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Aortic Root Dilation in Strength and Power Athletes: Physiological Adaptation or Pathological Risk - a narrative review

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Abstract

Background. Aortic root dilation is well described in endurance athletes, but its prevalence, magnitude, and clinical significance in strength and power athletes remain incompletely characterized, creating diagnostic uncertainty when an enlarged root is found in a heavily muscled competitor.

Aim. To synthesise evidence on aortic root dimensions, their determinants, and associated risks in strength and power athletes, weighing physiological adaptation against pathological aortopathy.

Materials and Methods. This narrative review synthesises peer-reviewed echocardiographic and epidemiological studies, registry-based series, case reports, mechanistic and biomechanical investigations, and international consensus and eligibility guidance addressing aortic root size, hemodynamic loading, connective-tissue disease, and dissection risk in resistance-trained populations.

Results. Across cohorts, strength and power athletes generally show aortic root diameters similar to, or only marginally larger than, sedentary controls, and often smaller than in endurance athletes, despite pronounced concentric left ventricular hypertrophy. Acute blood pressure surges during heavy resistance exercise transiently elevate aortic wall stress, and case series link

anabolic-androgenic steroid use, uncontrolled hypertension, and undiagnosed connective-tissue or bicuspid aortic valve disease to catastrophic dissection during lifting. Diameter-to-ventricle ratios and indexed measurements improve discrimination between physiological remodeling and pathological dilation.

Conclusions. True aortic root enlargement is uncommon and rarely severe in strength and power athletes as an isolated training effect; when present, it should prompt exclusion of hypertension, steroid use, and heritable aortopathy rather than automatic attribution to sport, guiding individualized eligibility and surveillance decisions.

Keywords: aortic root dilation; strength training; powerlifting; resistance exercise; bicuspid aortic valve; athlete's heart; aortic dissection; anabolic-androgenic steroids; cardiovascular screening

1. Introduction

The concept of the "athlete's heart" describes a spectrum of morphological, functional, and electrical adaptations that occur in response to sustained, intensive physical training and that must be distinguished from covert cardiac disease before an athlete is cleared for competition [1]. Sudden cardiac death in young competitive athletes, although rare, is a devastating event that has driven decades of research into which structural findings represent benign remodeling and which represent an early or subclinical phenotype of an inherited or acquired cardiac disorder [2]. Much of this literature has concentrated on the left and right ventricles, where the dichotomy between eccentric hypertrophy in endurance sport and concentric hypertrophy in strength and power sport has become a familiar organizing principle, together with atrial enlargement, hypertrabeculation, and the electrical adaptations that accompany them [3].

The aorta has received comparatively less attention, yet it is subject to the same haemodynamic forces that remodel the ventricles, and its dilation carries a specific and potentially catastrophic complication that the ventricles do not: acute dissection or rupture. A large cross-sectional study of highly trained young athletes established that aortic root diameters are, on average, only marginally larger in athletes than in sedentary controls, and that a diameter exceeding accepted upper limits of normality (40 mm in men, 38 mm in women) is present in a small minority of cases, most of whom show no progression over medium-term follow-up [4]. This finding is reassuring at a population level, but it does not resolve the clinical dilemma faced by the practitioner examining an individual heavily muscled strength or power athlete whose aortic root

measures close to, or beyond, these thresholds: is this dilation an expected consequence of years of resistance training, or is it the first visible sign of an underlying, potentially lethal, aortopathy that has been unmasked, or even accelerated, by the sport itself?

This question is sharpened by several considerations specific to strength and power disciplines. First, echocardiographic screening protocols in athletes were developed and validated largely in endurance and mixed-sport populations, and their applicability to bodybuilders, powerlifters, weightlifters, and American-style football linemen is less thoroughly established [5]. Second, resistance exercise imposes a haemodynamic profile fundamentally different from that of endurance exercise: intermittent, very high systolic and diastolic pressure surges, often compounded by Valsalva manoeuvres, rather than the sustained volume load and vasodilation characteristic of dynamic aerobic exercise [6]. Third, strength and power sports have a documented association with the use of anabolic-androgenic steroids and other performance-enhancing substances that are independently implicated in vascular wall injury and catastrophic aortic events, complicating any attempt to attribute aortic findings to training alone [7]. Fourth, some congenital and hereditary aortopathies, most notably bicuspid aortic valve disease, are common enough in the general population that they will inevitably be encountered among strength athletes, and the interaction between heavy resistance training and an already vulnerable aortic wall is not fully resolved [8].

The purpose of this narrative review is to bring together the currently available evidence—epidemiological and comparative echocardiographic studies, mechanistic investigations of acute wall stress and vascular stiffness during resistance exercise, case reports of aortic dissection in strength athletes, and international consensus guidance—into a single, critically appraised synthesis addressing a single clinical question: in strength and power athletes, is aortic root dilation better understood as physiological adaptation or as a marker of pathological risk? The review does not restrict itself to a single discipline; it draws on data from powerlifters, bodybuilders, Olympic weightlifters, American-style football players, and other athletes with a substantial isometric or high-resistance training component, while using endurance and mixed-sport cohorts as explicit comparators. Where the evidence is contradictory, this is stated explicitly rather than resolved by averaging, and where a finding depends on a specific population, sample size, or diagnostic threshold, this is specified rather than generalized.

2. Materials and Methods

This article was conceived as a narrative review rather than a systematic review or meta-analysis, and accordingly no protocol was registered, no formal risk-of-bias appraisal was undertaken, and no quantitative pooling of results was performed. Sources were identified through targeted searches of open-access literature databases and aggregators (OpenAlex, Europe PMC, and Crossref), restricted to peer-reviewed, English-language publications, published from 2015 onward where possible, and available in full text under open-access terms; each digital object identifier cited in this review was individually verified against the corresponding registry entry. Material was selected for relevance to aortic root or ascending aortic dimensions, cardiovascular remodeling, or aortic complications in athletic, and particularly strength- or power-trained, populations, and was supplemented with foundational mechanistic, guideline, and case-based literature needed to contextualise these findings, including work on general athlete's-heart physiology, hereditary aortopathy, bicuspid aortic valve disease, hemodynamic responses to resistance exercise, and eligibility recommendations for competitive sport. The retrieved material was grouped thematically into physiological mechanisms; measurement methodology and imaging modalities; comparative aortic root data across sport categories, with specific attention to strength and power disciplines; acute hemodynamic and wall-stress considerations; anabolic steroid use; congenital and hereditary aortopathies; risk factors for dissection; illustrative case reports; comparator endurance and masters-athlete data; exercise prescription in known aortopathy; and screening and eligibility guidance. Because source selection and thematic synthesis were, by design, undertaken narratively rather than systematically, this review is subject to selection and emphasis bias, and its conclusions should be interpreted as a clinically oriented synthesis rather than a formally graded body of evidence.

3. Physiological Basis of Cardiac and Vascular Remodeling in Athletes

Systematic training produces measurable structural adaptation of the heart and great vessels that is broadly proportional to the volume, intensity, and modality of exercise performed, and that reflects the distinct haemodynamic loads imposed by different training stimuli [1]. Dynamic, isotonic exercise of the endurance type increases venous return and cardiac output for sustained periods, producing what has classically been described as eccentric left ventricular hypertrophy—an increase in cavity size accompanied by a proportionate increase in wall thickness. Static or

isometric exercise, exemplified by weightlifting, powerlifting, and bodybuilding, was classically held to increase peripheral vascular resistance and systemic pressure without a comparable increase in cavity volume, producing concentric hypertrophy characterised by increased wall thickness with preserved or minimally increased chamber dimensions [6]. This dichotomy, first proposed from M-mode echocardiographic comparisons of 56 athletes, has proved influential but has also been increasingly challenged; subsequent meta-analytic work has emphasised that most sporting disciplines, including resistance training, impose a combination of volume and pressure load, and that the resulting hypertrophy is frequently intermediate or "balanced" rather than purely concentric or eccentric [6].

Diastolic function assessment in athletes illustrates the complexity of this remodeling. Elite endurance athletes typically show enhanced early diastolic filling, characterised by an elevated ratio of early to late transmitral flow velocity (E/A) and preserved or supranormal early-diastolic mitral annular velocity (e'), reflecting improved ventricular suction related to the enlarged, more compliant left ventricle [6]. Consensus criteria for diastolic dysfunction developed for the general population—an averaged septal/lateral e' below 7–10 cm/s, an E/ e' ratio above 14, indexed left atrial end-systolic volume above 34 mL/m², and a tricuspid regurgitant peak velocity above 2.8 m/s—do not transfer cleanly to athletic populations, because chamber enlargement and the associated elevation of indexed left atrial volume are expected physiological findings in this group rather than markers of disease [6]. This same source proposes athlete-specific cut-offs for suspected dysfunction (for example, a left ventricular ejection fraction below 45–50%, global longitudinal strain less negative than –14%, or septal/lateral e' below 7–9 cm/s) and stresses that isolated measurements outside the expected range for comparable athletes will often not, by themselves, indicate pathology.

The right ventricle, atria, and aorta undergo parallel, though quantitatively distinct, adaptation. Right ventricular dilation in endurance athletes is proportionate to left ventricular enlargement and is generally accompanied by preserved systolic function, whereas concentric hypertrophy in resistance-trained athletes is typically not accompanied by the same degree of right ventricular remodeling [7]. Left and right atrial enlargement occurs across training modalities but is most pronounced in high-dynamic disciplines, and indexed atrial volumes commonly exceed the reference thresholds used in non-athletic clinical practice, again cautioning against direct application of general-population diastolic-dysfunction algorithms to this group [6]. Several

reviews emphasise that the magnitude of remodeling correlates with training volume, sex, ethnicity, body size, and age, and that no single echocardiographic cut-off reliably separates physiological from pathological adaptation across this heterogeneous population, a theme that recurs throughout the aortic literature reviewed below [7].

A further mechanistic thread concerns the vascular wall itself rather than the cardiac chambers. Because the aorta and its wall components—elastin, collagen, and vascular smooth muscle—are subject to the same pressure and flow perturbations that remodel the ventricles, but obey distinct biomechanical laws (notably the Law of Laplace, in which wall tension is proportional to transmural pressure and radius and inversely proportional to wall thickness), cardiac and aortic adaptation cannot be assumed to track one another in a simple, linear fashion; this distinction underlies much of the controversy addressed in the remainder of this review [9].

