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The ABCs of cardiovascular disease prevention: communicating what we know in 2026

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ABSTRACT

Despite numerous breakthroughs in Preventive Cardiology, control of key cardiovascular disease (CVD) risk factors including blood pressure and diabetes has stagnated in the past decade, driven in part by the obesity epidemic and population aging. Improving dissemination and implementation of the latest guidelines and evidence-based recommendations is a key national priority for CVD prevention. The ABCs of CVD Prevention is a structured framework identifying actionable domains for CVD prevention, first published in 2001 and last updated in 2021, designed to maximize clarity and uptake of key preventive recommendations. In this State-of-the-Art review, we provide a comprehensively updated version of the ABCs reflecting what we know in 2026, with two practice pearls per domain and accompanying figures and materials to support dissemination. The ABCs of CVD Prevention are: **A**ssess CVD Risk, **A**ntiplatelets, **B**lood Pressure, **B**ody Fat, **C**holesterol, **C**igarettes & Alcohol, **D**iet, **D**iabetes & Cardiovascular-Kidney-Metabolic Syndrome, **E**xercise, **E**xtend Healthspan, Heart **F**ailure, and Atrial **F**ibrillation. The ABCs have the potential for population-level impact with clinical uptake and educational efforts across specialties and settings, supported by the evidence and materials herein.

1. Introduction

Cardiovascular disease (CVD) remains the leading cause of morbidity and mortality despite decades of progress in its prevention, particularly for atherosclerotic CVD (ASCVD) and heart failure [1,2]. With currently available tests, tools, and therapies, CVD risk can be identified and addressed at any life stage through lifestyle optimization and risk factor modification [3,4]. Despite this, national rates of obesity, blood pressure, and diabetes control have remained stagnant or declined over the past decade [5]. Preventive progress in the face of population aging and the obesity epidemic will require widespread high-fidelity implementation of contemporary guidelines across specialties and disciplines: a major challenge in the current healthcare environment. To improve outcomes and implementation of prevention guidelines, the Johns Hopkins Ciccarone Center has long advocated for a streamlined approach that can be reviewed at each visit, known as the ABCs of CVD Prevention.

The ABCs of CVD Prevention were first published in 2001 [6] as a structured framework to identify key actionable domains of prevention for both patients and clinicians. Multiple iterations with guideline-focused updates have been published, reflecting significant advances in the field of Preventive Cardiology [7–12]. Prior versions featured several major updates, including the introduction of risk estimators, primary and secondary prevention tools for both atrial fibrillation and heart failure, and expanded evidence on social and biological drivers of ASCVD [8–10]. In the years since the most recent updates, Preventive Cardiology has continued to expand, with notable advances including the development of more contemporary and accurate cardiovascular risk estimators [13,14], identification of risk enhancers [15,16], novel cardiometabolic [17–20] and lipid-lowering therapies (LLT) [21–23], large trials guiding preventive therapies and goals [21,24,25], and increased focus on the interconnection between cardiovascular, kidney, and metabolic health, reflecting the crucial link between adiposity and inflammation on CVD risk [26].

Several epidemiologic trends, including population aging and the obesity epidemic, have also resulted in a need for an updated approach. Rates of obesity, a key risk factor for both Cardiovascular-Kidney-Metabolic (CKM) Syndrome and CVD [5], are rising, with an expected prevalence of over 50% in the US in the next five years [27].

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Additionally, as the number of older adults with cardiovascular risk factors and CVD increases, in part due to continued advancements in CVD prevention, the proportion of patients with CVD over age 80 will double to nearly 25% by 2050 [28]. Although a suboptimal surrogate of biologic aging, chronologic age remains the strongest risk factor for development of CVD across prediction models [29]. To meet the needs of an aging population, insights from Geriatric Cardiology to personalize CVD prevention in older adults are increasingly essential to optimize patient-centered care [30,31].

Despite significant progress in Preventive Cardiology, many population-level gaps remain, including inadequate implementation of CVD risk factor screening and identification of those at elevated risk [32], suboptimal integration of clinical guidelines into practice [33], and insufficient initiation or intensification of proven pharmacotherapies [34]. For example, nearly 80% of adults in the United States have blood pressure readings above goal of <130/80 mmHg [35], approximately half of adults with diabetes achieve glycemic control [5], and almost 75% of patients with a prior ASCVD event have low-density lipoprotein cholesterol (LDL-C) above 70 mg/dL [5]. Given these challenges, in this State-of-the-Art review, we provide an updated framework and toolkit for CVD prevention based on the latest evidence, ready for implementation by any clinician in any setting.

2. Methods

We aim to provide a systematic approach to CVD prevention using a clearly defined framework updating the established ABCs of CVD prevention, targeting actionable areas of prevention to support dissemination of evidence-based recommendations across clinical settings [10]. This framework is intended for all clinicians caring for patients with cardiovascular risk factors and CVD. This can be useful to practitioners and patients in all clinical settings, including cardiology, internal medicine, family medicine, geriatrics, nephrology, endocrinology, and beyond. To maximize retention and recall, the framework includes two domains per letter and two practice pearls per domain.

3. ABCs of CVD prevention

To support dissemination and depth, the evidence and details of the ABCs of CVD Prevention are detailed below, accompanied by a figure (Fig. 1), flyer (Fig. 2), table (Table 1), educational slide deck (**Supplement 1**), and SmartPhrase template (**Supplement 2**).

4. Assess risk

Assessment of CVD risk in primary prevention has evolved with development and dissemination of various risk estimators [13,14,36]. In 2023, the Predicting Risk of CVD EVENTs (PREVENT™) Equations were introduced by the American Heart Association (AHA) [14], representing a major leap forward in CVD risk assessment.

