Sports medicine practitioners who care for a wide array of athletes and active individuals will consistently face issues regarding chronic cardiovascular diseases and their associated risk factors. Among these, hypertension, obesity, and dyslipidemia are common clinical conditions that may be encountered even amongst elite caliber athletes. Consequently, those entrusted with the care of this active population must recognize the presence of these disorders and feel comfortable with their management in the face of continued sports or exercise participation. This article reviews the pathophysiology of these conditions as they relate to athletes and outlines the value of continued exercise in the management of each of these entities while addressing the specific and unique treatment needs of active individuals.

**Hypertension**

Approximately 50 million Americans are hypertensive [1], and hypertension is the most common cardiovascular condition in competitive athletes [2]. A thorough understanding of the pathophysiology of the condition, especially as it relates to the effects of exercise, is essential for anyone who cares for active patients. With this understanding, physicians may ensure that active hypertensives continue to exercise and compete while guarding against the development of long-term complications.

**Pathophysiology of hypertension**

Hypertension is defined as blood pressure equal to or greater than 140/90 mm Hg with or without antihypertensive medication use. Classification criteria for the different stages of hypertension are provided in Table 1. Accurate, systematic,
and consistent blood pressure values are necessary to confirm the diagnosis. The evaluation of the hypertensive athlete should then include a thorough personal and family history, a physical examination with special attention to the cardiovascular system, kidneys, and thyroid, and appropriate laboratory and electrocardiographic tests, as listed below [2,3].

Components of a systematic evaluation for hypertension

Blood pressure measurement technique
- Patient seated, with arm bared and back supported
- No tobacco or caffeine within 30 minutes of measurement
- Patient rests 5 minutes before measurement
- Bladder of cuff encircles at least 80% of patient’s arm
- Multiple readings, when necessary, are separated by 2 minutes each and averaged

History
- Symptoms of hypertension-related conditions (chest pain, dyspnea, orthopnea, poor exercise tolerance)
- Family history of hypertension and its comorbidities
- Weight change
- Type and intensity of exercise
- Diet
- Alcohol and tobacco use
- Caffeine intake
- Illicit drug use (eg, cocaine, amphetamines, anabolic-androgenic steroids, erythropoietin)
- Use of sympathomimetic agents (eg, nasal decongestants), non-steroidal anti-inflammatory, or appetite suppressants or other stimulants (eg, ephedrine, ma huang)

Table 1

<table>
<thead>
<tr>
<th>Category</th>
<th>Systolic</th>
<th>Diastolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optimal</td>
<td>&lt; 120</td>
<td>&lt; 80</td>
</tr>
<tr>
<td>Normal</td>
<td>&lt; 130</td>
<td>&lt; 85</td>
</tr>
<tr>
<td>High-normal*</td>
<td>130–139</td>
<td>85–89</td>
</tr>
<tr>
<td>Stage 1* HTN</td>
<td>140–159</td>
<td>90–99</td>
</tr>
<tr>
<td>Stage 2* HTN</td>
<td>160–179</td>
<td>100–109</td>
</tr>
<tr>
<td>Stage 3* HTN</td>
<td>&gt; 180</td>
<td>&gt; 110</td>
</tr>
</tbody>
</table>

Abbreviation: HTN = hypertension.

* Elevated systolic or diastolic pressure alone is sufficient to meet the criterion.

Physical examination (for signs of end-organ damage or secondary causes)
- S4 gallop
- Arterial bruits, particularly renal
- Peripheral pulses
- Tachycardia
- Hypertensive retinopathy
- Exophthalmos
- Thyroid abnormalities
- Tremor

Diagnostic tests (in all hypertensive patients)
- Complete blood count
- Lipid profile
- Serum electrolytes, glucose, blood urea nitrogen, and creatinine
- Urinalysis for hematuria and proteinuria
- Electrocardiogram

Ninety-five percent of these individuals will be diagnosed with “essential” hypertension where no identifiable cause can be found. Their elevated blood pressure results from increases in total peripheral resistance mediated by plasma epinephrine and norepinephrine [4] and the renin-angiotensin system. In the other 5% of cases, a secondary cause of hypertension, as listed below, may be implicated.

