Current Atherosclerosis Reports (2025) 27:120
https://doi.org/10.1007/s11883-025-01370-3

REVIEW

Vademecum for the Physician Evaluating a Master Athlete

Andrea Palermi[1] · Marco Vecchiato[2] · Giuseppe Di Gioia[3] · Francesco Perone[4] · Gary Tse[5] · Sabina Gallina[1] · Mariarosaria De Luca[6] · Francesca Graziano[7] · Alessandro Zorzi[7] · Vasiliki Tsampasian[8,9] · Maria Marketou[10] · Alessandro Biffi[11] · Stefano Palermi[12]

Received: 1 October 2025 / Accepted: 16 November 2025

Abstract

Purpose of the Review The number of master athletes (MAs) is steadily increasing, reflecting broader societal trends in healthy aging and competitive sports participation beyond the age of 35. This work presents an up-to-date, evidence-based framework for evaluating the cardiovascular and general health of master athletes, integrating current guidelines with sport-specific considerations, and focusing primarily on cardiovascular prevention and risk management. It also acknowledges conditions that are more prevalent in this population—accelerated coronary calcification/coronary artery disease, endurance-related atrial fibrillation, and mild aortic enlargement—within the preventive assessment framework.

Recent Findings While regular exercise confers significant cardiovascular and metabolic benefits, aging athletes present unique clinical challenges requiring tailored assessment and management strategies. Current evidence highlights the

✉ Stefano Palermi
stefano.palermi@unicamillus.org

Andrea Palermi
andrea.palermi@outlook.com

Marco Vecchiato
marcovecchiato.md@gmail.com

Giuseppe Di Gioia
dottgiuseppedigioia@gmail.com

Francesco Perone
francescoperone1988@gmail.com

Gary Tse
gatse@hkmu.edu.hk

Sabina Gallina
sabina.gallina@unich.it

Mariarosaria De Luca
mariarosaria.deluca@unina.it

Francesca Graziano
Francesca.graziano@unipd.it

Alessandro Zorzi
alessandro.zorzi@unipd.it

Vasiliki Tsampasian
V.Tsampasian@uea.ac.uk

Maria Marketou
maryemarke@yahoo.gr

Alessandro Biffi
a.biffi@libero.it

[1] Department of Neuroscience, Imaging and Clinical Sciences, "G. d'Annunzio" University of Chieti-Pescara, Chieti 66100, Italy

[2] Sports and Exercise Medicine Division, Department of Medicine, University of Padova, Padova 35128, Italy

[3] Institute of Sport Medicine and Science, Italian National Olympic Committee, Largo Piero Gabrielli, 1, 00179 Roma, Italy

[4] Cardiac Rehabilitation Unit, Rehabilitation Clinic Villa delle Magnolie, 81020 Caserta, Italy

[5] Department of Population Health, School of Nursing and Health Sciences, Hong Kong Metropolitan University, Hong Kong, China

[6] Department of Translational Medical Sciences, Federico II University, Naples, Italy

[7] Department of Cardiac, Thoracic and Vascular Sciences and Public Health University of Padova, Padova 35128, Italy

[8] Norwich Medical School, University of East Anglia, Rosalind Franklin Road, Norwich NR4 7UQ, UK

[9] Department of Cardiology, Norfolk and Norwich University Hospital, Colney Lane, Norwich NR4 7UG, UK

[10] Department of Cardiology, Heraklion University General Hospital, University of Crete, Iraklio, Greece

[11] Med-Ex, Medicine & Exercise, Medical Partner Scuderia Ferrari, Rome 00187, Italy

[12] UniCamillus Saint Camillus International University of Health Sciences, Rome 00187, Italy

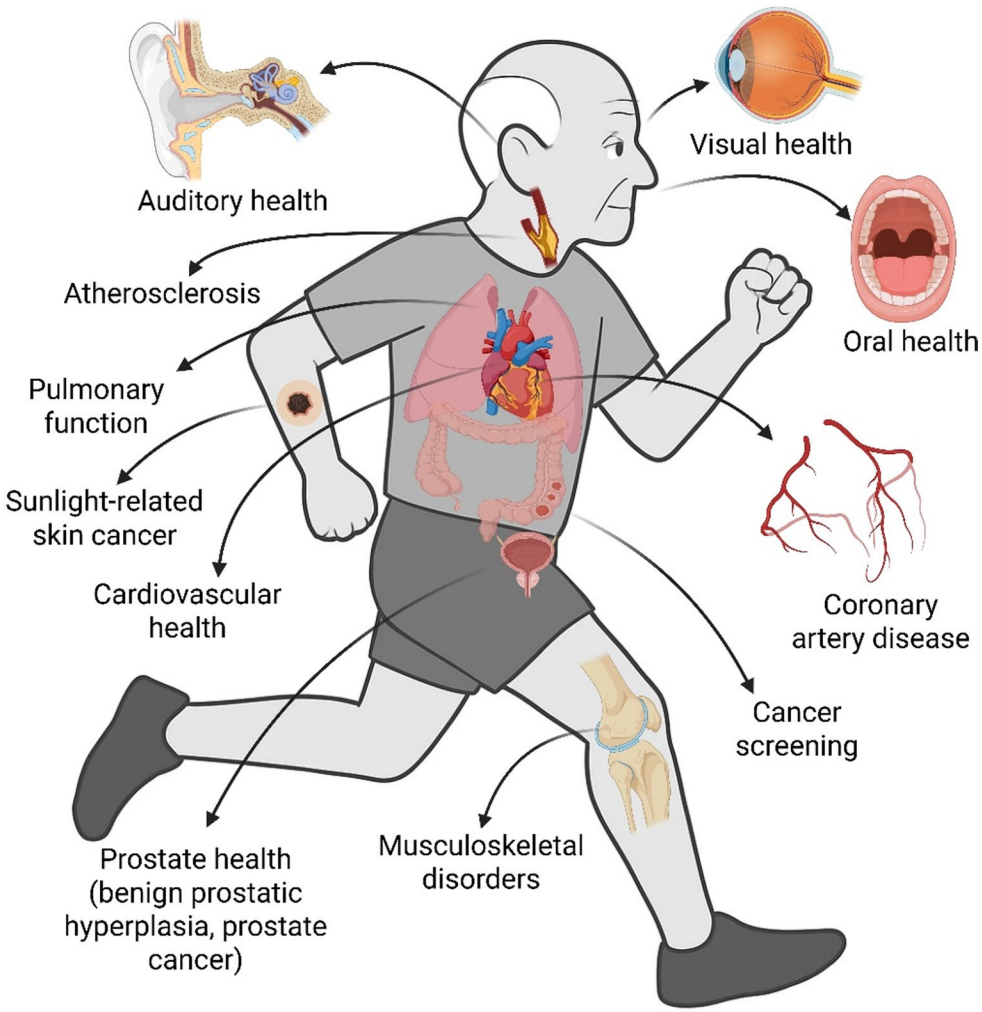
Published online: 03 December 2025

importance of recognizing both traditional and sport-specific risk factors, employing appropriate diagnostic modalities (including advanced imaging when indicated), and implementing an integrated approach combining lifestyle, pharmacological, and procedural interventions.

Summary MAs require individualized, multidisciplinary care to ensure safe and sustained participation in sports. Early detection and targeted management of cardiovascular and metabolic risk factors, along with ongoing surveillance, are essential for preserving health, performance, and quality of life in this growing population.

Graphical Abstract

The ageing athletes: what not to miss



Keywords Master athletes · Cardiovascular risk · Sports medicine · Hypertension · Dyslipidemia · Preventive cardiology

Introduction

In recent decades, there has been a significant rise in the number of men and women over the age of 35 to 40 participating in competitive and high-intensity endurance sports, commonly referred to as master athletes (MAs) [1].

This population is heterogeneous, including former professionals who wish to continue competing—often in different disciplines—individuals returning to structured training after a period of inactivity, and those who decide to begin systematic exercise in adulthood to improve health and fitness [1]. Endurance sports, such as cycling and running, show the highest prevalence of MAs [1, 2]. For instance, participation in the New York City Marathon between 1980 and 2009 markedly increased among runners over 40, with substantial performance improvements in men

over 65 and women over 45, while the gender gap in finish times decreased from 28.4% to 19.7% [3]. These trends highlight the progressive aging of the competitive sports population and pose new challenges for the sports cardiology community [1].

Compared to younger athletes, whose cardiovascular (CV) risk is primarily related to congenital or genetic conditions [4], MAs face a higher prevalence of acquired risk factors and age-related diseases, such as hypertension, dyslipidemia, diabetes, overweight, and coronary artery disease (CAD) [5]. Furthermore, non-CV conditions—including musculoskeletal degeneration, sensory decline, and metabolic disorders—must be considered when evaluating this population. Traditional risk stratification tools often underestimate the true risk profile of MA, who may present with excellent physical fitness despite underlying pathology. This calls for an appropriate diagnostic work-up [6, 7], often integrating advanced imaging techniques [4, 8].

Therefore, there is a need for an appropriate pre-participation screening (PPS) that addresses the increased risk of this population. The present vademecum aims to provide physicians with a practical, evidence-based framework for evaluating MA, integrating current guidelines with relevant considerations and decision-making tools. In the following sections, we will address the most relevant clinical conditions encountered in MA, with a particular focus on hypertension, dyslipidemia, hyperglycemia, and obesity. Each topic will be discussed in terms of diagnosis, risk stratification, and therapeutic implications, accompanied by practical tables and algorithms to guide physicians during preparticipation screening and follow-up.

Methods

This narrative review was conceived as a clinician-oriented vademecum, based on a structured but non-systematic literature search. Relevant articles were identified through PubMed/MEDLINE, Scopus, and the Cochrane Library, integrating guideline repositories from ESC, ESH, EAS, AHA/ACC, and the Italian COCIS. Searches were conducted up to November 2025, using combinations of key terms such as master athlete, aging athlete, sports cardiology, pre-participation evaluation, hypertension, dyslipidemia, diabetes, obesity, and sports eligibility. Additional sources were obtained by reviewing the reference lists of key articles and expert consensus documents.

The selection emphasized recent guidelines (2023–2025), major position papers, and clinical studies applicable to master or aging athletes (aged 35 years or older). When evidence specific to this population was limited, relevant data were extrapolated from general or younger athlete cohorts, and critically discussed from a practical perspective for sports physicians.

High Blood Pressure

Hypertension is one of the most frequently diagnosed conditions during PPS, particularly among MA [9]. A persistent systolic blood pressure (SBP) ≥ 140 mmHg and/or diastolic blood pressure (DBP) ≥ 90 mmHg is traditionally considered diagnostic, though classification varies based on the method of measurement [9].

Diagnosis

Accurate BP measurement is essential and should be performed in all athletes over 40 years of age [10]. Several modalities are available, each with distinct specific thresholds (Table 1) [9].