4.1. Calculate risk using PREVENT™ equations, personalize with risk enhancers, and reclassify borderline-intermediate risk with CAC

In comparison to the widely utilized Pooled Cohort Equations (PCEs), which were created using a population of ~25,000 adults and introduced in 2013 [13], the PREVENT™ equations were developed and validated using large and more contemporary data sets of ~6.5 million individuals aged 30 to 79 years old to estimate both 10- and 30-year (in persons up to age 60) risks of CVD, ASCVD, and heart failure [14]. In contrast to the PCE, the PREVENT™ calculator does not include race and incorporates new variables such as social deprivation index (zip code) and CKM syndrome risk factors [26]. While risk models provide a straightforward approach in evaluating risk at the population level,

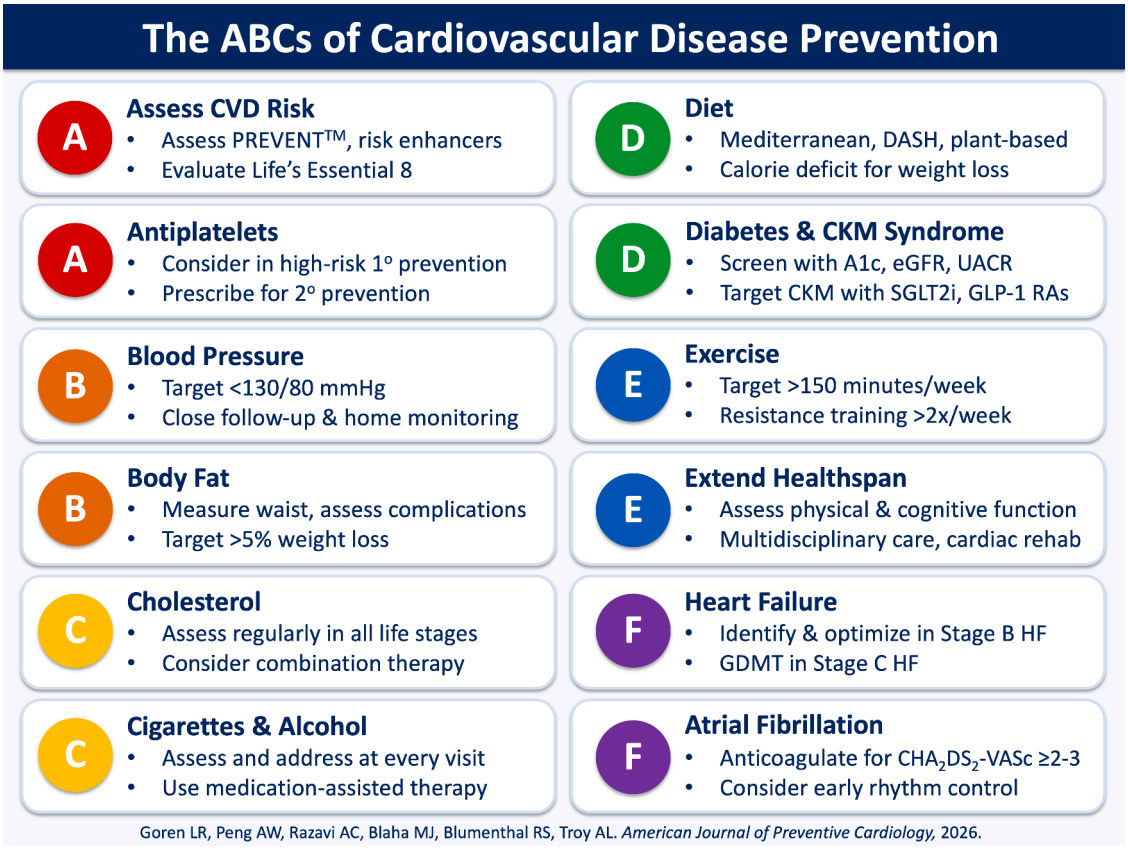

Fig. 1. Figure summarizing the ABCs of CVD prevention.

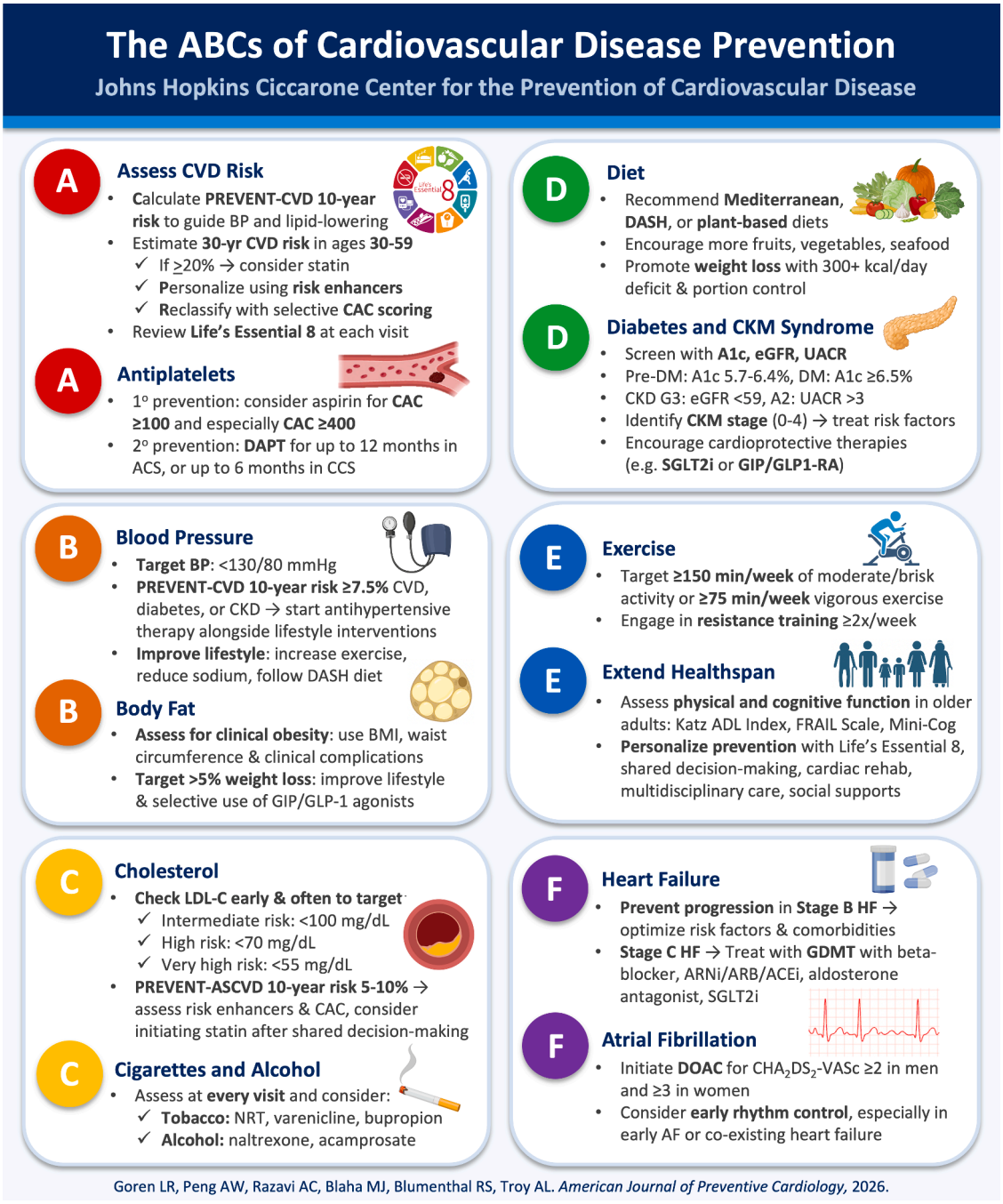

Fig. 2. Flyer summarizing the ABCs of CVD prevention.