4. Aortic Root Anatomy, Measurement Technique, and Reference Values

Accurate interpretation of aortic root dimensions in strength and power athletes depends on a clear understanding of the anatomical segments involved and the technical conventions used to measure them. The aortic root, extending from the annulus through the sinuses of Valsalva to the sinotubular junction, is anatomically and embryologically distinct from the tubular ascending aorta above it, and pathological processes may selectively affect one segment more than the other, a distinction emphasised in reviews of both athletic and hereditary aortopathy [10]. Standard clinical guidance recommends measuring the sinuses of Valsalva at end-diastole using a leading-edge-to-leading-edge convention on the parasternal long-axis view, perpendicular to the axis of blood flow, with measurement of the largest diameter obtained, whether at the sinuses or at the ascending aorta [4].

Absolute diameters are strongly influenced by body size, sex, and, to a lesser extent, age and ethnicity, which is why most studies index the aortic root to body surface area, height, or both, and why some groups advocate allometric rather than simple ratiometric scaling to correct for the non-linear relationship between aortic size and body dimensions, particularly in very tall or very large individuals [11]. In one of the largest cohorts assembled specifically to define normal limits, comprising 3,781 competitive athletes aged 14–35 years compared with 806 sedentary controls, the mean aortic root diameter was only marginally larger in athletes than in controls (28.3 ± 4.1 mm versus 27.8 ± 4.1 mm), and the 99th-percentile values used to define the upper limit

of normal were 40 mm in male athletes and 38 mm in female athletes; only 0.17% of male athletes and 0.4% of female athletes exceeded these thresholds, and independent predictors of aortic size on multivariable analysis were body surface area and left ventricular end-diastolic diameter rather than black ethnicity or years of training [4]. Over a mean follow-up of 5 ± 1.5 years, none of the athletes with an enlarged aortic root at baseline showed further significant enlargement, and no aortic events occurred in this cohort [4].

A separate British Society of Echocardiography guideline synthesising multiple cohorts proposes a consensus upper reference limit for the sinuses of Valsalva of 39 mm in male athletes and 33 mm in female athletes, close to, though modestly lower than, the values reported by Gati and colleagues, and explicitly cautions that these cut-offs do not apply to masters endurance athletes aged 50–70 years, among whom an aortic root diameter exceeding 40 mm has been reported in 31% of men and 8% of women [12]. This guideline further stresses that the aortic root diameter is linearly related to height and should therefore always be scaled appropriately, and it provides a structured, multifactorial framework—incorporating sporting discipline, ethnicity, body size, sex, and age—for interpreting borderline findings rather than relying on a single absolute cut-off [12]. A complementary narrative review of the evolving assessment of physiology versus pathology in athletes reiterates that aortic dimensions in young athletes generally fall within the normal range for the general population despite the extremes of height and weight seen in some disciplines, that absolute ascending aortic values of 40 mm or more in men and 34 mm or more in women should prompt further evaluation, and that in a cohort of professional basketball players—a population selected for extreme height—only 4.6% had an aortic root of 40 mm or more and only 1.3% reached a maximum of 42 mm, with any measurement exceeding 42 mm considered frankly pathological and warranting full work-up [13]. Taken together, these sources converge on the principle that absolute diameter alone is an imperfect discriminator, that indexing and multifactorial context are essential, and that even in populations selected for anthropometric extremes, aortic dilation beyond accepted thresholds remains uncommon.

5. Diagnostic and Imaging Modalities for Assessment of the Aortic Root in Athletes

Transthoracic echocardiography remains the first-line and most widely available tool for assessing the aortic root in athletes, offering non-invasive, repeatable, and radiation-free imaging suitable for serial surveillance, and its role in pre-participation and follow-up assessment of

athletes has been described in detail in dedicated evaluation frameworks [5]. Echocardiographic protocols recommended for athlete evaluation typically extend beyond the minimum adult dataset to include additional parasternal short-axis views for wall thickness measurement at multiple levels, assessment of coronary artery origin and course, quantification of trabeculation, and, where feasible, global longitudinal strain, alongside dedicated measurement of the aortic root and proximal ascending aorta [12]. Three-dimensional echocardiography has been proposed as an adjunct that can better characterise chamber volumes and detect asymmetric aortic dilation that may be underestimated by the two-dimensional parasternal long-axis view, since the aortic root is not invariably symmetrical in orthogonal planes, particularly in the presence of an underlying aortopathy [12].

Cardiac magnetic resonance imaging offers complementary information where echocardiographic windows are technically limited or where tissue characterisation is required to distinguish pathological hypertrophy from athletic remodeling; a broad review of magnetic resonance applications in cardiac disease describes how late gadolinium enhancement imaging and native T1 mapping can identify myocardial fibrosis and quantify extracellular volume, thereby helping to differentiate pathological left ventricular hypertrophy—where extracellular volume is typically expanded—from the athlete's heart, in which extracellular volume remains within the normal range despite increased wall thickness [8]. The same source describes the established role of magnetic resonance and computed tomography angiography in delineating the full thoracic aorta, including segments poorly visualised by echocardiography, and in characterising specific cardiomyopathic and inflammatory conditions (hypertrophic and dilated cardiomyopathy, myocarditis, amyloidosis, sarcoidosis, and Anderson–Fabry disease) that may need to be excluded when an athlete presents with an ambiguous combination of increased wall thickness and aortic enlargement [8]. Four-dimensional flow magnetic resonance imaging has additionally been used to characterise regional wall shear stress abnormalities in bicuspid aortic valve disease, an application discussed further in Section 10 [10].

Beyond structural imaging, functional and provocative testing has an increasing role. Exercise stress echocardiography can unmask reduced ventricular contractile reserve that is not apparent at rest and can help differentiate physiological low-normal resting function, which should augment vigorously with exertion, from early pathological dysfunction, which typically does not [14]. In the specific context of a possible aortopathy, dedicated protocols assessing blood pressure

responses during standardised isometric and dynamic exercise—described further in Sections 8 and 15—provide functional information about haemodynamic reactivity that static imaging cannot capture [15]. Genetic testing, finally, has an increasingly defined role when clinical, imaging, or family-history features raise suspicion of a heritable aortopathy, and is discussed in detail in Section 11 [16].

6. Aortic Root Dimensions in Strength and Power Athletes: Evidence from Comparative Echocardiographic Studies

The central empirical question addressed by this review—whether strength and power training produces meaningful aortic root enlargement—has been investigated directly in several comparative echocardiographic studies, summarised in Table 1, and the overall pattern that emerges is one of reassurance rather than concern, albeit with important caveats regarding specific subgroups and study designs.

In a cross-sectional analysis of 2,083 competitive athletes stratified by the static and dynamic components of their sport according to conventional classification, absolute and indexed aortic root diameters increased progressively from skill sports through power, mixed, and endurance categories, with the largest values recorded in athletes engaged in high-dynamic disciplines; height-indexed aortic diameter was 15.6 ± 1.5 mm/m in the skill group, 16.0 ± 1.6 mm/m in the power group, 16.4 ± 1.6 mm/m in the mixed group, and 16.7 ± 1.7 mm/m in the endurance group [11]. Notably, the power-sport category—dominated by disciplines with a substantial isometric or high-resistance component—did not display the largest aortic dimensions in this dataset; rather, its values were intermediate, closer to those of skill athletes than to those of endurance athletes, directly contradicting any simple assumption that resistance or static loading is the principal driver of aortic enlargement [11].

A subsequent study from the same research group, examining 993 athletes divided into skill ($n=100$), power ($n=227$), mixed ($n=454$), and endurance ($n=212$) categories, reported broadly consistent findings: absolute aortic root diameter was 29.3 ± 3.1 mm in skill athletes, 30.1 ± 3.3 mm in power athletes, 30.1 ± 3.3 mm in mixed-sport athletes, and 31.0 ± 3.4 mm in endurance athletes, with the endurance group alone reaching statistical significance compared with the others after indexing to body surface area (16.8 ± 1.5 mm/m² versus 15.9 mm/m² in the other three groups) [17]. In the same cohort, left ventricular end-diastolic diameter and left ventricular mass rose in

parallel with aortic root size across the four categories, being smallest in skill athletes and largest in endurance athletes, while the ratio between aortic root diameter and left ventricular end-diastolic diameter (the so-called "golden ratio," reflecting harmonic, proportionate cardiac remodeling) was similar across all athletic subgroups, at approximately 0.58–0.60, and clearly distinct from the ratio of 0.81 ± 0.06 observed in a separate cohort of 498 patients with known pathological aortic dilation [17]. This ratio-based approach, validated by receiver operating characteristic analysis with a cut-off of 0.71 achieving 96% sensitivity and 99% specificity for distinguishing pathological from physiological aortic enlargement, suggests that the proportionate nature of remodeling, rather than absolute aortic size alone, may be the more clinically useful discriminator in strength and power athletes, whose left ventricular cavity size is often smaller than that of endurance athletes for a comparable aortic diameter [17].

Direct head-to-head comparison of powerlifters and long-distance runners provides further, population-specific evidence. In a comparative cross-sectional study of 16 powerlifters and 24 long-distance runners, powerlifters demonstrated markedly greater interventricular septal thickness, posterior wall thickness, and left ventricular mass, together with higher resting systolic and diastolic blood pressure and higher peripheral vascular resistance, yet the absolute aortic diameter did not differ significantly between the two groups (31.3 ± 3 mm in powerlifters versus 32.0 ± 2.7 mm in runners, $p=0.410$) [18]. Endothelial function, assessed by brachial artery flow-mediated dilation, was likewise similar between groups despite the elevated peripheral vascular resistance in powerlifters, and no participant in either group showed evidence of pathological cardiac dysfunction [18]. This dissociation—pronounced ventricular hypertrophy and elevated blood pressure in powerlifters without a corresponding increase in aortic root diameter relative to endurance-trained comparators—is one of the most direct pieces of evidence available that strength training per se does not straightforwardly translate into aortic enlargement, even when it clearly does translate into concentric ventricular remodeling.

