Dilation of the thoracic aorta is the primary risk factor for aortic dissection, a highly morbid and mortal condition with in-hospital mortality over 25%. Causal and often synergistic mediators of aortic dilation include sex, age, body size, hypertension, and inherited/congenital conditions such as Marfan syndrome and bicuspid aortic valve. However, these risk factors fail to explain the full extent of aortic size variation, and patients often present with significant aortic dilation in the absence of a clear risk factor or cause. In this context, the question arises of whether aortic dilation might represent yet another example of cardiovascular remodeling in response to the hemodynamic forces associated with exercise and sport. The heart’s ability to adapt to exercise has long been a subject of clinical and scientific interest dating back to early reports of cardiomegaly among athletes in the late 19th century, followed by a broad contemporary literature documenting remodeling of the left ventricle (LV), right ventricle, and atria in response to sport-specific hemodynamic loads. Given these well-described changes, it has been hypothesized that the underlying exercise-induced hemodynamic factors, including increased stroke volume, high cardiac outputs, and elevated central aortic pressures, may also stimulate remodeling and dilation of the aorta. However, multiple large cross-sectional studies in active youthful athletes, including those at the extremes of body size such as professional basketball players, have failed to corroborate this by demonstrating that frank aortic dilation (typically beyond a cut point of 40 mm) is rare. Data comparing athletes to nonathletic controls are more limited, with the best estimates coming from a meta-analysis that reported an average difference of 3.2 mm. At present, clinical consensus is that athletic participation has minimal impact on aortic size and that the identification of significant aortic dilation in an athlete should be considered pathological and treated accordingly. However, all prior research has focused on young athletes with an average age below 30. This important limitation raises many important questions about aortic physiology and corollary health outcomes among former athletes and older active competitors.

Is Big Truly Bad?
Aortic Dilation in Former National Football League Players

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In this issue of Circulation: Cardiovascular Imaging, Gentry et al present valuable data on aortic size among former American-style football players. Using gated noncontrast computed tomography, the authors measured dimensions of the midlevel ascending aorta among a cohort of 206 middle-aged (mean age=57.1 years) former National Football League (NFL) players. Data generated from these former professional athletes were compared with those derived from the Dallas Heart Study (n=759, mean age=53.6 years), a population-based, cross-sectional cohort study with an ethnic distribution similar to the enrolled former players. Some important differences exist between the former NFL players and the comparator cohort including older age, larger body surface area (2.4 versus 2.1 m²) and body mass index (32.4 versus 30.0 kg/m²), and more favorable traditional cardiovascular risk factor profiles. In unadjusted analysis, former NFL players had larger aortic size than the Dallas Heart Study subjects, with midascending aortic diameters of 38±5 versus 34±4 mm, respectively (P<0.0001). This difference persisted in a multivariate model incorporating age and body surface area, the 2 most commonly cited risk factors for aortic enlargement, as well as hypertension, measured blood pressure, and other cardiovascular risk factors. Most strikingly, the prevalence of marked aortic dilation among former NFL players was 29.6% using a cut point of >40 mm and 9% using a cut point of ≥45 mm. Finally, the authors stratified the former NFL players by field position—either linemen or nonlinemen—and found that only linemen field position retained significance in a logistic regression model for predicting aortic size >40 mm.

To our knowledge, this is the first study to report aortic size among middle-aged former athletes, and the authors are to be congratulated for providing a valuable data set that advances our understanding of vascular phenotype among aging former elite athletes. The importance of their findings may best be highlighted by direct comparison with recent study reporting aortic dimensions among prospective NFL athletes. Among these much younger football players, studied during their transition from the collegiate to professional ranks (mean age=23.5 years), only 1 of 983 participants had an aorta larger than 40 mm. The striking difference in the prevalence of aortic dilation between prospective and former NFL players far exceeds what one would expect from aging in isolation and lends to a key explanatory hypotheses. Put simply, is incident aortic dilation among aging former NFL players driven by prior football exposure, postfootball factors including lifestyle choices and underappreciated cardiovascular disease, or some
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combination of both? Defining the relative, perhaps synergistic contributions of these 2 time periods and delineating the clinical relevance of aortic dilation in this population represent key areas of future work.