BP should be measured by a trained clinician, obtaining at least two resting measurements several minutes apart, in both arms, with the athlete seated and relaxed, back supported, and feet on the floor for at least five minutes beforehand [9]. Caffeine ingestion within 60 min, smoking within 30 min, and inadequate cuff size (common in athletes with large arms) can lead to falsely elevated readings [11]. A quiet and comfortable environment is also essential.

Transient factors such as stimulant use, certain medications, or supplements (e.g., anabolic steroids, growth hormone, NSAIDs, erythropoietin) should always be considered [11]. To minimize the "white coat" effect, measurements should be repeated after rest or confirmed via ambulatory blood pressure monitoring (ABPM) [11]. Elevated BP during PPS should be reassessed within 1–4 weeks [11].

A thorough evaluation should include dietary habits (sodium and alcohol intake), psychosocial factors (stress, work environment), and family history of hypertension or secondary causes such as pheochromocytoma or polycystic

Table 1 Diagnostic thresholds for hypertension according to different measurement modalities

	Office BP	Home BP	24 h ABPM	Daytime ABPM	Nighttime ABPM	Hypertensive response to exercise
Non-elevated BP	< 120/70	< 120/70	< 115/65	< 120/70	< 110/60	
Elevated BP	120/70 ≤ 140/90	120/70 ≤ 135/85	115/65 ≤ 130/80	120/70 ≤ 135/85	110/60 ≤ 120/70	
Hypertension	≥ 140/90	≥ 135/85	≥ 130/80	≥ 135/85	≥ 120/70	> 220/85 (M) > 200/80 (F)

BP blood pressure; *ABPM* ambulatory blood pressure monitoring; *M* male; *F* female

kidney disease [9]. Clinicians should actively screen for symptoms suggestive of secondary hypertension (e.g., muscle weakness, sweating spells, tachycardia, flank pain), features of sleep apnea, and signs of target-organ damage [9]. Assessment of sexual function, often underreported, may reveal medication side effects or vascular dysfunction [9]. Together, these elements guide both diagnostic stratification and tailored therapeutic decision-making [10, 12–14].

The physical examination should aim not only to confirm BP elevation but also to identify secondary causes and organ damage: abnormal peripheral pulses (suggesting coarctation), renal or carotid bruits, left ventricular hypertrophy, retinopathy, or neurological deficits [9]. Systematic evaluation of cardiac rhythm, fundoscopy, abdominal examination, and peripheral edema can provide further diagnostic clues [9].

Following diagnosis, athletes should undergo cardiovascular risk stratification (Table 2), supplemented by second- and third-line investigations such as blood tests, echocardiography, and carotid ultrasound [9]. These assessments should be repeated annually.

Treatment

The first-line management of hypertension in athletes consists of non-pharmacological interventions (Table 3; Fig. 1) [9]. Weight loss reduces SBP by approximately 1 mmHg per kg lost, with additional benefits achieved by maintaining a healthy BMI (20–25 kg/m²) and waist circumference (< 94 cm in men, < 80 cm in women) [15]. Healthy nutrition plays a crucial role in managing arterial hypertension. Among dietary strategies, the DASH diet—rich in fruits, vegetables, and low-fat dairy, and low in saturated fats—has proven effective, lowering systolic blood pressure by up to 11 mmHg [9]. Reduced sodium intake (< 1500 mg/day) and increased potassium intake (3500–5000 mg/day) also contribute to BP control [9]. Regular aerobic exercise (≥ 150 min/week of moderate intensity) decreases SBP by 5–8 mmHg [16], while resistance training provides additional benefits [17]. Alcohol moderation and smoking cessation further improve outcomes [9].

Pharmacological therapy is indicated in patients with confirmed hypertension (sustained office BP ≥ 140/90 mmHg) or when BP remains ≥ 130/80 mmHg after three months of lifestyle modification [9]. Although MAs often have lower hypertension prevalence than the general population due to high levels of physical activity, those with uncontrolled hypertension are at risk of left ventricular hypertrophy, myocardial fibrosis, and other complications. Resting SBP >200 mmHg and/or DBP >110 mmHg represent relative contraindications to exercise, as do exaggerated exercise BP responses (SBP >250 mmHg or DBP >115 mmHg) [18]. In elite athletes, however, very high BP values may reflect the extraordinary workloads achieved, making absolute cut-offs difficult to interpret. To address this limitation, the SBP/MET (Metabolized Energy Equivalent) slope has recently been proposed as a more physiologically sound parameter [19]. An excessive rise in systolic BP relative to the increase in workload may indicate vascular maladaptation and a higher risk of hypertension [19].

Drug choice should strike a balance between efficacy, side effects, and impact on athletic performance (Table 3).

Table 2 CV risk stratification of the hypertensive master athlete

Clinical condition		BP		
		Grade 1	**Grade 2**	**Grade 3**
		SBP 140-159 mmHg DBP 90-99 mmHg	SBP 160-179 mmHg DBP 100-109 mmHg	SBP ≥180 mmHg DBP ≥110 mmHg
Stage 1	0 CV risk factors*	Low	Moderate	High
	1-2 CV risk factors*	Moderate	High	High
	≥3 CV risk factors*	High	High	High
Stage 2	HMOD[a]	High	High	Very high
Stage 3	CV or renal disease[b]	Very high	Very high	Very high

BP blood pressure; *SBP* systolic blood pressure; *DBP* diastolic blood pressure; *CV* cardiovascular; *HMOD* hypertension-mediated organ damage

Table 3 Antihypertensive medications in athletes

Indication	Drugs	Contraindications	Sports eligibility/Effects on performance
0 line therapy	Non-pharmacological intervention: weight loss, reducing waist circumference, healthy diet, reducing sodium and increasing potassium intake, regular physical activity, limiting alcohol intake and stopping smoke		
I line therapy	ACE inhibitors (e.g. Ramipril 2.5–10 mg/day, Enalapril 5–20 mg/day, Zofenopril 7,5–60 mg/day)	Pregnancy (teratogenic risks), renal artery stenosis	Performance neutral (no effect on VO_2max, peak workload, or overall exercise duration)
I line therapy	Angiotensin receptor blockers (e.g., Losartan 50–100 mg/day, Valsartan 80–320 mg/day), Olmesartan 10–40 mg/day	Pregnancy (teratogenic risks)	Performance neutral (in some cases, has been associated with modest improvements in athletic performance).
II line therapy	Calcium channel blockers (DHP e.g. Amlodipine 5–10 mg/die, Lercanidipine 10–20 mg/die	DHP ◊ lower limb edema (side effect)	Performance neutral (VO_2max and endurance performance not impaired)
III line therapy	Alpha-blockers (e.g. Doxazosin 1–16 mg/die)	History of postural hypotension or syncope	Performance neutral (only modest blood pressure-lowering capacity). It may increase the risk of post-exercise orthostatic hypotension, potentially leading to episodes of presyncope or syncope
IV line therapy (generally not recommended)	Beta-blockers (e.g. Nebivolol 5 mg/die)	Bradycardia, Second- or third-degree atrioventricular (AV) block, Sick sinus syndrome	May reduce exercise capacity and are banned in certain skill-based competitive sports, such as shooting
IV line therapy (generally not recommended)	Diuretics (Thiazide and thiazide-like diuretics e.g. Hydrochlorothiazide 12,5–25 mg/die, Loop Diuretics e.g. Furosemide 25–50 mg/die, Mineralcorticoid receptor antagonists e.g. Spironolactone 12,5–25 mg/die)	Hypovolemia, Kidney failure, Serum electrolyte abnormalities	Banned across all competitive sports due to their potential for performance enhancement and masking effects

ACE Angiotensin-converting enzyme; *DHP* dihydropyridine; *NDHP* non-dihydropyridine; *VO_2max* maximal oxygen consumption

ACE inhibitors, ARBs, and long-acting dihydropyridine calcium channel blockers are generally preferred [11, 20]. Dosing is similar to that in the general population, with adjustments made based on clinical response. Early treatment phases require close monitoring (every 2 weeks), while long-term follow-up can be tailored to risk profile [9]. Diuretics and beta-blockers should generally be avoided due to performance impairment and restrictions by governing bodies [21].

Exercise-induced Hypertension

Exercise-induced hypertension (EIH) remains an underrecognized entity, particularly in MA. Although office or ambulatory BP measurements may fall within the normal range, several athletes display an abnormal pressor response during exercise testing. This finding has been associated with an increased risk of developing sustained hypertension, left ventricular hypertrophy, and adverse cardiovascular events [22].

Recent evidence also suggests a possible connection between EIH and arrhythmic risk. A multicenter analysis has demonstrated that athletes with exaggerated BP responses are more likely to exhibit ventricular arrhythmias during exercise [23]. While these studies are not specifically limited to MA, they highlight the importance of performing serial stress tests, both in untreated individuals and in those receiving antihypertensive therapy, to assess exercise BP control.

From a practical perspective, clinicians should be aware that achieving BP targets at rest does not necessarily translate into adequate control during exercise. This represents a clinical challenge, since there is no consensus on whether treatment goals should be extended to exercise BP and how to individualize therapy in this context. Further studies focusing specifically on MA are warranted to clarify the prognostic significance of EIH and to define optimal management strategies in this unique population.

Practical Implications for Sports Physicians

- Monitor blood pressure during exercise: Even in athletes with normal resting BP, assessing the pressor response during exercise testing can help identify individuals at risk of sustained hypertension and cardiovascular complications.
- Individualize pharmacological management: Achieving target BP at rest does not guarantee adequate control during exercise; consider therapy adjustments and tailored follow-up for athletes with exaggerated exercise BP responses.
- Post-exercise hypotension: Antihypertensive therapy (especially ACEi/ARB and calcium channel blockers) may potentiate post-exercise hypotension, increasing the risk of dizziness or syncope after training. Cool-down and adequate hydration should always be recommended.