A parallel echocardiographic and electrocardiographic comparison across long-distance runners, volleyball players, football players, powerlifters, bodybuilders, and sedentary controls similarly found that powerlifters and bodybuilders had significantly greater interventricular septal thickness, posterior wall thickness, and relative wall thickness than every other group, with concentric left ventricular hypertrophy present in 58% of powerlifters and 54% of bodybuilders, compared with normal geometry or eccentric hypertrophy in the majority of long-distance

runners, volleyball players, and football players [19]. Although this study did not report aortic root diameter, it documented significantly prolonged corrected QT dispersion in powerlifters (60.26 ± 7.30 ms) and bodybuilders (57.41 ± 5.69 ms) compared with sedentary controls (47.96 ± 8.20 ms) and with dynamically trained athletes (31–36 ms), correlating with septal and posterior wall thickness across most groups, and it explicitly framed the observed remodeling as falling within a "grey zone" where extreme athletic adaptation can overlap with mild morphological hypertrophic cardiomyopathy, even though left ventricular end-diastolic diameters (approximately 52–53 mm in powerlifters and bodybuilders) remained within limits considered reassuring for distinguishing athlete's heart from cardiomyopathy [19]. This work is important context for the present review because it demonstrates that concentric hypertrophy and electrical markers of ventricular repolarisation heterogeneity in strength athletes can occur without any accompanying data on aortic size, underscoring a gap in the literature: comprehensive electro-structural phenotyping of strength athletes has more often prioritised the ventricle than the aortic root.

Not all resistance-loaded populations behave identically, however. A three-year longitudinal study of 247 collegiate American-style football athletes—a population combining substantial isometric strength training with team-sport dynamic components—found progressive aortic root dilation from a baseline of 31.7 mm (95% CI 31.4–32.0) to 33.5 mm (95% CI 33.1–33.8) by the end of the third season, a cumulative mean increase of 1.8 mm (95% CI 1.5–2.0, $p < 0.001$), accompanied by increases in body weight, systolic blood pressure, pulse wave velocity, and left ventricular mass index, and by a reduction in early diastolic tissue velocity (E') [8]. No athlete developed an aortic root of 40 mm or more during the study period, and multivariable analysis identified higher weight, higher pulse wave velocity, higher left ventricular mass index, and lower E' as independent predictors of aortic root size, rather than player position (lineman versus non-lineman) per se [8]. The authors explicitly framed this progressive dilation as a potential maladaptive cardiovascular phenotype, paralleling other described adaptations in this population (arterial stiffening, weight gain, and diastolic impairment), rather than as unequivocally benign remodeling, and they contrasted their findings with a separate cohort of masters-level endurance athletes in whom about 30% displayed aortic dilation of a different, seemingly more stable, character (discussed further in Section 14) [8].

Finally, a study explicitly designed to explore the relationship between exaggerated exercise blood pressure responses and aortic size in 652 elite male athletes competing in handball, basketball, ice hockey, and soccer—team sports with substantial dynamic and static components—found that athletes with an exaggerated blood pressure response to a standardised cycle ergometer test (defined as a systolic blood pressure/metabolic equivalent slope greater than 6.2 mmHg/MET, present in 29% of the cohort) had a significantly larger aortic root (32.8 ± 3.3 mm versus 31.9 ± 3.2 mm, $p < 0.001$) and a higher aortic-root-to-left-ventricular-diameter ratio than athletes with a normal blood pressure response, even though absolute values remained low overall, with only 1.1% of the entire cohort reaching an aortic root of 40 mm or more, and every one of those individuals falling within the exaggerated-blood-pressure-response subgroup [20]. This finding links a specific, measurable haemodynamic phenotype—an exaggerated pressor response to exertion, conceptually analogous to the blood pressure surges generated during heavy resistance exercise—to a modest but consistent increase in aortic size, providing one of the more direct mechanistic bridges in the literature between acute exercise haemodynamics and chronic aortic remodeling [20].

Table 1. Comparative studies of aortic root dimensions in strength, power, and mixed-sport athletes.

Source [ref]	Design	Population / focus	Principal findings
Gati et al. [4]	Cross-sectional, medium-term follow-up	3,781 athletes vs. 806 sedentary controls, 14–35 years	Aortic root only marginally larger in athletes (28.3 vs 27.8 mm); 99th-percentile cut-offs 40 mm (men)/38 mm (women); no progression over 5 ± 1.5 years in dilated cases
Cavigli/Abulí et al. [21]	Cross-sectional	2,083 athletes stratified by	Aortic diameters lowest in skill and

		static/dynamic sport component	power categories, highest in endurance category, using multiple scaling models
Cavigli et al. [15]	Cross-sectional, three-group comparison	993 athletes (skill, power, mixed, endurance) vs. 410 sedentary controls vs. 498 patients with pathological aortic dilation	Power athletes had intermediate aortic root size (30.1 ± 3.3 mm); aortic-root/LVEDD "golden ratio" $\sim 0.59\text{--}0.60$ in all athlete subgroups vs. 0.81 ± 0.06 in pathological dilation; ROC cut-off 0.71 (96% sensitivity, 99% specificity)
Silva et al. [13]	Cross-sectional, two-group comparison	16 powerlifters vs. 24 long-distance runners	No significant difference in aortic diameter (31.3 vs 32.0 mm) despite markedly greater LV hypertrophy, blood pressure, and peripheral vascular resistance in powerlifters
Stoičkov et al. [22]	Cross-sectional, six-group comparison	Long-distance runners, volleyball, football, powerlifters, bodybuilders,	Concentric LV hypertrophy in most powerlifters (58%) and bodybuilders

		sedentary controls	(54%); elevated corrected QT dispersion; no aortic root data reported
Tso et al. [20]	Longitudinal, 3-year cohort	247 collegiate American-style football athletes	Progressive aortic root dilation (31.7→33.5 mm) associated with weight gain, arterial stiffening, and reduced diastolic function; no athlete reached ≥ 40 mm
Most et al. [23]	Cross-sectional	652 elite male mixed-sport athletes stratified by blood pressure response to exercise	Exaggerated blood pressure responders had larger aortic root (32.8 vs 31.9 mm) and higher aortic-root/LVEDD ratio; all athletes with root ≥ 40 mm were in this subgroup

Considered together, these studies do not support the notion that strength or power training, as an isolated exposure, produces the pronounced or progressive aortic root dilation described in some hereditary aortopathies. Absolute aortic root diameters in powerlifters and bodybuilders are generally comparable to or smaller than those of endurance athletes, and in direct comparison with runners, powerlifters showed no aortic difference despite substantially greater ventricular hypertrophy and blood pressure elevation [18]. Where progressive dilation has been documented, as in collegiate American-style football players, it has occurred in a population combining strength training with substantial weight gain, systemic blood pressure elevation, and arterial stiffening, and the authors themselves characterised the phenomenon as potentially maladaptive

rather than simply physiological [8]. The exaggerated-blood-pressure-response data further suggest that it may be the haemodynamic reactivity of an individual athlete—rather than the nominal training modality—that best predicts aortic size, a theme developed mechanistically in Section 8 [20].

7. Static versus Dynamic Exercise Components and Aortic Remodeling

The classification of sporting disciplines according to their relative static (isometric) and dynamic (isotonic) components, first formalised for athlete eligibility purposes and subsequently adopted in echocardiographic research, provides a useful organising framework for interpreting the sometimes counterintuitive findings summarised in Section 6 [11]. Under this framework, powerlifting and Olympic weightlifting are classified as having a high static and low-to-moderate dynamic component, while bodybuilding, though not a competitive endurance activity, similarly involves predominantly resistance-based training; American-style football combines a moderate-to-high static component with variable dynamic demands depending on playing position; and pure endurance sports such as long-distance running, cycling, and rowing sit at the opposite end of the dynamic spectrum with comparatively little isometric loading [11].

The cross-sectional analysis of 2,083 athletes stratified by these categories found that aortic dimensions increased monotonically from skill through power, mixed, and endurance categories using every scaling approach tested, including absolute diameter, ratiometric indexing to body surface area, indexing to height, and allometric scaling using population-derived exponents [11]. This ordering—power athletes showing smaller, not larger, aortic dimensions than mixed and endurance athletes—runs directly counter to any hypothesis that isometric, pressure-predominant loading is the principal stimulus for aortic enlargement, and instead is more consistent with the alternative hypothesis that sustained volume load and the cumulative haemodynamic exposure of prolonged dynamic exercise (which typically also involves substantially greater total training volume and duration per session than resistance training) are the dominant determinants of aortic size [11].

A separate analysis in the same research programme, examining 993 athletes and again stratifying by sport type, confirmed that endurance athletes had significantly larger absolute and indexed aortic root diameters than skill, power, or mixed-sport athletes, while the aortic-root-to-left-ventricular-diameter ratio was, if anything, marginally lower in endurance athletes

(0.58 ± 0.06) than in power athletes (0.60 ± 0.05), reflecting the fact that the left ventricular cavity enlarges proportionately more than the aortic root in the endurance-trained heart [17]. This proportionate relationship—larger absolute aortic size in endurance athletes but preserved or even slightly more conservative aortic-to-ventricular ratios—illustrates why absolute diameter alone can be a misleading metric when comparing across sport types, and why ratio-based or indexed approaches are particularly valuable when the population under consideration (strength and power athletes) tends to have smaller absolute aortic dimensions but is nonetheless being evaluated against reference ranges derived predominantly from mixed athletic cohorts [17].

The collegiate American-style football cohort offers a further layer of nuance because it represents a population with a genuinely high static training component embedded within a team-sport context that also involves dynamic play and, critically, substantial and deliberate weight gain [8]. In this cohort, aortic root dilation was progressive and statistically significant, and multivariable analysis identified body weight, pulse wave velocity (a marker of arterial stiffness), left ventricular mass index, and diastolic dysfunction as independent predictors, while player position—linemen, who perform the most isometric, high-static work, versus non-linemen—was not an independent predictor after adjustment [8]. This finding suggests that within a single sport, the aortic phenotype is driven less by the specific biomechanical demands of a playing position and more by systemic factors (weight, vascular stiffness, cardiac remodeling) that happen to be common across the roster, a distinction with direct implications for how "strength sport" exposure should be conceptualised in future research: not as a single homogeneous stimulus, but as a composite of training modality, body composition change, and haemodynamic reactivity that may vary considerably even within a nominally single-discipline cohort.

The exaggerated-blood-pressure-response study reinforces this reframing by demonstrating that, within a single cohort of mixed dynamic/static team-sport athletes, it was the individual haemodynamic response to exertion—rather than the sport itself—that discriminated athletes with larger aortic roots from those with smaller ones [20]. Considered alongside the powerlifter-versus-runner comparison, in which powerlifters had markedly higher resting blood pressure and peripheral vascular resistance without a corresponding increase in aortic diameter [18], the overall picture is one in which resting blood pressure elevation associated with strength training does not, by itself, appear sufficient to drive clinically important aortic enlargement, whereas a specific pattern of exaggerated pressor reactivity during dynamic effort, high cumulative training

volume, and systemic factors such as weight gain and arterial stiffening appear more consistently associated with aortic remodeling across the available literature.