further personalization is crucial to guide decision making, especially for patients who fall between low and high-risk categories. Therefore, shared decision-making surrounding initiation of LLT, anti-hypertensives, antiplatelet agents, and broader preventive pharmacotherapies (e.g. sodium-glucose cotransporter 2 inhibitors [SGLT2i] and incretin mimetics) requires Consideration of patient-specific benefits and risks, Listening to patient priorities, then incorporating those into a shared Decision [37].

As a result of the recalibration within the PREVENT™ model, 10-year risk estimates are generally around 40–50% lower than the PCE, highlighting the need for a personalized approach to avoid undertreating individuals who may otherwise benefit from escalation of therapies [38]. To further refine clinical decision making, assessing for risk enhancers is vital [15,26], which include elevated lipoprotein(a) [Lp(a)] and high sensitivity C-reactive protein [38], persistent LDL-*C* ≥160 mg/dL, family history of premature ASCVD, inflammatory conditions, sex-specific factors (e.g. preeclampsia), cardio-oncologic risk factors (e.g. anthracycline exposure), and geriatric risk factors (e.g. frailty) [39]. In patients with borderline (3 to <5% 10-year ASCVD risk) or intermediate (5 to ≤10% 10-year) ASVCD risk, incorporating risk enhancers supports the decision-making process. For borderline or intermediate risk individuals in whom uncertainty remains about whether to start or intensify pharmacotherapy, coronary artery calcium (CAC) scoring may provide further refinement in risk assessment and guide initiation or escalation of therapy [40]. For younger patients (30–59 years old), estimation of 30-year PREVENT-CVD risk may be helpful to guide risk discussion, with risks >10% considered elevated [41].

4.2. Evaluate Life's Essential 8 to create a personalized plan for cardiovascular health

In 2022, the AHA created an updated checklist of behaviors assessing cardiovascular health initially constructed in 2010, known as Life's Essential 8 [42]. This framework highlights 8 foundational domains for cardiovascular health, which include dietary patterns with whole foods, lean proteins, and fresh fruits and vegetables; engaging in physical activity of at least 2.5 h of moderate or 75 min of vigorous exercise per week; avoiding nicotine products; prioritizing at least 7 h of sleep per night; maintaining a healthy weight; and lowering lipids, glucose, and blood pressure to heart healthy levels [42]. This checklist should be reviewed at nearly all visits through a positive lens with the goal of shifting from treatment only to promoting cardiovascular health across the life span.

5. Antiplatelets

5.1. Consider antiplatelet medications in primary prevention for individuals at highest cardiovascular risk

Aspirin therapy for primary prevention should be considered for only high-risk individuals age 40 to 70 years of age without increased bleeding risk, as detailed in the 2019 ACC/AHA Guideline on the Primary Prevention of CVD [4]. CAC scoring is a valuable marker of coronary plaque burden to help identify those who are at highest risk and may benefit from aspirin initiation for primary prevention [43]. Specifically, the benefits of aspirin are likely to outweigh risks and should be considered for primary prevention for patients age ≤70 without high bleeding risk who have CAC ≥100, with even stronger consideration for those with CAC ≥400 [44]. There is clinical equipoise for the utilization of primary prevention aspirin therapy among individuals with elevated

Table 1
ABCs of CVD prevention [14,15,42,44,45,46,50,50,53,55,56,57,59,60,58,61,42,62,63,63–67,69,70,17–19,26,4,73,75–77,4,55,82,86–88,29,93,50,94,95,26,101,101–103,108,114,116,117].

	Domains	Pearls	Impact	References
A	**Assess Risk**	Calculate risk using PREVENT equations, personalize with risk enhancers, & reclassify borderline-intermediate risk with CAC	Individualized risk assessments & risk enhancers promote appropriate initiation of cardioprotective therapies & can further refine CVD risk	[14, 15]
		Evaluate Life's Essential 8 to create a personalized plan for cardiovascular health	Implementation of Life's Essential 8 shifts the clinical focus from disease management to health promotion, identifying preventive priorities across the lifespan	[42]
A	**Antiplatelets**	Consider antiplatelet medications in primary prevention for individuals at highest cardiovascular risk	CAC scoring & Lp(a) can identify high-risk patients in primary prevention who may benefit from aspirin to reduce MACE	[44. 45]
		Prescribe antiplatelet medications for secondary prevention, personalized by ischemic & bleeding risk	DAPT for ≥6-12 months after PCI reduces MACE and CVD mortality	[46]
B	**Blood Pressure**	Target blood pressure <130/80 mmHg using lifestyle & medications, particularly for those with clinical CVD, diabetes, CKD, or PREVENT-CVD ≥7.5%	Targeting BP <130/80 mmHg reduces risk of dementia, orthostatic hypotension, CAD, HF, AF, stroke, CKD, and mortality	[50]
		To achieve blood pressure targets, ensure close follow-up with team-based care & HBPM, & consider single-pill combination therapy for those with stage 2 hypertension	Team-based care improves BP control. Single-pill combinations improve adherence, reduce MACE and hospitalization.	[50]
B	**Body Fat**	Assess for clinical obesity with BMI, weight circumference & clinical complications	Obesity increases risk for HF, OSA, metabolic syndrome, hepatic steatosis & AF	[53, 55, 56]
		Target >5% weight loss using lifestyle, & selectively, use of GLP-1 agonists	10% weight loss reduces risk for MACE & 5-10% weight loss can improve hepatic steatosis or stress incontinence	[57]
C	**Cholesterol**	Check lipid levels early & regularly to prevent & manage ASCVD risk	Starting LDL-C reduction early with lifestyle and pharmacotherapy prevents atheroma and ASCVD	[59, 60]
		Stratify risk & intensify lipid-lowering using combination therapy for patients at high-risk or not reaching targets	Target-directed LLT (e.g. <55 mg/dL) for patients at high ASCVD risk reduces MACE and CVD mortality	[58, 61]
C	**Cigarettes & Alcohol**	Assess & address substance use, particularly tobacco & alcohol at each visit	Tobacco & alcohol use increases CVD risk, inflammation & endothelial dysfunction	[42, 62,63]
		Utilize evidence-based tools and therapies to support tobacco and alcohol cessation	Behavioral & pharmacotherapies are effective in reducing tobacco &/or alcohol consumption	[63-67]

