Menopause, cardiovascular health and the role of hormone therapy

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Background
The menopause transition marks a period of accelerated cardiovascular risk in women, highlighting the importance of opportunistic screening and intervention in midlife primary care.

Objective
This article summarises current evidence linking menopause with cardiovascular disease (CVD) and appropriate interventions, including lifestyle optimisation and menopausal hormone therapy (MHT).

Discussion
The midlife acceleration of CVD risk is driven by distinct cardiometabolic and vascular changes, with ovarian ageing independently associated with adverse lipid profiles, fat redistribution, vascular dysfunction and metabolic syndrome. Early, premature, surgical and symptomatic menopause further amplifies risk. Lifestyle optimisation and lipid-lowering therapy remain powerful strategies to mitigate these changes. MHT, although not recommended solely for primary or secondary CVD prevention, may be offered for symptom management and bone protection in appropriately selected women, and in cases of premature, early or surgical menopause. Midlife consultations provide a unique window for general practitioners to opportunistically risk stratify, initiate preventive measures and support informed MHT decision making, thereby shaping long-term cardiovascular trajectories.

CARDIOVASCULAR DISEASE (CVD) is the leading cause of death in Australians, yet important sex-specific differences exist.[1] For women, risk accelerates in the fifth decade of life, 7–10 years later than in men, which coincides with the menopause transition.[2,3]

Accumulating evidence links oestrogen deficiency with adverse cardiometabolic and vascular changes, including lipid derangement, fat redistribution, increased blood pressure, insulin resistance and vascular dysfunction.[3] For general practitioners (GPs), midlife consultations provide a critical opportunity to identify at-risk women and implement preventive strategies, improving long-term cardiovascular outcomes.

During this period, GPs are often tasked with managing bothersome vasomotor symptoms and guiding shared decision making surrounding menopausal hormone therapy (MHT). This can be challenging, particularly given the enduring fears from the 2002 Women's Health Initiative (WHI) study reporting cardiovascular and breast cancer risks, despite subsequent analyses demonstrating MHT's safety and benefit in appropriately selected women.[4] Clear, evidence-based and sex-specific guidance is crucial to support GPs in optimising women's cardiovascular health across the menopause transition.

Aim

This article explores the complex relationship between menopause and CVD. It summarises current evidence on the impact of MHT on cardiovascular risk and outlines strategies for risk assessment and prevention in primary care, recognising the pivotal role GPs have in empowering women and supporting their cardiovascular health.

Menopause and cardiovascular physiology

Menopause is a complex physiological transition with various manifestations among midlife women and corresponding implications for cardiovascular risk (Table 1). When compared with the mean age of natural menopause (51 years),

women who experience premature menopause (age <40 years) carry a 55% higher risk of CVD, whereas those with early menopause (age 40–44 years) face a 30% increased risk.[5] Current modelling demonstrates a 3% rise in incident CVD for each year menopause occurs earlier, reflecting the loss of endogenous oestrogen's cardioprotective effects.[6] This elevated risk encompasses multiple cardiovascular outcomes, including coronary artery disease (CAD), heart failure (HF) and atrial fibrillation (AF), and is associated with a 19% increase in CVD mortality.[7–9]

Surgical menopause incurs an even greater CVD risk, especially when performed earlier, with a 5% increase in incident CVD for each 1-year decrease in age of menopause.[6] Early initiation of MHT to replace oestrogen until the mean age of natural menopause mitigates many of these adverse outcomes, highlighting the importance of timely diagnosis and management of early, premature and surgical menopause in general practice.[3,4,9]

Vasomotor symptoms, affecting up to 80% of midlife women, are associated with sympathetic overactivity and an unfavourable cardiometabolic profile, with substantial increases in subclinical CVD and coronary heart disease (CHD) events.[2,3] Risk rises with frequent and persistent symptoms, as outlined in Table 1.[10] Other menopausal features, including sleep disturbance and depression, are similarly linked to adverse cardiovascular biomarkers and outcomes.[3]

The physiological processes responsible for the acceleration of CVD during the menopause transition are multifactorial, as summarised in Table 2. Menopause drives an adverse lipid profile shift whereby total cholesterol and low-density lipoprotein cholesterol (LDL-C) increases and the protective anti-atherogenic effect of high-density lipoprotein cholesterol (HDL-C) weakens.[2,3] Although overall midlife weight gain is largely age related, menopause promotes central and visceral fat redistribution, corresponding to subclinical atherosclerosis and likely contributing to increased sleep apnoea and metabolic syndrome prevalence.[3,11–13]