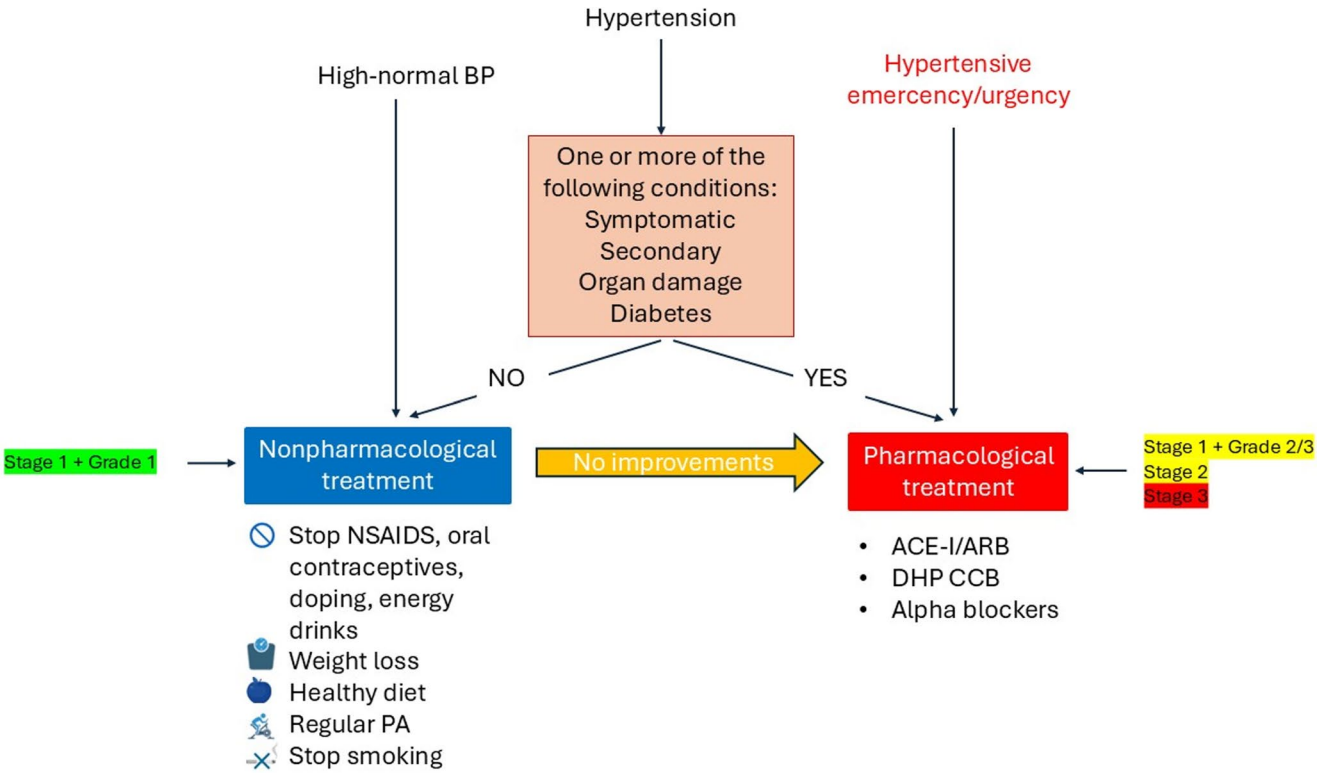


Fig. 1 How to manage high blood pressure in master athletes. ACE-I: Ace inhibitors; ARB: Angiotensin receptor blockers; DHP CCB: dihydropyridine calcium channels blockers; PA: physical activity; NSAIDS: not steroidal anti-inflammatory drugs

- Timing of therapy: Evening dosing can sometimes reduce excessive BP drops during morning exercise, but schedules should be individualized.
- Beta-blockers: Besides impairing performance, they blunt heart-rate response and can mask hypoglycemia, raising safety concerns in endurance athletes.
- Diuretics: Beyond regulatory bans, they increase dehydration risk in hot environments or prolonged outdoor training.
- Monitoring in practice: BP checks before and after training sessions are advisable in the early phases of therapy initiation or adjustment to assess exercise tolerance and safety.

Hypercholesterolemia

Dyslipidemia remains an underrecognized CV risk factor in athletes, particularly in MAs, who, despite maintaining high levels of physical activity, undergo age-related metabolic changes [24]. Epidemiological data indicate that approximately 30–35% of elite athletes present with dyslipidemia [25], with elevated low-density lipoprotein (LDL) cholesterol (≥ 115 mg/dL) being the most frequently encountered abnormality.

Interestingly, lipid profiles differ significantly depending on the type of sport [26, 27]. Endurance athletes typically show higher HDL-C and lower triglycerides (TG); power athletes (e.g., weightlifters, wrestlers) tend to have higher LDL-C and TG, partly related to higher BMI and sport-specific dietary patterns; skill-based athletes (e.g., shooters, archers) often display the highest TG levels, likely [26, 27].

Despite consistent physical training, genetic predispositions and dietary patterns can sustain elevated LDL-C in a subset of athletes [28]. Longitudinal evidence indicates that LDL-C elevations often persist over time: nearly 70% of athletes with elevated baseline LDL-C remain above target at follow-up [26, 27]. These data reinforce the need for structured screening and proactive management in MA.

Diagnosis

Non-fasting blood sampling for lipid profiling is recommended for general CV risk screening, as it provides prognostic information comparable to fasting measurements [24]. A standard panel should include total cholesterol, LDL-C, HDL-C, TG, non-HDL-C, apolipoprotein B (ApoB), and—at least once in a lifetime—lipoprotein(a) [Lp(a)] (Table 4) [26, 27].

Diagnostic assessment must be interpreted within the overall CV risk profile [24].

Non-HDL-C (TC − HDL-C) is useful when TG are elevated and in diabetes because it does not rely on TG for calculation [29]. ApoB reflects the number of atherogenic particles and may better estimate atherogenic burden than LDL-C alone [30]. Lp(a) is largely genetically determined and is an independent causal risk factor for ASCVD; measuring it once in life—especially with personal/family history of ASCVD—helps identify very high inherited levels (>180 mg/dL or >430 nmol/L) that approximate the lifetime risk of heterozygous familial hypercholesterolemia [31].

How To Define Risk Categories (for Table 4): according to contemporary ESC guidance [24, 32].

- **Very high risk**: documented ASCVD; diabetes with target-organ damage or ≥3 major risk factors; severe CKD (eGFR < 30 mL/min/1.73 m²); or markedly elevated calculated risk (e.g., SCORE2/SCORE2-OP very high).
- **High risk**: markedly elevated single RF (e.g., LDL-C ≥ 190 mg/dL); diabetes ≥ 10 years without target-organ damage; moderate CKD (eGFR 30–59); or high calculated risk.
- **Low-to-moderate risk**: individuals not meeting high/very-high definitions.

In general, aim for LDL-C < 100 mg/dL; for high risk, < 70 mg/dL; for very high risk, < 55 mg/dL; after a second CV event within 2 years, consider < 40 mg/dL [13]. TG should ideally be < 150 mg/dL. While endurance training often raises HDL-C, high HDL-C does not offset risk if LDL-C remains elevated [29].

Treatment

Lifestyle intervention is the cornerstone, especially in low- to moderate-risk athletes (Table 5) [20, 24] (Table 5). Key actions include a cardioprotective dietary pattern (Mediterranean, DASH, plant-forward, or low-carbohydrate), < 10% energy from saturated fat, elimination of trans fats, increased mono-/polyunsaturated fats, and soluble fiber, omega-3-rich foods, and plant sterols/stanols ~ 2 g/day [33, 34]. Structured physical activity (≥ 150 min/week moderate-to-vigorous aerobic + resistance training) and weight management (BMI < 25 kg/m²; reduced waist circumference) further improve the lipid profile [20, 24]. Nutraceuticals may be considered in mild dyslipidemia or where statin acceptance is low. Among these, red yeast rice (RYR) is the most frequently used in athletic settings [35]. However, evidence supporting the use of most dietary supplements—including red yeast rice, PUFAs, and other popular nutraceuticals—for LDL-C reduction or cardiovascular risk reduction is limited [36].

When lifestyle alone is insufficient—particularly in high or very high risk—initiate pharmacologic therapy (Fig. 2) [24]. The currently available lipid-lowering agents include HMG-CoA reductase inhibitors (statins), selective cholesterol absorption inhibitors (e.g., ezetimibe), bempedoic acid, PCSK9 inhibitors (e.g., alirocumab, evolocumab), siRNA specific for PCSK9 (e.g. inclisiran), bile acid sequestrants, fibrates, and omega-3 fatty acids [24]. A high-intensity statin should be initiated and titrated to the maximum tolerated dose to attain LDL-C goals according to the individual's risk profile [24]. In apparently healthy individuals under the age of 70 at very high risk, a ≥ 50% reduction in LDL-C from baseline is considered an appropriate therapeutic target [24]. The efficacy of therapy should be assessed by remeasuring LDL-C levels 4 to 6 weeks after treatment initiation or modification [24]. If target levels are not achieved, or if the current regimen is not tolerated, therapeutic adjustments, either through dose escalation, drug substitution, or combination therapy, should be considered, as outlined in Table 5; Fig. 2 [24, 37, 38]. Ezetimibe (10 mg/day) is often used as add-on therapy when statin monotherapy fails to meet LDL-C targets. Athletes, however, are at increased risk for statin-associated muscle symptoms and elevated creatine phosphokinase (CPK) levels, necessitating careful monitoring during treatment initiation and up-titration. Bempedoic acid, a novel oral cholesterol synthesis inhibitor, has recently been approved in several countries and may represent an alternative for statin-intolerant individuals [32]. In cases of familial hypercholesterolemia, confirmed statin intolerance or when LDL-C targets for the patient's risk category are not achieved despite optimal therapy, PCSK9 inhibitors may be considered, offering an additional ~ 60% reduction in LDL-C [24]. Inclisiran, a small interfering RNA (siRNA) targeting PCSK9 synthesis, represents an additional therapeutic option, providing a sustained LDL-C reduction with only biannual dosing.

Table 4 Diagnostic thresholds for hypercholesterolemia

Category of patients	LDL-c	Non-HDL-c	Apo-B	TG
Low-to-moderate risk	> 100 mg/dL	> 131 mg/dL	> 100 mg/dL	> 150 mg/dL
High risk	> 70 mg/dL	> 85 mg/dL	> 80 mg/dL	> 150 mg/dL
Very high risk	> 55 mg/dL	> 60 mg/dL	> 65 mg/dL	> 150 mg/dL

LDL low density lipoprotein; *HDL* high density lipoprotein; *Apo* apolipoprotein; *TG* triglycerids

Table 5 Pharmacological treatment of dyslipidemia in athletes

Indication	Drugs	Contraindications	Side-effects
0 line therapy	Non-pharmacological intervention: weight loss, regular physical activity, cardioprotective dietary patterns (Mediterranean diet, DASH diet, vegetarian or plant-forward diets, and low-carbohydrate regimens, reducing saturated fat, eliminating trans fats, increasing intake of mono- and polyunsaturated fats, and incorporating foods rich in soluble fiber, omega-3 fatty acids, and plant sterols or stanols), nutraceuticals (red yeast rice, berberine), reducing waist circumference		
I line therapy	Statins (e.g. Atorvastatin 10–80 mg/day, Rosuvastatin 10–40 mg/day)	Pregnancy/ Breastfeeding, Liver disease	Athletes are at high risk for developing statin-associated myopathy and elevated creatine phosphokinase levels
-In combination with statins when LDL-C targets are not achieved with maximally tolerated statin therapy -Monotherapy when a statin-based regimen is not tolerated at any dosage	Ezetimibe (10 mg/day)	Liver disease	Abdominal pain, diarrhea, flatulence
-In combination with statins and/or ezetimibe when LDL-C targets are not achieved with maximally tolerated therapy -Monotherapy when a statin- or ezetimibe-based regimen is not tolerated at any dosage	Bempedoic acid (180 mg/day)	The combination of ezetimibe with a statin is contraindicated in patients with active liver disease or with unexplained, persistent elevations in serum transaminase levels	Hyperuricemia
-In combination with statins and/or ezetimibe when LDL-C targets are not achieved with maximally tolerated therapy -Monotherapy when a statin- or ezetimibe-based regimen is not tolerated at any dosage	PCSK9 inhibitors (e.g. Alirocumab 75 mg/week-300 mg/month, Evolocumab 140 mg/two weeks −420 mg/month)	Pregnancy	Nasopharyngitis and upper respiratory tract infections
In patients taking statins who are at LDL-C goal with triglycerides > 200 mg/dL	Fibrates (e.g. Bezafibrate 400 mg/day)	Kidney failure	Gastrointestinal and hepatobiliary disorders, Muscle pain
Generally contraindicated	Niacin (e.g. Acipimox 250–750 mg/day)	Liver disease, active peptic ulcer disease	Flushing and impaired glucose control

DASH Dietary Approaches to Stop Hypertension; *LDL* low-density lipoprotein; *CPK* creatin phosphokinase

Practical follow-up in MA

- Re-check lipids 4–6 weeks after any therapy change; then every 3–6 months until at goal, and every 6–12 months thereafter.
- In statin/RYR users: symptoms + CK at baseline and after major training load changes; consider ApoB or non-HDL-C when TG are elevated.
- Consider Lp(a) once in life; if very high, intensify LDL-C lowering and global risk control.