(continued on next page)

Table 1 (*continued*)

D	**Diabetes/CKM Syndrome**	Screen A1c, eGFR & UACR, & personalize A1c targets	Screen for & treat albuminuria & CKD; target hemoglobin A1c of <7% for most patients to prevent microvascular complications & improve glycemic control	[69, 70]
		Identify CKM stage & encourage cardioprotective therapies, including SGLT2 inhibitors & GLP-1 agonists	SGLT2i improve CVD mortality, HF hospitalizations, & progression of CKD; GLP-1 agonists reduce CVD mortality & body fat	[17-19, 26]
D	**Diet**	Encourage Mediterranean, DASH, or plant-based diets	Dietary patterns consisting of lean proteins, fruits, vegetables & whole grains can reduce CVD risk, lower BP & improve glycemic and lipid control	[4, 73, 75-77]
		Focus on portion control & calorie deficit in weight-loss discussions	A 500 to 750 kcal/day deficit can be effective in promoting weight loss	[4, 55]
E	**Exercise**	Aim for >150 minutes/week or moderate/brisk activity or > 75 minutes/week of vigorous activity	ASCVD benefits from physical activity occur in a dose-response fashion	[82]
		Engage in resistance training at least 2x per week	Resistance training improves strength, mood, sleep, & reduces risk of CVD morbidity & mortality	[86-88]
E	**Extend Healthspan**	Assess physical & cognitive function in older adults with cardiovascular risk factors or disease	Frailty & dementia are bidirectionally associated with ASCVD, HF, AF, CV hospitalization, & MACE	[29, 93]
		Personalize prevention throughout the life course with Life's Essential 8, shared decision-making, cardiac rehab, and multidisciplinary care addressing health-related social needs.	BP, LDL-C, DM, & HF control, as well as cardiac rehab participation, improve frailty, cognition, MACE, & mortality.	[50, 94, 95]
F	**Failure**	Identify patients in Stage B heart failure & prevent progression to Stage C using neurohormonal & cardiometabolic therapies	Medication optimization, whole-scale preventive assessment & treatment of comorbidities (e.g. HTN, DM, obesity) can halt further progression of HF	[26, 101]
		Treat Stage C heart failure with GDMT to prevent hospitalizations & mortality	Rapid GDMT titration & close follow up improves symptoms, quality of life, HF hospitalizations, & mortality	[101-103]
F	**Fibrillation**	Initiate oral anticoagulation for CHA2DS2-VASc score ≥ 2-3	Reduces risk of stroke, with DOACs having lower stroke and bleeding risk than warfarin	[108]
		Consider early rhythm control, especially for adults with early atrial fibrillation &/or coexisting heart failure	Early rhythm control strategy reduces atrial remodeling, HF, stroke & atrial fibrillation related deaths	[114, 116, 117]

Legend: CAC = coronary artery calcium; CVD = cardiovascular disease; Lp(a) = lipoprotein(a); MACE = major adverse cardiovascular event; DAPT = dual antiplatelet therapy; PCI = percutaneous coronary intervention; CKD = chronic kidney disease; BP = blood pressure; CAD = coronary artery disease; HF = heart failure; AF = atrial fibrillation; HBPM = home blood pressure monitoring; BMI = body mass index; OSA = obstructive sleep apnea; GLP-1 agonist = glucagon-like peptide-1 receptor agonists; LDL-C = low-density lipoprotein C; ASCVD = atherosclerotic cardiovascular disease; LLT = lipid-lowering therapy; eGFR = estimated glomerular filtration rate; UACR = urine albumin-to-creatinine ratio; CKM = Cardiovascular-Kidney-Metabolic; SGLT2i = sodium-glucose cotransporter 2 inhibitors; DASH = Dietary Approaches to Stop Hypertension; CV = cardiovascular; DM = diabetes mellitus; HTN = hypertension; GDMT = guideline directed medical therapy; DOAC = direct oral anticoagulant.

Lp(a), though risk-benefit discussions may be helpful [45].

5.2. Prescribe antiplatelet medications for secondary prevention, personalized by ischemic and bleeding risk

Selection of antiplatelet therapies for secondary prevention requires an individualized approach based upon assessment of bleeding and thrombotic risk [46]. In patients who received percutaneous coronary intervention (PCI) for acute coronary syndrome (ACS), the 2025 AHA/ACC Guideline for the Management of Patients With Acute Coronary Syndromes recommend a default strategy of dual antiplatelet therapy (DAPT) for up to 12 months for most patients, with individualized consideration based on comorbidities, risk of bleeding and ischemic risk, and likelihood of adverse events [46].

For patients at higher bleeding risk following PCI, deescalation strategies involving shorter durations of DAPT (1–3 months) followed by P2Y12 inhibitor monotherapy may be considered. In patients with low bleeding risk and history of myocardial infarction, it may be reasonable to extend DAPT past 12 months and for up to 3 years to reduce major adverse cardiovascular events [47]. For patients already on anticoagulation and undergoing PCI, triple antithrombotic therapy is recommended for no more than 1 to 4 weeks followed by transition to oral anticoagulation and single antiplatelet therapy, typically clopidogrel for 6 months in chronic coronary disease and 12 months for ACS [46,48].

After this timeline, which can be personalized according to ischemic and bleeding risk, oral anticoagulation monotherapy is recommended. For individuals with chronic coronary disease or polyvascular disease without an indication for oral anticoagulation who are at high ischemic risk and low bleeding risk, the addition of low-dose rivaroxaban 2.5 mg twice daily to aspirin 81 mg may be considered [49].

6. Blood pressure

Hypertension is the most prevalent modifiable risk factor for incident and recurrent CVD events. The recently published 2025 AHA/ACC/Multisociety Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults provides key updates and management strategies for clinicians treating hypertension [50].