Oestrogen deficiency accelerates vascular ageing, characterised by endothelial dysfunction, arterial stiffening, atherosclerosis progression and coronary microvascular dysfunction.[2,3,14] Menopause is also associated with adverse cardiac remodelling, including increased pericardial fat deposition, left ventricular hypertrophy and diastolic dysfunction.[3,15] Both cardiac remodelling and coronary microvascular dysfunction are hypothesised to contribute

Table 1. Menopause features and associated cardiovascular risk

Feature	Cardiovascular disease (CVD) risk	Clinical takeaway for general practitioners
Primary ovarian insufficiency (POI, aged <40 years) and early menopause (EM, aged <45 years)	↑ CVD in a dose-dependent pattern graded by age at natural menopause (POI: ↑ 55%, EM: ↑ 30%)[5,6] Includes: ↑ coronary artery disease (CAD), atrial fibrillation (AF), heart failure (HF), aortic stenosis, venous thromboembolism (VTE) and ischaemic stroke[8,9]	Consider timing of menopause as a 'risk-enhancing' factor in Australian CVD risk (AusCVDRisk) calculation, helping to guide decision making, particularly for women at borderline or intermediate risk [23] Encourage lifestyle optimisation and recommend menopausal hormone therapy (MHT) until mean age of natural menopause, in the absence of contraindications
Surgical menopause	↑ CVD in a dose-dependent pattern graded by age at surgery (35–39 yrs: ↑ 91%; <35 yrs: ↑ 155%)[6] Includes: ↑ mitral regurgitation, VTE, HF and CAD[9]	Inform women considering elective bilateral oophorectomy of the increased CVD risk and incorporate this into the risk–benefit discussion After surgery, discuss MHT if there are no contraindications, especially in the case of early surgical menopause, and reinforce primary prevention strategies
Vasomotor symptoms (VMS)	↑ CVD events, especially with more frequent symptoms (≥6 days/2 weeks: ↑ 51%) and persistent symptoms (>33% of up to 16 attended visits over 22 years, corresponding to roughly 6 years of frequent VMS: ↑ 77%)[10] ↑ Autonomic dysfunction, lipid derangement, insulin resistance, hypertension and coronary artery calcification[2,3]	Address symptom burden: MHT is the most effective treatment; alternative nonhormone options include cognitive behavioural therapy (CBT), weight loss or other medications (including selective serotonin reuptake inhibitors, gabapentin, fezolinetant, oxybutynin)[21] Calculate AusCVDRisk[22]
Menopause-associated depression and anxiety	↑ Major depressive episodes and anxiety symptoms during the menopause transition[3] Depression is associated with subclinical CVD (↑ coronary artery calcium scores) and is an independent predictor of both CVD and all-cause mortality[3]	Screen, consider pharmacotherapy if symptoms fail to improve with MHT and behavioural modification, and consider referral for CBT or psychotherapy There is some evidence that MHT has antidepressant effects in perimenopausal women[2]
Sleep disturbance	↑ Metabolic syndrome and vascular dysfunction (carotid plaque, aortic calcification and arterial stiffness)[3]	Screen for sleep quality and sleep apnoea, address modifiable risk factors, counsel on sleep hygiene and lifestyle modifications, treat vasomotor symptoms if contributing, and recommend evidence-based management for insomnia such as CBT for insomnia (CBT-i)[38]

to the higher prevalence of heart failure with preserved ejection fraction (HFpEF) in postmenopausal women.[14,15]

Hypertension, the leading modifiable cardiovascular risk factor, is more prevalent in postmenopausal women, although it is not consistently linked with menopause itself.[2,3] Insulin resistance accelerates during and after menopause, with earlier menopause and severe vasomotor symptoms identified as sex-specific risk factors for progression to type 2 diabetes mellitus (T2DM).[16–18]

Prevention and risk reduction

Lifestyle habits often deteriorate during the menopause transition, contributing to increased CVD risk. Oestrogen deficiency is linked to several barriers to healthy behaviours, including bothersome vasomotor symptoms, hyperphagia, depression and disrupted sleep, which can reduce physical activity and overall energy expenditure.[2]