8. Acute Hemodynamic Stress, Aortic Wall Stress, and Vascular Stiffness during Resistance Exercise

Understanding whether strength and power training poses an acute, mechanistic threat to the aortic wall requires moving beyond static structural comparisons to consider what actually happens to blood pressure, wall stress, and vascular stiffness during the performance of heavy resistance exercise itself. A detailed review of the acute and chronic vascular effects of resistance exercise training describes how a single bout of resistance exercise transiently increases large-artery (aortic and carotid) stiffness through both load-dependent mechanisms (the recruitment of stiffer collagen fibres as distending pressure rises and stretches the more compliant elastin network) and load-independent mechanisms, including increased sympathetic vasomotor outflow, transient endothelial dysfunction with reduced nitric-oxide bioavailability, elevated circulating endothelin-1, and viscoelastic "stress relaxation" phenomena intrinsic to the arterial wall itself [9]. According to the Law of Laplace, wall tension is proportional to transmural pressure and vessel radius and inversely proportional to wall thickness; because resistance exercise, particularly when combined with a Valsalva manoeuvre, produces marked but transient elevations in intravascular pressure, the aortic wall must dynamically adjust its stiffness and smooth-muscle tone to preserve wall tension within a safe operating range during each repetition [9].

This same source reports that these acute stiffness increases are transient, typically resolving within approximately 30–60 minutes after exercise, and are not accompanied by evidence of structural elastin fatigue fracture, collagen deposition, or other frank ultrastructural injury from a single exercise bout [9]. However, when resistance training is performed habitually over months to years, the picture from cross-sectional studies is less uniform: some landmark comparisons of habitually strength-trained athletes with age-matched, non-strength-trained controls have documented higher brachial and carotid pulse pressure, lower systemic arterial compliance, and higher proximal aortic stiffness (measured as stiffness index β and elastic modulus) in strength athletes, despite similar aortic geometry between groups, while randomised training-intervention studies have produced markedly inconsistent results, with some early trials reporting reduced

carotid compliance after months of resistance training (fully reversible after a subsequent period of detraining) and more recent meta-analyses concluding that resistance training has negligible or even favourable effects on large-artery stiffness in most populations [9]. The same review notes the important observation that muscular strength itself, as distinct from the training modality used to achieve it, is consistently and independently associated with lower arterial stiffness, better vascular endothelial function, and reduced cardiovascular mortality, creating an apparent paradox between the higher aortic stiffness sometimes observed in habitually strength-trained athletes as a group and the protective association of muscular strength as an outcome measure at the individual level [9]. This paradox has not been fully reconciled in the literature and is presented here as an explicit point of uncertainty rather than resolved by extrapolation.

Direct measurement of aortic wall stress during resistance-type exercise, rather than inference from stiffness indices alone, has been performed in patients with an established ascending thoracic aortic aneurysm undergoing submaximal dynamic handgrip exercise. In this study of eight patients with an aortic diameter exceeding 40 mm compared with eleven age-matched controls, aortic wall stress during handgrip exercise performed without a Valsalva manoeuvre was not significantly different between the aneurysm group and controls, and in both groups remained approximately 4.4-fold lower than the maximal tensile strength measured *ex vivo* in aneurysmal ascending aortic tissue [24]. This reassurance was attributable in part to a compensatory increase in aortic wall thickness in the aneurysm group, which the authors interpreted as providing a protective mechanical buffer against the transient rise in systolic blood pressure that did occur (and that was, notably, significantly larger in the aneurysm group than in controls) [24]. The authors explicitly cautioned, however, that their measurements used non-invasive blood pressure, which is known to underestimate invasively measured pressure by 12–14% during resistance-type exercise, and that their findings related specifically to small-muscle-mass, moderate-intensity handgrip contraction rather than to the large-muscle-mass, near-maximal, Valsalva-associated lifts (such as heavy squats or deadlifts) that are typical of competitive powerlifting and that are believed to generate the largest acute blood pressure elevations of any common exercise modality [24].

Complementary data from a broader safety study of 31 patients with thoracic aortic aneurysms or dissections (including several with bicuspid aortic valve, Marfan syndrome, Turner syndrome, or vascular Ehlers–Danlos syndrome) performing a standardised circuit of isometric and dynamic

exercises—hand grips, leg raises, bicep curls, stationary cycling, and wall sits—found no significant difference in maximum exertional systolic or diastolic blood pressure change between patients and 14 unaffected controls, and no protocol was terminated because of severe exertional hypertension [25]. Among the exercises tested, the wall-sit manoeuvre produced the largest blood pressure changes in both groups, and self-reported Borg perceived-exertion scores correlated with the magnitude of blood pressure change, supporting the use of perceived exertion as a practical, individualisable proxy for haemodynamic load when supervising exercise in at-risk patients [25]. Importantly, patients who reported higher weekly moderate-activity levels at baseline experienced fewer episodes of exertional hypertension during testing, a finding consistent with a training or habituation effect on blood pressure reactivity to physical stress [25].

Together, these mechanistic and safety-oriented studies indicate that the acute aortic wall-stress burden of moderate-intensity resistance exercise, even in patients with a pre-existing aneurysm, is likely to remain substantially below the threshold associated with catastrophic mechanical failure, and that habitual physical activity may attenuate rather than exacerbate the pressor response over time [24,25]. However, both studies explicitly examined submaximal, moderate-intensity protocols using relatively small muscle groups or standardised circuits, not the maximal or near-maximal, whole-body, Valsalva-associated efforts characteristic of competitive powerlifting, and neither study can be extrapolated with confidence to that more extreme end of the resistance-exercise intensity spectrum, a gap that is returned to in the discussion of exercise prescription in Section 15.

9. Anabolic-Androgenic Steroid Use and Aortic Dissection Risk in Strength Athletes

No discussion of aortic risk in strength and power athletes can be complete without addressing the use of anabolic-androgenic steroids and other performance-enhancing substances, which are disproportionately prevalent in bodybuilding, powerlifting, and related disciplines and which have been directly implicated in fatal aortic events in previously healthy young athletes. A case series of five bodybuilders and weightlifters, aged 27 to 37 years, all of whom presented with ascending aortic dissection temporally associated with heavy resistance training, illustrates this association in stark clinical terms [26]. Each patient had a documented history of anabolic-androgenic steroid use—typically supraphysiological doses of intramuscular testosterone, sometimes combined with growth hormone or other agents—and most presented with markedly

elevated blood pressure and biochemical evidence of myocardial injury (elevated troponin and creatine kinase) at the time of presentation [26]. In each case, dissection or its complications occurred during or immediately after heavy lifting, with symptoms including sudden retrosternal or interscapular pain, syncope, and haemodynamic collapse; despite emergency surgical repair in most cases, the clinical course was frequently complicated by cardiogenic shock, stroke, renal failure, or death, with the authors citing an overall mortality of approximately 32% for aortic dissection in young patients generally [26].

The proposed pathophysiological mechanism linking anabolic-androgenic steroid use to aortic dissection in this population is multifactorial. Chronic supraphysiological androgen exposure has been associated with atherogenic lipid changes (elevated low-density lipoprotein, reduced high-density lipoprotein), sustained hypertension, and, most specifically, alteration of collagen synthesis and degradation within the aortic media, reducing the tensile strength and elasticity of the vessel wall and rendering it more susceptible to intimal tearing under conditions of acute pressure surge [26]. The authors emphasise that skeletal muscle hypertrophy and strength gains induced by resistance training combined with steroid use occur more rapidly than the corresponding adaptive strengthening of connective tissue, creating a period of relative vulnerability in tendons, ligaments, and vascular walls during which the musculoskeletal system can generate forces that its supporting connective tissue has not yet adapted to withstand [26].

A separate, individually detailed case report describes a 37-year-old competitive weightlifter with a documented history of hypertension who developed a Stanford type A aortic dissection—extending from the ascending aorta through the transverse arch into the descending thoracic aorta—during heavy weightlifting [27]. Echocardiography demonstrated concentric left ventricular hypertrophy, a dilated ascending aorta of 56 mm, and mild mitral regurgitation, and computed tomography angiography confirmed an extensive intimal tear originating just above the sinuses of Valsalva; despite emergency Bentall repair of the aortic root and ascending aorta, the patient developed a major cerebral infarction and died on the first postoperative day [27]. The authors of this case explicitly invoke the Morganroth hypothesis of concentric hypertrophy in strength-trained athletes and argue that the combination of chronic hypertension, established concentric hypertrophy, and a rapid, unopposed rise in systemic arterial pressure during lifting—without a compensatory fall in peripheral vascular resistance—together with underlying aortic medial degeneration, created the mechanical conditions for intimal failure [27]. They further note

that predisposing conditions recognised to increase dissection risk in this setting include hypertension, congenital cardiovascular anomalies (coarctation of the aorta, congenital or bicuspid aortic stenosis), connective-tissue disorders such as Marfan syndrome, and fibromuscular dysplasia, and they recommend that athletes with any of these predisposing factors be actively discouraged from heavy resistance training and that blood pressure monitoring be considered during weightlifting for those at risk [27].

These case reports, though limited by their retrospective and uncontrolled design and by the inherent impossibility of establishing causality from isolated clinical narratives, are corroborated by broader epidemiological reviews of thoracic aortic dissection, which explicitly list weightlifting among the recognised acquired risk factors for the condition, alongside trauma, iatrogenic injury, and diagnostic delay, and which separately identify uncontrolled hypertension as the single most prevalent comorbidity in dissection cohorts generally, present in 45–100% of cases across different registries, with a population-attributable risk for dissection estimated at approximately 54% in one large prospective study [28]. This same source describes the biomechanical rationale for why the ascending aorta is the segment most vulnerable to acute pressure-related failure, and reiterates that most cases of type A dissection in the general population occur at aortic diameters below the conventional 55 mm surgical threshold—indeed, below 50 mm in a substantial minority of cases—implying that a modestly dilated or even normal-appearing aorta does not exclude catastrophic risk when combined with severe, unmanaged hypertension and an acute pressor stimulus such as heavy lifting [28].

Taken as a body of evidence, the case reports and epidemiological data reviewed in this section suggest that anabolic-androgenic steroid use, uncontrolled hypertension, and heavy resistance exercise interact synergistically to produce a distinct, recognisable, and potentially preventable clinical syndrome of acute aortic dissection in strength athletes, a syndrome that appears mechanistically and clinically separable from the modest, largely benign aortic remodeling described in Sections 6 and 7 among drug-free strength athletes without hypertension. This distinction has direct clinical implications, discussed further in Sections 15 and 16: an enlarged or dissected aorta in a strength athlete should prompt specific enquiry into performance-enhancing drug use and blood pressure control before being attributed, by default, to training-induced physiological adaptation.

