 2 on page 11), while swimming with fins on the surface requires up to 40 percent less energy than
barefoot swimming. But the addition of scuba equipment increases drag on the swimmer and thus the energy cost of swimming. A 1996 paper in the journal Medicine & Science in Sports & Exercise showed that wearing just one scuba tank may increase a diver’s energy consumption by 25 percent over regular surface swimming at the same speed, and that using a drysuit may result in another 25 percent increase in energy consumption.

Most dives at neutral buoyancy and with no current require only short intervals of intermittent swimming at a slow pace and thus represent low-to-moderate intensity exercise. Exercise intensity is measured by a value known as metabolic equivalent (MET), with 1 MET representing the amount of energy consumed when at rest. (See page 11 for a detailed description of MET calculations.) It is suggested that divers be able to sustain exercise at 6 METs for a period of 20 to 30 minutes. Since people can sustain only about 50 percent of their peak exercise capacity for a protracted period, it is recommended that divers be able to pass an exercise stress test at 12 METs.

EFFECTS OF STRESS
Your autonomic nervous system (ANS)—the largely involuntary system that regulates internal functions, such as your heart rate, respiratory rate and digestion—is affected by diving, too. Among the components of the ANS are the sympathetic and parasympathetic systems; while the sympathetic system governs your body’s “fight-or-flight” response, the parasympathetic system governs resting functions and helps your body conserve energy. In healthy individuals, diving generally increases parasympathetic effects, preserving the heart rate and a measure known as heart rate variability. A dive that is perceived as stressful, however, pushes the ANS in the other direction, meaning sympathetic effects prevail—resulting in an increase in the heart rate, a decline in heart rate variability and an increase in the risk of arrhythmia.

SERIOUS ADVERSE EFFECTS
Most of the effects that diving has on your heart and circulatory system fall within your body's capacity to adapt, but sometimes serious adverse reactions can occur. A reaction known as bradyarrhythmia (a very slow and irregular heartbeat) can cause sudden death upon a diver’s entry into the water, especially in individuals with a preexisting rhythm anomaly. Conversely, tachyarrhythmia (a very rapid and irregular heartbeat) can also cause sudden death, especially in divers with structural or ischemic heart disease. And overexertion or the effects of stress may strain the heart and result in acute manifestations of previously undiagnosed ischemic heart disease.

Breath-hold diving can have particularly serious adverse cardiac effects; these effects occur in quick succession in a response known as the “diving reflex.” Its most significant elements include bradycardia (a slowing of the heart rate); the peripheral vasoconstriction reaction described above; and progressive hypoxia (or lack of an adequate supply of oxygen). To avoid bursting a lung, scuba divers must not hold their breath during ascent.
Statistics show that about one-third of all diving fatalities are associated with an acute cardiac event. In a recent study of DAN members, the incidence of diving-related deaths overall was determined to be 16 per 100,000 divers per year, and of diving-related deaths due to cardiac causes, to be nearly a third of that number—5 per 100,000 divers per year. It is of particular note that the risk of cardiac-related death while diving is 10 times higher in divers over age 50 than in those under 50. Indeed, the study of DAN members showed a continuous increase in risk with increasing age. While some suspected cardiac events may be provoked by dive-specific activities or situations, other cardiac events may not be caused by a dive at all—inasmuch as sudden cardiac death also occurs while engaged in surface swimming or land-based sporting activities of various sorts and even while at rest or during sleep.

Acute myocardial infarctions (commonly known as “heart attacks”) that are brought on by exertion—such as while swimming against a current, in heavy waves or under conditions of excessive negative buoyancy—are likely involved in some dive-provoked fatalities. Heart attacks are caused by an insufficient blood supply to the muscles of the heart; diving-related heart attacks typically occur in middle-aged males with undiagnosed coronary artery disease.

Diving (or just immersion) may also provoke acute arrhythmias, or disturbances of the heart’s rhythm, that can likewise result in sudden death. Arrhythmias are more likely to cause death in older divers. As Dr. Carl Edmonds explains in his book *Diving and Subaquatic Medicine*, and DAN data confirms, “The victim often appeared calm just before his final collapse. Some were unusually tired or resting, having previously exerted themselves, or were being towed at the time—suggesting some degree of exhaustion. Some acted as if they did not feel well before their final collapse. Some complained of difficulty in breathing only a few seconds before the collapse, whereas others underwater signaled that they needed to buddy breathe, but rejected the offered regulator. Explanations for the dyspnea include psychogenic hyperventilation, autonomic induced breathing stimulation and pulmonary edema—the latter being demonstrated at autopsy. In all cases there was an adequate air supply available, suggesting that their dyspnea was not related to equipment problems. Some victims lost consciousness without giving any signal to their buddy, whereas others requested help in a calm manner.”

The incidence of sudden cardiac death (SCD) also increases with age. Patterns of SCD are similar among divers and among the general population; nevertheless, it is important that divers not dismiss the possibility of a causative relationship between diving and SCD. Cases of SCD where there was no obvious external provoking factor occur more frequently in older divers. Postmortem examinations of SCD victims are more likely to reveal signs of previously unsuspected heart disease than a specific precipitating event. The best way to prevent SCD is thus to prevent heart disease and to maintain physical fitness and wellness as you age.
Your capacity for sustained physical activity depends on the amount of energy your body can produce in a process using oxygen called aerobic capacity. Your individual aerobic capacity depends on how well your cardiovascular system—your heart and blood vessels—works. It’s the system that moves your blood through your lungs, where it’s loaded with oxygen, and then distributes it to every part of your body, where the oxygen sustains life, nourishes your muscles and supports your ability to exercise. The “motor” of the circulatory system is the heart. The heart is a pump made of live tissue: muscles, supportive tissue and a conduction system that produces the electrical signals which stimulate your heart’s pumping action. An empty heart weighs an average of a little over half a pound (250 to 300 grams) in females and between two-thirds and three-quarters of a pound (300 to 350 grams) in males. It has four chambers: the right atrium, right ventricle, left atrium and left ventricle.

The atria receive blood at low pressure. The right atrium receives venous blood returning to the heart from all over the body after it’s been depleted of oxygen. The left atrium receives blood returning to the heart from the lungs after it’s been enriched again with oxygen. The ventricles do most of the pumping. The right ventricle pumps blood to and through the lungs, while the left ventricle maintains the circulation of blood throughout the body, to all its organs and tissues. Blood flows through the heart in only one direction, thanks to a system of valves that open and close at just the right time. How hard your heart has to work varies depending on many factors, including your activity level.

On average, a human heart pumps about 2.4 ounces (70 milliliters) of blood per heartbeat—a measure that’s known as “stroke volume.”

The heart of an individual at rest beats, on average, 72 times per minute (this is your “heart rate”), which results in a cardiac output as follows:

- 1.3 gallons (5 liters) of blood per minute.
- 1,900 gallons (7,200 liters) per day.
- 700,000 gallons (2,628,000 liters) per year.
- 48 million gallons (184 million liters) over an average life span of 70 years.

And that output is just to meet the body’s basic metabolic needs at rest: about 3.5 milliliters of oxygen per kilogram of body mass per minute. This resting metabolic rate is designated as one metabolic equivalent, which is expressed as “1 MET.” When you exercise, your body’s muscles require more oxygen, so your blood flow increases to meet that need; your heart rate may increase threefold and your stroke volume may double. This increases the cardiac output of a person of average fitness from about 1.3 gallons (5 liters) per minute to between 4 and 5 gallons (15 and 20 liters) per minute, and of a top athlete to as much as 10 gallons (40 liters) per minute. And not only does the blood flow increase, but more oxygen is extracted from each unit of blood. As a result of these changes, the metabolic level of a person of average fitness exercising at peak capacity increases to about 12 METs, and of a top athlete running a 4:17 mile (or a 22.5-kilometers-per-hour pace) may increase to 23 METs.
An individual’s ability to sustain a high level of exercise for a prolonged period of time decreases with age, even with healthy aging. This decline can be slowed by regular exercise, but it cannot be avoided completely. The decline is caused by a weakening of the functions of all the body’s systems, though the focus here is on the heart.

The heart has a pacing system that controls the heartbeat and regulates the electrical signals that stimulate the heart’s pumping action. Over time, this natural pacemaker loses some of its cells, and some of its electrical pathways may get damaged. These changes can result in a slightly slower heart rate at rest and a greater susceptibility to abnormal rhythms (the most common of which is known as “atrial fibrillation”).

With increasing age, all the structures of the heart also become more rigid. The muscles of the left ventricle get thicker, the heart may increase slightly in size and the volume of the left ventricle may decline. As a result, the heart may both fill and empty more slowly, thus putting less blood into circulation. The increase in one’s heart rate and cardiac output in response to physical activity is also diminished, and one’s maximum heart rate declines. The drop in maximum heart rate appears to be greater than average in sedentary individuals and in those with overt cardiovascular disease.

<table>
<thead>
<tr>
<th>AGE IN YEARS</th>
<th>20</th>
<th>30</th>
<th>40</th>
<th>50</th>
<th>60</th>
<th>70</th>
<th>80</th>
<th>90</th>
</tr>
</thead>
<tbody>
<tr>
<td>BEATS PER MINUTE (TRADITIONAL ESTIMATE)</td>
<td>200</td>
<td>190</td>
<td>180</td>
<td>170</td>
<td>160</td>
<td>150</td>
<td>140</td>
<td>130</td>
</tr>
<tr>
<td>BEATS PER MINUTE (HEALTHY NONSMOKERS+)</td>
<td>194</td>
<td>187</td>
<td>180</td>
<td>173</td>
<td>166</td>
<td>159</td>
<td>152</td>
<td>145</td>
</tr>
</tbody>
</table>

* The traditional formula for calculating maximum heart rate, proposed in the 1970s, was 220 less the individual’s age.

+ Tanaka and coauthors proposed an updated formula in 2001 for healthy nonsmokers of 208 less 7/10ths of the individual’s age.

Source: Modified from “Age-predicted maximal heart rate revisited” by H. Tanaka H et al. Journal of the American College of Cardiology; 2001; Vol. 37; pages 153-156

The autonomous nervous system changes with age, too. Normally, its parasympathetic component sets the level of the heart rate at rest, while its sympathetic component governs the heart in anticipation of and in response to physical activity—stimulating a timely and appropriate increase in blood flow to support the activity. Continuous adjustments between the sympathetic and parasympathetic systems result in minute variations in the heart rate (a factor known as “heart rate variability”) that are evident on a beat-to-beat basis—the kind of sensitive regulation that is a signature of a healthy control system. With increasing age, however, the contribution of the parasympathetic system wanes; the sympathetic system’s activity increases, even at rest; heart rate variability disappears; and the heart’s rhythm becomes more prone to disruption. This age-related falloff in heart rate variability and increase in resting heart rate (due to the decline in parasympathetic activity) are responsible for a 2.6-fold increased risk of SCD.
The intensity of any physical activity can be calculated directly—by measuring the amount of oxygen you use for energy metabolism (a factor that’s abbreviated as VO₂, short for “volume of oxygen”) per minute of exercise—or indirectly—by measuring your heart rate and using that value as an index of the strain your exertion is placing on your heart and lungs.

**DIRECT MEASUREMENT OF EXERCISE INTENSITY**

The amount of energy you use at any given time is proportional to the amount of oxygen your body requires. At rest, the average healthy person uses roughly 3.5 milliliters of oxygen per kilogram of body weight per minute; this is known as “resting metabolic rate.” The energy cost of a physical activity can be expressed as a multiple of resting metabolic rate; this is known as “metabolic equivalent of task,” or simply metabolic equivalent, and is abbreviated as MET.

An individual of average fitness can achieve about a 12-fold increase in metabolic rate (which is expressed as “12 METs”), while top athletes can exceed a 20-MET increase.

The table to the right lists examples of activities classified as being of light, moderate or vigorous intensity, based on the amount of energy required to do them.