6.1. Target blood pressure <130/80 mmHg using lifestyle and medications, particularly for those with clinical CVD, diabetes, chronic kidney disease, or PREVENT-CVD ≥7.5%

Elevated blood pressure is defined as systolic blood pressure (SBP) 120–129 mmHg and diastolic blood pressure (DBP) <80 mmHg. Hypertension is further classified into Stage 1 and Stage 2, defined by SBP 130–139 mmHg or DBP 80–89 mmHg and SBP ≥140 mmHg or DBP ≥90 mmHg, respectively. Clinicians should target blood pressure goals ≤130/80 mmHg and encourage a target of ≤120/80 mmHg via continued lifestyle optimization, especially in patients at increased cardiovascular risk to reduce the risk of CVD and mortality [50]. The decision to initiate anti-hypertensive pharmacotherapy should be based on both blood pressure and clinical risk, including the PREVENT™ estimated CVD risk [14]. Pharmacotherapy is recommended for patients with SBP ≥130 mmHg or DBP ≥80 mmHg and CVD, chronic kidney disease (CKD), diabetes, or increased 10-year CVD risk (≥ 7.5%) based on PREVENT™ [50], replacing the 10% threshold previously recommended using the PCEs. In adults with blood pressure ≥130/80 mmHg without clinical CVD and <7.5% 10-year PREVENT-CVD risk, a 3- to 6-month trial of lifestyle modifications should be considered prior to initiating medications [50]. In patients who are overweight or have obesity, clinicians should counsel patients on weight loss with a goal of at least 5% of their body weight [50]. A focus on heart-healthy dietary patterns, including the Dietary Approaches to Stop Hypertension (DASH) diet, mediterranean diet, and sodium reduction, as well as structured exercise programs are recommended [50].

6.2. To achieve blood pressure targets, ensure close follow-up with team-based care and home blood pressure monitoring, and consider single-pill combination therapy for those with stage 2 hypertension

Treating uncontrolled hypertension is a global challenge, requiring a multidisciplinary team, close interval follow-up, and home blood pressure monitoring [50]. In adults with stage 2 hypertension, starting 2 medications is recommended, prioritizing single-pill combination therapy with 2 first-line agents [50]. Patients with resistant hypertension should be screened for underlying causes of secondary hypertension, such as obstructive sleep apnea and primary hyperaldosteronism [50].

7. Body fat

Obesity, a chronic condition and powerful CVD risk factor [51], impacts over 40% of adults in the US, with obesity-associated mortality rising (1.8 per 100,000 in 2010 to 3.1 per 100,000 in 2020) [5,52], highlighting a critical need for strategies to achieve a healthy body weight.

7.1. Assess for clinical obesity with BMI, weight circumference, and clinical complications

The Lancet Diabetes and Endocrinology Commission issued updated guidelines in January 2025 outlining how to diagnose clinical obesity, defined by BMI ≥40 or confirmation of excess body fat via waist circumference or direct fat measurement [53]. Based on this updated definition, obesity can be further classified into clinical obesity, with associated organ dysfunction or functional limitation, and preclinical obesity, without associated deficits [53]. To screen for excess body fat, BMI should be calculated and waist circumference should be measured using a flexible, non-stretchable tape measured at the level of the iliac crest [54]. Once obesity is diagnosed, screening for clinical complications of obesity through detailed history and physical examination is vital. Clinical complications include heart failure, obstructive sleep apnea, metabolic syndrome, hepatic steatosis, venous thromboembolism, and atrial fibrillation [53,55,56].

7.2. Target >5% weight loss using lifestyle, and selectively, use of GLP-1 agonists

Specific weight loss goals for clinical benefit are provided in the 2025 American Association of Clinical Endocrinology Consensus Statement [57]. For example, a 10% weight reduction reduces the risk for major adverse cardiovascular events and sleep apnea, while a 5 to 10% reduction may improve hepatic steatosis or stress incontinence [57]. Lifestyle modifications and individualized counseling are recommended to achieve and maintain weight loss [4,57]. Pharmacotherapy with incretin-based therapy may be beneficial for individuals with clinical obesity and CVD or high cardiovascular risk [57]. Glucagon-like peptide-1 receptor agonists (GLP-1RAs) are the primary option given the cardioprotective effects in patients with and without type 2 diabetes [18–20]. Importantly, primary care clinicians, endocrinologists, and cardiologists are key collaborators in initiation and dose titration of GLP-1RAs.

8. Cholesterol

Dyslipidemia is the leading direct driver of ASCVD, and prevalent in over 25% of Americans [58]. Both the degree of LDL-C elevation and duration of elevated LDL-C drive plaque formation [59] and lower LDL-C is better for longer [60].

8.1. Check lipid levels early and regularly to prevent and manage ASCVD risk

Lipid panels should be assessed around age 9–11, age 19–21, and at least every 5 years thereafter, and Lp(a) levels should be measured at least once in a person's lifetime. Both the 2026 ACC/AHA/Multisociety Guideline on the Management of Dyslipidemia [58] and the 2025 Focused Update of the 2019 European Society of Cardiology/European Atherosclerosis Society (ESC/EAS) Guidelines for the Management of Dyslipidemias promote this approach, with a shift towards earlier and individualized management [61]. For primary prevention, a stepwise approach of lifestyle modification and statin initiation, lipid panel recheck 4 to 12 weeks after starting therapy, and addition of other therapies if goals are not reached is recommended, with more rapid and intensified lipid-lowering strategy in high risk primary prevention or secondary prevention patients [61]. For patients with LDL-*C* ≥160 mg/dL, family history of ASCVD, and/or ≥10% 30-year risk of ASCVD, LLT should be strongly considered [58].

8.2. Stratify risk and intensify lipid-lowering using combination therapies for patients at high-risk or not reaching targets

Personalized algorithms are recommended to classify CVD risk and

guide pharmacotherapy initiation. The 2026 ACC/AHA/Multisociety Dyslipidemia Guideline promotes a CPR approach, focused on Calculating risk, Personalizing estimated risk with patient-specific risk enhancers, Reclassifying with CAC scores if uncertainty remains, and Reassessing recommended treatments. For example, patients with PREVENT™ 10-year ASCVD risk ≥5% and <10% are classified as intermediate risk and statin therapy should be discussed in a shared decision-making conversation after consideration of risk enhancers and commonly a coronary artery calcium scan [58]. For men age ≥40 and women age ≥45, CAC scoring is a safe and validated marker of risk, especially useful for reclassifying patients in whom lipid-lowering decision-making is unclear. For very high, high, and borderline-intermediate risk, target LDL-C goals <55, <70, and <100 mg/dL, respectively [58]. In adults age 40–75 with diabetes or CKD stage 3–4, LLT should be initiated regardless of LDL-C. Reflective of the link between inflammation and CVD, all patients living with HIV who are at least 40 years old should be prescribed statin therapy that does not interfere with their retroviral therapy [58,61]. Use of early combination therapies with high-intensity statin, ezetimibe, and proprotein convertase subtillsin/Kexin Type 9 inhibitors (PSK9i) can aggressively lower LDL-C and newer agents such as bempedoic acid can also be considered [58,61].