Research indicates that 82% of CHD events in women could be avoided through lifestyle modification, including smoking cessation, healthy eating, regular exercise and moderation of alcohol consumption.[19] Strong adherence to these healthy behaviours has been shown to reduce CVD risk by 23% among all women and 52% in premature menopause, and weight loss is an evidence-based recommendation for the management of vasomotor symptoms.[20,21] Although smoking abstinence demonstrates the strongest inverse association, dietary and exercise interventions also slow the progression of dyslipidaemia, weight gain, glucose intolerance and subclinical atherosclerosis, with the potential to reverse components of metabolic syndrome during midlife.[3]

Routine CVD screening in midlife women is recommended (Table 3). In general practice, Medicare Benefit Schedule (MBS) item 699 (Heart Health Check) and item 695 (Menopause Health Assessment) can facilitate thorough assessment and risk mitigation counselling.

The Australian CVD risk (AusCVDRisk) calculator estimates 5-year cardiovascular risk and guides initiation of lipid-modifying and blood pressure–lowering therapies for primary prevention without incorporating sex-specific factors.[22] The American Heart Association recommends taking into account female 'risk-enhancing factors', including premature menopause, pre-eclampsia, preterm delivery, chronic inflammatory diseases (rheumatoid arthritis, lupus) or female family history of atherosclerotic CVD under the age of 65 years. These factors can further risk stratify and help guide investigations or interventions, such as a coronary artery calcium scoring or medication initiation, particularly in those with borderline or intermediate estimated risk.[23]

Menopausal hormone therapy

The relationship between MHT and CVD is complex. Historically, MHT was widely prescribed for CVD prevention until the 2002 WHI trial reported adverse cardiovascular events and increased all-cause mortality, prompting a sharp decline in uptake.[4]

Table 2. Cardiometabolic and vascular changes across the menopause transition

Cardiometabolic and vascular changes	Key physiological changes across menopause
Dyslipidaemia	↑ Total cholesterol, low-density lipoprotein cholesterol (LDL-C) and apolipoprotein B markedly within the 1 year before and after the final menstrual period (FMP)[3] ↓ High-density lipoprotein cholesterol (HDL-C) antiatherogenic function, with increased HDL-C associated with greater atherosclerosis progression after menopause[3]
Fat redistribution	↑ Central adiposity across the menopause transition, associated with subclinical atherosclerosis (visceral adipose tissue increased by 8.2% annually within 2 years preceding and 5.8% annually after the FMP, with no significant change prior)[11]
Sleep apnoea	↑ Obstructive sleep apnoea prevalence (21% premenopausal to 47% post menopause)[12]
Metabolic syndrome	↑ Cluster of insulin resistance, abdominal obesity and dyslipidaemia (↑ 254% post vs prior to menopause)[13]
Vascular dysfunction	↑ Atherosclerosis progression rates and arterial stiffness (↑ 7.5% within 1 year of the FMP)[3] ↑ Coronary microvascular dysfunction, rendering postmenopausal women more susceptible to heart failure with preserved ejection fraction (HFpEF)[14]
Cardiac remodelling	↑ Pericardial fat, serving as an independent predictor for cardiovascular disease[3] ↑ Left ventricular and atrial remodelling and diastolic dysfunction, contributing to increased HFpEF[15]
Hypertension	↑ Blood pressure resulting in higher hypertension prevalence when compared with age-matched men aged >65 years[2,3] Shift towards sympathetic autonomic predominance[39]
Insulin resistance	↑ Impaired glucose tolerance by 6% per year after menopause[16] Type 2 diabetes mellitus sex-specific risk factors include earlier menopause (early menopause: ↑ 12%; primary ovarian insufficiency: ↑ 53%)[17] and vasomotor symptoms (any: ↑ 18%; severe: ↑ 48%)[18]

Table 3. General practitioner recommendations for optimising cardiovascular health and preventing disease[A]