Practical Implications for Sports Physicians

- Sport-specific nutrition: High-protein, ketogenic, or supplement-rich diets can adversely affect lipid profiles. Nutritional counselling should be integrated into follow-up for MA.
- Statin-associated muscle symptoms (SAMS): Master athletes are at increased risk of muscle-related adverse effects. Start with low doses, prefer hydrophilic statins (rosuvastatin, pravastatin), and titrate gradually. CK monitoring is advised, especially during high training loads.
- Nutraceuticals (Red Yeast Rice): Frequently chosen by athletes reluctant to use statins. Demonstrates 15–25% LDL-C reduction with good tolerability, but as it contains monacolin K (a lovastatin analogue), CK monitoring remains essential.
- PCSK9 inhibitors: Neutral effect on performance, though adherence to injection schedules must be emphasized.
- Training periodization: Consider lipid-lowering drug titration or adjustment during lower training loads to minimize confounding muscle symptoms.

Hyperglycemia and Obesity

Disorders of glucose metabolism (impaired fasting glucose, impaired glucose tolerance, and type 2 diabetes) become more prevalent with age and may coexist with high fitness levels in MA [39]. While regular exercise improves insulin

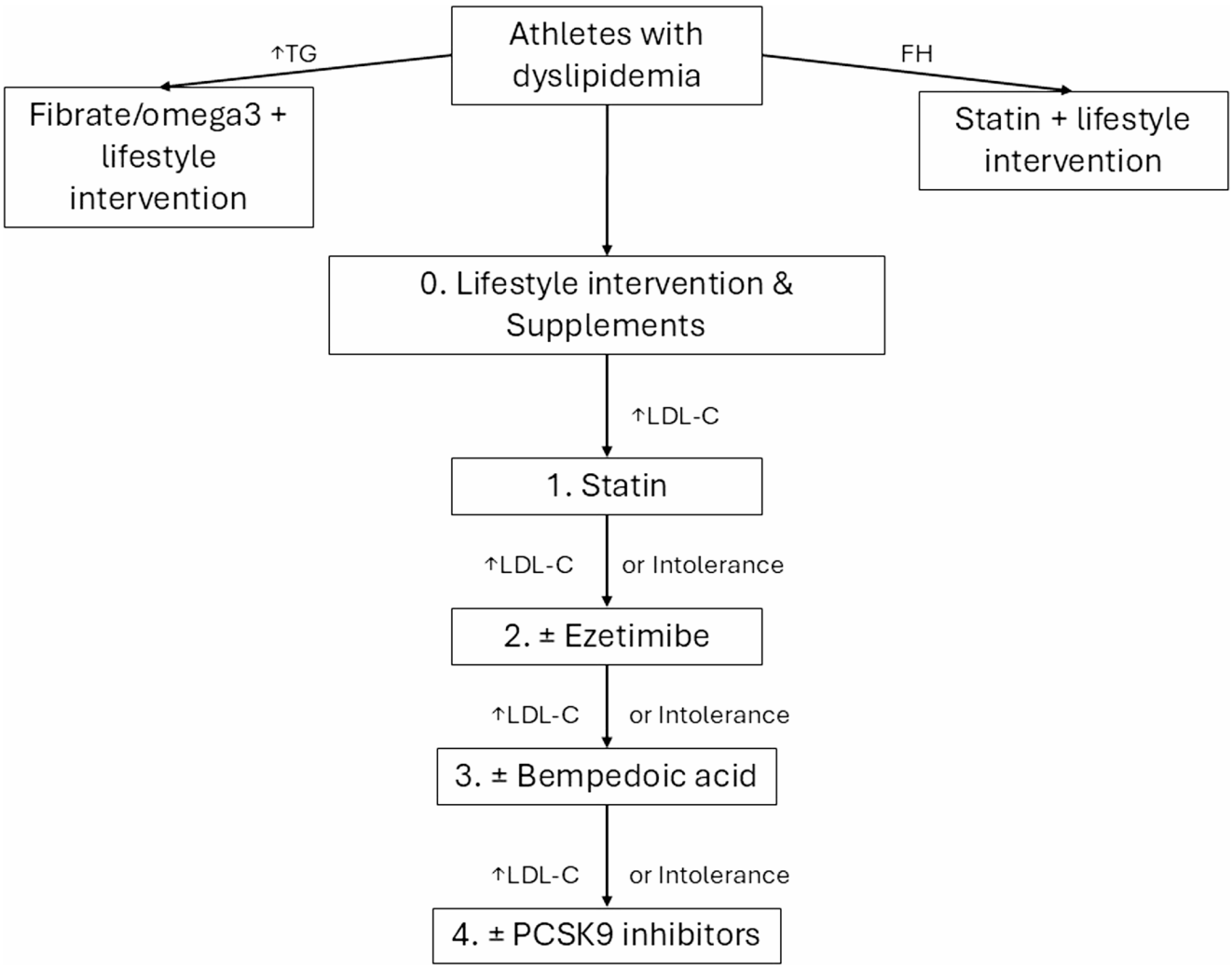


Fig. 2 Flowchart for managing dyslipidemia in master athletes. FH: familial hypercholesterolemia; LDL-C: low density lipoprotein; PCSK9: proprotein convertase subtilisin/kexin type 9; TG: tryglicerids; ↑: increase

sensitivity, genetics, visceral adiposity, suboptimal diet, sleep restriction, and concomitant cardiometabolic risk factors can sustain hyperglycaemia despite training [39]. From a sports-medicine perspective, recognizing dysglycemia is essential to: (i) refine CV risk stratification, (ii) prevent acute exercise-related complications (hypoglycemia with insulin/sulfonylureas; hyperglycemic crises in poorly controlled diabetes), and (iii) individualize training and nutrition plans [40, 41]. Moreover, in athletes with diabetes, the issue of chronic complications should not be underestimated, particularly diabetic neuropathy and diabetic foot, which may significantly impact performance, increase the risk of injuries, and compromise long-term participation in sports.

Obesity is defined as an abnormal or excessive accumulation of body fat that presents a health risk. The most widely used diagnostic criterion is the body mass index (BMI). According to the World Health Organization (WHO), obesity is diagnosed when BMI is ≥ 30 kg/m². However, obesity in MA may be underestimated when BMI is near-normal but visceral adiposity is increased ("normal-weight obesity"). Excess adiposity—especially abdominal—promotes hypertension, dyslipidemia, insulin resistance, sleep apnea, osteoarthritis, and reduced sport longevity [42]. Contemporary guidance frames obesity as a chronic disease requiring long-term, multimodal care [43, 44]. Consider also sarcopenic obesity in aging athletes, where reduced lean mass coexists with excess fat, impacting performance, injury risk, and metabolic health.

Diagnosis

Screen MAs for dysglycemia during PPS with fasting plasma glucose (FPG), HbA1c, and—when indicated—75-g Oral Glucose Tolerance Test (OGTT) (Table 6) [45, 46]. In asymptomatic individuals, confirm abnormal results on a separate day; in symptomatic athletes, random plasma

Table 6 Diagnostic thresholds for hyperglycemia

	HbA1c (%)	FPG (mg/dL)	2 h-oGTT (75 gr) (mg/dL)	Random Plasma Glucose (with classic symptoms) (mg/dL)
Normal	< 5.7	< 100	< 140	
IFG	5.7–6.4	100–125		
IGT	5.7–6.4		140–199	
Diabetes	≥ 6.5	≥ 126	≥ 200	≥ 200

HbA1c Hemoglobin A1c; *FPG* Fasting Plasma Glucose; *2 h-oGTT* Two-Hour Oral Glucose Tolerance Test; *IFG* Impaired Fasting Glucose;*IGT* Impaired Glucose Tolerance

glucose ≥ 200 mg/dL is diagnostic. Account for transient exercise-induced glucose elevations after intense/prolonged sessions and for confounders (acute illness, corticosteroids) [40, 41].

Assess adiposity using BMI + waist circumference (WC) or waist-to-height ratio (WHtR) to capture cardiometabolic risk; in athletic phenotypes with high lean mass, integrate WC, clinical risk, and—when needed—DXA rather than relying on BMI alone [42] (Table 7).

Therapy

Lifestyle intervention—medical nutrition therapy, structured physical activity, weight management, sleep optimization—remains first-line for both dysglycemia and obesity (Table 8) [39, 47]. Combine aerobic + resistance training (≥ 150 min/week aerobic + 2–3 resistance sessions/week) and tailor session timing and fueling to minimize hypoglycemia in in patients treated with insulin or oral hypoglycemic agents associated with risk of hypoglycemia [39, 47].

In MA:

- Prefer evenly distributed carbohydrate (CHO) across the day; match CHO timing to training intensity/duration.
- Use a continous glucose monitoring (CGM) system in insulin users or high-risk scenarios to support peri-exercise decision-making.
- Address sleep (7–9 h) and stress: both affect glycemic control and recovery.
- Education on hypoglycemia – Physicians should instruct patients to recognize early and advanced signs of hypoglycemia and to manage them promptly with fast-acting carbohydrates. Education should also cover prevention strategies, including therapy adjustments and carbohydrate planning before and during activity.
- Foot care – Reinforce the importance of proper footwear and regular foot inspection to prevent injuries and diabetic foot complications.

Pharmacologic therapy for hyperglycemia should be individualized to account for cardiovascular–renal risk and weight goals. In athletes, prioritize weight-neutral or weight-reducing agents with low hypoglycemia risk [40, 41]. Current guidelines prioritize SGLT2 inhibitors and/or GLP-1 receptor agonists (including dual GIP/GLP-1 agonists) in those with ASCVD, HF, or CKD, irrespective of baseline metformin use (Table 8) [40, 41].