**Sources:** “Compendium of physical activities: an update of activity codes and MET intensities”; “Oxygen consumption in underwater swimming”; and “Oxygen uptake studies of divers when fin swimming with maximum effort at depths of 6–176 feet” (see the list of further readings on page 53 for details on these sources).

**TABLE 2. AVERAGE METABOLIC ENERGY REQUIREMENTS FOR SELECTED PHYSICAL ACTIVITIES**

<table>
<thead>
<tr>
<th>PHYSICAL ACTIVITY</th>
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<tbody>
<tr>
<td>LIGHT-INTENSITY ACTIVITIES</td>
<td>&lt; 3.0</td>
</tr>
<tr>
<td>SLEEPING</td>
<td>0.9</td>
</tr>
<tr>
<td>WATCHING TELEVISION</td>
<td>1.0</td>
</tr>
<tr>
<td>WRITING, DESKWORK, TYPING</td>
<td>1.8</td>
</tr>
<tr>
<td>WALKING AT 2.5 MILES PER HOUR (4.0 KILOMETERS PER HOUR)</td>
<td>2.9</td>
</tr>
<tr>
<td>MODERATE-INTENSITY ACTIVITIES</td>
<td>3.0 TO 6.0</td>
</tr>
<tr>
<td>WALKING AT 3.4 MILES PER HOUR (5.5 KILOMETERS PER HOUR)</td>
<td>3.6</td>
</tr>
<tr>
<td>UNDERWATER SCUBA SWIMMING AT 20 YARDS PER MINUTE (0.6 KNOTS)</td>
<td>5.0</td>
</tr>
<tr>
<td>SURFACE SWIMMING AT A LEISURELY PACE</td>
<td>6.0</td>
</tr>
<tr>
<td>VIGOROUS-INTENSITY ACTIVITIES</td>
<td>&gt; 6.0</td>
</tr>
<tr>
<td>JOGGING, GENERAL</td>
<td>7.0</td>
</tr>
<tr>
<td>UNDERWATER SCUBA SWIMMING AT 34 YARDS PER MINUTE (1 KNOT)</td>
<td>7.5</td>
</tr>
<tr>
<td>SURFACE SWIMMING, CRAWL, AT 50 YARDS PER MINUTE</td>
<td>8.0</td>
</tr>
<tr>
<td>WALKING AT 5.0 MILES PER HOUR (8.0 KILOMETERS PER HOUR)</td>
<td>8.3</td>
</tr>
<tr>
<td>SURFACE SWIMMING, CRAWL, AT 75 YARDS PER MINUTE</td>
<td>11.0</td>
</tr>
<tr>
<td>UNDERWATER SCUBA SWIMMING AT 40 YARDS PER MINUTE (1.2 KNOTS)</td>
<td>11.0</td>
</tr>
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An individual’s peak aerobic capacity is expressed as maximum oxygen uptake while engaged in all-out exercise (which is abbreviated as “VO₂ max”). Measuring VO₂ max accurately requires following strict protocols in a sports-performance lab—a procedure known as a “maximal exercise test.” Conducting such tests is time-consuming and expensive, so they are used only in special situations.

INDIRECT ESTIMATION OF EXERCISE INTENSITY

It is also possible to make a relative estimate of an activity’s intensity by measuring its effects on your heart rate and respiration rate. This can be done in several ways.

Talk test: If an average healthy person can talk but not sing while exercising, that activity is considered to be of moderate intensity. A person engaged in vigorous-intensity activity is not able to say more than a few words without pausing for a breath. If you must gasp for air and cannot talk during what is generally considered to be moderate-intensity exercise, it means that your physical capacity is below average.

Heart rate test: Your heart rate rises in a regular fashion as the intensity of your activity increases (though the maximum heart rate you’re able to achieve will decline as you age). You can figure the average maximum heart rate for healthy individuals your age by subtracting your age from 220. For example, the maximum heart rate for a 50-year-old would be calculated as follows: 220 - 50 = 170 beats per minute (bpm). You can then use your actual heart rate to estimate the relative intensity of various activities you engage in and to indirectly estimate your maximum exercise capacity. Experts often recommend reaching and sustaining a certain heart rate to improve or maintain fitness.

Submaximal exercise test: A submaximal exercise test can be used to figure your maximum exercise capacity without exceeding 85 percent of the estimated maximum heart rate for your age. Conducting such a test calls for gradually increasing your exercise intensity, based on a defined protocol, while your heart rate is being monitored. When you reach the target heart rate, you stop exercising and your maximum exercise capacity can then be extrapolated using various methods. However, because of variations in the relationship between heart rate and exercise intensity due to age, fitness level and other factors, an indirect estimation of maximum aerobic capacity has limited value. Nevertheless, the test is still a valuable clinical tool to assess an individual’s tolerance for exercise and likelihood of having ischemic heart disease.
Adults need two types of regular activity to maintain or improve their health—aerobics and strength training. The Centers for Disease Control and Prevention's 2008 Physical Activity Guidelines for Americans recommends at least two and a half hours a week of moderate-intensity aerobic exercise to achieve health benefits, and five hours a week to achieve additional fitness benefits. And just as important as engaging in aerobic exercise is doing muscle-strengthening activities at least two days a week. Physical activities are commonly classified by intensity into one of the following four categories:

- **SEDENTARY BEHAVIOR**: Sedentary behavior refers to activities that do not substantially increase one's heart rate or energy expenditure above the resting level; included in this category are activities like sleeping, sitting, lying down and watching television. Such activities involve an energy expenditure of 1.0 to 1.5 METs.

- **LIGHT-INTENSITY PHYSICAL ACTIVITY**: Light physical activity—which is often grouped with sedentary behavior but is, in fact, a distinct activity level—involves an energy expenditure of between 1.6 and 2.9 METs and raises the heart rate to less than 50 percent of one's maximum heart rate. It encompasses such activities as slow walking, deskwork, cooking and washing dishes.

- **MODERATE-INTENSITY PHYSICAL ACTIVITY**: Physical activity that increases the heart rate to between 50 percent and 70 percent of one's maximum heart rate is considered to be of moderate intensity. For example, 50-year-olds have an estimated maximum heart rate of 170 beats per minute (bpm), so the 50-percent and 70-percent levels would be 85 bpm and 119 bpm. That means a moderate-intensity activity for 50-year-olds is one that keeps their heart rate between 85 bpm and 119 bpm. By contrast, 30-year-olds have an estimated maximum heart rate of 190 bpm, making a moderate-intensity activity one that raises their heart rate to between 95 bpm and 133 bpm.

- **VIGOROUS-INTENSITY PHYSICAL ACTIVITY**: A vigorous-intensity activity is one that increases the heart rate to between 70 percent and 85 percent of one's maximum heart rate. For 60-year-olds, that would be between 122 bpm and 136 bpm; for 25-year-olds, it would be between 136 bpm and 167 bpm.

Detailed physical activity recommendations can be found at [cdc.gov/physicalactivity/everyone/guidelines](https://www.cdc.gov/physicalactivity/everyone/guidelines).
PRE-ACTIVITY SCREENING

Engaging in physical activity is beneficial for one’s health, but making the transition from a sedentary lifestyle to being physically active, or increasing one’s accustomed level of activity, may be associated with increased risk—especially in individuals with preexisting heart disease. Scuba diving typically involves moderate-intensity physical activity, but situations can occur that require high-intensity activity. In addition, scuba diving challenges the cardiovascular system in a variety of ways that may be life-threatening for individuals with heart disease or a low capacity for exercise.

A common pre-activity screening tool is the Recreational Scuba Training Council (RSTC) Medical Statement and Guidelines. The RSTC questionnaire asks about your medical history, as well as symptoms and signs of chronic and acute diseases. If prospective divers have any of the listed conditions, they are advised to consult with a physician to obtain a medical evaluation of their fitness to dive. Most dive operators use the RSTC form to screen customers, and if you check any conditions that call for medical evaluation but cannot present documentation of a recent exam that has cleared you for diving, you may be unable to dive. So you should complete the RSTC Medical Statement in advance of any trip during which you plan to dive and, if necessary, obtain a written evaluation from a physician knowledgeable about diving medicine—and take it with you on your trip.

And remember that it is of the utmost importance that you be honest in filling out the questionnaire: You hold the keys to your safe participation in any physical activity, including scuba diving.

In addition, regardless of their medical condition, men age 45 and older and women age 50 and older are advised to review their health annually with their primary-care physician. And all divers with any risk factors for cardiac disease should see their primary-care physician before engaging in diving and should be sure to follow any advice they’re given.
In general, engaging in regular physical activity reduces an individual's risk of death due to heart disease—but in susceptible individuals, vigorous activity can increase the risk of an acute myocardial infarction (heart attack) or of sudden cardiac death (SCD). Individuals with advanced atherosclerosis—a disorder that involves a narrowing of the arteries due to a buildup of fatty deposits on their inner walls—are especially susceptible to such risks.

The incidence of both acute myocardial infarction and SCD is greatest in generally sedentary individuals, especially those who engage in unaccustomed physical activity. A paper published in the *New England Journal of Medicine* found that habitually sedentary men were 56 times more likely to experience cardiac death during or after vigorous exercise than when at rest; by contrast, very physically active men were only five times more likely to die during or after vigorous exercise than when at rest. Another *New England Journal of Medicine* paper reported that an acute myocardial infarction was 50 times more likely during or soon after vigorous physical exercise in the least active than in the most active subjects.

So while sedentary individuals are advised to change their lifestyle and take up regular physical exercise—starting with low-intensity activities and gradually increasing the intensity at which they exercise—they may require pre-activity screening. Individuals with any health limitations need both medical clearance and, preferably, a professional fitness coach. Individuals identified as being at high risk for cardiac problems should abstain from certain activities. For relevant guidelines, read “When to consult a health-care provider before engaging in physical activities.”

It is important to emphasize, however, that even the most restrictive practices will never be able to completely prevent cardiovascular events associated with exercise. It is thus essential that individuals who exercise recognize and report the symptoms that often precede a cardiac event; these are known as “prodromal symptoms” and may include one or more of the following:

- Chest pain (known as “angina”).
- Increasing fatigue.
- Indigestion, heartburn or other gastrointestinal symptoms.
- Excessive breathlessness.
- Ear or neck pain.
- A feeling of vague malaise.
- Upper respiratory tract infections.
- Dizziness, palpitations or a severe headache.

Such symptoms have been shown to be present in 50 percent of joggers, 75 percent of squash players, 81 percent of distance runners and 60 percent of scuba divers who die while exercising. People who exercise must be aware of these facts, and physicians should query patients during medical exams about their exercise habits and their knowledge of prodromal symptoms. Divers who experience any of the symptoms above during exercise should obtain a medical evaluation before they resume diving.
Chapter 2: Risk Factors for Cardiovascular Disease

It behooves divers to be aware of the risk factors for cardiovascular disease, especially atherosclerosis, and of specific measures they can take to mitigate them. Atherosclerosis—popularly known as "hardening of the arteries"—is the most common affliction of the heart. Its prevalence increases with age, and it causes premature death in many people. Indeed, it is often assumed to be associated with normal aging. However, the disorder can be prevented—or at least slowed down—and a physically active lifestyle extended well into older age.

Coronary heart disease is a leading cause of morbidity and mortality among adults in both North America and Europe.
The most common manifestations of acquired (rather than congenital) cardiovascular disease are coronary heart disease, stroke and peripheral artery disease. Coronary heart disease is a leading cause of morbidity and mortality among adults in both North America and Europe.

The likelihood that a given individual will acquire cardiovascular disease and suffer a life-threatening cardiovascular event depends on many risk factors. Some risk factors—such as family history, gender, ethnicity and age—cannot be changed. Other risk factors are modifiable—including some involuntary health conditions and some lifestyle-related factors. Involuntary conditions such as high blood pressure, high cholesterol and diabetes can be treated with medication, as well as with diet and lifestyle adjustments. Lifestyle-related risk factors include tobacco use, an unhealthy diet, physical inactivity and excessive alcohol consumption—all of which can be voluntarily changed.