Risk factor	Assessment and screening	Targets and recommendations
Physical activity	Assess physical activity (frequency, duration and intensity) and sedentary behaviour every 2 years	• Aim for regular, sustainable physical activity, ideally ≥2.5 hours of moderate-intensity or ≥1.25 hours of vigorous aerobic exercise weekly (or an equivalent combination)
Weight	Opportunistically and sensitively calculate body mass index (BMI) and measure waist circumference in adults who are not pregnant and do not have an eating disorder, maintaining awareness of weight stigma	• Recommend lifestyle modification • Target BMI <25 kg/m^2 (European), <23 kg/m^2 (Asian, Middle Eastern, Black African or African Caribbean) • Target waist circumference: females <80 cm
Diet	Opportunistically assess diet including fruit, vegetable, fat and salt intake	• Follow the Australian Dietary Guidelines: plenty of vegetables, fruit and wholegrains; a variety of protein-rich foods; unflavoured dairy; and healthy fats/oils • Limit saturated fat, added salt and added sugars
Smoking	Opportunistically assess smoking status, amount, dependence and readiness to quit	• Recommend cessation • Offer behavioural intervention referral combined with approved pharmacotherapy where clinically indicated
Alcohol	Screen every 2 years for unhealthy alcohol use	• Limit intake to ≤10 standard drinks per week and ≤4 on any one occasion • Provide behavioural counselling interventions for risky or hazardous use
Dyslipidaemia	Measure lipids and apply Australian CVD risk (AusCVDRisk) calculator in all patients aged 45–79 years (aged 35–79 years if living with diabetes, aged 30–79 years for Aboriginal and Torres Strait Islander people). Repeat 5-yearly if low risk or 2-yearly if higher risk/near thresholds, unless risk factors worsen. Consider coronary artery calcium score if treatment decisions remain uncertain.[22]	• Recommend lifestyle modification • Initiate lipid-modifying therapy according to AusCVDRisk.[41] • Recommended low-density lipoprotein cholesterol (LDL-C) targets:[42] – <2 mmol/L for primary prevention – <1.8 mmol/L for secondary prevention, although some recent international guidelines recommend a lower LDL-C target (<1.4 mmol/L) in the secondary prevention setting • Aim for ≥50% LDL-C reduction in secondary prevention and ≥35% reduction in primary prevention[41] • Statins are the first-line treatment; use additional agents if LDL-C targets are not met
Hypertension	Opportunistically measure blood pressure (BP) in all adults, consider secondary causes and white coat hypertension, and perform ambulatory measurement as appropriate	• Recommend lifestyle modification • Individualise treatment targets on the basis of comorbidities; aim for BP ≤130/85 mmHg in patients at normal risk[43] • Initiate stepwise antihypertensive pharmacotherapy for primary prevention according to AusCVDRisk recommendations[22]
Diabetes	General population: use Australian Type 2 Diabetes Risk (AUSDRISK) tool every 3 years if aged >40 years without risk factors High risk: perform fasting blood glucose (FBG) or HbA1c every 3 years (or every 12 months if previous impaired glucose tolerance or for Aboriginal or Torres Strait Islander people) Perform oral glucose tolerance test if results equivocal	• Recommend lifestyle modification • Initiate stepwise pharmacotherapy if glycaemic targets are not met after 3 months (metformin used as first-line therapy) • Target HbA1c ≤7%, FBG 4–7 mmol/L and 2-hour postprandial blood sugar level 5–10 mmol/L, without hypoglycaemic episodes, for most non-pregnant adults[44] • More stringently target HbA1c ≤6.5% for people with short disease duration, long life expectancy and no significant CVD[44] • Screen for complications
Obstructive sleep apnoea (OSA)	Screen symptomatic individuals (eg daytime sleepiness, disrupted sleep) and high-risk groups (eg male, aged >50 years, postmenopausal women, overweight, excessive alcohol intake, smoking) using a sleep questionnaire (eg Epworth Sleepiness Scale, OSA50, STOP-Bang Questionnaire), confirm with diagnostic sleep study and refer to sleep specialist as required[45]	• Recommend lifestyle modification (weight loss and alcohol reduction) • Initiate continuous positive airway pressure therapy for symptomatic moderate-to-severe OSA or mild OSA with significant hypoxaemia and/or excessive daytime sleepiness[45] • Consider alternative therapies, including positional therapy or mandibular advancement splints in mild or moderate cases, or surgery if indicated[45] • Assess driving safety and advise as per Austroads guidelines and screen for complications

[A] Recommendations are based on The Royal Australian College of General Practitioners' *Guidelines for preventive activities in general practice* (Red Book),[40] unless otherwise specified.