Pharmacologic therapy for obesity is appropriate at BMI ≥ 30 kg/m² or ≥ 27 kg/m² with comorbidity (or elevated WC with comorbidity per policy) [48]. GLP-1 and dual GIP/GLP-1 agents provide the greatest weight loss and cardiometabolic gains; monitor GI effects, hydration, and fuel availability for training [48]. Metabolic/bariatric surgery may be considered for class III obesity, or class II with significant comorbidity/refractory T2D, with staged return-to-sport planning and micronutrient surveillance [48] (Table 9).

Type 1 Diabetes

In MA with type 1 diabetes, remarkable advances in pharmacological approaches and the widespread use of diabetes technologies—such as CGM and automated insulin delivery systems—have substantially improved survival and quality of life. Despite these achievements, managing physical exercise in this population remains challenging. Exercise is associated with rapid and often unpredictable changes in insulin sensitivity and glucose requirements, which can be challenging to match with subcutaneous insulin delivery

Table 7 Diagnostic thresholds for excess adiposity in adults

	Normal	Overweight	Obesity I	Obesity II	Obesity III
BMI (kg/m2)	18.5–24.9	25.0–29.9	30.0–34.9	35.0–39.9	≥ 40
Waist circumference (cm)	Men < 94; Women < 80	Men ≥ 94 (increased risk); Women ≥ 80 (increased risk)	Men ≥ 102 (substantial risk); Women ≥ 88 (substantial risk)		
Waist-to-height ratio	< 0.5	≥ 0.5			

BMI Body Mass Index; *WC* Waist Circumference

Table 8 Therapeutic lines for hyperglycemia and obesity in master athletes

	Hyperglycemia			Obesity		
	Indications	Options	Athletic specific notes	Indications	Options	Athletic specific notes
Line 0 – lifestyle	IFG/IGT; all individuals with diabetes	Individualised nutrition plan; ≥150 min/week moderate–vigorous aerobic activity + 2–3 resistance sessions/week; structured weight management; optimise sleep and limit alcohol	Exercise substantially improves HbA1c and insulin sensitivity; combine aerobic and resistance; adjust training schedule to minimise hypoglycaemia risk; consider CGM for high-risk athletes	All with excess adiposity	Structured lifestyle programme: 500–750 kcal/day deficit; Mediterranean, DASH, or plant-based patterns; reduce saturated fat, eliminate trans fats; ≥150–300 min/week aerobic + 2–3 resistance sessions/week; stress and sleep optimisation	Exercise alone rarely produces large weight loss but is essential for long-term maintenance and cardiometabolic benefits; combine aerobic and resistance training; adapt programme for competition
Line 1 – drugs	Most with new T2D; A1C above target after lifestyle	If ASCVD/HF/CKD: prioritise SGLT2 inhibitor and/or GLP-1 RA (including dual GIP/GLP-1) with proven benefit, regardless of metformin use; otherwise metformin remains a reasonable initial choice, with early combination if targets unmet	GLP-1 RA and GIP/GLP-1 agents deliver robust glycaemic and weight benefits; SGLT2i lower HF and CKD risk. Monitor hydration with intense exercise/heat; educate on euglycaemic DKA warning signs	BMI ≥ 30, or BMI 27–29.9 with ≥1 weight-related comorbidity; elevated WC + comorbidity may also qualify per policy	GLP-1 RA / dual GIP-GLP-1 (e.g., semaglutide; tirzepatide); orlistat; others per availability/coverage	Recent trials show 5–22.5% mean weight loss with medications + lifestyle; GLP-1/GIP–GLP-1 agents provide the largest effect with added cardiometabolic benefit. Monitor GI effects, hydration, and training fuel availability
Line 2 – add on/alternative	ASCVD/CKD/HF, or need for weight loss, or A1C above target	GLP-1 RA / GIP–GLP-1 (e.g., semaglutide, tirzepatide); SGLT2 inhibitors; basal insulin for severe hyperglycaemia	Select agents with weight-neutral or weight-reducing effects and low hypoglycaemia risk; titrate insulin cautiously on training days	Class III obesity, or BMI ≥ 35 with comorbidity, or selected BMI 30–34.9 with refractory T2DM after comprehensive care	Metabolic/bariatric surgery (sleeve gastrectomy, gastric bypass) under specialist supervision	Post-operative athletes require staged return to sport, adequate protein intake, and micronutrient monitoring
Line 3 – additional agents	Persistent hyperglycaemia	DPP-4 inhibitors, TZDs, basal insulin, or other agents per current guidelines	Customise peri-event glucose management (snacks, medication adjustments, cooldown strategies) based on training type and competition demands			

IFG Impaired Fasting Glucose; *IGT* Impaired Glucose Tolerance; *T2DM* Type 2 Diabetes Mellitus; *A1c* Glycated Hemoglobin; *ASCVD* Atherosclerotic Cardiovascular Disease; *HF* Heart Failure; *CKD* Chronic Kidney Disease; *SGLT2 inhibitor* Sodium–Glucose Cotransporter 2 Inhibitor; *GLP-1 RA* Glucagon-Like Peptide-1 Receptor Agonist; *GIP* Glucose-Dependent Insulinotropic Polypeptide; *WC* Waist Circumference; *CGM* Continuous Glucose Monitoring; *DKA* Diabetic Ketoacidosis; *TZD* Thiazolidinedione; *DPP-4 inhibitor* Dipeptidyl Peptidase-4 Inhibitor; *DASH* Dietary Approaches to Stop Hypertension

and may exceed the adaptive capacity of closed-loop systems. Automated devices, although highly effective in daily management, often face additional challenges during exercise, including the physiological lag of glucose sensors, the risk of dislodgement of infusion sets or sensors due to sweating or movement, and mechanical damage in contact activities. Moreover, the fine-tuning of insulin delivery algorithms—whether through basal rate reductions, temporary suspensions, or predictive adjustments—requires careful anticipation, while individual responses vary considerably depending on age, comorbidities, and the type and intensity of exercise. These factors underscore the need

Table 9 Peri-exercise glucose management in master athletes with diabetes [41]

Step	Key Actions	Practical Targets / Notes
1. Pre-Exercise Check	Measure BG 15–30 min before starting	Target start range: 90–250 mg/dL; delay if BG < 90 mg/dL (treat first) or >250 mg/dL with ketones; avoid >300 mg/dL until corrected
2. Carbohydrate Intake	Adjust pre/during CHO for insulin/ sulfonylurea users	< 90 mg/dL: 15–30 g rapid CHO before, then 30–60 g/h during;90–150 mg/dL: 0–15 g before, 30–60 g/h during if >60 min;150–250 mg/dL: no CHO pre; hydrate;>250 mg/dL: no CHO pre; hydrate; monitor every 30–45 min
3. Medication Adjustment	Modify insulin or sulfonylurea dose based on training load	Basal insulin: ↓ 20–50% before long endurance; Bolus: ↓ 25–75% if meal < 3 h pre-exercise; Pump: temporary basal ↓ 20–80% 60–90 min pre; Sulfonylureas: omit/halve dose on high-volume days; SGLT2i: maintain hydration; monitor for ketoacidosis risk
4. Monitoring During & After	Track BG and symptoms	Check every 30–45 min for ≥ 60 min sessions; use CGM trend arrows; overnight monitoring after intense/prolonged sessions; consider bedtime snack (15–30 g CHO + protein) if high risk for nocturnal hypoglycaemia
5. Competition & travel planning	Adapt strategies for events, heat, altitude, and time zones	Rehearse fueling/insulin plan during training; adjust basal insulin across time zones; in heat/humidity: prioritize hydration/electrolytes; in altitude: anticipate ↑ insulin resistance; keep rapid CHO and testing devices accessible
6.Post-exercise strategy	Prevent late-onset hypoglycemia and rebound hyperglycemia	For pump users: temporary basal ↓10–20% for 2–6 h post-session (if prone to hypoglycemia) or ↑10–20% (if rebound hyperglycemia); recheck BG 6–15 h post high-intensity/long exercise

BG Blood Glucose; *CHO* Carbohydrate; *SGLT2i* Sodium–Glucose Cotransporter 2 Inhibitor; *CGM* Continuous Glucose Monitoring

for specialized diabetic assessment and structured follow-up, aimed at personalizing therapeutic strategies, optimizing device settings, and ensuring both safety and efficacy in incorporating physical activity into the lifestyle of adults and elderly individuals with type 1 diabetes.

Beyond Cardiovascular Risk

While CV screening remains a cornerstone of PPS evaluation in MAs, a truly comprehensive medical assessment must extend beyond the heart and vessels [49]. As with advancing age, athletes become vulnerable to a spectrum of physiological changes and medical conditions that may not immediately affect sport performance but significantly influence long-term health, quality of life, and safe sport participation [50]. Proactively identifying and managing these conditions enables effective prevention strategies, fosters healthy aging, and sustains athletic involvement. Integrating such assessments into routine sports medicine evaluations is therefore essential (Table 10). For completeness, each organ-system domain listed in Table 10 can be cross-referenced to the most recent international recommendations from the relevant societies; the table is intended as a pragmatic checklist rather than a substitute for specialty guidelines.

Beyond conventional risk stratification, specific cardiovascular findings are particularly prevalent among master endurance athletes. The most frequent is atrial fibrillation (AF), whose incidence increases with long-term high-volume aerobic exercise, showing a dose–response relationship with cumulative training load. This "exercise-induced AF" is usually paroxysmal, often vagally mediated, and occurs in the context of otherwise normal cardiac structure and function, reflecting adaptive atrial remodeling rather than overt pathology [51].

Other common findings include accelerated but predominantly calcified coronary atherosclerosis—typically with low inflammatory activity and limited plaque vulnerability—and mild, symmetrical aortic enlargement due to chronic haemodynamic stress. In these cases, distinguishing physiological adaptations from clinically relevant disease remains essential for accurate risk assessment and counselling [20].