It is important to understand that having any of these risk factors does not mean that you will definitely develop cardiovascular disease. However, the more risk factors you have, the greater is the likelihood that you will develop cardiovascular disease—unless you control your involuntary health conditions and adopt a healthy lifestyle.

The following percentages of deaths caused by cardiovascular disease can be attributed to these specific risk factors:

- High blood pressure: 13%
- Tobacco use: 9%
- High blood sugar: 6%
- Physical inactivity: 6%
- Overweight and obesity: 5%
Hypertension, or high blood pressure, is a common medical condition in the general population, as well as among divers. Blood pressure is a measure of the force with which blood pushes outward on the arterial walls. A blood-pressure reading is a ratio of two numbers. The top number is the systolic pressure, when your heart is beating, and the bottom number is the diastolic pressure, when your heart is resting between beats. The unit of measurement for a blood-pressure reading is millimeters of mercury, which is abbreviated as “mmHg”; a normal reading is 120/80 mmHg, often referred to as “120 over 80.”

The criteria for a diagnosis of hypertension vary slightly from country to country and even from one reference to another. The table below shows the most common criteria used in the United States.

<table>
<thead>
<tr>
<th>BLOOD PRESSURE CATEGORY</th>
<th>SYSTOLIC PRESSURE</th>
<th>DIASTOLIC PRESSURE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Less than 120 mmHg</td>
<td>over</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120 to 139</td>
<td>80 to 89</td>
</tr>
<tr>
<td>Stage 1 Hypertension</td>
<td>140 to 159</td>
<td>90 to 99</td>
</tr>
<tr>
<td>Stage 2 Hypertension</td>
<td>160 or higher</td>
<td>100 or higher</td>
</tr>
<tr>
<td>Hypertensive crisis</td>
<td>Higher than 180</td>
<td>over</td>
</tr>
<tr>
<td>(emergency care needed)</td>
<td></td>
<td>Higher than 110</td>
</tr>
</tbody>
</table>

*Source: American Heart Association*

**STATISTICS**
- 78 million American adults (or 31%—almost 1 in 3) have hypertension.
- 69% of those who have a first heart attack, 77% of those who have a first stroke, and 74% of those with chronic heart failure have hypertension; it is also a major risk factor for kidney disease.
- 348,000 American deaths in 2009 were attributed, as either a primary or contributing cause, to hypertension.
- $47.5 billion annually is spent on direct medical expenses related to hypertension.
- $3.5 billion annually is lost in productivity due to hypertension.
- Only 47% (less than half) of those with hypertension have the condition under control.
- 30% of American adults have prehypertension.

*Sources: U.S. Centers for Disease Control and Prevention and American Heart Association*
Two kinds of complications face a person with hypertension: short-term and long-term. Short-term complications generally result from extremely high blood pressure; the most significant is the risk of a stroke (also called a “cerebrovascular accident”) due to the rupture of a blood vessel in the brain. Long-term detrimental effects are more common; they include coronary artery disease, kidney disease, congestive heart failure, eye problems and cerebrovascular disease.

Mild hypertension can often be controlled with diet and exercise; however, medication may be necessary to keep blood pressure within tolerable limits. Many classes of drugs are used to treat hypertension, and they have varying side effects. Some individuals must change medications after one drug appears to be or becomes ineffective. Others might need to take more than one drug at a time to keep their blood pressure under control.

A class of antihypertensive drugs known as beta blockers may cause a decrease in maximum exercise tolerance and may also have some effect on the airways. These side effects normally pose no problem for the average diver. Another class of antihypertensives, known as angiotensin-converting enzyme (ACE) inhibitors, may be preferred for divers, though a persistent cough is a possible side effect of ACE inhibitors. Calcium channel blockers are another choice, but a potential side effect of these drugs is lightheadedness upon going from a sitting or supine to a standing position.

Diuretics—drugs that promote the production of urine—are also frequently used to treat hypertension. Their use requires careful attention to maintaining adequate hydration and to monitoring electrolyte levels in the blood.

**EFFECT ON DIVING**

As long as an individual’s blood pressure is under control, the main concerns regarding fitness to dive are side effects of any medication(s) and evidence of damage to the major organs. Most antihypertensive drugs are compatible with diving, as long as side effects are minimal and the diver’s performance in the water is not significantly compromised. In addition, a diver with long-standing hypertension should be monitored for evidence of associated damage to the heart and kidneys.

Divers who demonstrate adequate control of their blood pressure and who show no significant decrease in their performance in the water due to drug side effects should be able to dive safely. However, it is important that such divers have regular physical examinations, including screening for long-term consequences of hypertension, such as coronary artery disease.
Cholesterol—a soft, waxy substance—is one of the lipids found in the blood and, indeed, in all the cells of the body. Important to the healthy functioning of our bodies, cholesterol is a part of our cells’ membranes and is used in the production of hormones.

The cholesterol in the human body may originate from foods rich in cholesterol—like meat, eggs and dairy products—or it can be made internally by our bodies. The body can also produce cholesterol from foods that do not themselves contain cholesterol but that do contain saturated fat—like palm oil and coconut oil—or from trans fats—like fried food in restaurants and commercial cakes or cookies. Cholesterol by itself does not dissolve in blood; it has to be combined with proteins to form soluble lipoprotein particles. Lipoproteins come in two forms: low-density lipoprotein (LDL) and high-density lipoprotein (HDL).

LDL is considered “bad cholesterol” because too much of it leads to a narrowing and stiffening of the arteries due to a buildup of cholesterol, which accumulate in deposits called “plaques” on the arteries’ inner walls. This condition is called atherosclerosis. It contributes to hypertension and causes peripheral artery disease, coronary artery disease, heart attack and stroke—as well as erectile dysfunction in men.

In contrast, HDL cholesterol is considered “good cholesterol” because it reduces the risk of cardiovascular disease by transporting cholesterol away from the bloodstream and back to the liver, which facilitates its removal from the body. HDL thus helps to prevent the buildup of cholesterol plaques on the walls of the arteries. An individual’s HDL cholesterol level is to some extent a factor of one’s genetic makeup. But HDL levels can be lowered by type 2 diabetes; certain drugs, such as beta blockers and anabolic steroids; smoking; being overweight; and being sedentary. On the other hand, estrogen, a female hormone, raises HDL levels, partially explaining why cardiovascular disease is less prevalent in premenopausal women.

Triglycerides are another factor in hyperlipidemia. Triglyceride is the most common type of fat in the body. Normal triglyceride levels vary by age and sex. High triglyceride levels combined with high levels of LDL cholesterol increase one’s risk of cardiovascular disease.

Your cholesterol level is a composite measure of all these lipids, in either milligrams per deciliter of blood (mg/dl) or millimoles per liter of blood (mmol/L).

MANY AMERICAN EXPERTS RECOMMEND THE FOLLOWING CHOLESTEROL LEVELS:

- Total cholesterol: 200 mg/dl (5.2 mmol/L)
- LDL cholesterol: from below 70 mg/dl (1.8 mmol/L) to 129 mg/dl (3.3 mmol/L), depending on your health status
- HDL cholesterol: above 60 mg/dl (1.6 mmol/L)
- Triglycerides: below 150 mg/dl (3.9 mmol/L)

Source: American Heart Association

The American Heart Association recommends that all adults age 20 and older have their cholesterol and other risk factors for hyperlipidemia checked every four to six years and also work with their health-care providers to determine their risk for cardiovascular disease and stroke.
The terms overweight and obesity refer to a body weight in relation to height that is greater than is considered healthy; both conditions often (but not necessarily) result in a higher proportion of body fat, known as adipose tissue, compared to lean muscle mass. Overweight is applied to those with a somewhat elevated weight, and obesity to those who are extremely overweight.

**STATISTICS**
- 69% of adult Americans (more than two-thirds) are either overweight or obese.
- Adult obesity rates have more than doubled in just over 30 years, from 15% in 1976–1980 to 36% percent in 2009–2010.
- 10 years ago, the obesity rate was significantly higher among women than men; currently, the rates are essentially the same—within a few decimal places of 36% for both men and women.

Body mass index (BMI) is a common way of expressing the ratio between weight and height. The following equations are used to calculate BMI:

**WHEN USING IMPERIAL WEIGHT AND HEIGHT MEASUREMENTS:**
\[
BMI = \frac{(\text{WEIGHT IN POUNDS}) \times 703}{(\text{HEIGHT IN INCHES}) \times (\text{HEIGHT IN INCHES})}
\]

**WHEN USING METRIC WEIGHT AND HEIGHT MEASUREMENTS:**
\[
BMI = \frac{\text{WEIGHT IN KILOGRAMS}}{(\text{HEIGHT IN METERS}) \times (\text{HEIGHT IN METERS})}
\]

BMI is an important measure for understanding population trends, but it does have some limitations, as follows:
- It may overestimate the proportion of body fat in athletes and others with a muscular build.
- It may underestimate the proportion of body fat in older persons and others who have lost muscle mass.

Accordingly, BMI is just one of many factors that should be considered in evaluating whether an individual is at a healthy weight—along with waist size, waist-to-hip ratio and a measurement known as “skin-fold thickness.”
Metabolic syndrome is a disorder that affects how the body uses and stores energy. According to the American Heart Association, a diagnosis of metabolic syndrome requires the presence of three or more of these conditions:

1. Abdominal obesity—defined as a waist circumference of 40 inches (102 centimeters) or above for men and 35 inches (89 centimeters) or above for women.

2. A triglyceride level equal to or greater than 150 mg/dL (3.9 mmol/L).

3. An HDL cholesterol level below 40 mg/dL (1.0 mmol/L) for men and below 50 mg/dL (1.3 mmol/L) for women.

4. A blood pressure equal to or greater than 130/85 mmHg or the use of medication for hypertension.

5. A fasting blood glucose level equal to or greater than 100 mg/dL (5.6 mmol/L) or the use of medication for hyperglycemia.

Metabolic syndrome is associated with an elevated risk of cardiovascular disease. Other disorders associated with metabolic syndrome include endothelial dysfunction and chronic low-grade inflammation.

Measuring the circumference of your waist to detect abdominal obesity, meaning more fat is at your waist than at your hips, is a good start in assessing whether you may have metabolic syndrome. This is important because abdominal obesity represents a higher risk for heart disease and type 2 diabetes, and the risk increases progressively as waist size increases beyond the dimensions noted above. The implications of these factors are shown in the chart below.

<table>
<thead>
<tr>
<th>BMI</th>
<th>DISEASE RISK* RELATIVE TO NORMAL WEIGHT AND WAIST CIRCUMFERENCE+</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;40 in (102 cm) for Men</td>
<td>&gt;40 in (102 cm) for Men</td>
</tr>
<tr>
<td>≤35 in (88 cm) for Women</td>
<td>&gt;35 in (88 cm) for Women</td>
</tr>
<tr>
<td>UNDERWEIGHT</td>
<td>&lt;18.5</td>
</tr>
<tr>
<td>NORMAL</td>
<td>18.5–24.9</td>
</tr>
<tr>
<td>OVERWEIGHT</td>
<td>25.0–29.9</td>
</tr>
<tr>
<td>OBESE</td>
<td>30.0–34.9</td>
</tr>
<tr>
<td></td>
<td>35.0–39.9</td>
</tr>
<tr>
<td>EXTREMELY OBESE</td>
<td>≥40.0</td>
</tr>
</tbody>
</table>

* For type 2 diabetes, hypertension and cardiovascular disease.
+ An increased waist circumference also can be a marker for increased risk, even in persons of normal weight.