Timing of commencement

Commencing MHT aged <60 years or <10 years since menopause onset

Retrospective age-stratified analysis of the WHI data gave rise to the timing hypothesis: initiation of MHT in women within 10 years of the final menstrual period (FMP) or under 60 years of age does not lead to increased cardiovascular risk.[24]

This concept has since been supported by landmark trials including the Danish Osteoporosis Prevention Study, Early Versus Late Intervention Trial With Estradiol, and Kronos Early Estrogen Prevention Study.[4] As summarised in a 2015 Cochrane review, initiation of oral MHT (oestrogen, with or without progestogen) within 10 years of menopause onset lowers overall mortality by 30% and coronary artery disease by roughly half without significantly increasing stroke risk.[25] However, venous thromboembolism (VTE) risk remains elevated (Table 4).

Commencing MHT aged >60 years or >10 years after menopause onset

In contrast, initiating oral MHT more than 10 years after menopause onset carries an increased risk of stroke and VTE. However, the 2015 Cochrane review and 2019 and 2020 meta-analyses demonstrated a null effect on all-cause mortality and CHD.[25–27]

Although there is a paucity of randomised controlled trials, observational studies have consistently shown lower VTE risk with transdermal formulations.[3,4] Transdermal oestrogen might also lower stroke risk, although more evidence is required.[28]

Relationship with cardiovascular disease

Coronary heart disease

Oestrogen may exert plaque-destabilising effects in the context of advanced atherosclerosis.[4] Hence, established coronary or peripheral artery disease is generally considered a contraindication for MHT.[4] Ideally, comorbid conditions such as hypertension, diabetes and hyperlipidaemia should be controlled before initiation, and transdermal formulations are preferred in such cases.[4]

However, balancing multiple cardiovascular risk factors in the absence of established disease can be challenging in women experiencing bothersome symptoms. A risk stratification tool has been developed to guide MHT use in women at low (<5%), intermediate (5–10%) or high (≥10%) CVD risk based on the American 10-year Atherosclerotic Cardiovascular Disease (ASCVD) calculator.[4] The AusCVDRisk calculator could be used similarly to support decision making (Table 5).[22]

Venous thromboembolism

A personal history of VTE is considered a contraindication for oral MHT on the basis of the multitude of studies demonstrating increased thromboembolic risk.[9,29] Women experiencing significant bothersome symptoms with a history of provoked VTE

Table 4. Summary of findings from 2015 Cochrane review[25]

	Commencing oral MHT aged <60 years or <10 years since menopause onset	Commencing oral MHT aged >60 years or >10 years after menopause onset
Death (all causes)	RR 0.70 (95% CI: 0.52–0.95)	RR 1.06 (95% CI: 0.95–1.18)
Coronary heart disease (death from cardiovascular causes and non-fatal myocardial infarction)	RR 0.52 (95% CI: 0.29–0.96)	RR 1.07 (95% CI: 0.96–1.20)
Stroke	RR 1.37 (95% CI: 0.80–2.34)	RR 1.21 (95% CI: 1.06–1.38)
Venous thromboembolism	RR 1.74 (95% CI: 1.11–2.73)	RR 1.96 (95% CI: 1.37–2.80)

CI, confidence interval; MHT, menopausal hormone therapy; RR, risk ratio.

Table 5. Menopausal hormone therapy (MHT) decision-making tool using the Australian cardiovascular disease risk (AusCVDRisk) score and years since menopause onset[4,22]

AusCVDRisk score	<10 years since menopause onset	≥10 years since menopause onset
Low (<5%)	MHT appropriate	Consider alternatives MHT acceptable with individualised shared decision making
Intermediate (5–10%)	MHT acceptable Preference transdermal formulations in the presence of cardiovascular or thromboembolic risk factors (including diabetes, hypertension, hyperlipidaemia, obesity, metabolic syndrome)	Consider alternatives Persistent severe vasomotor symptoms warrant individualised shared decision making
High (≥10%)	Consider alternatives Persistent severe vasomotor symptoms warrant individualised shared decision making	Avoid systemic MHT Persistent severe vasomotor symptoms warrant individualised shared decision making

or thrombophilia could be further evaluated for appropriateness of transdermal MHT under haematologist guidance.[4] Women with a family history of VTE should undergo a thrombophilia screen prior to being prescribed MHT. For individuals with thromboembolic risk factors, including obesity, hyperlipidaemia or advanced age, low-dose transdermal oestrogen (<50 µg/day), combined with micronised progesterone if required, appears to be the safest choice.[4,29]

Stroke

Stroke risk increases with MHT use, and prior history of a cerebrovascular event is considered a contraindication.[4,9]

Spontaneous coronary artery dissection

There is a presumed association between spontaneous coronary artery dissection (SCAD) and female sex hormones, with MHT posed as a potential SCAD trigger. Therefore, experts recommend avoiding systemic MHT initiation or continuation in SCAD survivors where possible, while recognising the need for individualised decision making.[30]

Alternative hormone formulations

Tibolone is increasingly becoming a popular alternative to traditional MHT because of its oestrogenic, progestogenic and androgenic properties, which may improve libido. A Cochrane review found no increased risk of cardiovascular and VTE events, although much of the evidence was considered low quality.[31] There is conflicting evidence regarding increased stroke risk, particularly in women aged over 60 years.

