Cardiovascular Issues in Boxing and Contact Sports

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• Cardiovascular risks • Cardiac evaluation • Boxing
• Cardiac contusion • Coronary heart disease

Despite the inherent traumatic nature of boxing and contact sports, there are relatively few reports of boxing-associated cardiovascular (CV) events. This may be related to the type of force that is applied to the chest with a gloved hand and the anatomy of the heart in the chest. The heart, as opposed to the brain, is mobile and padded in the thoracic cavity and less prone to acceleration-deceleration injuries that routinely occur during a boxing match. The age and general condition of the participants is also different from most recreational athletes. Despite a recent increase in boxing as CV exercise training, the general population is young and well trained. On the other end of the age spectrum, participation in the more vulnerable pre-teen and teenage athlete (such as is present in baseball) is more limited and controlled. The duration of exercise in boxing is limited and although there is evidence of hyper adrenergic activity because of the competition, the period of sustained elevation of heart rate (HR) and blood pressure (BP) are limited. The extensive conditioning necessary for competitive boxing reduces the chances of a subclinical pathology from reaching the competitive arena. Environmental factors are also limited, as boxing is generally performed in a controlled environment, without wide extremes of either temperature or humidity.

Even with these caveats, boxing requires a high level of physical effort that may tax the abilities of an abnormal or susceptible heart. The metabolic demand of 1 hour of boxing is equivalent to running 9 km in 60 minutes on the treadmill.[1] The chest trauma incurred from a blow or fall may damage even this protected vital organ. This article discusses the nature of intrinsic and traumatic CV risk with boxing with a focus on the underlying anatomy and physiology of cardiac, vascular, and arterial damage.

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BACKGROUND

The heart sits protected by the rib cage and the thoracic musculature. It is suspended by attachments to the posterior chest wall, however is mobile within the pericardial sac and thorax during normal function. The coronary arteries lie embedded in grooves in the surface of the heart. Fatty tissue is usually present that also reduces the risk of direct injury to the coronary arteries.

Atherosclerotic disease of the coronary arteries may begin in the second decade of life but is usually nonobstructive, only involving the subintimal region of the artery. Most myocardial infarctions (MI) occur because of rupture of the subintimal atherosclerotic plaque and most commonly occur (70%) in arteries that are not severely obstructed (<75%). This may occur at any time, but exercise has been documented to increase the risk of plaque rupture. This occurs less in the well-trained aerobically fit than in sedentary individuals.

Sudden cardiac death (SCD) is generally an arrhythmic event with the development of ventricular tachycardia or other hemodynamically unstable rhythm. Several types of genetic predispositions have been identified, including abnormalities in myocardial membrane channels and anatomic abnormalities of the right ventricle and conduction system.[2] These abnormalities are generally rare, however may become clinically apparent in the second and third decades of life when competitive athletic participation is greatest.

CARDIOVASCULAR RISKS OF EXERCISE

Cardiovascular events are catastrophic, occurring in otherwise healthy people frequently in the prime of their life and are met with publicity and blame. Because of the relatively low incidence of CV events with exercise, there are widely divergent rates reported in the literature. Among the millions of teenage athletes competing in the United States, approximately 10 to 13 such cases are reported every year.[3] The rates differ by age, gender, and geography, occurring more commonly in older athletes and men. In the older athlete, atherosclerotic disease becomes the predominate risk. Sudden cardiac death or myocardial infarction during exercise is usually associated with rupture of an atherosclerotic plaque with subsequent obstruction of the coronary artery. As competitive boxers are generally younger athletes, we will focus on the risks for those younger than 35 years old.

There are numerous physical and physiologic adaptations by the heart to the demands of chronic intense exercise. These changes, although considered normal variations and not pathologic, are similar to abnormalities that may result in injury. In general, athletes who train with significant resistance loads develop hypertrophy of the left ventricle (LV) with minimal increase in LV volume, whereas prolonged low-resistance training will result in higher LV volume and cardiac output without as large an increase in ventricular mass and thickness. The training required for competitive boxing frequently entails both resistance and prolonged aerobic exercise. The changes that occur may mimic many of the abnormalities discussed and make screening and diagnosis more difficult.

In autopsy studies of death in young athletes, 95% demonstrate a structural abnormality.[4] Inheritable cardiomyopathies and congenital coronary artery anomalies account for most of the events, with valvular heart disease, Marfan syndrome, dilated cardiomyopathy, and myocarditis being the remainder of the myocardial abnormalities and premature coronary atherosclerosis and myocardial bridging affecting the coronary circulation. The remaining 5% of events without a structural anomaly probably represent arrhythmogenic events related to long QT syndrome, Brugada syndrome,