Source: National Heart, Lung, and Blood Institute
Having healthy heart valves is essential if your heart is to properly pump and circulate blood throughout your body. Some people are born with structural anomalies in their heart valves or in the walls. Many such disorders are diagnosed early in life and corrected, restoring the affected individuals’ exercise capacity and enabling them to dive safely. However, some inborn structural disorders, like a condition known as patent foramen ovale, may not become obvious until after an affected individual has taken up diving—and may result in an increased risk of certain diving injuries. In addition, some people are impacted later in life by acquired valvular damage that may affect their fitness to dive.

Divers who suffer decompression sickness have a patent foramen ovale (PFO) prevalence twice that of the population in general.
The heart has four main valves that facilitate the pumping activity of the heart:

1. The tricuspid valve, between the right atrium and the right ventricle.
2. The pulmonary valve, between the right ventricle and the pulmonary artery.
3. The mitral valve, between the left atrium and the left ventricle.
4. The aortic valve, between the left ventricle and the aorta.

Each valve consists of a set of flaps (also called “leaflets” or “cusps”) that open and close to enable blood to flow in the correct direction. The function of the valves may be compromised by either congenital or acquired abnormalities. Damage to the valves can occur due to infection, rheumatic fever or aging. For example, the opening in a valve may narrow (a condition known as “stenosis”), meaning the heart has to work harder to get blood through the opening; this generates higher pressure within the heart and eventually causes the cardiac muscle to overdevelop. Another common valvular problem is incomplete closure, which allows the blood to flow backward through the valve (a condition known as “regurgitation”); this overloads the heart with blood, eventually resulting in enlargement (or “dilatation”) of the heart’s cavities.

The two most common valvular disorders in older adults are aortic stenosis and mitral regurgitation. The symptoms of valvular disorders vary depending on which valve is affected, as well as on the type and severity of the change. Mild changes may cause no symptoms; a heart murmur—detected when the heart is examined with a stethoscope—is often the first sign of valve damage. In aortic stenosis, however, exertion can cause chest pain (known as “angina”) or a feeling of tightness in the chest, shortness of breath, fainting or heart palpitations. Sudden death in otherwise healthy athletes is sometimes caused by aortic stenosis. Regurgitation can also cause detectable symptoms, such as shortness of breath or wheezing when lying down; these complaints may be intensified by exercise, increased resistance to breathing and immersion.

Treatment for valvular disorders generally involves surgery. Defective valves may be either repaired or replaced by prosthetic valves.

Preventing valvular damage is, of course, the best approach. Routine physical exams may uncover evidence of early valvular disease. In such cases, close, regular medical surveillance is advised to identify, and hopefully slow, progression of the damage.

**EFFECT ON DIVING**

Significant valvular anomalies may preclude diving until they can be corrected. Even after corrective surgery, there must be an assessment of such factors as exercise capacity, the presence of any residual regurgitation and the need for anticoagulation. Such an assessment should include a detailed examination of the heart and of the individual's ability to exercise at a level consistent with diving, without evidence of ischemia, wheezing, cardiac dysfunction or a problem known as “right-to-left shunting.”
Mitral Valve Prolapse (MVP) may also be referred to as “click-murmur syndrome” or “floppy-valve syndrome.” It is a common condition, especially in women. The problem arises as a result of excess tissue and loose connective tissue in the heart’s mitral valve, so that part of the valve protrudes down into the left ventricle during each contraction of the heart.

An individual with MVP may have absolutely no symptoms or may exhibit symptoms ranging from occasional palpitations or an unusual feeling in the chest when the heart beats, to chest pain or a myocardial infarction (or heart attack). MVP is also associated with a slightly increased risk of small strokes (known as “transient ischemic attacks”) or a transient loss of consciousness.

Beta blockers—drugs commonly used to treat high blood pressure—are occasionally prescribed for mitral valve prolapse. They often cause a drop in maximum exercise capacity and may also affect the airways. These side effects normally pose no problem for the average diver, but they may be significant in emergency situations.

**EFFECT ON DIVING**

Frequently, MVP results in no changes in blood flow that would prevent an individual from diving safely. A diver with MVP who has no symptoms (that is, no chest pain, alteration in consciousness, palpitations or abnormal heartbeats) and who takes no medication for the problem should be able to safely participate in diving. But anyone with MVP who exhibits an abnormal cardiac rhythm, which can produce palpitations, should not dive unless the palpitations can be controlled with a low dose of antiarrhythmic medication.

---

**NORMAL**

Valve leaflets close and prevent backflow into the atrium.

**PROLAPSE**

Valve leaflets balloon upward as the ventricle contracts.

**REGURGITATION**

Valve leaflets do not properly close, forcing blood back into the atrium.
Patent foramen ovale (PFO) is a fairly common, congenital, generally benign hole between the heart’s left and right atria (see illustration).

While a fetus is developing in utero, the wall separating the left and right atria of the heart develops from the septum primum, which grows up, and septum secundum, which grows down. The septa overlap, creating a sort of trap door (known as the “foramen ovale”), which allows oxygenated blood from the mother’s placenta that has entered the fetus’ right atrium to pass through to its left atrium. At birth, the baby’s lungs expand, and the resulting pressure in the left atrium closes the foramen ovale. Typically, shortly after birth, this former opening fuses shut—but in about 27 percent of babies, it fails to fuse completely and results in a PFO.

A PFO often causes no symptoms, and most people who have one are never aware of the fact. PFO is diagnosed by injecting a small amount of air into a vein and observing its passage through the heart using echocardiography. There are two methods of echocardiography. Transthoracic echocardiography (TTE) is easy and noninvasive—it involves simply placing an ultrasound probe on the outer wall of the chest—but it detects a PFO in only 10 percent to 18 percent of the population—about half of those who probably have one. Transesophageal echocardiography (TEE)—which involves local anesthesia and intravenous sedation, so the probe can be passed into the esophagus—detects a PFO in 18 percent to 33 percent of the population. However, even though TEE is more sensitive than TTE, there are still many false-negative results with both techniques; a properly conducted TTE may in fact be more reliable than a TEE.

One of the most common treatments for PFO is a procedure called transcatheter closure; it involves threading a catheter through the groin and up the femoral vein into the heart, where a device called an occluder is implanted across the PFO. Occluders come in various shapes and forms, but most act like a double umbrella that opens on each side of the atrial wall and seals the hole. With time, tissue grows over the occluder and completely covers its surface. The implantation is performed under local anesthesia and intravenous sedation, and the patient remains conscious. It takes less than an hour and can be performed on an outpatient or one-night-stay basis. Most patients can return to their normal activities in two days, but they must take anticoagulant and/or antiplatelet drugs for three to six months. Other postoperative restrictions include no elective dental care (such as cleanings) for three months, no contact sports for three months and no heavy lifting for one week. A diver who undergoes a transcatheter PFO closure must abstain from diving for three to six months.
No data is available on the outcome of PFO closure in divers. But the following outcomes were recorded in patients who underwent PFO closure for the prevention of stroke (note, however, that these patients have underlying medical conditions that may contribute to a greater than average risk of adverse outcomes):

- **Efficacy:** Complete closure of the opening was achieved in 95 percent of cases and incomplete closure in 4 to 5 percent of cases; no improvement was shown in only 1 percent of cases.

- **Complications:** Overall mortality was less than 1/10th of 1 percent (0.093 percent). The need for a follow-up operation due to an adverse event associated with the device was less than 1 percent (0.83 percent).

- **Serious complications:** The incidence of death, stroke, infection, bleeding or blood vessel injury was 0.2 percent; of device movement or dislodgement, 0.25 percent; of clot formation on the device, 0.3 percent; of major complications in the perioperative period, 1.2 percent; and of minor midterm complications, 2.4 percent.

**EFFECT ON DIVING**

Divers who suffer decompression sickness (DCS) have a PFO prevalence twice that of the population in general. And in divers who exhibit neurological DCS symptoms, PFO prevalence is four times greater. The risk of DCS seems to increase with the size of the PFO. Based on these facts, it is assumed that divers with a PFO are at greater risk of DCS than those without a PFO; however, the only prospective study designed to directly measure the relative risk of DCS in divers with a PFO is still ongoing.
Heart disease develops 7 to 10 years later in women than in men.

Ischemia is a term meaning that an inadequate supply of blood is reaching a part of the body. Ischemic heart disease thus means not enough blood is getting to the heart muscle. It is almost always caused by atherosclerosis (a narrowing of the arteries due to fatty deposits on their inner walls) in the coronary arteries (the arteries that supply the heart muscle), and it is the most common cause of heart disease. The prevalence of ischemia increases with age. The first manifestation of ischemic heart disease is sometimes a fatal heart attack, but the condition’s presence may be signaled by symptoms that should prompt lifesaving actions. Knowing these symptoms can mean living longer. And preventing heart disease in general means living happier—without symptoms or functional limitations.
Atherosclerosis is popularly referred to as “hardening of the arteries.” It’s the result of cholesterol and other fatty material being deposited along the inner walls of the arteries. The condition has different manifestations, depending on which arteries are affected; it causes coronary artery disease (CAD) in the heart, cerebrovascular atherosclerosis in the brain and peripheral artery disease (PAD) in the limbs.

The walls of the arteries, in response to the deposition of fatty material, also thicken. The result is a progressive reduction in the flow of blood through the affected vessels. These effects are especially damaging in the heart; CAD is the leading cause of death in the United States and other industrialized countries.

Many factors contribute to the development of atherosclerosis, including a diet high in fat and cholesterol, smoking, hypertension, increasing age and a family history of the condition. Women of reproductive age are generally at lower risk of atherosclerosis due to the protective effects of estrogen.

Medications typically used to treat atherosclerosis include nitroglycerin (which is also used in the treatment of angina, or chest pain) and calcium channel blockers and beta blockers (which are also used in the treatment of high blood pressure, or hypertension; see page 51 for more on these drugs). Sometimes, individuals with CAD may need what’s known as a revascularization procedure, to re-establish the blood supply—typically a coronary artery bypass graft or angioplasty. If such a procedure is successful, the individual may be able to return to diving after a period of healing and a thorough cardiovascular evaluation (see “Issues involving coronary artery bypass grafts” on page 31).

**EFFECT ON DIVING**

Symptomatic coronary artery disease is not consistent with safe diving: don’t dive if you have CAD. The condition results in a decreased delivery of blood—and therefore oxygen—to the muscular tissue of the heart. Exercise increases the heart’s need for oxygen. Depriving your heart of oxygen can lead to abnormal heart rhythms and/or myocardial infarction, (a heart attack). The classic symptom of CAD is chest pain, especially following exertion. But unfortunately, many people have no symptoms before they experience a heart attack.

A history of stroke—or of “mini strokes” known as transient ischemic attacks (TIAs)—are also, in most cases, not consistent with safe diving.

Cardiovascular disease is a significant cause of death among divers. Older divers and those with significant risk factors for coronary artery disease should have regular medical evaluations and undergo appropriate screening studies, such as a treadmill stress test.
When any of the arteries supplying the heart become blocked, a myocardial infarction, or heart attack, will occur if the blockage (or “infarct”) is not eliminated quickly. The heart muscle supplied by that artery then becomes starved for oxygen and eventually dies. If the infarct is large enough, the heart’s ability to pump blood is compromised, and circulation to all the body’s other critical organs is affected. The heart’s electrical system may also be adversely affected, resulting in an abnormal rhythm known as ventricular fibrillation.

The main cause of myocardial infarction is coronary artery disease (CAD), or a gradual narrowing of the arteries that supply the heart with blood. Eventually, a piece of the fatty plaques affixed to the arteries’ inner walls may break free and lodge in a smaller vessel, resulting in total occlusion. CAD affects 3 million Americans and kills over 700,000 each year; it is the most common life-threatening disease. A blockage that results in a myocardial infarction can also be caused by a bubble of gas or a clot within a blood vessel. But, simply stated, whatever the cause of the occlusion, it means the oxygen required by the heart muscle can no longer be supplied through the blocked vessel.