9. Cigarettes and alcohol

9.1. Assess and address substance use and readiness, particularly tobacco and alcohol at each visit

Tobacco use and exposure, including second-hand smoke and inhalation of nicotine products through electronic cigarettes or vaping, should be assessed at every visit, as cigarette smoke is a critical modifiable risk factor for the reduction of CVD. Alcohol use should also be reviewed, as excessive alcohol consumption, i.e. binge or heavy drinking, has negative CVD impacts, including systemic inflammation, endothelial dysfunction, and adverse cardiac remodeling [62]. For patients with ongoing tobacco and/or alcohol use, clinicians should assess cessation readiness at each visit, offer treatment options, including both behavioral and pharmacotherapy support, and connect patients with appropriate resources [63]. For patients who are not ready to quit, close follow-up and reassessment are critical. Preparation and planning may lead to increased cessation success, so Setting a quit date, Choosing a cessation method, Deciding on psychosocial support or pharmacotherapies, and Preparing for the Quit day are key and should be discussed in clinical visits [42].

9.2. Utilize evidence-based tools and therapies to support tobacco and alcohol cessation

Patients should be counseled on behavioral therapies and pharmacologic strategies that align with individualized goals and preferences. For both tobacco and alcohol cessation, cognitive behavioral therapy may be effective [64,65]. Motivational interviewing techniques are commonly used in combination, a strategy which reinforces behavior change through patient-centered counseling and relies on patients to identify their own motivation to change [63]. For patients with tobacco use interested in pharmacotherapies, first line options include nicotine replacement therapy (via nicotine patch, lozenge, gum, inhaler, or nasal spray), varenicline, or bupropion, which are all safe for patients with CVD [63]. Telephone quit lines, such as 1–800-QUIT-NOW are additional resources that can be utilized to assist in smoking cessation [66]. For patients interested in alcohol cessation pharmacotherapy, naltrexone and acamprosate are first line options, and disulfiram, gabapentin, or topiramate may also be considered [67].

10. Diabetes/CKM syndrome

10.1. Screen A1c, eGFR, and UACR, and personalize A1c targets

The American Diabetes Association recommends screening asymptomatic adults without risk factors for diabetes every 3 years beginning at age 35 years in patients without prediabetes or diabetes [68]. Risk factors include history of CVD or hypertension, physical inactivity, first-degree relatives with diabetes, and clinical conditions associated with insulin resistance [68]. While several screening laboratory tests for diabetes exist, hemoglobin A1c testing is typically the most convenient. Prediabetes is defined as hemoglobin A1c 5.7–6.4% or fasting glucose 100–125 mg/dL, whereas diabetes is defined as hemoglobin A1c ≥6.5% or fasting glucose ≥125 mg/dL [68]. The target hemoglobin A1c for most patients with diabetes is <7%, with an A1c goal of 6.5% for select patients with low risk of hypoglycemia and long life expectancy, and a less stringent goal of 8% for patients with significant comorbidities, limited life expectancy, or concerns that harms of treatment may outweigh the benefits [69]. In all patients with type 2 diabetes, a spot urine albumin-to-creatinine ratio (UACR) and estimated glomerular filtration rate (eGFR) should be assessed, regardless of treatment [70]. For patients with type 2 diabetes who have progressed to CKD, UACR and eGFR should be monitored 1–4 times yearly depending on CKD stage, with more severe CKD necessitating more frequent monitoring [70]. Initiation of either an angiotensin-converting enzyme inhibitor (ACEi) or angiotensin receptor blocker (ARB) is recommended for patients with moderate albuminuria (UACR 30–299 mg/g creatinine) and strongly recommended for patients with severe albuminuria (UACR ≥ 300 mg/g creatinine) and/or eGFR <60 mL/min/1.73 m^2 to delay further progression of CKD and reduce cardiovascular risk [70]. SGLT2i should be considered for patients with eGFR ≥20 mL/min/1.73 m^2 with or without the presence of type 2 diabetes to reduce progression of CKD and improve cardiovascular outcomes [71].

10.2. Identify CKM stage and encourage cardioprotective therapies, including SGLT2 inhibitors and GLP-1 agonists

The increasing prevalence of diabetes and obesity has resulted in a rising recognition of CKM syndrome, defined as a systemic disorder reflecting the connections between CKD, the cardiovascular system, and metabolic disease, resulting in risk factors for CVD or clinical CVD [26]. To improve population-level CKM health, the AHA released a Presidential Advisory defining a CKM staging system, ranging from Stage 0 (no risk factors) to Stage 4 (clinical CVD in CKM syndrome) [26]. Clinicians should identify CKM stage and use PREVENT™ for stratification of risk and identification of targeted therapies based on risk across each stage [14]. Stage 0 through 3 CKM focuses on CVD prevention, while stage 4 CKM focuses on CVD management. Notably, this staging system also identifies subclinical CVD (stage 3), through incorporation of imaging tools including CAC scoring and echocardiogram abnormalities. For patients in CKM stage 2 (those with established risk factors but without CVD), the focus should be placed on treating metabolic risk factors and CKD, while preventing progression to Stage 3 or 4 (those with progression to subclinical or clinical CVD, respectively). Given the complex physiology underlying the development of CKD and diabetes, combination cardioprotective therapies targeting both type 2 diabetes and CKD progression, such as SGLT2i and finerenone, can improve cardiovascular risk and should be prioritized [17,72]. GLP1-RAs should also be considered especially for patients with hemoglobin A1c ≥9% or elevated BMI ≥35 kg/m^2 [26]. In patients with Stage 3 or 4 CKM, the focus should be on prevention of CVD progression, as well as management of CKD and metabolic risk factors.

11. Diet

11.1. Encourage mediterranean, DASH, or plant-based diets

A diet rich in whole grains, lean proteins, fruits, and vegetables is beneficial in preventing CVD [73]. In contrast, dietary intake of processed foods, refined carbohydrates, and saturated fats are associated with increased CVD risk, obesity, and inflammation [74]. Dietary approaches such as the Mediterranean and plant-based diets have demonstrated superiority in preventing major cardiovascular events [75–77]. The Mediterranean diet, plant-based diets, and DASH diet have also demonstrated improvement in glycemic control, reduction in cholesterol, weight loss, and decrease in CVD risk [4]. The DASH diet in particular is also an effective intervention for lowering blood pressure, with an expected mean reduction in SBP of 5 to 8 mmHg in patients with hypertension [50].