Secondary causes of hypertension
- Androgen or growth hormone use
- Coarctation of the aorta
- Cushing’s disease
- Erythropoietin use
- Excessive alcohol intake
- Hyperaldosteronism
- Hyperthyroidism
- Illicit drug use (eg, cocaine, ephedrine amphetamines)
- Pheochromocytoma
- Renal artery stenosis
- Renal parenchymal disease
- Vasoconstrictive drug use (eg, decongestants)

Physiologic blood pressure responses to exercise are addressed elsewhere in this issue. Briefly, in contrast to normotensive athletes, hypertensive individuals, with both dynamic and static exercise, generally demonstrate exercise-induced increases in total peripheral resistance and impaired vasodilation, with a resultant increase in myocardial oxygen consumption and exaggerated blood pressure elevations [5]. Over time, these abnormal responses may lead to pathologic left
ventricular hypertrophy (LVH) with thickened, stiff, and poorly compliant ventricular walls, particularly during cardiac filling in diastole. This impaired filling response leads to diastolic dysfunction that may actually decrease cardiac output during exercise and significantly impair performance and exercise capacity. An altered vasodilatory response to exercise in hypertensive athletes may also predispose to heat illness by impairing heat dissipation and potentially leading to seriously elevated core body temperatures, excessive water loss, and hypokalemia.

Chronic pressure overload and microvascular trauma are the major pathophysiologic mechanisms for the development of hypertension-related end-organ disease. Such complications include an increased annual incidence of cerebrovascular, retinovascular, peripheral arterial, and renal parenchymal diseases with chronic blood pressure elevations above 160/95 mm Hg [6–8]. Sustained high blood pressure also significantly raises the risk of coronary artery disease (CAD) and LVH, with a resultant increase in cardiac morbidity and mortality, heart failure, and sudden cardiac death.

The management of high blood pressure in active patients and athletes should include exercise, lifestyle modifications, and, if blood pressure is still inadequately controlled, medication. Nonpharmacologic therapy may be sufficient for controlling blood pressure in borderline or mildly hypertensive patients and should be used concomitantly even if medication is needed. One of the most important messages for hypertensive active patients and athletes is that exercise should be continued, because it is safe for most patients and helps control blood pressure.

Relative postexercise hypotension [9] was first noted by Kaul [10] over 30 years ago as a normal physiologic response to moderate-intensity dynamic exercise. The largest absolute and relative blood pressure reductions are seen in hypertensive subjects [9,11], with mildly hypertensive athletes deriving the greatest benefit, as exercise reduces sympathetic neural activity, leading to lower heart rate and cardiac output [4]. Maximal decreases of 18 mm to 20 mm Hg systolic and 7 mm to 9 mm Hg diastolic have been reported among stage 1 hypertensives. This lowering effect may persist for up to 16 hours after exercise, thus allowing stage 1 patients to be normotensive for most of the day. [9,12–14].

Three prior meta-analyses have validated dynamic exercise as an effective means of managing high blood pressure [15–17]. In an additional recent meta-analysis of 54 studies investigating over 2400 patients, aerobic exercise over at least 4 weeks of time was found to modestly reduce blood pressure in both hypertensive and normotensive persons [18]. Overall, blood pressure was lowered by 3.84 mm Hg systolic and 2.58 mm Hg diastolic, but hypertensive individuals demonstrated even larger benefits, with systolic and diastolic lowering of 7 mm and 6 mm Hg, respectively. These findings were independent of changes in body weight. Given the results of these numerous studies, aerobic physical activity should be considered an important component of both the prevention and treatment of high blood pressure. This is also valuable information in light of our increasing understanding of the beneficial effects of even small degrees of blood pressure lowering on the risk for cardiovascular complications.
With respect to acceptable levels of activity in the face of hypertension, moderate-intensity exercise, in keeping with current American College of Sports Medicine (ACSM) guidelines [19], is safe for most hypertensive patients. Training studies have shown that the acute effects of exercise on blood pressure are a low-threshold phenomenon observed after energy expenditures requiring only 40% maximal capacity [9,11,12] and after only three sessions of aerobic activity [12,20,21]. Similarly, data from over 40 studies of low to moderate-intensity endurance training (20 to 60 minutes at 40% to 70% of VO_{2max}, or 50% to 70% maximum age-predicted heart rate, 3 to 5 days per week) found effective decreases in both systolic and diastolic blood pressures [19]. There is little evidence to justify higher-intensity exercise (>70% VO_{2max}) for lowering blood pressure [22–30], as the beneficial effects of moderate-intensity exercise are equal or superior to those of higher-intensity exercise [31]. This is an important consideration in limiting the cardiovascular and musculoskeletal risks of exercise, particularly in older patients.