Sport Eligibility According To Cardiovascular Risk

Resting 12-lead ECG remains a cornerstone of PPS. Once debated, its role is now supported by the AHA/ACC, which recognizes ECG as valuable when expert interpretation and resources are available [52]. In MAs, ECG interpretation can refer to the International Criteria [53], although these were validated in younger competitive athletes (12–35 years) and should therefore be applied with caution in masters, considering age-related changes and clinical context [54]. Moreover, while ECG is sensitive for inherited conditions,

Table 10 Practical recommendations for master athletes' non-CV screening

Organs	Problems	Screening strategy
Visual health	Common conditions such as presbyopia, cataracts, glaucoma, or macular degeneration can affect coordination, depth perception, and reaction time	A baseline ophthalmologic evaluation is recommended every 2–3 years, or more frequently in symptomatic individuals or those in high-risk sports (e.g., cycling, sailing, racquet sports)
Auditory function	Noise-induced hearing loss or age-related decline may impair team communication, balance, and spatial awareness	Audiometry should be performed every 3–5 years, especially in athletes exposed to loud environments (e.g., motorsports, water polo)
Oral and dental health	Periodontitis and dental infections can promote systemic inflammation and increase CV risk	Regular dental check-ups (every 6–12 months) are advised, particularly in athletes using mouthguards or with high-carbohydrate diets
Pulmonary function	Lifelong sport does not eliminate the age-related decline in pulmonary reserve	Screening with spirometry should be considered in smokers, ex-smokers, or those with unexplained dyspnea, fatigue, or prior lung conditions. Chest imaging may be warranted based on symptoms or occupational exposures
Urological health (men)	Prostate-related symptoms can reduce quality of life and training adherence	PSA screening and digital rectal exam are advised in men > 50 years or with family history of prostate cancer
Musculoskeletal integrity	Degenerative joint disease, tendinopathies, and overuse injuries are common	A targeted musculoskeletal evaluation, including joint mobility, posture, gait analysis, and previous injury history, should be performed annually. Bone mineral density (BMD) assessment is also recommended in athletes with low BMI, prior fractures, or those at risk of osteoporosis (e.g., postmenopausal women, chronic corticosteroid users)
Cancer screening	Master athletes should undergo age- and sex-specific screening in accordance with national guidelines	▪ Colorectal (FOBT/colonoscopy) > 50 years ▪ Breast (mammography) > 40 years ▪ Prostate (PSA + DRE) > 50 years ▪ Dermatological check for skin cancer annually in high sun-exposure sports (e.g., runners, cyclists, rowers)

it performs poorly in detecting coronary artery disease (CAD), the leading cause of sudden death in MA [55]. For this reason, the ESC advocates a comprehensive approach that includes maximal exercise stress testing (EST), particularly in sedentary or high-risk individuals aiming for vigorous activity, and in selected cases even for moderate exercise [20]. In the presence of symptoms or abnormal findings (e.g., ST-segment depression, high-risk premature ventricular beats [PVBs]), further imaging such as coronary CT angiography (CCTA) is warranted [54].

Italy represents a unique case where pre-participation cardiovascular screening (including a resting ECG) is mandated by law and regulated by national protocols [56–59]. In other countries, such as the United States, routine ECG-based screening is not universally required; therefore, the proposed flowchart and recommendations (Fig. 3) should be viewed as practical considerations that may be adapted to local policies and resources, rather than universal mandates. The most recent Italian recommendations advise considering personal and family history, performing a physical examination, conducting blood tests, obtaining a resting ECG, and undergoing maximal exercise testing, as well as risk stratification using SCORE2 or SCORE2-OP, depending on age [11]. EST plays a central role, not only for CAD detection but also for assessing exercise capacity, chronotropic and BP responses, and arrhythmia inducibility [60].

In MAs at low-to-moderate CV risk with a negative EST, eligibility is generally granted. However, additional testing (echocardiography, carotid ultrasound, CCTA) should be considered in individuals with familial hypercholesterolemia, a family history of premature cardiovascular events, or three or more risk factors (male sex, smoking, hypertension, dyslipidemia, impaired fasting glucose, obesity) [11]. Exercise stress echocardiography (ESE) enhances diagnostic accuracy in cases that are inconclusive [6, 61, 62].

A positive EST raises suspicion of CAD, particularly when traditional risk factors coexist [63]. Yet even in MAs, high-risk PVBs during exercise may indicate other substrates (fibrosis, channelopathies), prompting further investigation with cardiac MRI or genetic testing. It should also be recognized that PVB prevalence increases with age, including benign forms [64].

A novel proposal is the Lipid Athlete Score (Table 11), which stratifies risk based on lipid profile and additional factors [27]. While promising, it remains experimental and requires prospective validation before it can be widely adopted.

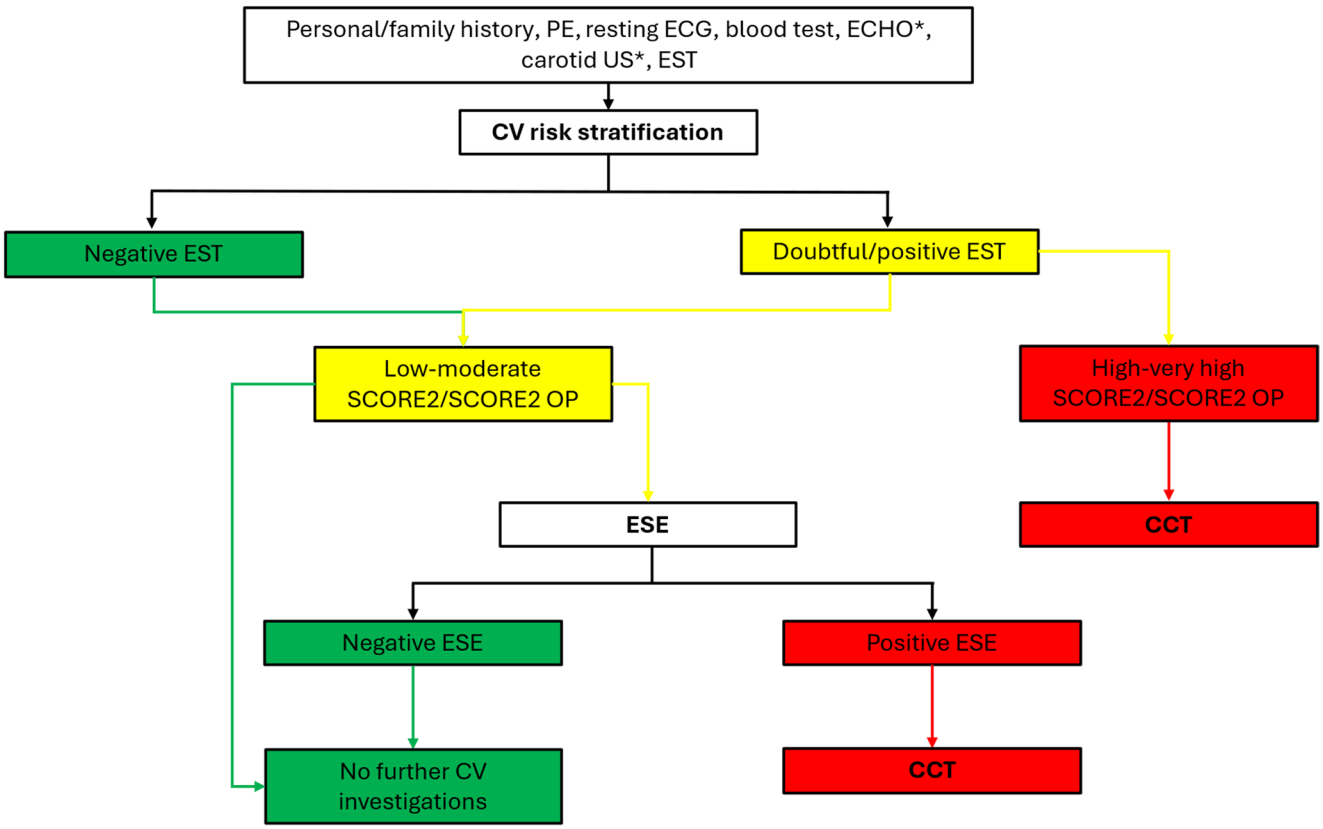


Fig. 3 Sports eligibility in master athlete. *ECHO and carotid ultrasound to be performed in specific condition (see text). CV history: CAD family history, coronary revascularization familiar history, SCD familiar history (1st -grade relatives who died suddenly < 55 aged if male or < 65 aged if female). CV risk factors: male sex, smoking habits, abnormal LDL (> 55 mg/dL if < 70 age, > 100 if > 70 age), high BP, IFG (110–126 mg/dL), BMI > 30 kg/m2, waist circumference ≥ 102 if male, ≥ 102 if female. Echocardiography: eccentric LV hypertrophy. Carotid ultrasound: atherosclerotic plaques (> 1.5 mm thickened). CV systemic indicators: GFR < 60 mL/min, long-standing diabetes mellitus (> 10 years). ECHO: echocardiography; EST: exercise stress test; CV: cardiovascular; US: ultrasound; ESE: exercise stress echocardiography; CAD: coronary artery disease.; SCD: sudden cardiac death; LDL: Low-density lipoprotein

Vademecum

The increasing participation of adults and older individuals in competitive sports necessitates a reevaluation of traditional PPS protocols. MAs represent a unique population: while they may exhibit physiological adaptations similar to younger athletes, they often carry CV and systemic risk profiles that are comparable to—or even exceeding—those of the general aging population.

This vademecum provides a concise, practical, and evidence-based guide for sports physicians, cardiologists, and healthcare professionals evaluating individuals over 40 years of age for competitive athletic activity. The proposed approach is stepwise, individualized, and multidisciplinary. It goes beyond cardiovascular assessment to include metabolic, oncological, urological, respiratory, sensory, and musculoskeletal domains, aiming to support safe sport participation and sustainable athletic longevity (Table 12). The

Table 11 Lipid athlete score. High risk: 2 M + ≥ 3 m; medium risk: 2 M + 1–2 m or 1 M + ≥ 2 m; low risk: 2 M or 1 M + 1 m or ≥ 2 m; and no risk: 1 M or 1 m

Major criteria (M)	LDL ≥ 115 mg/dL
	LDL/HDL ratio ≥ 1.90
Minor criteria (m)	Male sex
	BMI > 30 or fat mass > 22% for males and 32% for females
	Familiarity for dyslipidemia
	Cardiovascular risk factors (smoke, hypertension, and diabetes)

LDL low density lipoprotein; *HDL* high density lipoprotein; *BMI* body mass index