A 1% testosterone cream (AndroFeme 1%, Lawley Pharmaceuticals) has more recently become available for the treatment of hypoactive sexual desire dysfunction in postmenopausal women. Evidence on the long-term CVD effects of testosterone therapy is limited.[32] Therefore, it should be used with caution in those with established or at high risk of CVD.

Duration of therapy

Among women who initiate MHT within 10 years of menopause onset, extended-duration therapy may be appropriate for persistent bothersome vasomotor symptoms or bone protection.[4,9] This must be balanced against the progressive increase in breast cancer risk, particularly with combined oestrogen and progesterone therapy.[9] Frequent vasomotor symptoms persist for 7.4 years on average; hence, it is reasonable to discuss lowering or discontinuing hormone therapy after several years of use.[4,9]

Although the WHI reported neutral cardiovascular and thrombotic risk following MHT cessation, subsequent observational data suggested a possible early increase in cardiac and stroke mortality within the first year. Ongoing clinical surveillance and cardiovascular risk optimisation remain crucial after ceasing MHT, as well as consideration of alternative bone-protection strategies.[3]

Effects on cardiovascular and metabolic health

MHT ameliorates several cardiovascular and metabolic changes observed in menopause, although its effect varies by formulation, dose, route and timing of initiation.[33] Systemic oestrogen has a favourable effect on lipid profile, glucose metabolism, insulin sensitivity, vascular health and overall CVD risk, with the addition of progesterone potentially attenuating these benefits.[3,33] Oral MHT improves several components of metabolic syndrome, reducing T2DM incidence by up to 30% and improving lipid parameters including LDL-C and HDL-C by approximately 15%.[34,35] However, triglycerides also increase; hence, transdermal forms are preferred for patients with hypertriglyceridaemia.[4,35] At higher doses in recently postmenopausal women (<6 years since the FMP), MHT may slow atherosclerosis progression and coronary artery calcification, surrogate markers of CHD.[36]

Current recommendations

MHT is recommended in early, premature and surgical menopause until the mean age of natural menopause primarily to prevent bone loss, as well as for its protective impact on heart disease, cognitive decline and overall mortality.[9] In women aged under 60 years or within 10 years of menopause without contraindications, the risk–benefit profile, including improved CHD risk and all-cause mortality, favours MHT use for symptom management and bone protection.[3,9]

After this window, MHT commencement is not prohibited but requires thorough risk–benefit discussion, shared decision making and periodic re-evaluation. There are limited data examining extended therapy and discontinuation timing; however, benefits should be balanced against the progressive increase in breast cancer, stroke and VTE risk.[9]

Individualised decision making

Despite existing recommendations and frameworks, women's risk profiles are often nuanced, and applying MHT guidance in clinical practice remains complex. The development of evidence-based eligibility criteria, similar to the UK Medical Eligibility Criteria for Contraceptive Use, may help GPs navigate this complex evidence.[37] However, decisions should ultimately be guided by individualised risk–benefit assessment, symptom severity and patient preferences. Acceptable risk may vary between practitioners and patients, and women should be empowered to make informed personalised decisions. When patient complexity exceeds a GP's expertise, referral can be directed to another GP, gynaecologist or cardiologist with a special interest in menopause, or to the growing number of women's heart clinics. The Her Heart 'Find a Female Cardiologist' tool (https://herheart.org/find-a-female-cardiologist) is a practical resource for locating specialists and offers an abundance of patient-friendly resources and educational tools to support shared decision making.

Conclusion

The menopause transition is a pivotal period in women's cardiovascular health. GPs play a crucial part in screening, educating and managing at-risk women. Although MHT is not recommended principally for CVD primary or secondary prevention, it remains a valuable tool for bothersome symptoms and bone protection in appropriately selected women. As evidence continues to evolve, particularly in relation to sex-specific risk factors and therapy formulations, an individualised, risk-stratified approach is increasingly critical to optimise both quality of life and long-term cardiovascular outcomes.