Chronic exercise training produces greater blood pressure reductions than do acute bouts of exercise [32]. Such a program should be at least 1 to 3 months in duration to reach the stable stage, and training should be maintained indefinitely, because the hypotensive effect persists only as long as regular endurance exercise is maintained. Generally, maximum limits of blood pressure lowering are reached after 3 months of training; no further blood pressure reduction occurs thereafter, except in rare instances.

Most studies examining the different modes of exercise in the management of hypertension have focused on walking, running, or cycling. Obviously, each hypertensive athlete’s sporting activities must be assessed for relative value and safety with respect to blood pressure management. Walking and running do not cause a sustained increase in blood pressure and perhaps represent the most suitable endurance exercises for hypertensive patients. Moderate swimming (30- to 45-minute sessions, 3 days a week) can lower systolic but not diastolic blood pressure at rest. Swimming can be an alternative exercise for patients who are obese, have exercise-induced asthma, or have orthopedic injuries. Vigorous activities done with rhythmic high force, such as sprinting or rowing, are generally unsuitable for uncontrolled hypertensive patients. Downhill skiing can elevate blood pressure, and mountain sports may exaggerate an elevated blood pressure response from the cold and decreased partial pressure of oxygen. Even resistance training (8 to 10 major-muscle-group exercises, two to three times per week), especially circuit weight training [33,34], may help lower blood pressure when the exercises are done at 40% to 50% of the patient’s one-repetition maximum [35]. Resistance training, however, is not recommended as the only approach to lowering blood pressure.

Despite their high level of exercise performance, athletes may also need to correct poor lifestyle habits to obtain optimal blood pressure control. Interventions that have proven to be beneficial include weight reduction, moderation of alcohol and sodium intake, high potassium and increased calcium intake, and smoking cessation [36]. No convincing data support the use of increased magnesium intake,
<table>
<thead>
<tr>
<th>Class</th>
<th>Agents</th>
<th>Mechanism of action</th>
<th>Adverse effects</th>
<th>Effects on aerobic capacity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angiotensin-converting</td>
<td>Benazepril hydrochloride</td>
<td>Prevent production of angiotensin II, a potent vasoconstrictor</td>
<td>Cough, renal dysfunction, hyperkalemia</td>
<td>None</td>
</tr>
<tr>
<td>enzyme (ACE) inhibitors</td>
<td>Captopril</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Enalapril maleate</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Lisinopril</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Quinapril hydrochloride</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Calcium channel blockers</td>
<td>Amlodipine</td>
<td>Decrease vascular smooth muscle contractility; cause negative inotropic and chronotropic effects on myocardium</td>
<td>Bradycardia, constipation, peripheral edema; for short-acting dihydropyridines, increased cardiac mortality</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>Diltiazem hydrochloride</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Isradipine</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Verapamil hydrochloride</td>
<td></td>
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<tr>
<td>Alpha-1-receptor blockers</td>
<td>Doxazosin mesylate</td>
<td>Cause decreased vascular contractility by blocking alpha-1 receptors in smooth muscle</td>
<td>Orthostatic hypotension, tachycardia</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>Terazosin</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Prazosin</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Central alpha-receptor</td>
<td>Clonidine hydrochloride</td>
<td>Act on CNS alpha-2 receptors to block sympathetic stimulation</td>
<td>Many CNS effects, including dry mouth, dizziness, sedation; postexercise hypotension</td>
<td>None, but poor first-line choice because of CNS effects and risk of postexercise hypotension</td>
</tr>
<tr>
<td>antagonists</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Beta-blockers*</td>
<td>Propranolol hydrochloride</td>
<td>Block cardiac beta-receptors, leading to decreased heart rate, myocardial contractility,</td>
<td>Bradycardia, depression, exacerbation of asthma, and impotence</td>
<td>Decreased aerobic capacity</td>
</tr>
<tr>
<td>Nonselective</td>
<td>Nadolol</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Same as above (except labetalol has beta-1, beta-2, and alpha-1 blocking activity)</td>
<td>Bradycardia, depression, and impotence</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>Atenolol</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Metoprolol</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Labetalol hydrochloride</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Same as above (except labetalol has beta-1, beta-2, and alpha-1 blocking activity)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardioselective</td>
<td>Hydrochlorothiazide</td>
<td>Decrease circulatory volume</td>
<td>Hypokalemia, hyponatremia, volume depletion, dehydration</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>Furosemide</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diuretics**</td>
<td>Losartan potassium</td>
<td>Block angiotensin II receptor site, preventing vasoconstriction</td>
<td>Renal dysfunction, hyperkalemia. (No cough, however.)</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>Valsartan</td>
<td></td>
<td></td>
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<tr>
<td>Angiotensin receptor</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>blockers</td>
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Table 2
A profile of common antihypertensive medications: mechanism of action, adverse effects, and effects on aerobic capacity
high-protein diets, other special dietary measures, relaxation techniques, or biofeedback in the prevention or control of hypertension [19].