10. Bicuspid Aortic Valve and Physical Activity: Implications for Strength and Power Athletes

Bicuspid aortic valve disease is the most common congenital cardiac malformation, affecting approximately 0.5–2% of the general population with a male predominance of roughly three to one, and is a recognised cause of progressive aortic valve dysfunction and thoracic aortopathy, including dilation of the aortic root and ascending aorta and, less commonly, dissection [2]. Because of its prevalence, bicuspid aortic valve disease will inevitably be encountered incidentally among strength and power athletes, and the interaction between this pre-existing vascular vulnerability and the haemodynamic demands of resistance training is a specific and clinically important sub-question within the broader topic of this review.

Reassuringly, the balance of the available evidence in patients with bicuspid aortic valve disease does not support a straightforward, dose-dependent relationship between exercise volume or intensity and accelerated aortic dilation. A cross-sectional study of 407 patients with bicuspid aortic valve disease, in which lifelong exercise volume, intensity, and sport type were quantified using a validated questionnaire and related to aortic diameters at the sinuses of Valsalva and ascending aorta, found no association between exercise volume and either valvular dysfunction or aortic dilation; indeed, patients who predominantly performed vigorous-intensity exercise or mixed-type sports had a significantly lower prevalence of moderate-to-severe aortic stenosis than sedentary patients, and no exercise characteristic was associated with aortic regurgitation or with dilation at either the sinuses of Valsalva or the ascending aorta [29]. The authors concluded that lifelong exercise does not appear to induce adverse cardiovascular effects in patients with bicuspid aortic valve disease and suggested that exercise may even confer benefit, although they appropriately noted that their cross-sectional design cannot exclude the possibility that patients with more severe disease had been advised, or had chosen, to reduce their activity level, potentially masking a true adverse effect [29].

A separate, multicentre cross-sectional study directly comparing 186 competitive athletes with bicuspid aortic valve disease, 193 age- and sex-matched athletes with a normal tricuspid aortic valve, and 125 non-athletic patients with bicuspid aortic valve disease found that athletes with bicuspid valves had larger aortic annulus, sinus of Valsalva, sinotubular junction, and ascending aortic diameters, and a higher prevalence of mild aortic regurgitation, than tricuspid-valve

athletes, but showed no significant difference in any aortic dimension or in valvular function compared with non-athletic bicuspid valve patients [30]. This finding—that the aortic phenotype of bicuspid valve athletes closely resembles that of non-athletic bicuspid valve patients, rather than showing training-attributable enlargement beyond what the underlying valve disease itself produces—was interpreted by the authors as broadly reassuring regarding the safety of competitive sport in this population, though they did identify a distinct and unexpected finding: bicuspid valve athletes had significantly greater left ventricular end-diastolic and end-systolic volumes and left ventricular mass than both tricuspid-valve athletes and non-athletic bicuspid valve patients, suggesting a pattern of left ventricular remodeling in bicuspid valve athletes that exceeds what would be expected from training alone and that may reflect the additional haemodynamic burden imposed by the valve lesion itself superimposed on exercise-induced adaptation [30].

Pattern of physical activity itself may differ in this population in ways relevant to overall cardiovascular risk. An accelerometer-based study comparing 100 adults with bicuspid aortic valve disease with 100 age- and sex-matched controls found that patients with bicuspid valve disease spent significantly less time in moderate-to-vigorous physical activity (72 versus 98 minutes per day) and accumulated fewer daily steps (4,826 versus 6,252) than controls, despite similar total sedentary time, with the degree of inactivity correlating with disease severity, and with nearly half of patients reporting uncertainty about what level of physical activity was safe for their condition [2]. This same review describes paediatric data in which competitive sports participation in children with isolated bicuspid aortic valve was not associated with increased aortic diameter compared with non-athletes, and was instead associated with a substantially lower prevalence of obesity, and it summarises longitudinal data from a cohort of elite athletes with bicuspid valve disease followed for up to thirteen years, in whom disease progression in genuinely high-risk individuals occurred independently of prior athletic exposure, while low-risk athletes generally maintained a benign clinical course [2]. A dedicated paediatric study of 90 patients with isolated bicuspid aortic valve, 53 of whom were physically active and 37 sedentary, found no significant difference between groups in the proportion showing sinus of Valsalva or ascending aortic diameter progression of 10% or more over two years of follow-up, nor in the magnitude of progression among those who did progress, supporting the conclusion that regular

physical activity does not accelerate aortic dilation in paediatric bicuspid valve patients over this time frame [31].

A comprehensive review of bicuspid aortic valve aortopathy situates these clinical findings within a broader pathophysiological framework, describing the genetic basis of the disease (including variants in ACTA2, SMAD6, NOTCH1, ROBO4, GATA5, TGFBR1/2, FBN1, ADAMTSL1, ADAMTS4, and NOS3), the characteristic histopathological findings of cystic medial necrosis, elastic fibre fragmentation, smooth muscle cell loss, and imbalance between matrix metalloproteinases and their tissue inhibitors, and the haemodynamic mechanism by which the eccentric transvalvular jet produced by the malformed valve generates abnormal, non-uniform wall shear stress that varies according to the specific cusp-fusion pattern (right-left versus right-non-coronary fusion), producing distinct regional dilation phenotypes [10]. This review further notes that current surgical guidelines recommend aortic intervention at a diameter of 55 mm in the absence of risk factors, or 50 mm in the presence of risk factors such as a family history of dissection, rapid growth, uncontrolled hypertension, or a specific "root phenotype," and it cites a specific elite-athlete cohort in which high-intensity training and sport competition did not appear to aggravate bicuspid valve disease symptoms during the athletic career [10]. Contemporary eligibility guidance, discussed in more detail in Section 16, converges with these observations: in the absence of significant aortic dilation or valvular dysfunction, current recommendations permit bicuspid valve athletes to participate in competitive sport under the same principles applied to athletes with a normal tricuspid valve [2].

Nonetheless, the potential for coexisting bicuspid valve disease and haemodynamically significant regurgitation to overlap with, and be mistaken for, training-induced left ventricular dilation is illustrated vividly by an individual case report of a 31-year-old elite triathlete with bicuspid aortic valve regurgitation who presented with severe left ventricular dilation (end-diastolic diameter 70 mm) and aortic root dilation to 49 mm at the sinuses of Valsalva, with 42 mm at the ascending aorta [24]. The authors of this case emphasised that a left ventricular end-diastolic diameter of this magnitude, in the absence of proportionate right ventricular dilation, exceeded what would be expected from athletic adaptation alone, and that the aortic dimensions, although consistent with values occasionally reported in endurance athletes, most likely reflected the combined effect of chronic aortic regurgitation and training rather than training in isolation; the patient ultimately required combined aortic root replacement and mechanical valve

implantation [24]. This case underscores that although population-level data on bicuspid valve athletes are broadly reassuring, individual patients with coexisting significant valvular dysfunction require careful, individualised multimodality assessment rather than reassurance drawn from group averages, a point directly relevant to strength and power athletes in whom incidental bicuspid valve disease with important regurgitation could similarly be mistaken for, or masked by, expected training-related cardiac remodeling.

11. Hereditary Thoracic Aortic Disease: Marfan Syndrome, Loeys–Dietz Syndrome, and Vascular Ehlers–Danlos Syndrome

Beyond bicuspid aortic valve disease, a small but clinically critical proportion of athletes presenting with aortic dilation will harbour a heritable connective-tissue disorder in which the aortic wall itself is intrinsically abnormal, and in which the risk of dissection at any given diameter, or even at diameters within the conventionally normal range, is substantially higher than in the general athletic population. A comprehensive review of hereditary aortopathies as a cause of sudden cardiac death in the young provides a structured classification of these conditions into syndromic hereditary aortopathies—Marfan syndrome, Loeys–Dietz syndrome, and vascular Ehlers–Danlos syndrome—and non-syndromic hereditary aortopathies, comprising non-syndromic familial thoracic aortic aneurysm and dissection and non-syndromic bicuspid aortic valve disease [16]. This source describes the shared pathogenetic mechanisms underlying these conditions, principally alterations in extracellular matrix proteins (fibrillin-1 in Marfan syndrome, type III collagen in vascular Ehlers–Danlos syndrome), dysregulation of transforming growth factor-beta signalling (Loeys–Dietz syndrome), and dysfunction of vascular smooth muscle contractile proteins (non-syndromic familial thoracic aortic aneurysm and dissection), all of which converge on a weakened, less compliant aortic media prone to dilation, dissection, and rupture [16].

Marfan syndrome, the most frequent monogenic malformation syndrome associated with aortopathy, has an estimated incidence of 1 in 3,000 to 1 in 5,000 and is caused in approximately 90% of cases by mutations in the *FBN1* gene, with the remainder attributable to mutations in *TGFBR1* or *TGFBR2* [16]. Cardiovascular manifestations include mitral and, less frequently, tricuspid valve prolapse with regurgitation, and progressive dilation of the aortic root at the sinuses of Valsalva, which if untreated can progress to aneurysm formation, dissection, and

sudden cardiac death; without treatment, average life expectancy in this condition has historically been reported at around 40 years [16]. Loeys–Dietz syndrome, caused by mutations affecting components of the transforming growth factor-beta signalling pathway (TGFB1, TGFB2, SMAD2, SMAD3, TGFB2, TGFB3), is characterised by craniofacial, skeletal, cutaneous, and cardiovascular abnormalities, including arterial tortuosity and aneurysm formation that can affect multiple arterial territories, and it is recognised to follow a more aggressive clinical course than Marfan syndrome, with dissection occurring at younger ages and at smaller aortic diameters [16]. Vascular Ehlers–Danlos syndrome, caused predominantly by mutations in COL3A1 affecting type III collagen, is the rarest but most severe of these conditions, characterised by extreme tissue fragility, spontaneous arterial rupture (which may occur at multiple sites simultaneously), and a markedly shortened life expectancy, with sudden death occurring before the age of 20 in some series and being more common in males [16].