The classic symptoms of myocardial infarction include radiating chest pain (angina) or pain in the jaw or left arm. Other symptoms include heart palpitations; dizziness; indigestion; nausea; sweating; cold, clammy skin; and shortness of breath.

If a myocardial infarction is suspected, it is essential that emergency medical care be called and the affected individual evacuated to a hospital. In the meantime, keep the individual calm and administer oxygen. At the hospital, the treatment options include conservative medical management, anticoagulation drugs, heart catheterization or stenting or even coronary artery bypass surgery.

Preventing myocardial infarction calls for addressing any risk factors, such as obesity, diabetes, hypertension or smoking. A healthy diet and regular exercise are also important preventative.

**EFFECT ON DIVING**

Anyone with active ischemic CAD should not dive. The physiologic changes involved in diving, as well as the exercise and stress of a dive, may initiate a cascade of events leading to a myocardial infarction or to unconsciousness or sudden cardiac arrest while in the water. Divers who have been treated and evaluated by a cardiologist may choose to continue diving on a case-by-case basis; essential aspects of such an evaluation include the individual’s exercise capacity and any evidence of ischemia while exercising, of arrhythmias or of injury to the heart muscle.
A coronary artery bypass is the surgical correction of a blockage in a coronary artery; it is accomplished by attaching (or “grafting”) onto the damaged vessel a piece of a vein or artery from elsewhere in the body, so as to circumvent the blockage.

Doctors perform this procedure many hundreds of times a day, all around the country—more than half a million times a year. If a bypass is successful, the individual should be free of the symptoms of coronary artery disease, and the heart muscle should once again receive a normal supply of blood and thus oxygen.

A blocked coronary artery can also be treated with a less invasive procedure, coronary angioplasty. It consists of inserting a catheter with a tiny balloon on its tip into the area of the blockage, then inflating the balloon to open the artery. This procedure does not require opening the chest and can be performed in an outpatient setting.

EFFECT ON DIVING

Individuals who have had a coronary artery bypass graft or coronary angioplasty may have suffered significant cardiac damage prior to having surgery. Their postoperative cardiac function is what determines their fitness for a return to diving.

In particular, those who have had open-chest surgery need to have a thorough medical evaluation prior to diving again. After a period of stabilization and healing (6 to 12 months is the usual recommendation), such individuals should have a complete cardiovascular evaluation before being cleared to dive. They should be free of chest pain and have a normal tolerance for exercise, as evidenced by a normal stress EKG test (at 13 METs, as described on page 11). If there is any doubt about the success of the procedure, or how open the coronary arteries are, the individual should refrain from diving.
Heart disease is the leading cause of death in women, and myocardial infarction (heart attack) is the leading cause of their hospitalization. The characteristics of the disease in women may differ from those in men; the age of onset, presence of risk factors, probability of aggressive diagnosis and even likelihood of appropriate treatment vary in men and women.

For example, heart disease develops 7 to 10 years later in women than in men (possibly because of the protective effect of estrogen). Myocardial infarction is less frequent in young women than in young men, but young women who have a heart attack are at greater risk of dying within 28 days of their attack. The common risk factors for heart disease have a similar predictive value for men and women; however, men more frequently have smoking as risk a factor, whereas women more frequently have hypertension, diabetes, hyperlipidemia or angina. And although women typically smoke less than men, the relative risk for myocardial infarction in women who do smoke is 1.5 to 2 times greater than in men who smoke, especially in those less than 55 years old. A higher prevalence of diabetes also contributes to higher mortality rates from heart attacks among women.

Women receive fewer advanced diagnostic tests like coronary angiography and fewer interventions like coronary artery bypass grafts. These differentials may be due to the fact that acute heart attacks are likely to occur at an older age in women, or to the presence of other associated diseases, but could also be due to delays in admitting women to the hospital.

The symptoms of a heart attack in women are usually the same as those in men, with chest pain (angina) being the leading symptom. However, women are more likely to attribute their symptoms to acid reflux, the flu or normal aging. In addition, the chest pain that women experience does not necessarily occur in the center of the chest or the left arm; instead, women may feel pressure in their upper back—a sensation of squeezing or as if a rope is tied around them.

Although 90 percent of women who suffer a heart attack later admit that they intuitively knew that was the cause of their symptoms, at the time they often discount them, attribute them to something else, take an aspirin or just delay calling 911. This decreases the opportunity to preserve their heart from damage and lowers their chance of survival.

**These are the most common symptoms of a heart attack in women:**

1. Uncomfortable pressure, squeezing, fullness or pain in the center of the chest; it lasts more than a few minutes or goes away and comes back.

2. Pain or discomfort in one or both arms, the back, neck, jaw or stomach.

3. Shortness of breath, with or without chest discomfort.

4. Other signs, such as breaking out in a cold sweat, nausea or lightheadedness.

5. As with men, women’s most common heart attack symptom is chest pain or discomfort—but women are somewhat more likely than men to experience some of the other common symptoms, particularly shortness of breath, nausea/vomiting or back or jaw pain.

*Source: American Heart Association*
By 2050, it is estimated that atrial fibrillation (AFib) will affect between 5.6 million and 12 million Americans.

The electrical wiring of your heart—which controls the rate at which your heart beats, every minute, hour and day, 365 days a year—is one of the most sophisticated and enduring pieces of nature’s engineering. However, there are some irregularities that can occur in that wiring, as well as damage that can be caused by disease, all of which can cause symptoms and increase the risk of premature death. Divers, and any physicians who treat them, should be familiar with arrhythmias and their effects on the safety of scuba divers.
The term “arrhythmia” (or, sometimes, “dysrhythmia”) means an abnormal heartbeat. It is used to describe manifestations ranging from benign, harmless conditions to severe, life-threatening disturbances of the heart’s rhythm.

A normal heart beats between 60 and 100 times a minute. In well-trained athletes, or even select nonathletic individuals, the heart may beat at rest as slowly as 40 to 50 times a minute. Even entirely healthy, normal individuals experience occasional extra beats or minor changes in their heart’s rhythm. These can be caused by drugs (such as caffeine) or stress or can occur for no apparent reason. Arrhythmias become serious only when they are prolonged or when they do not result in proper contraction of the heart.

Physiologically significant extra heartbeats may originate in the upper chambers of the heart (this is called “supraventricular tachycardia”) or in the lower chambers of the heart (this is called “ventricular tachycardia”). The cause of these extra beats may be a short circuit or an extra conduction pathway in the heart’s wiring, or it may be the result of some other cardiac disorder. People who have episodes or periods of rapid heartbeat are at risk of losing consciousness during such events. Other people have a fairly stable arrhythmia (such as “fixed atrial fibrillation”), but in conjunction with additional cardiovascular disorders or other health problems that exacerbate the effect of their rhythm disturbance. A too-slow heartbeat (or a heart blockage) may cause symptoms, too.

EFFECT ON DIVING

Serious arrhythmias, like ventricular tachycardia and many types of atrial arrhythmia, are incompatible with diving. The risk for any person who develops an arrhythmia during a dive is, of course, losing consciousness while underwater. Supraventricular tachycardia, for example, is unpredictable in its onset and may even be triggered simply by immersing one’s face in cold water. Anyone who has had more than one episode of this type of arrhythmia should not dive.

Most arrhythmias that require medication also disqualify the affected individual from safe diving. Exceptions may be made on a case-by-case basis, in consultation with a cardiologist and a diving medical officer.

An individual who has any cardiac arrhythmia needs a complete medical evaluation by a cardiologist prior to engaging in diving. In some cases, electrophysiologic studies can identify an abnormal conduction pathway, and the problem can be corrected. Recently, clinicians and researchers have determined that people with some arrhythmias (such as certain types of Wolff-Parkinson-White syndrome, which is characterized by an extra electrical pathway) may safely participate in diving after a thorough evaluation by a cardiologist. Also, in select cases, people with stable atrial arrhythmias (such as uncomplicated atrial fibrillation) may dive safely if a cardiologist determines that they have no other significant health problems.
Syncope is an abrupt loss of consciousness, followed by a relatively quick recovery. The causes of syncope range from relatively benign to life-threatening. It is seldom overlooked and usually precipitates a visit to a medical professional.

Syncope that occurs in or around the water poses particular challenges. Drowning often results when a diver loses consciousness and remains in the water. A rapid response is required to bring an unconscious diver to the surface and prevent death. Syncope can also occur upon exiting the water, due to such factors as exertion, dehydration and normal return of blood volume to the lower extremities.

The initial response to syncope should focus on the ABCs of basic life support: airway, breathing and circulation. Advanced cardiac life support may be called for. Often, placing syncopal patients flat on their back in a cool environment will quickly restore them to consciousness. If syncope occurs following a dive, it is important to consider decompression sickness, pulmonary over-inflation and immersion pulmonary edema in addition to the usual causes of the condition. Although both syncope and cardiac arrest result in a loss of consciousness, they can usually be clearly differentiated.

The list of possible causes of syncope is extensive, but a good medical history can help eliminate the majority of them. The patient’s age, heart rate, family history, medical conditions and medications are key in identifying the cause. If syncope is accompanied by convulsions (known as “tonic-clonic movements”), it may have been precipitated by a seizure. If it occurs upon exertion, a serious cardiac condition may be preventing the heart from keeping up with the demands of the physical activity; chest pain may be associated with this type of syncope. If standing up quickly results in syncope, that points to a cause known as “orthostatic hypotension.” And pain, fear, urination, defecation, eating, coughing or swallowing may cause a variation of the condition known as “reflex syncope.”

A medical evaluation after an incident of syncope should include a thorough history and physical—plus interviews with witnesses who observed the individual's collapse and who can accurately relay the sequence of events. A few cases may require more extensive investigation, and some result in no conclusion.

EFFECT ON DIVING

While a medical evaluation is being conducted, it is recommended that the affected individual refrain from any further diving. The cause of a given syncopal episode can be elusive but must be pursued—especially if the individual hopes to return to diving. Once the underlying factors have been determined, a diving medical officer and appropriate specialists should consider whether diving can be resumed safely.
Heart beats that occur outside the heart’s regular rhythm are known as “extrasystoles.” They often arise in the ventricles, in which case they are referred to as “premature ventricular contractions,” or sometimes “premature ventricular complexes,” abbreviated as PVCs. The cause of such extra beats can be benign or can result from serious underlying heart disease.

PVCs are common even in healthy individuals; they have been recorded in 75 percent of those who undergo prolonged cardiac monitoring (for at least 24 hours, that is). The incidence of PVCs also increases with age; they have been recorded in over 5 percent of individuals more than 40 years old who undergo an electrocardiogram (or ECG, a test that typically takes less than 10 minutes to administer). Men seem to be affected more than women.

The extrasystole itself is usually not felt. It is followed by a pause—a skipped beat—as the heart’s electrical system resets itself. The contraction following the pause is usually more forceful than normal, and this beat is frequently perceived as a palpitation—an unusually rapid or intense beat. If extrasystoles are either sustained or combined with other rhythm abnormalities, affected individuals may also experience dizziness or lightheadedness. Heart palpitations and the sensation of missed or skipped beats are the most common complaints of those who seek medical care for extrasystole.

A medical examination of the condition begins with a history and physical and should also include an ECG and various laboratory tests, including the levels of electrolytes (such as sodium, potassium and chloride) in the blood. In some cases, doctors may recommend an echocardiogram (an ultrasound examination of the heart), a stress test and/or the use of a Holter monitor (a device that records the heart’s electrical activity continuously for a 24- to 48-hour period). Holter monitoring may uncover PVCs that are unifocal—that is, they originate from a single location. Of greater concern are multifocal PVCs—those that arise from multiple locations—as well as those that exhibit specific patterns known R-on-T phenomenon, bigeminy and trigeminy.