If other interventions fail to control blood pressure, pharmacologic therapy is warranted. The goals are to (1) decrease elevated resting blood pressure, (2) lower increases in systolic and diastolic blood pressure during exercise, and (3) preserve central hemodynamics and exercise capacity [37]. The choice of pharmacologic agents is especially important in active patients and athletes because inappropriate medications can impair performance, whereas optimal choices can enhance it by reducing elevated blood pressure at rest and thus limiting exercise-induced hypertensive responses [2]. According to the Sixth Joint National Committee report (JNC VI) on the management of hypertension, patients using beta-blockers and diuretics to control uncomplicated hypertension have lower mortality rates than those who use other medications [3]. The medications typically used in treating hypertensive athletes are presented in Table 2.

In general, agents that decrease total peripheral resistance have the least effect on exercise performance. All commonly used antihypertensive agents except for nonselective beta-blockers and diuretics allow for essentially normal exercise capacity. Noncardioselective beta-blockers (e.g., propranolol, nadolol) are contraindicated in athletes because they significantly reduce maximal aerobic capacity. Their potentially deleterious effects on performance, however, as well as those of diuretics, must be weighed against the mortality benefit noted above. The physician must also be sure that the use of a particular antihypertensive agent is allowed by a sport’s governing body. Restrictions regarding the use of beta-blockers and diuretics are included in Table 2.

Cardioselective beta-1 blockers without intrinsic sympathomimetic activity (e.g., atenolol, metoprolol tartrate) lead to the greatest reduction in exercise-induced blood pressure and heart rate increases and have little effect on performance. Thus, some authorities [37] consider these medications to be the best choice for hypertensive athletes. Even the slightest performance decrement may be unacceptable to elite athletes, however. Alternative agents that may be more suitable choices for such athletes include angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARB), calcium channel blockers, and alpha-1 blockers. These alternative medications are well tolerated and have few negative effects on exercise performance.

Recommendations for sports participation

The physician’s role in evaluating hypertensive athletes and managing their condition is vital. Although athletes should not be exposed to undue
risks, physicians should be cautious not to give recommendations for sports participation that are more conservative than is consistent with scientific evidence. For example, hypertensive people who exercise are not at increased risk for hemorrhagic stroke or sudden cardiac death [2,5]. In addition, resistance training has not been associated with increased morbidity in hypertensive individuals [19] despite the significantly elevated blood pressure that static exercise may cause.

Keeping these findings in mind, physicians may use the JNC VI staging system for hypertension (see Table 1) as a framework for making exercise recommendations that are based on the degree of hypertension and the presence or absence of complicating factors.

**Stages 1 and 2**

Patients may participate fully in dynamic and static sports if there is no evidence of end-organ damage, including heart disease [2]. For those with mild hypertension (a resting diastolic pressure of 90 mm to 105 mm Hg and systolic pressure of less than 160 mm Hg), physical activities should be primarily dynamic. Some authors have suggested that strength training and high-static and high-intensity activities, though not absolutely restricted, should be avoided until better blood pressure control is obtained [35]. Examples of suitable sports include walking, hiking, jogging, cycling, swimming, softball, volleyball, and cross-country skiing. Sports to avoid include rowing, diving, tennis, competitive ball sports, and strenuous physical training (above 80% of maximum age-predicted heart rate) [37]. Blood pressure should be checked every 2 to 4 months to ensure that it remains in an acceptable range.