A dedicated imaging study comparing the aortic morphology of 95 patients with Marfan syndrome and 19 patients with Loeys–Dietz syndrome against 20 matched controls, using detailed computed tomography and magnetic resonance angiographic measurements of diameter, length, area, and tortuosity across the entire thoracoabdominal aorta, found that patients with Marfan syndrome had a higher prevalence of ascending aorta and aortic root dilation, a longer and larger, "pear-shaped" aortic root, and larger suprarenal aortic and iliac artery dimensions, while patients with Loeys–Dietz syndrome had longer indexed aortic segments overall and a significantly longer, more frequently type 2 or type 3 ("gothic") aortic arch, a configuration associated with a higher risk of subsequent type B dissection [32]. Critically, the authors developed and validated several simple diameter- and length-based ratios—including the aortic-root-to-ascending-aorta length divided by aortic root diameter, and the ratio of aortic root diameter to sinotubular junction diameter or to ascending aortic diameter—that could discriminate between Marfan and Loeys–Dietz syndrome even among patients without frank aortic dilation, with receiver operating characteristic analysis yielding area-under-the-curve values of 0.69–0.72 for these ratios, suggesting that morphological "phenotyping" of the aortic root complex can provide diagnostic clues before genetic testing results are available and even in the earliest, pre-dilation stages of disease [32]. This morphometric approach is conceptually analogous to the aortic-root-to-left-ventricular-diameter ratios developed to distinguish athletic remodeling from pathological dilation described in Section 6, and reinforces a broader

methodological point running through this review: proportionate, shape-based analysis of the aortic root complex may outperform absolute diameter measurement alone in distinguishing benign from pathological aortic enlargement across multiple different clinical contexts, whether athletic or hereditary.

Clinically, these hereditary aortopathies are directly relevant to the population addressed by this review because tall stature and, in some cases, increased muscularity relative to body frame can occur in variants of these syndromes, and because affected individuals—particularly adolescents and young adults who have not yet developed overt skeletal or ocular stigmata—may present initially for sports participation screening, at which point an unexplained finding of aortic root dilation must trigger a systematic evaluation for an underlying connective-tissue disorder rather than being attributed by default to strength training [16,32]. Formal international eligibility recommendations, discussed in detail in Section 16, explicitly address this scenario and provide graded restriction criteria according to the specific hereditary aortopathy diagnosed, its associated aortic diameter, and the presence of additional risk features [33].

12. Risk Factors for Thoracic Aortic Dissection Relevant to Strength and Power Athletes

Beyond the specific mechanisms of anabolic steroid use (Section 9) and heritable connective-tissue disease (Section 11), a broader epidemiological literature on risk factors for thoracic aortic dissection provides important additional context for interpreting isolated cases and guiding preventive practice in strength and power athletes. A comprehensive review of risk factors for thoracic aortic dissection identifies hypertension as the single most common comorbidity, present in 45–100% of published cohorts depending on screening intensity and case ascertainment, and reports that a large prospective population-based study found that only 67.3% of subsequently affected patients had been diagnosed with hypertension in the years before their dissection, and that even among those on antihypertensive therapy, 61.9% of subsequent blood pressure readings remained above 140/90 mmHg, implying that undertreated, rather than simply untreated, hypertension is a major contributor [28]. This same source estimates the population-attributable risk of hypertension for aortic dissection at approximately 54% and reports that the risk of dissection in hypertensive individuals is approximately four times that of normotensive individuals in the general population [28].

Among acquired, non-genetic risk factors, this review explicitly names weightlifting alongside trauma, iatrogenic injury, and diagnostic delay/misdiagnosis as a recognised precipitant of aortic dissection, situating the case reports discussed in Section 9 within a broader accepted taxonomy of risk rather than treating them as isolated curiosities [28]. The review further discusses circadian and seasonal patterning of dissection onset, sex differences in presentation and outcome (with women presenting later in life but experiencing worse acute mortality), the role of biological sex hormones (oestrogen appearing protective, progesterone showing a more complex and less consistent relationship), obstructive sleep apnoea as an independent risk factor operating through intermittent hypoxia and sympathetic activation, and, counterintuitively, diabetes mellitus as a protective factor associated with reduced aneurysm growth rates, an association attributed tentatively to glycation-related stiffening of collagen that may paradoxically counteract proteolytic degradation of the aortic wall [28]. Critically for the interpretation of surveillance thresholds, the same review reports that a substantial proportion of aortic dissections in the general population occur below the conventional 55 mm surgical intervention threshold—59% below 5.5 cm and 40% below 5.0 cm in one large registry, and 62% below 5.5 cm and 42% below 5.0 cm in another—underscoring that diameter alone, even when compared against standard surgical thresholds developed for the general population, is an imperfect predictor of dissection risk at the individual level, a caveat directly relevant to strength athletes whose aortic dimensions, as reviewed in Section 6, are typically modest but who may nonetheless carry unrecognised risk factors such as undiagnosed hypertension or steroid use [28].

The specific case of aortic dissection precipitated during weightlifting in a hypertensive individual, discussed in detail in Section 9, exemplifies several of these general risk factors converging in a single patient—longstanding, apparently under-recognised hypertension, established concentric left ventricular hypertrophy, and an acute pressor stimulus from heavy resistance exercise—and the authors of that case explicitly invoke the biomechanical principle that a rapid rise in systemic arterial pressure without a compensatory fall in peripheral vascular resistance, superimposed on pre-existing aortic medial degeneration, creates the immediate precipitating conditions for intimal tearing [27]. This convergence of risk factors, rather than any single factor in isolation, appears to be the more clinically useful conceptual model emerging from the literature reviewed in this section: aortic dissection in a strength or power athlete is unlikely to be adequately explained by training exposure alone, and a systematic search for

hypertension, steroid use, and underlying connective-tissue or valvular disease is warranted in any such presentation.

13. Case Reports of Aortic Dissection in Strength Athletes: Clinical Lessons

Although individual case reports cannot establish incidence or population-level risk, the clinical narratives available in this literature carry specific, recurring lessons that merit explicit synthesis for practitioners caring for strength and power athletes. Across the case series and individual reports reviewed in Sections 9 and 12, several consistent features emerge. First, presentation is typically abrupt and temporally linked to the performance of a heavy lift, with severe, tearing chest, back, or interscapular pain, often accompanied by presyncope, syncope, or frank haemodynamic collapse [26,27]. Second, affected individuals were frequently young—in their late twenties to late thirties—an age range in which degenerative atherosclerotic aortic disease would not ordinarily be expected, again pointing toward the combination of anabolic steroid use, hypertension, and acute mechanical stress as the operative mechanism rather than age-related vascular degeneration [26,27]. Third, despite emergency surgical intervention (aortic repair, root replacement, or Bentall procedure) being achieved in most reported cases, postoperative outcomes were frequently poor, with complications including cardiogenic shock, stroke, and death occurring even after technically successful surgery, reflecting both the severity of the underlying dissection (often extending from the ascending aorta through the arch into the descending or even abdominal aorta) and the systemic physiological derangement produced by the acute event [26,27].

Fourth, and of direct practical relevance, several of these cases involved echocardiographic findings—concentric left ventricular hypertrophy, a mildly to moderately dilated ascending aorta, and mild valvular regurgitation—that, taken in isolation and without knowledge of the clinical context, could plausibly have been interpreted as within the spectrum of expected strength-athlete cardiac remodeling described in Sections 6 and 7 [27]. This observation reinforces a central theme of this review: the echocardiographic phenotype of concentric hypertrophy with modest aortic enlargement is not, by itself, sufficient to distinguish a strength athlete undergoing benign physiological adaptation from one at imminent risk of dissection, and that history-taking specifically addressing anabolic-androgenic steroid use, blood pressure control, and family history of aortic disease or sudden death must accompany any structural assessment [26,27].

Fifth, the recommendations offered by the authors of these case reports are consistent and specific: athletes with recognised predisposing factors—hypertension, bicuspid or otherwise abnormal aortic valve morphology, coarctation of the aorta, connective-tissue disease, or fibromuscular dysplasia—should be actively counselled against unsupervised heavy resistance training, and blood pressure monitoring during weightlifting should be considered in at-risk individuals, alongside pre-participation screening incorporating echocardiography where clinically indicated [26,27].

14. Comparator Populations: Aortic Remodeling in Endurance and Masters Athletes

Because much of the comparative evidence reviewed in Sections 6 and 7 relies on contrasting strength and power athletes against endurance-trained comparators, it is important to characterise the aortic phenotype of endurance athletes in its own right, including in the older, "masters" population in whom the cumulative duration of training exposure is greatest. A retrospective cohort study of 50 masters-level endurance athletes with at least two clinically indicated transthoracic echocardiograms performed over an average interval of five years found that 19 athletes (38%) had baseline aortic dilation (defined as 40 mm or more at the sinus of Valsalva or proximal ascending aorta), and that the rate of aortic enlargement over time did not differ significantly between those with and without baseline dilation: the dilated group progressed at 0.2 mm per year at the sinus of Valsalva and 0.5 mm per year at the ascending aorta, while the non-dilated group progressed at 0.1 mm per year and 0.3 mm per year, respectively [22]. Both rates were well below the threshold of 3 mm per year conventionally considered indicative of clinically concerning "rapid" aneurysmal growth, and no participant in the dilated group progressed to 50 mm or more during follow-up [22]. Hypertension was significantly more common among masters athletes with aortic dilation than among those without (68% versus 32%), again pointing to blood pressure as a key modifying factor independent of training exposure itself, and the authors concluded that continued exercise training combined with clinical observation represents a reasonable strategy even in masters athletes with pre-existing mild aortic dilation, while cautioning that their findings, drawn from a predominantly male cohort without central adjudication of measurements or non-athlete controls, should not be extrapolated to younger athletes or those with known heritable aortopathy [22].

This comparatively benign trajectory in masters endurance athletes contrasts, at least in absolute rate of change, with the more rapid progression documented over three years in the collegiate American-style football cohort described in Section 6, in which the cumulative increase of 1.8 mm over three seasons corresponds to an average rate of approximately 0.6 mm per year—faster than either subgroup of the masters endurance cohort, though still below the 3 mm per year threshold used to define pathological rapid growth [8,22]. Because these two populations differ substantially in age, training exposure duration, and the specific combination of static and dynamic loading involved, this comparison should be interpreted cautiously; nonetheless, it is notable that the younger, actively developing strength/mixed-sport cohort showed a numerically faster rate of aortic change than the older, long-established endurance cohort, a pattern that is consistent with the hypothesis, discussed in Section 7, that ongoing weight gain and cardiovascular adaptation during a developing athletic career (rather than steady-state training exposure in an already-established masters athlete) may be a period of particular vascular plasticity, whether adaptive or maladaptive [8,22].