Why might football exposure lead to aortic dilation? The physiological stressors inherent in the game of football are significant. Football players engage in several seconds-long bursts of high-intensity static activity and may do so hundreds of times each day during training and competition. This form of activity translates into a repetitive and marked afterload challenge for the LV and central arterial vasculature. Importantly, however, the complex physiological milieu of football varies by field position, as nonlinemen are additionally exposed to considerable amount of concomitant dynamic stress as defined by increased and sustained cardiac output. The impact of this differential physiology on the cardiovascular system has been demonstrated by our group through numerous previous studies of collegiate football athletes documenting position-specific changes in blood pressure, LV morphology and function, and vascular stiffness. More specifically, linemen who are exposed to high static loads without accompanying dynamic loads are more likely to develop hypertension, concentric LV hypertrophy, central aortic stiffening, and reduced LV systolic strain in as little as 90 days of collegiate football participation. Consideration of this potentially concerning constellation of subclinical pathology raises important questions about cardiovascular health later in life and may begin to explain why aortic dilation among former NFL players seems most prominent among former linemen. But the degree to which components of football exposure, including sport physiology but also incident hypertension and weight gain, factors common among active football linemen, contribute to later life aortic dilation remains unclear.

At present, we remain uncertain how much of the observed aortic dilation among former players occurs during years of active play versus after career completion. The average length of an NFL playing career hovers just above 3.3 years, and the vast majority of players have transitioned to their postfootball lives by the age of 35. With a mean age of 57 years among former athletes, aortic dilation among former players occurs during years of active play versus after career completion. The average length of an NFL playing career hovers just above 3.3 years, and the vast majority of players have transitioned to their postfootball lives by the age of 35. With a mean age of 57 years among former athletes, it remains possible if not probable that the interceding postcareer decades may play an important role in contributing to aortic dilation. Why might aortic dilation develop once the helmet has been shelved and the cleats hung up for the last time? Speculative explanations include former players’ continuation of high-intensity static and dynamic activity to maintain lean muscle mass and aerobic fitness, concomitant undiagnosed hypertension that may be related to obesity or disordered sleep breathing, and perhaps passive and continual dilation of a fragile ascending aorta resulting from injury occurring during active playing years. Such injury, if it occurs, may be attributable to the static physiology detailed above and the deceleration stresses that occur when an athlete moving at high speeds is tackled, stopped suddenly in his tracks, or thrown hard to the ground.

Finally, we must consider the issue of clinical relevance. Cross-sectional data are incapable of establishing causality but set the stage for the generation of hypotheses. The most important area of uncertainty generated by the current article is whether aortic dilation among former NFL athletes, and perhaps athletes from other sporting disciplines, carries the same propensity for dissection as in the general population. The conservative null hypothesis is that aortic dimensions among athletes and nonathletes alike are all created equal with respect to long-term risk. Alternatively, perhaps mild to moderate aortic dilation among highly active people and competitive athletes represents a relatively benign phenotype much like the eccentric LV hypertrophy that is common among endurance athletes. These represent critical hypotheses that await rigorous scientific assessment. Clarifying the physiology and prognostic relevance of aortic dilation among former athletes will take time, cooperation, and scientific rigor. The first logical missing piece of this puzzle is to obtain data, similar to those generated by Gentry et al., coinciding with the immediate termination of the athletes’ professional careers and ideally including longitudinal study of the brief transitional period, beginning at career conclusion and lasting months to years, during which athletes determine the lifestyle choices, social connectivity patterns, and health habits that define the rest of their lives. We suspect this short but critical period of time plays a key role in establishing behavioral patterns and risk factors that drive subsequent cardiovascular morbidity and mortality. This work will help us begin to disentangle the relative contributions of active playing years from the postcareer phase as they relate to the temporal emergence of aortic dilation. Next and of paramount importance is the need for longitudinal clinical outcomes data. This will require cooperation between former players, clinical researchers, and ultimately the governing organizations that oversee health care for both active and former players. Such an integrative approach to studying health in former players will require carefully defined serial measurement time points, advanced phenotyping, responsible handling of data, and ultimately individualized clinical care. Ultimately, addressing these areas of uncertainty will have important implications that extend beyond former football athletes. NFL players are emerging as a paradigm of the diverse positive and negative health impacts of sports participation. Further study of their nuanced aortic physiology and pathology will deepen our understanding of the relationship between sport and health not just among these former warriors of the gridiron but also across highly active people and dedicated exercisers of all shapes and sizes.

Disclosures

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References

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