**Stage 3**

Patients who have a resting diastolic pressure above 110 mm to 115 mm Hg should discontinue exercise until blood pressure is well-controlled, especially if they are involved in high static or isometric sports [37]. Because activities such as boxing, cycling, rowing, bodybuilding, weight lifting, wrestling, and gymnastics have a static component, they should be avoided until acceptable blood pressure levels are established [2]. Although prudence would suggest that all types of exercise be limited until blood pressure is controlled, strenuous dynamic exercise, even among severely hypertensive patients, has never been shown to increase the risk of progression of hypertension or sudden death [38–40].

**Hypertension with complications**

For patients who have conditions such as pathologic LV hypertrophy, hypertensive nephropathy or retinopathy, or peripheral vascular disease, the type and severity of the condition and the nature of the sport determine the patient’s ability to participate safely [2]. Again, high static or isometric sports and dynamic exercise of more than moderate intensity should likely be avoided.
Obesity

Obesity and overweight have become increasing health concerns worldwide over the last several decades. Few sports medicine practitioners will encounter frankly obese athletes competing at elite levels. Much more commonly, the sports medicine physician may play a vital role in the creation of exercise and diet strategies for the management of overweight athletes at lower skill levels or obese patients from the general population. As the complications of obesity become more apparent, there are few more important tasks that must be assumed than to advocate the establishment and maintenance of healthful weight.

The most widely used measure of relative body size is the body mass index (BMI), which is determined by weight in kilograms divided by height in meters squared (kg/m$^2$). BMI is age- and sex-dependent but independent of ethnicity [41]. Per current National Institutes of Health (NIH) guidelines [42], ideal BMI is less than 19 to 25. “Overweight” is defined as a BMI 25 to 29.9, and “obese” corresponds to a BMI greater than 30. The prevalence of obesity has risen more than 50% within the past 10 to 15 years, with nearly half of adult Americans now classified as overweight or obese by these criteria [43]. Rates of obesity are even higher in non-Hispanic black women and Mexican-American women. Alarmingly, the numbers of Americans whose BMIs classify them as overweight or obese are rising in both categories.

Although much attention has been paid to this growing problem, there is much that is unknown regarding the causative factors. What is understood is that the determination of body weight is a complex process with neural, humoral, metabolic, and psychological contributing features [44]. It is likely that elements of both “nature” and “nurture” play roles in a unique interaction of genotypic versus environmental factors [45,46]. One common theory implicates genetic metabolic programming that favors a tendency toward obesity. Although genetic predispositions do determine resting metabolic rate (RMR) [47,48], the available data have failed to show that low RMR is a major factor in the etiology of human obesity [49–51]. A societal theory for the high prevalence of obesity in the United States is a positive energy balance (and resultant weight gain) that results from the promotion and increasing acceptance of a high dietary energy intake and, at a variety of levels, the discouragement of regular physical activity.

At the cellular level, obese patients demonstrate disordered metabolic responses of adipose tissue to insulin and to catecholamines. Insulin normally stimulates lipoprotein lipase (LPL) activity to control triglyceride metabolism and fat deposition. In obese patients, the hormonal control of adipocyte triglyceride turnover is altered and is most marked in patients with central obesity [52]. There is resistance to insulin stimulation of LPL and abnormal hepatic sensitive lipase (HSL) activity as well. Alterations in catecholamine-induced lipolysis lead to enhanced activity in visceral fat but decreased activity in subcutaneous fat [53], leading in turn to abnormal fat deposition and further weight gain.

Whether activity is actively discouraged or not, data at this point support the notion that our society as a whole is becoming less active. According to the
Behavioral Risk Factor Survey (BRFSS) [54], 64% of high school students reported vigorous (hard or very hard) activity for at least 20 minutes on 3 or more days per week. Boys were more active than girls, and whites were more active than blacks or Hispanics. Unfortunately, physical activity declined with higher grade in school, especially in girls. Among adults, only 28% of men and women achieve moderate or vigorous levels of physical activity. Twenty-seven percent of men and 31% of women report no regular physical activity at all outside of work. Although fewer than 20% of college graduates reported being inactive, nearly half of the high school educated population is inactive. These are disturbing trends with rising consequences.