Key points

- Menopause is a period of accelerated cardiometabolic and vascular decline, with characteristics such as timing, type and symptom severity increasing risk and warranting closer monitoring.
- Midlife consultations present an opportunity to screen for and address cardiometabolic health, including addressing smoking status, physical activity, dietary choices, weight, blood pressure and lipid control to improve long-term outcomes.
- Tools such as Heart Health Checks (MBS item 699), Menopause Health Assessments (MBS item 695) and the AusCVDRisk calculator can support effective risk stratification, preventive planning and MHT decision making in general practice.
- Early initiation of MHT until the mean age of natural menopause mitigates many adverse outcomes in premature, early and surgical menopause, highlighting the importance of timely diagnosis and management.
- MHT is recommended for vasomotor symptom management and bone protection in women aged under 60 years or within 10 years of the FMP if not contraindicated, with risk–benefit discussions and periodic re-evaluation guiding individualised, informed decision making beyond these parameters.

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References

References are available online only.

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References

1. Australian Bureau of Statistics (ABS). Causes of death, Australia. ABS, 2023. Available at www.abs.gov.au/statistics/health/causes-death/causes-death-australia/2023 [Accessed 2 October 2025].
2. Maas AHEM, Rosano G, Cifkova R, et al. Cardiovascular health after menopause transition, pregnancy disorders, and other gynaecologic conditions: A consensus document from European cardiologists, gynaecologists, and endocrinologists. Eur Heart J 2021;42(10):967–84. doi: 10.1093/eurheartj/ehaa1044.
3. El Khoudary SR, Aggarwal B, Beckie TM, et al; American Heart Association Prevention Science Committee of the Council on Epidemiology and Prevention; and Council on Cardiovascular and Stroke Nursing. Menopause transition and cardiovascular disease risk: Implications for timing of early prevention: A scientific statement from the American Heart Association. Circulation 2020;142(25):e506–32. doi: 10.1161/CIR.0000000000000912.
4. Cho L, Kaunitz AM, Faubion SS, et al; ACC CVD in Women Committee. Rethinking menopausal hormone therapy: For whom, what, when, and how long? Circulation 2023;147(7):597–610. doi: 10.1161/CIRCULATIONAHA.122.061559.
5. Zhu D, Chung HF, Dobson AJ, et al. Age at natural menopause and risk of incident cardiovascular disease: A pooled analysis of individual patient data. Lancet Public Health 2019;4(11):e553–64. doi: 10.1016/S2468-2667(19)30155-0.
6. Zhu D, Chung HF, Dobson AJ, et al. Type of menopause, age of menopause and variations in the risk of incident cardiovascular disease: Pooled analysis of individual data from 10 international studies. Hum Reprod 2020;35(8):1933–43. doi: 10.1093/humrep/deaa124.
7. Muka T, Oliver-Williams C, Kunutsor S, et al. Association of age at onset of menopause and time since onset of menopause with cardiovascular outcomes, intermediate vascular traits, and all-cause mortality: A systematic review and meta-analysis. JAMA Cardiol 2016;1(7):767–76. doi: 10.1001/jamacardio.2016.2415.
8. Shin J, Han K, Jung JH, et al. Age at menopause and risk of heart failure and atrial fibrillation: A nationwide cohort study. Eur Heart J 2022;43(40):4148–57. doi: 10.1093/eurheartj/ehac364.
9. Faubion S, Crandall CJ, Davis L, et al. "The 2022 Hormone Therapy Position Statement of The North American Menopause Society" Advisory Panel. The 2022 hormone therapy position statement of The North American Menopause Society. Menopause 2022;29(7):767–94. doi: 10.1097/GME.0000000000002028.
10. Thurston RC, Aslanidou Vlachos HE, Derby CA, et al. Menopausal vasomotor symptoms and risk of incident cardiovascular disease events in SWAN. J Am Heart Assoc 2021;10(3):e017416. doi: 10.1161/JAHA.120.017416.