Table 12 Practical vademecum for the evaluation of master athletes

Step	Focus	Key Actions	Recommended Tools/Tests
1	Clinical and Athletic History	- Assess CV symptoms (chest pain, dyspnea, palpitations, syncope, fatigue) - Screen family history (CAD, SCD, hypercholesterolemia) - Identify traditional risk factors (HTN, smoking, diabetes, dyslipidemia, obesity) - Define athletic profile (sport type, training load, years active)	- Structured questionnaire - Clinical interview - Risk factor checklist
2	Physical Examination	- Measure BP in both arms - Cardiac auscultation - Assess BMI and waist circumference - Inspect for xanthomas/xanthelasmas	-BP cuff - Stethoscope - Tape measure - Clinical inspection
3	First-Line Diagnostics	- Identify ECG abnormalities (arrhythmias, blocks, repolarization) - Perform transthoracic echocardiography (if indicated) - Perform EST to detect ischemia or arrhythmias and assess BP response	− 12-lead ECG - Echocardiography - Exercise Stress Test (EST)
4	Laboratory Testing	- Evaluate lipid profile (TC, LDL-C, HDL-C, TG, Lp(a)) - Screen glucose metabolism (fasting plasma glucose, HbA1c ± OGTT if indicated) - Assess renal function (creatinine, eGFR, ACR)	- Blood sample (non-fasting acceptable) - Urine sample
5	Second- and Third-Line Investigations (if indicated)	- Consider in high-risk profiles or equivocal results - Perform ESE if borderline EST - Perform CCTA in intermediate–high risk or abnormal ESE	- Stress echocardiography (ESE) - Coronary CTA I
6	Risk Stratification & Eligibility	- Calculate 10-year CV risk (SCORE2/SCORE2-OP) - Match risk level with sport demands - Refer to current guidelines	- SCORE2 calculator -Carotid US, additional labs
7	Extra-Cardiovascular Evaluation	- Vision: screen for presbyopia, cataract, glaucoma (q 2–3 years) - Hearing: audiometry in high-risk sports (q 3–5 years) - Oral health: assess periodontal status (q 6–12 months) - Pulmonary function: spirometry if smoking history or dyspnea - Prostate (males): PSA + DRE > 50 years or with family history - Musculoskeletal health: evaluate joint mobility, previous injuries, posture, gait; consider BMD - Cancer screening: follow age- and sex-specific guidelines (colorectal, breast, prostate, skin)	- Ophthalmologic and audiologic exams - Dental check-ups - Spirometry, chest imaging (if needed) - PSA, DRE - BMD, gait analysis - Dermatological exam - Cancer screening protocols
8	Follow-up & reassessment	- Reassess CV risk and eligibility every 1–2 years depending on baseline risk - Repeat EST/ESE in high-risk athletes or when symptoms develop - Monitor therapy effects (antihypertensive, lipid-lowering, antidiabetic) on training and recovery	- Structured follow-up program - Periodic ECG/EST - Repeat labs and imaging as indicated

BP blood pressure, *BMI* body mass index, *ECG* electrocardiogram, *EST* exercise stress test, *ESE* exercise stress echocardiography, *CCTA* coronary computed tomography angiography, *MRI* magnetic resonance imaging, *SCORE2* Systematic Coronary Risk Estimation 2, *SCORE2-OP* SCORE2 for Older Persons, *LDL-C* low-density lipoprotein cholesterol, *HDL-C* high-density lipoprotein cholesterol, *TG* triglycerides, *Lp(a)* lipoprotein(a), *eGFR* estimated glomerular filtration rate, *ACR* albumin-to-creatinine ratio, *PSA* prostate-specific antigen, *DRE* digital rectal examination, *BMD* bone mineral density

stepwise approach is intended to be adapted to jurisdiction-specific screening frameworks and to individual risk, sport demands, and resource availability.

Limitations

This vademecum is a narrative review; it does not include a quantitative meta-analysis and is therefore susceptible to selection and publication bias. Evidence specifically focused on master athletes remains limited; many recommendations are extrapolated from general populations or younger athletes and translated to older, competitive individuals with clinical judgment. Reported thresholds (e.g., BP or lipid targets) reflect contemporary guideline ranges and may require individualization by sport demands and comorbidities. Some recommendations (e.g., exercise-induced hypertension management, lipid targets with high training loads, and the use of statins versus non-statin agents in performance settings) are informed by indirect or emerging data and should be interpreted with caution. Finally, regulatory aspects (e.g., doping bans, national eligibility rules) are jurisdiction-dependent and may change over time; readers should verify local policies at the time of application.

Conclusion and Future Perspectives

The growing participation of middle-aged and older individuals in competitive and recreational sport requires physicians to adopt a structured and individualized approach to

cardiovascular and metabolic evaluation. This vademecum summarizes the primary evidence and guideline-based recommendations concerning hypertension, dyslipidemia, dysglycemia/obesity, and extra-cardiovascular health in master athletes. Physicians should focus on identifying and controlling modifiable risk factors, applying appropriate exercise testing and imaging based on pre-test probability, and tailoring pharmacologic and lifestyle interventions to both safety and performance goals, always within current anti-doping and national eligibility frameworks. Despite the increasing scientific attention to this population, many recommendations still rely on extrapolation from younger or general cohorts. Future studies should aim to validate sport-specific thresholds for blood pressure response, lipid management, and cardiometabolic risk in aging athletes, and to clarify the long-term impact of intensive exercise on cardiovascular remodeling and event rates. A more integrated, longitudinal, and multidisciplinary model—combining clinical assessment, imaging, and digital tools—will be essential to ensure safe, enjoyable, and sustainable sport participation throughout healthy aging.

Key References

- Graziano F, Bondarev S, Corrado D *et al.* The Challenges of Screening Master Athletes. *Cardiology (Switzerland)* 2024:1–4.

  ◦ Provides a focused discussion on the unique challenges of cardiovascular screening in master athletes, highlighting unmet needs in this growing population.

- Palermi S, Cavarretta E, D'Ascenzi F *et al.* Athlete's Heart: A Cardiovascular Step-By-Step Multimodality Approach. *Rev Cardiovasc Med* 2023;**24**:151.

  ◦ Offers a comprehensive multimodality imaging framework for assessing athlete's heart, directly relevant to distinguishing physiologic from pathologic adaptations in older athletes.

- McEvoy JW, McCarthy CP, Bruno RM *et al.* 2024 ESC Guidelines for the management of elevated blood pressure and hypertension. *Eur Heart J* 2024, https://doi.org/10.1093/EURHEARTJ/EHAE178.

  ◦ Represents the most recent European consensus on hypertension management, a critical issue given its prevalence and prognostic impact in master athletes.

- Mancia G, Kreutz R, Brunström M *et al.* 2023 ESH Guidelines for the management of arterial hypertension The Task Force for the management of arterial hypertension of the European Society of Hypertension: Endorsed by the International Society of Hypertension (ISH) and the European Renal Association (ERA). *J Hypertens* 2023;**41**:1874–2071.

  ◦ Provides updated evidence-based recommendations on arterial hypertension, complementing ESC guidelines with broader international endorsement

- Zeppilli P, Biffi A, Cammarano M *et al.* Italian Cardiological Guidelines (COCIS) for Competitive Sport Eligibility in athletes with heart disease: update 2024. *Minerva Med* 2024;**115**, https://doi.org/10.23736/S0026-4806.24.09519-3.

  ◦ Delivers specific Italian recommendations on sport eligibility for athletes with cardiovascular disease, with direct implications for competitive master athletes.

- Pelliccia A, Sharma S, Gati S *et al.* 2020 ESC Guidelines on sports cardiology and exercise in patients with cardiovascular disease. *Eur Heart J* 2021;**42**:17–96.

  Represents the cornerstone European guideline on sports cardiology, addressing risk assessment and safe exercise participation in individuals with cardiovascular disease

- Mach F, Koskinas KC, Roeters van Lennep JE, et al. 2025 Focused Update of the 2019 ESC/EAS Guidelines for the management of dyslipidaemias. Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk: The Task Force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and European Atherosclerosis Society (EAS). *Eur Heart J* 2020;**41**:111–88.

  ◦ Defines the standards for dyslipidaemia management in Europe, highly relevant for cardiovascular risk stratification in aging athletic populations.

- Kim JH, Baggish AL, Levine BD *et al.* Clinical Considerations for Competitive Sports Participation for Athletes With Cardiovascular Abnormalities: A Scientific Statement From the American Heart Association and American College of Cardiology. *Circulation* 2025.

◦ Provides North American expert consensus on sports participation in athletes with cardiovascular abnormalities, offering a global perspective complementary to ESC/COCIS guidance.

Author Contributions All authors contributed equally to the conception, design, literature review, and writing of the manuscript. All authors critically revised the work, approved the final version, and agree to be accountable for all aspects of the manuscript.

Funding The authors did not receive support from any organization for the submitted work.

Data Availability No datasets were generated or analysed during the current study.

Declarations

Human and Animal Rights and Informed Consent This article does not involve studies with human participants or animals conducted by the authors. All figures, tables, and the graphical abstract are original and were created by the authors for this manuscript.

Competing Interests The authors declare no competing interests.