The degree to which lack of regular physical activity creates or promotes obesity remains in debate. There are substantial data to suggest that differences in the amount of individual physical activity do contribute to differences in body weight and body fatness and may play a role in whether obesity develops. When evaluating cross-sectional and population-based studies, there emerges an inverse relationship between level of physical activity and indices of obesity, such as BMI [55–57]. Similarly, cohort studies have revealed that high levels of physical activity do appear protective against obesity [57,58]. Although there are no prospective studies that have looked at this issue, there is overwhelming indirect evidence via observational studies that declining amounts of physical activity have contributed importantly to a rising prevalence of overweight and obesity.

Taken in total, these data support a relationship between the level of physical activity and body fatness. This should not be surprising, in light of the fact that exercise is one of the most potent physiologic stimuli for lipolysis, with a higher rate of fat metabolism during exercise in trained individuals than that reported during either critical illness [59] or starvation [60]. With adaptation to chronic endurance aerobic activity, muscle develops a higher oxidative enzyme activity and is better equipped to burn fat through the use of free fatty acids as the energy substrate instead of glycogen stores. Activation of HSL is also increased [61].

Many significant medical conditions have been linked either directly or indirectly to obesity. The exact correlations between obesity and a number of these conditions remain unclear. Obesity-related sedentary behaviors and associated poor dietary habits may exert independent effects on health that are difficult to discern from obesity alone. Nevertheless, from the standpoint of identification and management of these conditions, they can at a minimum be safely viewed as obesity-associated.

Weight gain during adult life is associated with an increased risk of heart disease and death [62]. Indeed, most studies show an increase in mortality rate associated with a BMI of 30 or more [42]. There exists a J-shaped relationship between BMI and relative morbidity/mortality, with a primary concentration in the cardiovascular diseases [63–70], presumably from the effects of obesity on cardiovascular disease risk factors. In particular, the android fat distribution pattern with prominent central/abdominal adipose deposition is now associated with a variety of metabolic derangements, including dyslipidemia [71–73], hypertension [43], hyperinsulinemia [74,75], and glucose intolerance/diabetes [68,76,77], with a 25%
increase in diabetes risk for each additional unit of BMI over 22 kg/m² [46,78]. Excess weight is also associated with increased risk for stroke, gall bladder disease, sleep apnea and respiratory problems, and for a number of cancers, including endometrial, breast, prostate, and colon cancer [46,79].

Similar associations have been found between increasing body weight and increased risk for osteoarthritis (OA) [80,81]. Specifically, age, female sex, and BMI are independent predictors of disabling knee OA [82]. Persons with a BMI >30 have a markedly increased risk for knee OA compared with those with a more modest BMI (25–29) [83]. As a result of increases in both cardiac preload and afterload, obesity may create a hypertensive effect and may also predispose to heat illness in athletes as a result of impaired heat exchange.

The value of regular exercise for the prevention or management of obesity cannot be overstated. Each encounter with the obese patient should take into consideration the information noted above with regard to rising prevalence and increasing concern about obesity-associated morbidity. Substantial work in the area of exercise physiology now allows for a focused approach to obesity management based on predictable responses to the prescribed interventions.

Even modest reductions in body weight (5%–10%) will significantly improve health. Overweight individuals can realize substantial improvements in a number of health parameters by achieving the minimum public health recommendation for physical activity and by improving their level of cardiorespiratory fitness [84–87]. Indeed, regular exercise participation is known to improve the health of all participants, regardless of size [42,85,88–93]. Being physically active and fit reduces obesity-related chronic diseases and decreases risk for early death. In a large observational study, unfit men of normal weight actually had a higher all-cause mortality than men who were fit but overweight (BMI >27.8) [94]. Although a combination of cardiovascular fitness and at least modest weight loss is the goal for almost all overweight or obese patients, the pursuit of fitness even in the absence of substantial weight loss is clearly beneficial and should be aggressively sought. Normalization of body weight is also not necessary to allow exercise to improve the health of obese individuals with bodyweight-related metabolic disorders such as diabetes [88]. In the athlete population, a multifaceted approach including dietary measures and training alterations should be undertaken to positively influence body composition, especially in athletes with a BMI >30 kg/m².