11.2. Focus on portion control and calorie deficit in weight-loss discussions

In discussions with patients, healthy eating and lifestyle changes should be emphasized and framed in a positive light. A 500 to 750 kcal/day deficit is recommended to promote weight loss in patients with obesity [4,55]. Types of food consumed and portion control also matter. For example, clinicians can recommend filling half of the plate with vegetables, a quarter with protein, and a quarter with carbohydrates. There is not one ideal diet, but instead the best choice is a healthy food pattern to which a patient will adhere over time [57]. Additionally, barriers contributing to nutritional insecurity, such as financial strain, digital isolation, homebound status, and transportation disadvantages should be addressed [78].

12. Exercise

12.1. Aim for >150 min/week of moderate/brisk activity or >75 min/week of vigorous activity

Lack of regular physical activity is a significant risk factor for the development of CVD [79,80] and strategies are needed to improve physical activity in adults [81]. Clinicians should promote regular moderate to brisk physical activity at each visit. Current guidelines recommend at least 150 min per week of moderate intensity aerobic exercise or 75 min per week of vigorous activity [4]. For individuals unable to meet these goals, physical activity should still be encouraged, as ASCVD reduction benefits occur in a dose-response fashion [82]. Implementation of physical activity tracking technologies may be a successful strategy in meeting these goals [83]. For patients with a recent cardiovascular event, chronic coronary artery disease, and/or heart failure, cardiac rehabilitation should be encouraged, and is an essential, multidisciplinary strategy which improves mortality, functional status, and quality of life [84,85].

12.2. Engage in resistance training at least 2x per week

In addition to aerobic activity, resistance training at least twice per week is recommended to improve functional status, glycemic control, lipids, and body composition [86]. Resistance training, which involves muscular contraction against an external force, not only maintains or improves strength, but is also associated with lower risk of CVD morbidity and mortality [87]. Resistance training does not need to be time intensive, with at least two 30-minute sessions per week demonstrating benefit [87]. Resistance training also improves nontraditional CVD risk factors, such as enhanced sleep quality and improved mood [88,89].

13. Extend healthspan

With global population aging, extending cardiovascular healthspan (years spent in good health) is a central goal of Preventive Cardiology [90]. Exercise, healthy diet, and lifelong lipid and blood pressure lowering, for example, attenuate vascular aging processes and support vascular resilience [91]. Moreover, the evidence in older populations is growing to guide patient-centered prevention to enhance longevity and quality of life [31].

13.1. Assess physical and cognitive function in older adults with cardiovascular risk factors or disease

Physical and cognitive function intersect bidirectionally with cardiovascular health [29]. These domains can be evaluated in busy outpatient clinics using brief (<3-minute) validated tools such as the Katz Activities of Daily Living Index and Duke Activity Status Index for function [92], Clinical Frailty Scale or FRAIL Scale for frailty [93,94], and basic 2-item screens, Mini-Cog, or AD8 for cognition [95]. Biologic aging processes and cardiovascular risk factors contribute to disability, frailty, and cognitive impairment, and these geriatric conditions strongly influence cardiovascular outcomes and the benefit-risk profiles of cardiovascular medications and procedures [29,95].

13.2. Personalize prevention throughout the life course with Life's Essential 8, shared decision-making, cardiac rehab, and multidisciplinary care addressing health-related social needs

Lifestyle interventions, particularly aerobic and resistance exercise, Mediterranean diet, adequate sleep, and smoking cessation, directly target cardiovascular aging processes and synergistically improve physical, cognitive, and cardiovascular health [42,94–97]. For preventive medications and procedures, shared decision-making using a Consider, Listen, Decide framework and decision aids when possible, should incorporate physical and cognitive status [31,98]. For example, maintaining blood pressure <130/80 mmHg is protective against cognitive impairment and orthostatic hypotension as well as other adverse cardiovascular events [50]. However, in the setting of moderate-severe frailty, disability, or cognitive impairment, evidence for net benefit is more limited and pharmacokinetics vary, supporting a thoughtful, individualized approach with close home blood pressure monitoring [93,99]. Cardiac rehabilitation improves cardiovascular healthspan and longevity yet remains underused, although covered by Medicare for patients with stable angina, chronic heart failure with reduced ejection fraction (left ventricular ejection fraction [LVEF] <35%), and those post-myocardial infarction, revascularization, valve repair/replacement, or transplant [100]. As social drivers of health continue to accelerate cardiovascular aging, the clinical and social complexity of the aging cardiovascular patient population is increasing and multidisciplinary care teams that assess and address cardiovascular, geriatric, and health-related social needs are essential to optimize cardiovascular health and independence [78].

14. Failure: Heart failure

14.1. Identify patients in stage B pre-heart failure and prevent progression to stage C using neurohormonal and cardiometabolic therapies

Asymptomatic patients with structural heart disease, evidence of increased filling pressures, or increased levels of natriuretic peptide or cardiac troponin as well as risk factors for heart failure are considered to have stage B pre-heart failure [101]. Identification and treatment of patients with pre-heart failure is a key opportunity to delay or prevent transitioning to heart failure through prompt, complete preventive assessment and optimization [119]. The 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure recommends ACEi/ARBs and

beta blockers for patients with LVEF ≤40% to prevent symptomatic heart failure (stages C-D) and reduce mortality [101]. Comorbidities should also be assessed and treated promptly given the overlap between hypertension, diabetes, and obesity with CKM [26]. For example, clinicians should consider ARBs or mineralocorticoid antagonists (MRAs) for patients with hypertension or CKD, whereas SGLT2 and GLP1-RAs should be discussed for patients with diabetes, CKD, and/or obesity.

14.2. Treat stage C heart failure with GDMT to prevent hospitalizations and mortality

Stage C heart failure is defined as structural heart disease with symptoms of volume overload or hypoperfusion [101]. For patients with stage C HF, the pillars of guideline directed medical therapy (GDMT) are recommended to reduce mortality and hospitalizations. Specifically, SGLT2i (empagliflozin or dapagliflozin) and MRA (spironolactone, eplerenone, or finerinone) are recommended for all patients with HF, with beta blockers (sustained release metoprolol, carvedilol, or bisoprolol) and angiotensin receptor-neprilysin inhibitors, ACEi, or ARB being indicated for HFrEF as well [101]. For most patients, a rapid GDMT initiation and up-titration strategy is supported [102,103]. Additionally, diuretics are recommended for patients with fluid retention to improve congestive symptoms and prevent hospitalization [101]. Multidisciplinary care has also been shown to improve survival [104].