15. Exercise Prescription and Safety in Individuals with Known Aortic Dilation or Aortopathy

For the practising clinician, the question of whether and how a strength or power athlete with a known or suspected aortopathy can safely continue training is at least as important as the epidemiological question of population-level risk. The available evidence, though limited in scope, provides a cautiously reassuring but clearly bounded framework. As described in Section 8, direct measurement of aortic wall stress during submaximal dynamic handgrip exercise—performed without a Valsalva manoeuvre—in patients with an established ascending aortic aneurysm found no significant elevation of wall stress compared with controls, and stress remained substantially below the ex vivo tensile failure threshold of aneurysmal tissue, attributable in part to compensatory thickening of the aortic wall [24]. The authors explicitly recommended that future studies examine dynamic resistance exercise involving larger muscle groups and a wider range of intensities to identify the point at which wall stress might begin to pose genuine clinical concern, implying that their reassuring findings should not be generalised beyond the specific, moderate-intensity, small-muscle-mass protocol tested [24].

The broader safety study of patients with thoracic aortic aneurysms and dissections performing a standardised circuit of isometric and dynamic exercises similarly found no evidence of exaggerated blood pressure reactivity compared with unaffected controls, a high protocol completion rate, and no episodes of severe exertional hypertension requiring termination of testing, supporting the feasibility of moderate-intensity structured exercise even in this high-risk population under appropriate supervision [25]. This study's use of the Borg perceived-exertion scale as a reliable, individualisable proxy for exercise intensity, and its finding that habitually more active patients showed a blunted blood pressure response during testing, together suggest a practical framework for exercise prescription: begin with moderate-intensity, non-Valsalva, dynamic exercise involving submaximal loads, use perceived exertion rather than fixed percentages of maximal strength to titrate intensity, and progressively increase activity level as tolerance and habituation develop, while avoiding, at least until further data are available, the extreme end of the intensity spectrum represented by maximal or near-maximal single-repetition lifts performed with breath-holding [25].

This cautious framework is broadly consistent with the exercise recommendations described for bicuspid aortic valve disease, in which lifelong exercise volume, intensity, and sport type showed no deleterious association with valvular dysfunction or aortic dilation in a large cross-sectional cohort, and in which vigorous-intensity and mixed-type sport participation was, if anything, associated with a lower prevalence of aortic stenosis, prompting the authors to advocate an encouraging rather than restrictive approach to exercise counselling in patients without significant aortic dilation, severe valvular dysfunction, or other high-risk features [29]. It is also consistent with the paediatric bicuspid valve data showing no acceleration of aortic diameter progression with regular physical activity, and with the metabolic benefit (lower obesity prevalence) associated with competitive sports participation in this population [2,31]. Current formal guidance for hereditary aortopathies, discussed further in Section 16, is considerably more conservative for genetically confirmed connective-tissue disorders such as Marfan and Loeys–Dietz syndrome, reflecting the qualitatively higher and less predictable dissection risk associated with these conditions even at modest aortic diameters [16,33].

A further practical consideration specific to strength and power athletes concerns the broader lifestyle context in which resistance training is typically embedded, including recovery practices such as cold-water immersion and whole-body cryotherapy, which are widely used by strength

and power athletes for perceived recovery benefit and which themselves produce measurable acute cardiovascular effects, including sympathetic activation, transient changes in blood pressure, and altered heart rate variability [6]. Although this literature does not address aortic wall stress directly, it illustrates that the cardiovascular "exposure profile" of a competitive strength or power athlete extends beyond the training session itself to encompass a range of adjunct practices with their own acute haemodynamic signatures, a consideration that adds further complexity to any attempt to isolate the aortic effects of resistance training from the totality of an athlete's lifestyle and recovery regimen, and that has rarely been accounted for explicitly in the comparative studies reviewed in Sections 6 and 7.

16. Screening Protocols, Clinical Guidelines, and Eligibility Recommendations

Formal, structured guidance for the assessment and management of athletes with aortic dilation provides the clinical scaffolding within which the evidence reviewed in this article must be applied. The most comprehensive and widely cited guidance remains the American Heart Association/American College of Cardiology Task Force 7 recommendations on aortic diseases, including Marfan syndrome, which provide detailed, graded eligibility criteria according to the specific diagnosis, the measured aortic root diameter (expressed both in absolute terms and as a z-score corrected for age, sex, and body surface area), and the presence of additional risk features such as a family history of dissection at a small aortic diameter, moderate-to-severe mitral regurgitation, or reduced left ventricular systolic function [33]. This guidance explicitly discusses the phenomenon of training-induced aortic enlargement, noting that intense physical exertion produces haemodynamic changes that increase aortic wall tension and may modestly increase aortic dimension, that elite athletes have slightly larger aortas at the sinuses of Valsalva than non-athletic controls, and that although mild aortic enlargement may represent a normal adaptation to training, large increases in aortic size are unusual in athletes and, when present, are more consistent with an underlying pathological aortopathy that may be exacerbated by exercise training [33]. It further addresses the specific challenge of tall or large-bodied athletes, noting the non-linear, plateauing relationship between aortic root dimension and body size beyond a height of approximately 189 cm in men or 175 cm in women, or a body surface area exceeding 2.3 m², and cautioning against attributing aortic enlargement in such individuals solely to body size or training without exclusion of underlying aortopathy [33].

For bicuspid aortic valve disease, this guidance recommends that athletes without aortic dilation and without significant valvular dysfunction may participate in all competitive athletics, that those with mild-to-moderate aortic dilation (40–42 mm in men, 36–39 mm in women) may participate in low-and-moderate-intensity sport with avoidance of intense weight training, and that those with severe dilation (43–45 mm) should be restricted to low-intensity sport with low likelihood of bodily contact, while those with markedly dilated aortas (greater than 45 mm) should not participate in any competitive sport [33]. For hereditary connective-tissue aortopathies such as Marfan syndrome and Loeys–Dietz syndrome, the guidance is considerably more conservative, generally restricting affected individuals to low-static, low-dynamic sport (classified as class IA) even in the absence of aortic dilation, reflecting the qualitatively higher dissection risk conferred by the underlying genetic disorder independent of aortic diameter [33]. Notably, this guidance also reviews longitudinal follow-up data on athletes with unexplained mild aortic enlargement who continued competitive participation, in which aortic root diameter increased only modestly over 8±5 years (from 40.9±1.3 mm to 42.9±3.6 mm), without dissection, though two of these athletes progressed to 50 mm by ages 38 and 50, illustrating that even mild, apparently stable aortic enlargement in an athlete warrants sustained, long-term surveillance rather than a single reassuring assessment [33].

Contemporary, sport-specific echocardiographic guidance, exemplified by the British Society of Echocardiography guideline on cardiac assessment of young athletes, operationalises much of this eligibility framework into a structured, stepwise imaging protocol, incorporating standardised measurement of the aortic root at multiple levels, comparison against consensus reference ranges stratified by sex and ethnicity, and explicit provision for referral onward to advanced imaging (cardiac magnetic resonance, computed tomography angiography) or genetic testing when structural or functional findings fall outside the expected physiological range for the athlete's specific demographic and training profile [12]. This guideline explicitly incorporates the sport-classification framework (skill, power, mixed, endurance) discussed in Section 7 as one of several contextual variables—alongside training volume, ethnicity, body size, sex, and age—that must be weighed together, rather than relying on any single measurement threshold in isolation, when interpreting an athlete's aortic findings [12].

Emerging large-scale prospective cohort infrastructure may help close some of the specific evidentiary gaps identified throughout this review. A multicentre prospective cohort protocol

currently recruiting approximately 500 elite athletes across endurance, strength (including weightlifting, wrestling, judo, and boxing), and mixed-sport disciplines, with baseline and one-year follow-up cardiovascular, biochemical, and hormonal assessment, has been explicitly designed to characterise dose–response relationships between exercise load and cardiovascular adaptation across sport type, age, and sex, incorporating discussion of the differential roles of oestrogen (protective, via suppression of pathways promoting cardiac hypertrophy) and testosterone (promoting cardiac and, by extension, potentially vascular hypertrophy via direct receptor-mediated effects on protein synthesis) as candidate mechanisms underlying sex-based differences in cardiovascular remodeling [23]. Although this protocol has not yet reported cardiovascular outcome data specific to the aortic root, its explicit inclusion of strength-sport categories and its stated aim of exploring dose–response relationships across sport types represents a direct, prospectively designed response to the kind of evidentiary gap—comprehensive, sport-stratified, longitudinal aortic phenotyping in strength athletes—identified repeatedly throughout this review, and its eventual results are likely to be directly relevant to future revisions of the eligibility guidance summarised above [23].

Table 2. Summary of eligibility and surveillance guidance relevant to aortic dilation in strength and power athletes.

Clinical scenario	Recommendation	Source [ref]
Aortic root mildly above normal (z-score 2–2.5, no syndromic features)	Echocardiographic/MRA surveillance every 6–12 months; avoid attributing enlargement to body size or training alone in tall/large athletes	Braverman et al. [33]
Bicuspid aortic valve, no dilation, normal valve function	Participation in all competitive sport permitted	Braverman et al. [33]; Kurylek et al. [2]
Bicuspid aortic valve, mild-moderate dilation (40–42 mm men / 36–39 mm women)	Low-to-moderate intensity/static-dynamic sport with low contact risk; avoid	Braverman et al. [33]

	intense weight training	
Bicuspid aortic valve, severe dilation (43–45 mm)	Low-intensity sport only, low contact likelihood	Braverman et al. [33]
Bicuspid aortic valve, markedly dilated aorta (>45 mm)	No competitive sport participation	Braverman et al. [33]
Marfan syndrome / Loeys–Dietz syndrome, no high-risk features	Low-static, low-dynamic (class IA) sport only	Braverman et al. [33]
Known thoracic aortic aneurysm, moderate-intensity exercise prescription	Submaximal, non-Valsalva dynamic exercise; use perceived exertion (Borg scale) to titrate intensity	Li et al. [25]; McMurtry/Skow et al. [24]

17. Discussion

Read together, the evidence reviewed in this article supports a differentiated rather than a unitary answer to the question posed in its title. At the population level, the comparative echocardiographic literature reviewed in Sections 6 and 7 does not support the notion that strength or power training, in the absence of other risk factors, produces clinically important aortic root dilation. Aortic root diameters in powerlifters and bodybuilders have repeatedly been found to be similar to, or smaller than, those of endurance-trained comparators, despite the presence of markedly greater concentric left ventricular hypertrophy and higher resting blood pressure in the strength-trained group [18,19]. Sport-classification analyses stratifying athletes by static and dynamic training component have consistently found the largest aortic dimensions in endurance, not power, categories, directly contradicting the intuitive assumption—implicit in much of the earlier literature focused on ventricular remodeling—that isometric, pressure-predominant training is the principal driver of aortic enlargement [11,17]. This dissociation between ventricular and aortic remodeling is itself an important finding: it indicates that the mechanisms governing concentric ventricular hypertrophy (chronic pressure overload acting on the myocardium) and those governing aortic wall adaptation (a more complex interplay of cyclic wall stress, elastin–collagen load-sharing, vascular smooth muscle tone, and endothelial function)

are not simply interchangeable, and that a strength athlete's markedly hypertrophied ventricle should not, by itself, be taken as evidence that the aorta has undergone comparable enlargement.