If serious structural disorders, such as coronary artery disease or cardiomyopathy (a weakening of the heart muscle), can be ruled out—and the patient remains asymptomatic—the only “treatment” required may be reassurance. But for symptomatic patients, the course is less clear, as there is controversy regarding the effectiveness of the available treatment options. Two drugs commonly used to treat high blood pressure—beta blockers and calcium channel blockers—have been used in patients with extrasystole, with some success. Antiarrhythmics have also been prescribed for extrasystole but have met with mixed reviews. A procedure known as cardiac ablation may be an option for symptomatic patients, if the location where their extra beats arise can be identified; the procedure involves threading tiny electrodes into the heart via catheters, then zapping the affected locations to rewire the heart’s faulty circuits.

EFFECT ON DIVING

Although PVCs are present in a large percentage of otherwise normal individuals, they have been shown to increase mortality over time. If PVCs are detected, it is important that they be investigated and that known associated conditions be ruled out. Divers who experience PVCs and who are found to also have coronary artery disease or cardiomyopathy will put themselves at significant risk if they continue to dive. Divers diagnosed with R-on-T phenomenon, nonsustained runs of ventricular tachycardia or multifocal PVCs should likewise refrain from diving. Divers who experience PVCs but remain asymptomatic may be able to consider a return to diving; such individuals should discuss with their cardiologist their medical findings, their desire to continue diving and their clear understanding of the risks involved.
Atrial fibrillation (AF or AFib), the most common form of arrhythmia, is characterized by a fast and irregular heartbeat. It results from a disturbance of the electrical signals that normally make the heart contract in a controlled rhythm. Instead, chaotic and rapid impulses cause uncoordinated atrial filling and ventricle pumping action. This leads to a decrease in overall cardiac output, which can affect one’s exercise capacity or even result in unconsciousness. In addition, AF causes blood to pool in the atria, which promotes the formation of blood clots that may break loose and enter the circulatory system; if this occurs, it may result in a stroke.

Recent U.S. studies have shown a rising incidence of AF overall, as well as significant racial differences in its prevalence. The lifetime risk of AF (at 80 years of age) was recently found to be 21 percent in white men and 17 percent in white women, but only 11 percent in African-Americans of both sexes. By 2050, it is estimated that AF will affect between 5.6 million and 12 million Americans. These figures are significant, because AF is associated with a fourfold to fivefold higher risk of ischemic stroke. Individuals with AF, after adjustment for other risk factors, also have a twofold higher risk of dementia.

The most common causes of AF are hypertension and coronary artery disease. Additional causes include a history of valvular disorders, hypertrophic cardiomyopathy (a thickening of the heart’s muscle), deep vein thrombosis (DVT), pulmonary embolism, obesity, hyperthyroidism (also called “overactive thyroid”), heavy alcohol consumption, an imbalance of electrolytes in the blood, cardiac surgery and heart failure.

Some people with AF experience no symptoms and are unaware they have the condition until it’s discovered during a physical examination. Others may experience symptoms such as the following:

- Palpitations (a racing, uncomfortable, irregular heartbeat or a flip-flopping sensation in the chest)
- Weakness
- Reduced ability to exercise
- Fatigue
- Lightheadedness
- Dizziness
- Confusion
- Shortness of breath
- Chest pain
The occurrence and duration of atrial fibrillation usually falls into one of three patterns:

- **OCCASIONAL (OR “PAROXYSMAL”):** The rhythm disturbance and its symptoms come and go, lasting for a few minutes to a few hours, and then stop on their own. Such events may occur a couple of times a year, and their frequency typically increases over time.

- **PERSISTENT:** The heart’s rhythm doesn’t go back to normal on its own, and treatment—such as an electrical shock or medication—is required to restore a normal rhythm.

- **PERMANENT:** The heart’s rhythm can’t be restored to normal. Treatment may be required to control the heart rate, and medication may be prescribed to prevent the formation of blood clots.

Any new case of AF should be investigated and its cause determined. An investigation may include a physical exam; an electrocardiogram; a measurement of electrolyte levels, including magnesium; a thyroid-hormone test; an echocardiogram; a complete blood count; and/or a chest X-ray.

Treating the underlying cause of AF can help control the fibrillation. Various medications, including beta blockers, may help regulate the heart rate. A procedure known as cardioversion—which can be performed with either a mild electrical shock or medication—may prompt the heart to revert to a normal rhythm; before cardioversion is attempted, it is essential to ensure that a clot has not formed in the atrium. Cardiac ablation, which is described in the section on extrasystole (see page 36), may also be used to treat AF. In addition, anticoagulant drugs are often prescribed for individuals with AF to prevent the formation of clots and thus reduce their risk of stroke. It is also of note that the neurological effects of an embolic stroke associated with AF can sometimes be confused with the symptoms of decompression sickness.

**EFFECT ON DIVING**

A thorough medical examination should be conducted to identify the underlying cause of the atrial fibrillation. It is often that underlying cause which is of most concern regarding fitness to dive. But even atrial fibrillation itself can have a significant impact on cardiac output and therefore on maximum exercise capacity. Individuals who experience recurrent episodes of symptomatic AF should refrain from further diving. The medications often used to control atrial fibrillation can present their own problems, by causing other arrhythmias and/or impairing the individual’s exercise capacity. It is essential that anyone diagnosed with AF have a detailed discussion with a cardiologist before resuming diving.
Sudden cardiac arrest (SCA)—a cessation of the heart’s beating action, with little or no warming—is an acute medical emergency. During the arrest, blood stops circulating to the body’s vital organs, including the brain, the kidneys and the heart itself. Cut off from oxygen, these organs die within minutes. If the arrest is not corrected quickly, the affected individual will not survive.

The causes of SCA include myocardial infarction (heart attack), heart failure, drowning, coronary artery disease, electrolyte abnormalities, drugs, abnormalities in the heart’s electrical conduction system, cardiomyopathy (a weakening of the heart muscle) and embolism (a clot that has lodged in a major vessel).

SCA accounts for 450,000 deaths in the United States each year and for 63 percent of cardiac deaths in Americans more than 35 years old. The risk of sudden cardiac death in adults increases as much as sixfold with increasing age, paralleling the rising incidence of ischemic heart disease. The risk of SCA is greater in those with structural heart diseases, but in 50 percent of sudden cardiac deaths, the victim had no awareness of having heart disease, and in 20 percent of autopsies conducted following such deaths, no structural cardiovascular abnormalities were found.

Though there is typically little warning before a sudden cardiac arrest, occasionally the individual may experience lightheadedness, difficulty breathing, palpitations or chest pain.

Immediate treatment should be focused on restoring circulation quickly using chest compressions or CPR and defibrillation. Following resuscitation, the victim should be transported to a hospital as soon as possible. Subsequent treatment may consist of efforts to eliminate the underlying cause of the arrest through administration of medication, surgery or the use of implanted electrical devices.

Preventive strategies include learning to recognize the warning signs of SCA, in case they occur; identifying, eliminating or controlling any risk factors that may affect you; and scheduling regular physical exams, as well as appropriate testing, when it is indicated.

**EFFECT ON DIVING**

Divers with any symptoms of cardiovascular disease should be evaluated by a cardiologist and a dive-medicine specialist regarding their continued participation in diving. In asymptomatic individuals, the risk of SCA may be evaluated by using known cardiovascular risk factors like smoking, high blood pressure, high cholesterol, diabetes, lack of exercise and overweight. For example, people who smoke have two and a half times the risk of suffering sudden cardiac death than do nonsmokers.
A pacemaker is a small battery-operated device that helps an individual's heart beat in a regular rhythm. It does this by generating a slight electrical current that stimulates the heart to beat. The device is implanted under the skin of the chest, just below the collarbone, and is hooked up to heart with tiny wires that are threaded into the organ through its major vessels. In some individuals, the heart may need only intermittent help from the pacemaker, if the pause between two beats becomes too long. In others, however, the heart may depend completely on the pacemaker for regular stimulation of its beating action.

**EFFECT ON DIVING**

Every case involving a pacemaker must be evaluated individually. The two most important factors to take into account are the following:

1. Why is the individual dependent on a pacemaker?

2. Is the individual's pacemaker rated to perform at depths (in other words, pressures) compatible with recreational diving—plus an added margin of safety?

The reason for the second factor is that a pacemaker is implanted in tissues just under the skin and thus is exposed during a dive to the same ambient pressures as the diver. For safe diving, a pacemaker must be rated to perform at a depth of at least 130 feet (40 meters) and must also operate satisfactorily during conditions of relatively rapid pressure changes, such as would be experienced during ascent and descent.

As with any medication or medical device, the underlying problem that led to the implantation of the pacemaker is the most significant factor in determining someone's fitness to dive. The need to have a pacemaker implanted usually indicates a serious disturbance in the heart's own conduction system.

If the disturbance arose from structural damage to the heart muscle itself, as is often the case when someone suffers a major heart attack, the individual may lack the cardiovascular fitness to dive safely.

Some individuals, however, depend on a pacemaker not because the heart muscle has been damaged but simply because the area that generates the impulses which make the heart muscle contract does not function consistently or adequately. Or the circuitry that conducts the impulses to the heart muscle may be faulty, resulting in improper or irregular signals. Without the assistance of a pacemaker, such individuals might suffer episodes of syncope (fainting). Others may have suffered a heart attack mild enough that they sustained minimal residual damage to their heart muscle, but their conduction system remains unreliable and thus needs a boost from a pacemaker.

If a cardiologist determines that an individual's level of cardiovascular fitness is sufficient for safe diving, and the individual's pacemaker is rated to function at a pressure of at least 130 feet (40 meters), that individual may be considered fit for recreational diving. But once again, it cannot be emphasized strongly enough that any divers with cardiac issues check with their doctor before diving.
Your lungs have many functions in your body, beyond just oxygenating your blood. One of their other important roles is filtering the venous blood that returns from the body. The venous system is characterized by slower blood flow than the arterial system, which contributes to the occasional formation of a blood clot (known as a “peripheral venous thrombosis”), which could be transported into the lungs and could even cause a pulmonary embolism (or blockage in the vessels of the lungs).
Deep vein thrombosis (DVT) is a condition in which a blood clot (a “thrombus”) forms in one or more of the body’s deep veins, usually in the legs. If a clot breaks free and travels through the circulatory system, it can lead to life-threatening conditions. For example, if a clot lodges in the lungs, it is known as a pulmonary embolism (PE) and affects the lungs’ ability to oxygenate the blood (see page 44 for more on PE). Collectively, DVT and PE are sometimes referred to as venous thromboembolisms (VTEs).

A clot that originates as a DVT can also cause a stroke in individuals with a patent foramen ovale (PFO, a hole in the wall between their atria—see page 26 for details about this condition); in such a case, the clot travels through the veins to the right atrium of the heart, passes through the PFO to the left atrium and thence travels through the arteries to the brain.

DVT is not related to diving, but divers often travel, and travel is a significant risk factor for DVT. In about half of all cases of DVT, the individual experiences no noticeable symptoms before the onset of the condition. Most often, it starts in the calf. Symptoms may include the following:

- Swelling in the affected leg, ankle or foot.
- Pain in the calf that spreads to the ankle or foot.
- Warmth in the affected area.
- A change in the color of the skin—to pale, red or blue.

Most VTEs related to air travel occur within two weeks of the flight and are resolved within eight weeks. If untreated, a DVT that starts in the calf will spread up into the thigh and pelvis in about 25 percent of cases. And an untreated DVT of the thigh and pelvis has about a 50-percent risk of leading to a PE, which is the most serious complication of DVT. Many cases of DVT are asymptomatic and resolve spontaneously. However, DVT often recurs in an individual who has had one episode of the condition.