Advice to eat a healthful diet and increase physical activity to enhance overall energy expenditure and assist with gradual weight loss is appropriate for almost all individuals who are above a healthy weight [95,96]. Longer duration, low-intensity training (at 30%–50% VO₂max) favors the utilization of adipose tissue over muscle glycogen and thus fosters weight loss [97]. Intermittent bouts of exercise provide largely the same benefit as continuous for obtaining weight loss; this may be advantageous for those who dislike continuous exercise or perceive barriers to continuous exercise. Exercise goals for these patients should include the progression of overweight adults to 200 to 300 minutes of exercise per week or expenditure of >2000kcal/week [98]. They should be progressed to higher
levels of exercise gradually over time, and a variety of behavioral strategies should be used to facilitate adoption of this level of exercise. Setting a reasonable goal of a 5% to 10% reduction in weight is appropriate because many diseases clinically improve with weight loss in this range [99]. Maintenance of a regular exercise program may be one of the best predictors of long-term weight control [100,101]; data increasingly show that those able to continue exercising are able to maintain body weight loss for many years [92,93,100,102–105]. An excellent example of the success of such an approach is the National Weight Control Registry, which tracks patients with reduced body weight for at least 1 year. These studied individuals have maintained a minimum weight loss of 13.6 kg for an average of 5 years. Their success is largely due to consuming only 1800 kcal/day and expending about 400 kcal/day [106].

The most widely accepted, safe, and accessible means of increasing physical activity for weight control is walking. It appears clear from previous study that low-intensity endurance activities are capable of producing beneficial metabolic effects that are similar to those produced with high-intensity exercise. The clinician should focus on improvements in the metabolic profile rather than on weight loss alone, and a realistic goal of 0.5 to 1 pound per week of weight loss should be set and actively sought. Little benefit has been derived from resistance training for absolute weight loss [107–111].

By aggressively addressing overweight and obesity as major negative factors for the overall health of our patients, sports medicine-oriented physicians may foster vital changes in the management of this population. With obesity’s associated comorbidities, few conditions should be viewed as more urgent in their management.

Dyslipidemia

Dyslipidemia is a common clinical entity worldwide. Although experts now largely view the “conservative” measures of diet, weight loss, and exercise as adjuncts to powerful pharmacotherapy in the management of elevated lipids, data do demonstrate a positive effect of exercise on the lipid profile. Even a single exercise session has been shown to acutely reduce triglycerides (TG) and increase high-density lipoprotein (HDL) cholesterol [32]. A number of physiologic characteristics must be taken into consideration when assessing the potential effects of exercise on elevated lipids. Key patient-related factors include the baseline physical fitness of the subjects, subjects’ pre-exercise lipid levels, and the intensity and duration of exercise performed [32]. Primary care physicians and sports medicine practitioners should feel comfortable with the rationale for the use and prescription of exercise activity for the control of dyslipidemic patients. Similarly, it is also essential to feel comfortable managing elevated lipids in athletes and highly active individuals in the face of continued participation.

The acute effects of exercise on lipids are greatest with respect to its HDL-elevating properties. HDL has been found to increase 4% to 43% in various studies
and these changes have been seen in both moderately fit [114,122] and highly trained [117] subjects. They have been reported after expenditures of 350 to 400 and 1000 kcal, respectively, in a single exercise session. A reduction in TG also occurs 18 to 24 hours after an acute bout of exercise and persists for up to 72 hours [112–122]. This effect is greatest in those with the highest pre-exercise TG values [115], and does not appear to require a threshold of exertion to be demonstrated [116].

Single exercise sessions can also reduce postprandial lipemia, and in so doing, reduce the risk of coronary artery disease (CAD). The magnitude of this risk reduction is related to total energy expenditure rather than exercise intensity. Acute reductions in total cholesterol are much more difficult, requiring very prolonged exercise with a large amount of energy expenditure (>1100 kcal) [117,123]. A number of studies, in fact, have shown no acute exercise-induced change in plasma total cholesterol levels [124–130]. Low-density lipoprotein (LDL) may also acutely decrease, but the changes are modest, with declines of only 5% to 8% in hypercholesterolemic men [114,115,119]. No acute effects on apolipoprotein subset levels have been demonstrated [123,131–135].

When evaluating the chronic effects of exercise, endurance athletes provide the largest group for analysis. As a result of their high level of fitness, endurance athletes have serum HDL cholesterol concentrations 10 to 20 mg/dL or 40% to 50% higher than their sedentary counterparts [120,136–138]. Their triglyceride levels are 20% lower, and LDL cholesterol concentrations are approximately 5% to 10% lower as well [120,136,137]. Untrained individuals are generally incapable of the exertion required to foster these lipid adaptations, thus demonstrating the importance of developing aerobic fitness to maximize the lipid-lowering effect [32].