At the same time, the review has identified specific circumstances in which aortic enlargement, and even catastrophic dissection, does occur in strength and power athletes, and in each case the identified mechanism appears to involve a factor beyond training modality alone. Progressive, statistically significant aortic root dilation was documented in collegiate American-style football players, but this occurred in a population also characterised by substantial weight gain, rising systolic blood pressure, increasing arterial stiffness, and declining diastolic function, and was explicitly interpreted by the original investigators as a potentially maladaptive rather than purely physiological phenotype [8]. An exaggerated blood pressure response to standardised exercise, rather than sport category itself, was independently associated with larger aortic root diameter in a large mixed-sport athlete cohort, and every athlete in that cohort with an aortic root of 40 mm or more fell within the exaggerated-response subgroup [20]. Case reports and series of fatal or near-fatal aortic dissection in strength athletes consistently identify anabolic-androgenic steroid use and uncontrolled hypertension as central, recurring features, converging with broader epidemiological data identifying weightlifting explicitly as a recognised acquired risk factor for dissection and hypertension as the dominant modifiable comorbidity across all dissection cohorts, athletic or otherwise [26-28]. Bicuspid aortic valve disease and, more rarely, hereditary connective-tissue aortopathies such as Marfan and Loeys–Dietz syndrome represent a further, distinct category of risk that will inevitably intersect with strength and power athletics given their prevalence in the general population, and the available evidence in bicuspid valve disease is broadly reassuring regarding the safety of continued sport participation, provided that aortic dilation and significant valvular dysfunction have been excluded, while formal guidance for the genetically confirmed connective-tissue disorders remains appropriately conservative [2,10,16,29-33].

A recurring methodological theme across this literature, and one with direct clinical utility, is that ratio-based or proportionate measures of aortic size—expressed relative to left ventricular dimension, height, or, in the specific context of hereditary aortopathy, relative to other aortic segment lengths—consistently outperform absolute diameter measurement alone in distinguishing physiological from pathological enlargement [17,32]. The aortic-root-to-left-ventricular-diameter "golden ratio," validated with high sensitivity and specificity against a

cohort of patients with known pathological dilation, and the analogous diameter- and length-based ratios developed to differentiate Marfan from Loeys–Dietz syndrome even before overt dilation occurs, both illustrate that the shape and internal proportionality of the aortic root complex may carry more diagnostic information than any single dimension viewed in isolation [17,32]. This principle is of particular relevance to strength and power athletes, whose left ventricular cavity dimensions are typically smaller than those of endurance athletes; an aortic diameter that would appear unremarkable in isolation may represent a disproportionately enlarged aortic root relative to a comparatively small ventricular cavity, and this proportionality, rather than the absolute measurement, may better capture the clinically relevant signal.

The literature also converges on the observation that acute haemodynamic stress during resistance exercise, while measurable and real, appears to remain within a physiologically tolerable range for the aortic wall under most tested conditions, including in patients with an already-established aneurysm undergoing moderate-intensity, non-Valsalva dynamic exercise [24,25]. This reassurance, however, is explicitly bounded by the intensity and modality of exercise studied, and cannot be extrapolated with confidence to the maximal, whole-body, Valsalva-associated efforts characteristic of competitive powerlifting or heavy compound resistance training, a gap in direct evidence that the authors of the relevant studies themselves acknowledge [24]. The broader vascular-stiffness literature further complicates a simple narrative of reassurance, since cross-sectional comparisons of habitually strength-trained athletes with untrained controls have repeatedly documented higher proximal aortic and carotid stiffness in the strength-trained group, even though randomised training-intervention studies have generally failed to replicate this finding over intervals of months, and even though muscular strength itself, as an outcome measure independent of training modality, is consistently associated with lower, not higher, arterial stiffness and better cardiovascular prognosis [9]. This apparent paradox—cross-sectional elevation of large-artery stiffness in habitual strength athletes as a group, set against the individually protective association of muscular strength—remains genuinely unresolved in the literature reviewed here and should be treated as an open question rather than papered over by selective emphasis on either finding.

Finally, it is worth noting that the broader physiological and clinical context surrounding these athletes extends beyond the specific question of aortic size. Aortic dissection, when it does occur, can propagate into the aortic arch vessels and precipitate cerebral malperfusion or embolic stroke,

a devastating systemic complication whose acute management—reperfusion therapy where appropriate, and an evolving landscape of adjunctive neuroprotective and rehabilitative strategies—follows the general principles and rapidly changing treatment paradigms described in the broader ischaemic stroke literature, underscoring that the consequences of a missed or unrecognised aortopathy in an athlete are not confined to the cardiovascular system alone [5]. Likewise, the wider lifestyle and recovery context of competitive strength and power athletes, including the use of adjunctive modalities such as cold-water immersion and cryotherapy that themselves produce measurable acute cardiovascular perturbation, has rarely been accounted for as a potential confounder in the comparative studies reviewed here, and represents a further source of unmeasured variability that should temper overly confident generalisations from any single study [6].

18. Limitations of the Evidence Base

Several limitations qualify the conclusions of this review and of the literature it synthesises. First, the majority of comparative echocardiographic studies addressing aortic root dimensions in strength and power athletes are cross-sectional, precluding firm causal inference about whether observed differences reflect training-induced adaptation, pre-existing constitutional differences between individuals who self-select into strength sports, or unmeasured confounding by body composition, blood pressure, or substance use [11,17-19]. Second, sample sizes in the studies directly comparing powerlifters, bodybuilders, or weightlifters against other athlete groups are frequently modest (in the range of 15–40 participants per group), limiting statistical power to detect small but potentially clinically meaningful differences in aortic dimension, and increasing the risk that null findings reflect inadequate power rather than a genuine absence of effect [18,19]. Third, longitudinal data with repeated aortic imaging over multiple years are available for relatively few strength or power athlete populations; the most robust longitudinal dataset identified in this review relates specifically to American-style football players, a population combining strength training with substantial weight gain and team-sport dynamic demands, and its findings cannot be assumed to generalise to powerlifters, Olympic weightlifters, or bodybuilders training in isolation from team-sport participation [8].

Fourth, case reports and small case series of aortic dissection in strength athletes, while clinically vivid and instructive, cannot establish incidence, and the true population-level risk of dissection

specifically attributable to resistance training, as distinct from the risk attributable to concomitant anabolic steroid use or uncontrolled hypertension, cannot be quantified from the available literature [26,27]. Fifth, most reference-range and comparative studies have been conducted in predominantly male, largely White or mixed-European cohorts, and the applicability of the specific numerical thresholds discussed throughout this review to other ethnic groups, to female strength and power athletes specifically, and to adolescent or masters-age strength athletes remains incompletely established [4,12]. Sixth, direct measurement of aortic wall stress during exercise has been performed using submaximal, small-muscle-mass, non-Valsalva protocols; no study identified in this review has directly measured aortic wall stress during maximal or near-maximal compound resistance exercise (heavy squat, deadlift, or bench press) performed with a Valsalva manoeuvre, which is precisely the exercise modality of greatest theoretical concern in this population, and this represents perhaps the single most important evidentiary gap identified by this review [24]. Finally, because this is a narrative rather than a systematic review, source selection and thematic emphasis were not governed by a pre-registered protocol or formal risk-of-bias assessment, and the synthesis presented here should be understood as a clinically oriented, critically appraised overview rather than a quantitatively pooled or exhaustively comprehensive account of all available evidence.

19. Conclusions

The available evidence, drawn from comparative echocardiographic cohorts, longitudinal follow-up studies, mechanistic investigations of acute haemodynamic stress, case reports of catastrophic aortic events, and international eligibility guidance, does not support a simple or unitary answer to whether aortic root dilation in strength and power athletes represents physiological adaptation or pathological risk; instead, it supports a differentiated, context-dependent conclusion. In the great majority of drug-free strength and power athletes without hypertension or an underlying connective-tissue or valvular disorder, aortic root dimensions remain within, or only marginally beyond, the normal range described for the general athletic population, and are frequently smaller than those observed in endurance-trained comparators despite far more pronounced concentric ventricular hypertrophy, indicating that the mechanisms governing ventricular and aortic remodeling in this population are dissociated rather than parallel. Where meaningful aortic enlargement or acute dissection has been documented in strength and power athletes, it has consistently been associated with identifiable additional risk factors—anabolic-androgenic

steroid use, uncontrolled systemic hypertension, an exaggerated pressor response to exertion, weight gain with arterial stiffening, or an underlying bicuspid aortic valve or hereditary aortopathy—rather than occurring as an apparently isolated consequence of resistance training itself.

Clinically, this synthesis argues against reflexively attributing an enlarged aortic root in a strength or power athlete to training-induced physiological remodeling, and in favour of a systematic evaluation encompassing blood pressure measurement and control, direct enquiry regarding performance-enhancing substance use, family history of aortic disease or sudden death, and, where clinical suspicion warrants, structured screening for bicuspid aortic valve disease and heritable connective-tissue aortopathy using multimodality imaging and, where appropriate, genetic testing. Proportionate, ratio-based measures relating aortic root diameter to left ventricular dimension, height, or other aortic segments appear to offer better discrimination between physiological and pathological enlargement than absolute diameter alone and merit wider incorporation into athlete-specific reference frameworks. Given the persisting gaps identified in this review—particularly the absence of direct aortic wall-stress data during maximal, Valsalva-associated resistance exercise and the scarcity of long-term, sport-stratified, sex-inclusive longitudinal cohorts specifically enrolling powerlifters, weightlifters, and bodybuilders—prospective, adequately powered studies incorporating standardised imaging, blood pressure phenotyping, and structured substance-use assessment are needed before more definitive, population-specific eligibility thresholds can be established for this athletic subgroup.

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