15. Fibrillation: Atrial fibrillation

Atrial fibrillation is the most common cardiac arrythmia with rising prevalence despite modifiability of risk factors such as hypertension, tobacco use, and obesity [105–107]. Optimization of these risk factors is foundational to reduce the morbidity and mortality associated with atrial fibrillation. In particular, stroke remains a devastating complication of atrial fibrillation and clinicians should continue to assess stroke risk, target modifiable risk factors, treat comorbidities, and manage symptoms [108].

15.1. Initiate oral anticoagulation for CHA_2DS_2-VASc score ≥2–3

For patients diagnosed with atrial fibrillation, thrombotic event risk stratification should be performed using a validated risk scores [108–111], such as CHA_2DS_2-VASc. Anticoagulation is generally not recommended in patients with non-valvular atrial fibrillation and CHA_2DS_2-VASc scores of 0 or 1 in males and females, respectively [108]. For males with CHA_2DS_2-VASc ≥2 or females with CHA_2DS_2-VASc ≥3, anticoagulation is recommended due to increased thrombotic risk [108]. For patients with intermediate risk (males with CHA_2DS_2-VASc score of 1 and females with CHA_2DS_2-VASc of 2), further consideration of individual risk factors, such as hypertension control and atrial fibrillation burden, can provide an additional personalized risk assessment [108]. Direct oral anticoagulants are recommended over warfarin for patients without moderate to severe rheumatic mitral stenosis or mechanical heart valves given decreased risks of stroke and bleeding [108]. For patients with a prior severe bleeding event or risk of serious bleeding due to fall risk, left atrial appendage occlusion is reasonable [108,112].

15.2. Consider early rhythm control, especially for adults with early atrial fibrillation and/or coexisting heart failure

Large trials and meta-analyses have demonstrated that a rhythm control strategy, which involves antiarrhythmic medications and/or catheter ablation, is favored over rate control alone, especially in patients with heart failure [113–115]. An early rhythm control strategy and restoration of sinus rhythm is favored to prevent adverse atrial remodeling as well as incident heart failure, stroke, and atrial fibrillation-related mortality [114,116,117]. In patients with reduced left ventricular function and persistent atrial fibrillation, a trial of rhythm control is recommended to assess whether atrial fibrillation is contributing to the reduced ventricular function [108]. Choice of rhythm control therapy should be individualized, with early electrophysiology referral recommended for consideration of catheter ablation and advanced antiarrhythmic agents such as dofetilide.

16. Conclusion

The ABCs of CVD Prevention is a practical tool for clinicians to incorporate contemporary evidence and guidelines into practice (Table 1). This framework can inform clinical decision making and individualize patient-centered care. We highlight important clinical updates and recognize that many questions in CVD prevention exist and opportunities for future research are vast:

- **Assess Risk:** With clear guideline recommendations that incorporate PREVENT™ estimated risk, the next step is improving nation-wide dissemination and implementation into clinical care and electronic medical record systems.
- **Antiplatelets:** Comorbidities such as atrial fibrillation and increased bleeding risk create additional complexity in antiplatelet management. Further studies are needed to determine and confirm optimal selection and durations of therapy, including dual antiplatelet therapy and antiplatelet plus oral anticoagulant therapy for secondary prevention and chronic coronary disease.
- **Blood Pressure:** Nearly 80% of adults with hypertension have blood pressures above guideline-recommended targets. Improving national rates of blood pressure control will require improved implementation of team-based care, optimal monitoring, and single-pill combination use.
- **Body Fat:** As the clinical definition of obesity continues to be refined and the prevalence of obesity increases, developing universal physiology- and evidence-based obesity measures will be crucial to guide optimal population-level use of GLP1-RAs and other weight loss pharmacotherapies.
- **Cholesterol:** Recent updates in lipid guidelines support earlier LDL-C lowering and for longer, which will require a shift in management towards more intensive and earlier treatment with combination therapy. As novel and more intensive LLT, such as PSK9i, become more affordable and as novel therapies targeting Lp(a) are potentially introduced, a new generation of evidence will be required to guide the optimal use of these therapies in clinical practice.
- **Cigarettes:** As marijuana use and vaping become more prevalent with evolving regulations, understanding the impacts of these substances on cardiovascular health will be crucial in developing optimal patient guidance.
- **Diabetes/CKM Syndrome:** As GLP1-RAs and other cardiometabolic therapies evolve, deeper understanding and implementation of CKM syndrome staging and its implications for optimization of cardiometabolic therapies will grow.
- **Diet:** With the 2025–2030 Dietary Guidelines for Americans both overlapping and diverging from Mediterranean and DASH dietary patterns [118], further study is needed to elucidate the cardiovascular impacts of the new national recommendations.
- **Exercise:** Suboptimal rates of physical activity in the US continue to contribute to high rates of CVD. Emerging wearable technologies may motivate individuals to engage in physical activity and provide more granular data to develop tailored physical activity recommendations to improve cardiovascular health across the nation.
- **Extend Healthspan:** As the global population ages, the development of optimal clinical models and workflows integrating physical and cognitive assessments and interventions into clinical practice will be required to meet the needs of the geriatric population.
- **Heart Failure:** With the CKM syndrome epidemic and population aging, the prevalence of stage B heart failure will continue to rise, necessitating prompt recognition of patients in this stage and

initiation of appropriate therapies. Future research is required to guide therapy for patients with stage B heart failure, especially for those with overlapping comorbidities, such as obesity or diabetes.

- **Atrial Fibrillation:** Ongoing studies investigating utility of novel screening methods including wearables, benefits and risks of innovative anticoagulants such as Factor XI inhibitors, and impacts of risk factor control will shape the management of atrial fibrillation in coming years.

CRediT authorship contribution statement

Lea R. Goren: Conceptualization, Writing – original draft, Writing – review & editing. **Allison W. Peng:** Writing – review & editing, Visualization. **Alexander C. Razavi:** Writing – review & editing, Visualization. **Michael J. Blaha:** Writing – review & editing. **Roger S. Blumenthal:** Conceptualization, Writing – review & editing. **Aaron L. Troy:** Conceptualization, Supervision, Visualization, Writing – review & editing.

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Supplementary materials

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