Our present understanding of the physiologic changes that occur in lipid metabolism in the face of exercise supports the concept that frequent repetition of isolated exercise sessions produces long-term adaptations in lipid metabolism. Endurance exercise reduces TG and increases HDL, and these effects are likely related to total energy expenditure and to baseline TG concentration [126,139,140]. Ferguson et al [117] found that lipoprotein lipase (LPL) activity was increased in trained runners 24 hours after treadmill running at 70% VO2,max with an energy expenditure >1100 kcal. Such exercise is of sufficient volume and intensity to deplete intramuscular triglyceride stores and increase LPL muscle production and release, with a positive effect on lipid metabolism.

Exercise cessation studies confirm that higher HDL levels in very active individuals are not due solely to acute exercise effects [32]. Rather, HDL responds to aerobic training and increases by 2 to 8 mg/dL in a dose-dependent fashion, with increased energy expenditure over time [126,141–143]. Wood et al [144] reported that the beneficial effects of training on HDL elevation are seen at 12 weeks or more and not seen at 10 weeks or less. Increased training volume predictably yielded greater results [126,130,142,143,145,146]. Wood et al [147] also combined caloric restriction and exercise training and found greater resultant body composition and HDL changes. In the Health, Risk Factors, Exercise
Training and Genetics (HERITAGE) Family Study [148], 200 men were enrolled in a 20-week endurance training program to study exercise effects on lipid metabolism. A variety of triglyceride and HDL cholesterol patient subsets were followed. Regular endurance exercise training was found to be particularly helpful in men with low HDL cholesterol, elevated TGs, and central/abdominal obesity.

Data regarding the effects of chronic exercise on LDL and total cholesterol lowering are less definitive. Prolonged exercise generally produces small reductions in LDL; the decrease in males has been shown to be approximately 8% [135]. The addition of physical activity to a modestly weight reducing, low-fat diet does significantly enhance the LDL-lowering effect of the diet [149]. For a reduction in plasma total cholesterol to occur with chronic exercise, a reduction in body weight, body fat, or dietary fat must accompany the exercise training program.

In a meta-analysis by Halbert, et al [150], 31 trials were assessed, including 1833 previously sedentary hyperlipidemic and normolipidemic patients. Regular aerobic exercise resulted in small but statistically significant decreases in TC, LDL, TG and an increase in HDL. A comparison of intensities of training gave inconsistent results, but more frequent exercise did not appear to result in greater improvements in the lipid profile than exercise three times per week. The effects of resistance training were also inconclusive in this study, although most data find no consistent benefit from resistance training in lowering lipoprotein levels.

Of special note, in athletes with dyslipidemia severe enough to warrant therapy, the use of statin lipid-lowering agents presents special concerns to the treating physician. In addition to symptomatic muscle complaints that may arise with statin use, these athletes may also develop asymptomatic elevations of creatine phosphokinase (CPK). Elevations in CPK may be further exacerbated by eccentric exercise. Although elevations up to 21,000 U/L are generally tolerated without sequelae, rarely athletes will develop frank rhabdomyolysis with vigorous exercise. The risk for such a significant complication is raised by dehydration and the concomitant use of statins with fibric acid derivatives, niacin, macrolide antibiotics, and azole derivative antifungals.

Few of the elite caliber athletes that sports medicine physicians care for have significant dyslipidemia. Many of our patients who are attempting to manage one if not several major risk factors for cardiovascular disease do suffer with dyslipidemia, however, and are being counseled to exercise regularly. An understanding of the response of lipids to both acute and chronic exercise is essential to guiding patients toward appropriate decisions in the management of dyslipidemia. Sport and exercise activities can be safely continued, as no large-scale studies exist to suggest that patients with dyslipidemia are at increased risk for adverse cardiovascular events as a result of their exercise activities.

References

[2] Kaplan NM, Deveraux RB, Miller Jr HS. 26th Bethesda conference: recommendations for de-


[147] Wood PD, Stefanick ML, Williams PT, et al. The effects on plasma lipoproteins of a prudent...