Most DVTs occur in individuals with pre-existing risk factors for DVT who remain motionless for a long time—such as when traveling a long distance by plane, car or train; when doing deskwork over a period of many hours; or when bedridden. This is because immobility slows down the blood flow in the veins (a condition known as “venous stasis”); in addition, pressure on the calf from an inadequate seat can injure the vein walls. If you sit still for 90 minutes, the blood flow in your calf drops by half, and that doubles your chance of developing a blood clot. For every additional hour you spend sitting, your risk of a blood clot increases by 10 percent.
The incidence of DVT in the general population is one-tenth of one percent, but it is higher in those who have risk factors and those who travel often. Long-distance air travel may double, or even quadruple, the risk of suffering a VTE. Although DVT is often called the “economy class disease,” business-class travelers are susceptible, too. The risk of a DVT occurring on a flight lasting more than four hours is between 1 in 4,650 flights and 1 in 6,000 flights; this is lower than the risk in the general population, but that’s because people who take long trips are likely to be healthier than average. The incidence of DVT among travelers with a low to intermediate pre-existing risk for VTE who take a journey longer than eight hours was found to be 0.3 percent for symptomatic cases and 0.5 percent when including asymptomatic cases as well.

Risk factors for DVT include the following:

- Older age (the risk rises after age 40).
- Obesity (defined as a body mass index greater than 30).
- Estrogen use (either hormonal contraceptives or hormone replacement therapy).
- Pregnancy (including the postpartum period).
- Thrombophilia (an abnormally increased tendency of the blood to clot).
- Previous VTE or a family history of VTE.
- Active cancer.
- Serious medical illness.
- Recent surgery, hospitalization or trauma.
- Limited mobility.
- Central venous catheterization (the presence of a catheter in one’s chest, for use in administering medication or nutrients and/or drawing blood samples).

Between 75 percent and 99 percent of those who develop a travel-related VTE had more than one of these risk factors.

Height is also a factor in one’s risk of developing a travel-related DVT. People who are either very short—less than 5 feet, 3 inches (1.6 meters)—or very tall—more than 6 feet, 3 inches (1.9 meters)—appear to be at increased risk as a result of their inability to adjust their seats sufficiently to accommodate their height. In addition to effects of immobility, shorter passengers may suffer greater than usual seat-edge pressure on the backs of their knees, and taller passengers may be cramped due to insufficient leg room. All of these factors can contribute to injury of deep veins, venous stasis and activation of the blood’s clotting mechanisms.

Those who are at increased risk of DVT should wear compression socks whenever they fly or drive long distances and should consult their primary-care provider regarding the possible benefit of taking a clot-preventative like aspirin. Although the risk of DVT for healthy people is small, everybody should be aware of the factors that can precipitate the condition—and avoid long periods of immobility. The best way to prevent DVT is to get up and walk around from time to time. It also helps to flex your feet and calf muscles regularly if you must remain seated for any length of time. Finally, it is also helpful in preventing DVT to stay well hydrated.

**EFFECT ON DIVING**

Any individual who has been diagnosed with acute DVT or who is taking anticoagulants should refrain from diving. It may be possible to return to safe diving after having a DVT, but the evaluation of fitness to dive must be made on an individual basis.
A pulmonary embolism (PE) is an obstruction (or “embolus”) that lodges in the vasculature of the pulmonary system, or lungs. The embolus may be air, fat or a blood clot (or “thrombus”). If a PE is caused by a thrombus, the clot typically originated in the deep vein system of the legs—a condition known as deep vein thrombosis (DVT); see the preceding section for a discussion of DVT. The resulting obstruction in the flow of blood to the lungs typically causes a drop in cardiac output and a significant drop in blood pressure.

The onset of PE can be acute or chronic. Acute PE often causes symptoms evident to the individual, while chronic-onset PE frequently reveals its presence only with very subtle findings that went unnoticed by the affected individual. An untreated PE has a high mortality rate. An especially grim prognosis applies to individuals who have a concurrent DVT, right ventricular thrombus or right ventricular dysfunction. An estimated 1.5 percent of all deaths are diagnosed as being due to PE.

Risk factors for DVT—and thus for PE—include recent surgery; a stroke; a diagnosis of autoimmune disease, malignancy or heart disease; obesity; smoking; hypertension; and a previous DVT.

Symptoms of PE include chest pain (also known as “dyspnea”), pain or swelling of the calf (signaling a DVT), hypotension (abnormally low blood pressure), an altered level of consciousness and syncope (fainting). Distension of the neck veins in the absence of other conditions—such as pneumothorax (a buildup of air in the membrane surrounding the lungs, sometimes referred to as a collapsed lung) or heart failure—may also be observed in individuals suffering a PE.

PE should be one of the first conditions considered when attempting to make a diagnosis in someone exhibiting acute onset of any of the symptoms listed above and any of the associated risk factors. Appropriate diagnostic tests may include measurement of the individual’s levels of a hormone called brain natriuretic peptide (BNP) and of a protein known as cardiac troponin, as well as a CT angiogram of the lungs.

Treatment should focus initially on managing the significant cardiopulmonary impairments that are usually involved in a PE. Such care may include breathing support from an artificial ventilator and fluid management. The use of anticoagulant medication is also important, both to treat the embolus and to stop the development of another thrombus. Thrombolysis (known as “clot-busting”), embolectomy (surgical removal of the embolus) or the placement in the vena cava (one of the large vessels in the chest) of a filter designed to prevent any future clots from reaching the lungs may also be considered—especially in anyone who goes into shock, because mortality in such cases approaches 50 percent. Similar measures may be called for in cases of PE caused by a venous gas bubble. Hyperbaric oxygen therapy may be indicated as well, if the individual’s condition does not improve or deteriorates even after the application of supportive measures.

**EFFECT ON DIVING**

Despite many medical advances, five-year all-cause mortality in individuals who have suffered a PE due to underlying risk factors remains over 30 percent. And pulmonary hypertension—elevated pressure in the arteries that carry blood from the heart to the lungs, a condition that limits one’s exercise capacity—often persists in individuals who have had a PE, even after successful treatment. Thus any determination of fitness for diving by those who have had a PE must include an evaluation of their lung function, underlying conditions, anticoagulation status, exercise capacity and cardiac status.
Immersion pulmonary edema (IPE) is a form of pulmonary edema—an accumulation of fluid in the tissues of the lungs—that specifically affects divers and swimmers. Immersion at depth is a key factor in the development of IPE. That's because immersion in an upright position causes a significant shift of fluid from the peripheral to the central circulatory system, resulting in higher pressure in the capillaries of the pulmonary system. Elements of the diving milieu that contribute to IPE's occurrence include the fact that divers breathe gases that are denser than air at sea level which means more negative pressure within chest is needed to inhale; the likelihood of gas bubbles becoming trapped in the vasculature of the lungs; the cold underwater environment; and the potential in underwater settings for exertion or panic, which can exacerbate elevated capillary pressure.

Maintaining a proper fluid balance in your lung tissue and its vasculature requires a dynamic combination of various opposing forces. Unopposed changes in any of these forces can result in a buildup of excess fluid—or edema—in your pulmonary tissue. The main variables involved in regulating this fluid balance are the following:

- Oncotic pressure (a form of pressure exerted by proteins) in the pulmonary capillaries, the tiniest vessels of the circulatory system.
- Oncotic pressure in the pulmonary system's interstitial fluid (fluid in the cavities of your lung tissue).
- Permeability of the pulmonary capillaries.
- Hydrostatic pressure (the pressure of a fluid at rest) in the pulmonary capillaries.
- Hydraulic pressure (the pressure of a fluid that is being compressed or pumped) in the interstitial fluid.
- Pressure in the alveoli, the tiny air sacs of the lungs.

These factors, which collectively are known as “Starling forces,” can all be quantified and placed in an equation that can then be used to calculate the net differential of the forces.

Pulmonary edema is caused by changes in these forces—such as a drop in the levels of key proteins in the blood; leakage from the pulmonary capillaries due to sepsis (a life-threatening complication of infections); an increase in hydrostatic pressure in the pulmonary capillaries due to heart failure; and negative pressure in the alveoli due to resistance from breathing through a faulty regulator. Additional issues that can contribute to the development of pulmonary edema include side effects of some cardiovascular drugs; ARDS (acute respiratory distress syndrome, a life-threatening condition that prevents oxygen from getting to the lungs); reperfusion (a procedure that restores circulation after a heart attack or stroke); cardiomyopathy (a weakening of the heart muscle); high-altitude pulmonary edema; a pulmonary embolus (a blood clot lodged in a vessel in the lungs); re-expansion (the re-inflation of a collapsed lung); pulmonary hypertension (elevated pressure in the arteries that carry blood from the heart to the lungs); lung cancer; hemorrhage (uncontrolled bleeding); and various...
disorders of the nervous system. Other factors can include overhydration by well-intentioned divers who’ve heard the conventional wisdom that dehydration is a risk factor for decompression sickness, as well as poor physical conditioning, which can result in increased negative pressure in the alveoli during deep inspiration.

The symptoms of IPE include chest pain; dyspnea (discomfort or difficulty breathing); wheezing; and pink, frothy sputum while submerged or shortly after emerging from the water. Most people who suffer an episode of IPE had no significant history or signs that would indicate a susceptibility to the condition; nevertheless, the risk of IPE does rise with age, obesity and elevated blood pressure.

Once pulmonary edema occurs, hypoxia (lack of an adequate supply of oxygen) leads to constriction of the pulmonary vasculature, which worsens the cascade of ill effects. The situation can be further aggravated by the accompanying dyspnea, which, when experienced underwater, can induce panic and uncontrolled ascent to the surface—leading to overinflation of the lungs and even near-drowning.

To help differentiate immersion pulmonary edema from other conditions with similar symptoms (such as near-drowning, pulmonary decompression sickness and pulmonary overinflation syndrome), it is important to keep in mind that IPE’s onset can occur either at depth or upon reaching the surface. And it is not necessarily precipitated by aggressive diving, a rapid ascent or the aspiration of water.

Treatment for IPE should begin with removal of the affected individual from the water (to relieve the compression of the vessels in the lower extremities, allowing centrally pooled fluids to return to the extremities) and with administration of oxygen (beginning at 100 percent and later at a reduced concentration). A diuretic such as Lasix may help to reduce excess intravascular fluid, although diuresis—the body’s natural excretion of fluid—may already be under way as a result of hormonal influences. The condition usually resolves quickly in a healthy diver. Prolonged hospitalization is rarely required; if it is necessary, it’s usually due to contributing factors, such as an underlying cardiac problem.

EFFECT ON DIVING
Some divers have one episode of IPE and never experience the condition again, but repeated episodes are likely. Any individuals who suffer a first episode of IPE are advised to undergo a detailed examination to rule out any medical conditions that may have caused the edema and then to have a thorough discussion with their physician regarding the risks of continuing to dive. And all divers are urged to have regular maintenance on their regulators, to refrain from overhydration and to attend to proper dive planning in order to avoid exertion and panic—as well as to keep such conditions as obesity and hypertension under control.
Divers who take drugs to treat cardiovascular diseases often worry about the drugs’ compatibility with diving. However, in most cases, it is the underlying condition and not the drug that should cause concern.

Some drugs may have side effects that preclude diving. Divers should be very familiar with the side effects of any drugs they take and should discuss them with their physician—and be sure their physician knows about their diving activities. A one-time clearance to dive does not preclude progression of a disease, so any changes in your health status should prompt another medical examination before you dive again.
Antiplatelets and anticoagulants are two classes of drugs—popularly known as “blood thinners”—that reduce the risk of clot formation and thus the risk of deep vein thrombosis, pulmonary embolism, heart attack and stroke. They may also be prescribed for individuals who have been diagnosed with atrial fibrillation or for those who have had heart-valve surgery or who have received a stent, an implanted pacemaker or an implanted defibrillator. (See other chapters for detailed descriptions of these conditions.)

Clots form when blood cells known as platelets stick together, and then proteins in the blood bind them together into a solid mass. Clotting is a normal function that limits and stops bleeding when a blood vessel is injured. However, if a clot grows out of control or starts to travel within the circulatory system, it then poses a danger. Clots may get lodged in a pulmonary artery and cause a pulmonary embolism; in the arteries of the heart and cause a heart attack; or in the vessels of the brain and cause a stroke. All of these events can be life-threatening.