References

1. Graziano F, Bondarev S, Corrado D, Zorzi A. The challenges of screening master athletes. Cardiology, pp. 1–4. 2024. https://doi.org/10.1159/000538326.
2. Churchill TW, Baggish AL. Cardiovascular care of masters athletes. J Cardiovasc Transl Res. 2020;13:313–21.
3. Lepers R, Cattagni T. Do older athletes reach limits in their performance during marathon running? Age (Omaha). 2012;34:773–81.
4. Palermi S, Cavarretta E, D'Ascenzi F, et al. Athlete's heart: A cardiovascular Step-By-Step multimodality approach. Rev Cardiovasc Med. 2023;24:151.
5. Cavarretta E, Pingitore A, Peruzzi M, et al. Cardiovascular screening of master athletes: insights from the master athletes screening study. Eur J Prev Cardiol. 2023;30:884–6.
6. Pescatore PV, Grassi M, Palermi S, et al. Enhancing cardiovascular screening in master athletes: the role of exercise stress echocardiography. Minerva Cardiol Angiol. 2025. https://doi.org/10.23736/S2724-5683.25.06747-X.
7. Palermi S, Sperlongano S, Mandoli GE, et al. Exercise stress echocardiography in athletes: applications, methodology, and challenges. J Clin Med. 2023;12. https://doi.org/10.3390/JCM12247678.
8. D'andrea A, Sperlongano S, Russo V, et al. The role of multimodality imaging in athlete's heart diagnosis: current status and future directions. J Clin Med. 2021;10. https://doi.org/10.3390/JCM10215126.
9. McEvoy JW, McCarthy CP, Bruno RM, et al. 2024 ESC guidelines for the management of elevated blood pressure and hypertension. Eur Heart J. 2024. https://doi.org/10.1093/EURHEARTJ/EHAE178.
10. Mancia G, Kreutz R, Brunström M, et al. 2023 ESH guidelines for the management of arterial hypertension the task force for the management of arterial hypertension of the European society of hypertension: endorsed by the international society of hypertension (ISH) and the European renal association (ERA). J Hypertens. 2023;41:1874–2071.
11. Zeppilli P, Biffi A, Cammarano M, et al. Italian cardiological guidelines (COCIS) for competitive sport eligibility in athletes with heart disease: update 2024. Minerva Med. 2024;115. https://doi.org/10.23736/S0026-4806.24.09519-3.
12. Williams B, Mancia G, Spiering W, et al. 2018 ESC/ESH guidelines for the management of arterial hypertension: the task force for the management of arterial hypertension of the European society of cardiology (ESC) and the European society of hypertension (ESH). Eur Heart J. 2018;39:3021–104.
13. Schweiger V, Niederseer D, Schmied C, et al. Athletes and hypertension. Curr Cardiol Rep. 2021;23:1–11.
14. Caselli S, Vaquer Sequì A, Lemme E, et al. Prevalence and management of systemic hypertension in athletes. Am J Cardiol. 2017;119:1616–22.
15. McCarthy CP, Bruno RM, Brouwers S, et al. 2024 ESC guidelines for the management of elevated blood pressure and hypertension. Eur Heart J. 2024;45:3912–4018.
16. Liguori G, Feito Y, Fountaine C, et al. ACSM's guidelines for exercise testing and prescription. Wolters Kluwer (ed.). Wolters Kluwer; 2021.
17. Palermi S, Bragazzi NL, Cular D, et al. How chest press-based exercises can alleviate the burden of cardiovascular diseases. Hum Mov. 2022;23:88–98.
18. Sharman JE, La Gerche A, Coombes JS. Exercise and cardiovascular risk in patients with hypertension. Am J Hypertens. 2015;28:147–58.
19. Di Gioia G, Ferrera A, Maestrini V, et al. Correlation between workload-indexed blood pressure response to exercise (SBP/MET slope) and clinical and echocardiographic parameters among normotensive olympic athletes. Int J Cardiol. 2025;429. https://doi.org/10.1016/j.ijcard.2025.133171.
20. Pelliccia A, Sharma S, Gati S, et al. 2020 ESC guidelines on sports cardiology and exercise in patients with cardiovascular disease. Eur Heart J. 2021;42:17–96.
21. Vecchiato M, Baioccato V, Palermi S. Antidoping testing and testicle cancer diagnosis: an unexpected helper. J Sci Med Sport. 2024;27. https://doi.org/10.1016/J.JSAMS.2024.08.001.
22. Würzburger L, Wiech P, Rossi VA, et al. Hypertensive response to exercise in athletes: unremarkable finding or relevant marker for future cardiovascular complications? Int J Hypertens. 2022;2022. https://doi.org/10.1155/2022/8476751.
23. Di Gioia G, Ferrera A, Maestrini V, et al. Abnormal blood pressure response to exercise and ventricular arrhythmias: A suspicious association in athletes. J Hypertens. 2025;43:513–20.
24. Mach F, Koskinas KC, Roeters van Lennep JE, et al. 2025 Focused Update of the 2019 ESC/EAS Guidelines for the management of dyslipidaemias. Eur Heart J. 2025;46(42):4359–78. https://doi.org/10.1093/eurheartj/ehaf190.
25. D'ascenzi. Cardiovascular risk profile in olympic athletes: an unexpected and underestimated risk scenario. Br J Sports Med. 2018;0:1–7.
26. Di Gioia G, Buzzelli L, Maestrini V, et al. Long-Term evaluation of lipid profile changes in olympic athletes. Int J Sport Nutr Exerc Metab. 2024;34:267–74.
27. Di Gioia G, Buzzelli L, Maestrini V, et al. Lipid profile in olympic athletes: proposal for a lipid athlete score as a clinical tool to identify high-risk athletes. J Clin Med. 2023;12. https://doi.org/10.3390/JCM12237449.
28. Castelletti S, Zorzi A, Ballardini E, et al. Molecular genetic testing in athletes: why and when a position statement from the Italian society of sports cardiology. Int J Cardiol. 2022;0. https://doi.org/10.1016/J.IJCARD.2022.05.071.
29. Stanton KM, Kienzle V, Dinnes DLM, et al. Moderate-and High-Intensity exercise improves lipoprotein profile and cholesterol efflux capacity in healthy young men. J Am Heart Assoc. 2022;11:23386.

30. Soffer DE, Marston NA, Maki KC, et al. Role of Apolipoprotein B in the clinical management of cardiovascular risk in adults: an expert clinical consensus from the National lipid association. J Clin Lipidol. 2024;18:e647–63.
31. Reyes-Soffer G, Ginsberg HN, Berglund L, et al. Lipoprotein(a): A genetically Determined, Causal, and prevalent risk factor for atherosclerotic cardiovascular disease: A scientific statement from the American heart association. Arterioscler Thromb Vasc Biol. 2022;42:E48–60.
32. Visseren F, Mach F, Smulders YM, et al. 2021 ESC guidelines on cardiovascular disease prevention in clinical practice. Eur Heart J. 2021;42:3227–337.
33. Nestel PJ, Mori TA. Dietary patterns, dietary nutrients and cardiovascular disease. Rev Cardiovasc Med. 2022;23:17.
34. Palermi S, Vecchiato M, Brugin E, et al. The role of omega-3 fatty acids in sports nutrition: an overview. Med Dello Sport. 2025;77. https://doi.org/10.23736/S0025-7826.25.04523-5.
35. Buzzelli L, Segreti A, Di Gioia D, et al. Alternative lipid lowering strategies: state-of-the-art review of red yeast rice. Fitoterapia. 2024;172. https://doi.org/10.1016/j.fitote.2023.105719.
36. Mach F, Koskinas KC, van Roeters JE, et al. 2025 focused update of the 2019 ESC/EAS guidelines for the management of dyslipidaemias: developed by the task force for the management of dyslipidaemias of the European society of cardiology (ESC) and the European atherosclerosis society (EAS). Eur Heart J. 2025. https://doi.org/10.1093/EURHEARTJ/EHAF190.
37. Katsiki N, Mikhailidis DP, Bajraktari G, et al. Statin therapy in athletes and patients performing regular intense exercise – Position paper from the international lipid expert panel (ILEP). Pharmacol Res. 2020;155:104719.
38. Christou GA, Kouidi EJ, Deligiannis AP, et al. Diagnosis and treatment of dyslipidaemias in athletes. Curr Vasc Pharmacol. 2017;15:238–47.
39. Pharmacologic approaches to glycemic treatment. Standards of care in diabetes—2025. Diabetes Care. 2025;48:S181–206.
40. Vecchiato M, Zanardo E, Battista F, et al. The effect of exercise training on Irisin secretion in patients with type 2 diabetes: a systematic review. J Clin Med. 2022;12. https://doi.org/10.3390/JCM12010062.
41. Palermi S, Iacono O, Sirico F, et al. The complex relationship between physical activity and diabetes: an overview. J Basic Clin Physiol Pharmacol. 2021. https://doi.org/10.1515/jbcpp-2021-0279.
42. Busetto L, Dicker D, Frühbeck G, et al. A new framework for the diagnosis, staging and management of obesity in adults. Nat Med. 2024;30:2395–9.
43. Ross R, Neeland IJ, Yamashita S, et al. Waist circumference as a vital sign in clinical practice: a consensus statement from the IAS and ICCR working group on visceral obesity. Nat Rev Endocrinol. 2020;16:177.
44. Palermi S, Vecchiato M, Brusiani S, et al. Distribution of cardiovascular modifiable risk factors in a corporate wellness program: A case study of occupational cardiology in the Ferrari company. High Blood Press Cardiovasc Prev. 2025. https://doi.org/10.1007/S40292-025-00722-Z.
45. Echouffo-Tcheugui JB, Selvin E. Pre-Diabetes and what it means: the epidemiological evidence. Annu Rev Public Health. 2021;42:59.
46. Committee ADAPP, ElSayed NA, McCoy RG, et al. 2. Diagnosis and classification of diabetes: standards of care in Diabetes—2025. Diabetes Care. 2025;48:S27–49.
47. Committee ADAPP, ElSayed NA, McCoy RG, et al. 8. Obesity and weight management for the prevention and treatment of type 2 diabetes: standards of care in Diabetes–2025. Diabetes Care. 2025;48:S167–80.
48. Tchang BG, Aras M, Kumar RB, et al. Pharmacologic treatment of overweight and obesity in adults. In: Feingold KR, Ahmed SF, Anawalt B, et al., editors. Endotext. South Dartmouth (MA): MDText.com, Inc.; 2024.
49. Palermi S, Annarumma G, Spinelli A, et al. Acceptability and practicality of a quick musculoskeletal examination into sports medicine Pre-Participation evaluation. Pediatr Rep. 2022;14:207–16.
50. Catapano M, Ahmed M, Breslow RG, et al. The aging athlete. PM R. 2022;14:643–51.
51. Palermi A, Molinari LV, Ricci F, et al. Practical guidance for management of atrial fibrillation in sports cardiology. Curr Probl Cardiol. 2025;50(4):102995. https://doi.org/10.1016/j.cpcardiol.2025.102995.
52. Kim JH, Baggish AL, Levine BD, et al. Clinical considerations for competitive sports participation for athletes with cardiovascular abnormalities: A scientific statement from the American heart association and American college of cardiology. Circulation. 2025;151:e716–61.
53. Zorzi A, Vio R, Bettella N, et al. Criteria for interpretation of the athlete's ECG: A critical appraisal. Pacing Clin Electrophysiol. 2020;43:882–90.
54. Graziano F, Tonelli R, Balla D, et al. Modern interpretation of electrocardiogram and premature ventricular beats in athletes. Cardiol Discovery. 2025. https://doi.org/10.1097/CD9.0000000000000164.
55. Panhuyzen-Goedkoop NM, Wellens HJ, Verbeek ALM, et al. ECG criteria for the detection of high-risk cardiovascular conditions in master athletes. Eur J Prev Cardiol. 2020;27:1529–38.
56. Corrado D, Zorzi A, Sarto P. Pre-participation screening for safe sports activity. Eur Heart J. 2023;44:2258–9.
57. Zorzi A, Vessella T, De Lazzari M, et al. Screening young athletes for diseases at risk of sudden cardiac death: role of stress testing for ventricular arrhythmias. Eur J Prev Cardiol. 2020;27:311–20.
58. Vessella T, Zorzi A, Merlo L, et al. The Italian preparticipation evaluation programme: diagnostic yield, rate of disqualification and cost analysis. Br J Sports Med. 2020;54:231–7.
59. Sarto P, Zorzi A, Merlo L, et al. Serial versus single cardiovascular screening of adolescent athletes. Circulation. 2021;143:1729–31.
60. Sirico F, Fernando F, Di Paolo F et al. Exercise stress test in apparently healthy individuals – where to place the finish line? The Ferrari corporate wellness programme experience. 2019;26:731–8. https://doi.org/10.1177/2047487318825174.
61. Palermi S, Sperlongano S, Mandoli GE, et al. Exercise stress echocardiography in athletes: Applications, methodology, and challenges. J Clin Med. 2023;12(24):7678. https://doi.org/10.3390/jcm12247678.
62. Gaibazzi N, Tuttolomondo D, Guaricci AI, et al. Stress-echocardiography or coronary computed tomography in suspected chronic coronary syndrome after the 2019 European guidelines? A practical guide. J Cardiovasc Med (Hagerstown). 2022;23:12–21.
63. Graziano F, Cozza E, Millin A, et al. Prevalence and characteristics of coronary artery disease in master athletes with ST-segment depression or high-risk premature ventricular beats at pre-participation exercise testing. Eur Heart J Open. 2025;5:90.
64. Pizzolato M, Graziano F, Bondarev S, et al. Incidence and characteristics of premature ventricular beats at exercise testing for preparticipation screening. J Sports Med Phys Fitness. 2024;64:846–8.