Antiplatelets and anticoagulants keep blood from clotting as quickly or as effectively as usual by preventing the platelets from adhering to one another and by preventing the clotting proteins from binding together. They can even help to break up clots that have already formed.

Antiplatelets—such as aspirin and clopidogrel (also known by the brand name Plavix)—work by preventing the platelets from adhering to one another.

Anticoagulants—such as heparin and warfarin (Coumadin)—inhibit the action of the clotting proteins and thus slow down the chemical reactions that lead to the formation of a clot. There were also several new anticoagulants approved between 2010 and 2012, including rivaroxaban (Xarelto), dabigatran (Pradaxa) and apixaban (Eliquis).

The major side effect of all antiplatelets and anticoagulants is excessive bleeding. Those taking such drugs—especially at too high a dosage—may bleed or bruise easily or may experience bleeding that does not stop as quickly as usual.

SPECIAL CAUTION REGARDING WARFARIN

Individuals who take warfarin (Coumadin) are generally advised to avoid any activities that may cause abrasions, bruising or cuts—such as contact sports. They are also urged to exercise caution while brushing their teeth and shaving. Even such trivial injuries as insect bites may cause complications in anyone taking warfarin.

There are additional risks involving warfarin particular to diving. Most significantly, there is an appreciable chance of serious injury in any diving environment, despite one’s best efforts to mitigate the risk. Cuts and bruises are unavoidable, for example. And in anyone taking warfarin, a decompression injury or difficulty equalizing ear pressure could cause bleeding in the ears or the spinal cord that would otherwise not occur.

In addition, both travel and any resulting dietary disruption can interfere with the action of warfarin in dangerous ways. Furthermore, the health-care capabilities in many popular dive destinations may not be up to providing the care that would be required in case of an adverse event.

For all these reasons, anyone taking warfarin is generally advised not to dive. Nevertheless, many people who take warfarin are able to dive without major complications. The keys to safe diving while using warfarin are strict adherence to monthly blood tests and regular surveillance by a physician. With good control of blood-thinning, the risk of a bleeding complication is quite low.

According to Dr. Alfred Bove, a dive-medicine specialist, “For divers, the most important question is whether the condition which requires the use of Coumadin or Plavix prohibits diving. In many cases, the illness is over, or chronic but well adjusted, and does not interfere with safe recreational diving. Safe diving with Coumadin or Plavix depends on the absence of illness which would limit diving, careful control of clotting time, avoiding ear or sinus squeeze, and a thorough education on drugs and foods which cause changes in the effects of Coumadin. There are many divers using Coumadin and Plavix safely, but a special effort must be made to understand how to avoid problems of excess or not enough anticoagulation.”
Statins are a class of drugs prescribed to lower high blood cholesterol and thus prevent heart attack and stroke. They reduce both LDL cholesterol (“bad cholesterol”) and inflammation in the arteries. Statins work by inhibiting a liver enzyme that is involved in the production of cholesterol. Though they are most effective at lowering LDL cholesterol, they may also contribute to raising HDL cholesterol (“good cholesterol”).

Common statins include the following—listed first by their generic name and, in parentheses, their brand name:

- Atorvastatin (Lipitor)
- Cholestipol (Cholestid)
- Colesevelam hydrochloride (Welchol)
- Fluvastatin (Lescol)
- Lovastatin (Mevacor)
- Ezetimibe (Zetia)
- Ezetimibe combined with simvastatin (Vytorin)
- Fenofibrate (Tricor)
- Pravastatin (Pravachol)
- Rosuvastatin (Crestor)
- Simvastatin (Zocor)

Clinical trials sponsored by the companies that manufacture these drugs have found rare and mild side effects. In a carefully designed trial known as IDEAL, however, almost 90 percent of subjects reported side effects, almost half of them serious. Adverse effects of statins that have been noted in the medical literature and that could interfere with diving include the following:

- Dyspnea (discomfort or difficulty breathing).
- Muscle pain.
- Tendon complications.
- Digestive problems.
- Rush or flashing.
- Increased blood sugar or type 2 diabetes.
- Cognitive dysfunction (some studies report that up to 75 percent of those on statins experienced cognitive dysfunction determined to be probably or definitely related to the statin therapy; the severity of the cognitive deficits were clearly related to statin potency).
- Fatigue (almost half of those in a 2012 study reported a significant increase in fatigue while taking statins).

The most common statin side effect is muscle pain. It occurs in about 20 percent of those taking statins. This pain may feel like aches, soreness, tiredness or weakness in your muscles. The pain is sometimes described as mild discomfort, but it is sometimes severe enough to make daily activities difficult. Scientists suspect the occurrence of pain is due to the fact that statins block production of a molecule the body uses to generate energy, called CoQ10; clinical trials are currently exploring whether taking CoQ10 supplements can prevent this side effect. Routine use of CoQ10 supplementation is not recommended, however, even though there are few safety concerns with such supplementation.

Very rarely, statins can lead to a kind of life-threatening muscle damage called rhabdomyolysis; it causes severe muscle pain and may result in liver damage, kidney failure and death. Rhabdomyolysis is especially likely to occur in those who take statins in combination with other drugs such as antibiotics and antidepressants or in those who take a high dose of statins.

Some people who take statins may develop nausea, gas, diarrhea or constipation. These side effects are rare.

A rash or flushing can also occur after taking a statin. This is more likely to occur in individuals who take a statin and niacin together, either in a combination pill such as Simcor or as two separate medications.

The FDA warns on statin labels that some people taking statins have developed memory loss or confusion; these effects are reversed when the medication is halted. Conversely, there has also been evidence that statins may help with brain function—in patients with dementia or Alzheimer’s, for example. This effect is still being studied.
But no matter what side effects individuals taking statins may experience, it is important that they not stop taking the medication without talking to their doctor. It is also important that those who take statins minimize changes in their lifestyle, diet and over-the-counter medications, especially during dive-related travel.

Risk factors for statin side effects include the following:

- Taking multiple cholesterol-lowering medications.
- Being 65 or older, female or having a smaller body frame.
- Having kidney or liver disease or type 1 or 2 diabetes.
- Drinking too much alcohol (more than two drinks a day for men age 65 and younger or more than one drink a day for women of all ages and men older than 65).

In addition, problems are more likely in those who take both statins and the following drugs:

- Antimalarials, such as chloroquine and hydroxychloroquine (Plaquenil).
- Thyroid medications.

**EFFECT ON DIVING**

Though the side effects of some statins may interfere with diving, they may nevertheless offer overall health benefits.

One of the effects of statins is an increase in the body’s production of nitric oxide. This helps to preserve the integrity of the endothelium (the inner lining of the blood vessels), to reduce injury from ischemia and/or reperfusion (a procedure that restores circulation after a heart attack or stroke) and to depress interdependent inflammatory and coagulation activity—all of which could provide protection against decompression sickness (DCS). On the other hand, a study of healthy divers who took either a statin or a placebo several days before a dive found no difference in their risk of postdive venous gas bubbles. Thus taking statins specifically to prevent DCS does not appear to offer any benefit, especially in view of the possibility of adverse side effects.

If you are over age 45 and are already taking statins for medical reasons, you need to answer “yes” to at least two questions on the Recreational Scuba Training Council (RSTC) Medical Statement (see page 14 for details regarding this form):

**Q:** Are you presently taking prescription medications? (with the exception of birth control or anti-malarial)

**Q:** Are you over 45 years of age and can answer YES to one or more of the following?

- Have a high cholesterol level

These two positive responses signal a pre-existing condition that may affect your safety while diving and that calls for a thorough medical examination to check for the presence of other risk factors or signs of cardiovascular disease. Indeed, anyone who is over age 45, who is at high risk of cardiac problems or who has any signs of cardiovascular disease should see a physician at least once every year.

It is also of note that one-time clearance to dive does not preclude progression of a disease, so any changes in your health status should prompt another medical examination before you dive again. Divers should also be mindful of the fact that they may be required to fill out a new RSTC Medical Statement before any dive, and that they may be denied permission to dive based on their responses. However, most dive operators will accept evidence of recent medical clearance for diving. If you are ever in any doubt about your fitness to dive, discuss your status with your dive operator in advance.
There are a number of drugs that can be used to lower hypertension (which is also referred to as high blood pressure). Their side effects vary, so some are more suited than others for use by divers.

### BETA BLOCKERS
Beta blockers are commonly prescribed to treat hypertension, but they have a big drawback for divers: they can reduce the heart's capacity for exercise. If a medication restricts the heart’s function during exercise, then there is an increased risk of loss of consciousness—which could prove fatal while diving.

Because of this effect, doctors often recommend that those who take beta blockers undergo a stress test before diving. According to Dr. Alfred Bove, a dive-medicine specialist, divers who take beta blockers but who can achieve a strenuous level of exercise without severe fatigue can be cleared for diving. Bove also points out that although diving does not usually represent the maximum workload on an individual’s heart, anyone who takes beta blockers should avoid extreme exercise because their maximum exercise capacity may be reduced.

### ACE INHIBITORS
Drugs known as ACE (angiotension-converting enzyme) inhibitors have less effect on exercise capacity than beta blockers, so many doctors prescribe them for people who exercise frequently. But although ACE inhibitors seem to have fewer adverse effects, they can lead to a cough or to airway swelling—conditions that could cause severe problems underwater. If a cough related to ACE-inhibitor use persists, many physicians will recommend a different medication. ACE inhibitors should also be avoided by anyone with kidney disease.

### CALCIUM CHANNEL BLOCKERS
Calcium channel blockers don’t typically pose problems for divers; they relax the walls of the blood vessels, reducing resistance to the flow of blood and thus lowering blood pressure. However, some individuals who take calcium channel blockers, especially in moderate doses, find that a change of position from sitting or lying down to standing causes a drop in blood pressure and thus momentary dizziness. This effect may be cause for concern in divers, but calcium channel blockers appear to have no other adverse implications for diving.

### DIURETICS
Diuretics reduce the amount of excess water and salt in the body; the decline in the volume of bodily fluids results in a lowering of the blood pressure. Divers seem to have very little trouble with diuretics, although in very warm environments, they may cause excessive water loss and thus dehydration. Because dehydration seems to contribute to the risk of decompression sickness, divers may want to reduce their diuretic dosage on days that they engage in diving—though they should check with their doctor before doing so.
Antiarrhythmics are designed to help your heart maintain a stable rhythm. Dr. Alfred Bove, a dive-medicine specialist, warns that some antiarrhythmics, when combined with exercise and lowered levels of potassium, could increase the risk of injury to the heart. Although such drugs do not normally interfere with diving, the arrhythmia for which the drug is being taken may itself preclude safe diving. A thorough consultation with a cardiologist and a dive-medicine specialist is essential if you take medication to control an abnormal heart rate and wish to consider diving.
Further Readings and Sources


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“How much physical activity do adults need?” Centers for Disease Control and Prevention.


“Primary and secondary prevention of coronary artery disease” by T.N. Mohamad. Medscape.com; September 2014.


THE EARS & DIVING:
Ear injuries are the leading cause of injury among scuba divers. Many of these injuries can be easily prevented. The Ears & Diving reference book examines the complex anatomy of the ear, proper equalization techniques, symptoms of injury, medical conditions and the importance of good aural hygiene in the preventative care and management of this vital organ.

HAZARDOUS MARINE LIFE:
While exciting, observing marine life in their environment comes with a risk. Injuries, though rare, may occur as a result of an uninformed swimmer or diver's actions. The Hazardous Marine Life reference book examines the most common hazardous marine life that water enthusiasts may encounter and introduces the mechanisms of injury, techniques for injury prevention and application of first aid.

DECOMPRESSION SICKNESS:
Decompression sickness (DCS) is an unwanted outcome of diving. Measures to mitigate the risk of DCS have to be a part of every dive. This booklet provides updated concepts of causes and mechanisms, typical manifestations, standard management and prevention of DCS.