11. Samargandy S, Matthews KA, Brooks MM, et al. Abdominal visceral adipose tissue over the menopause transition and carotid atherosclerosis: The SWAN heart study. Menopause 2021;28(6):626–33. doi: 10.1097/GME.0000000000001755.
12. Dancey DR, Hanly PJ, Soong C, Lee B, Hoffstein V. Impact of menopause on the prevalence and severity of sleep apnea. Chest 2001;120(1):151–55. doi: 10.1378/chest.120.1.151.
13. Hallajzadeh J, Khoramdad M, Izadi N, et al. Metabolic syndrome and its components in premenopausal and postmenopausal women: A comprehensive systematic review and meta-analysis on observational studies. Menopause 2018;25(10):1155–64. doi: 10.1097/GME.0000000000001136.
14. Sickinghe AA, Korporaal SJA, den Ruijter HM, Kessler EL. Estrogen contributions to microvascular dysfunction evolving to heart failure with preserved ejection fraction. Front Endocrinol (Lausanne) 2019;10(442):442. doi: 10.3389/fendo.2019.00442.
15. Ying W, Post WS, Michos ED, et al. Associations between menopause, cardiac remodeling, and diastolic function: The CARDIA study. Menopause 2021;28(10):1166–75. doi: 10.1097/GME.0000000000001815.
16. Wu SI, Chou P, Tsai ST. The impact of years since menopause on the development of impaired glucose tolerance. J Clin Epidemiol 2001;54(2):117–20. doi: 10.1016/S0895-4356(00)00284-5.
17. Anagnostis P, Christou K, Artzouchaltzi AM, et al. Early menopause and premature ovarian insufficiency are associated with increased risk of type 2 diabetes: A systematic review and meta-analysis. Eur J Endocrinol 2019;180(1):41–50. doi: 10.1530/EJE-18-0602.
18. Gray KE, Katon JG, LeBlanc ES, et al. Vasomotor symptom characteristics: Are they risk factors for incident diabetes? Menopause 2018;25(5):520–30. doi: 10.1097/GME.0000000000001033.
19. Stampfer MJ, Hu FB, Manson JE, Rimm EB, Willett WC. Primary prevention of coronary heart disease in women through diet and lifestyle. N Engl J Med 2000;343(1):16–22. doi: 10.1056/NEJM200007063430103.
20. Pant A, Gibson AA, Marschner S, et al. Age of menopause, healthy lifestyle and cardiovascular disease in women: A prospective cohort study. Heart 2025;111(6):262–68. doi: 10.1136/heartjnl-2024-324602.
21. The North American Menopause Society. The 2023 nonhormone therapy position statement of The North American Menopause Society. Menopause 2023;30(6):573–90. doi: 10.1097/GME.0000000000002200.
22. Heart Foundation. Guideline for assessing and managing CVD risk and Australian CVD risk calculator. Heart Foundation, 2023. Available at www.heartfoundation.org.au/for-professionals/guideline-for-managing-cvd [Accessed 10 October 2025].
23. Arnett DK, Blumenthal RS, Albert MA, et al. 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease. Circulation 2019;140(11):e596–646. doi: 10.1161/CIR.0000000000000678.
24. Rossouw JE, Prentice RL, Manson JE, et al. Postmenopausal hormone therapy and risk of cardiovascular disease by age and years since menopause. JAMA 2007;297(13):1465–77. doi: 10.1001/jama.297.13.1465.
25. Boardman HM, Hartley L, Eisinga A, et al. Hormone therapy for preventing cardiovascular disease in post-menopausal women. Cochrane Database Syst Rev 2015;2015(3):CD002229. doi: 10.1002/14651858.CD002229.pub4.
26. Kim JE, Chang JH, Jeong MJ, et al. A systematic review and meta-analysis of effects of menopausal hormone therapy on cardiovascular diseases. Sci Rep 2020;10(1):20631. doi: 10.1038/s41598-020-77534-9.
27. Nudy M, Chinchilli VM, Foy AJ. A systematic review and meta-regression analysis to examine the 'timing hypothesis' of hormone replacement therapy on mortality, coronary heart disease, and stroke. Int J Cardiol Heart Vasc 2019;22:123–31. doi: 10.1016/j.ijcha.2019.01.001.
28. Renoux C, Dell'aniello S, Garbe E, Suissa S. Transdermal and oral hormone replacement therapy and the risk of stroke: A nested case-control study. BMJ 2010;340:c2519. doi: 10.1136/bmj.c2519.
29. Oliver-Williams C, Glisic M, Shahzad S, et al. The route of administration, timing, duration and dose of postmenopausal hormone therapy and cardiovascular outcomes in women: A systematic review. Hum Reprod Update 2019;25(2):257–71. doi: 10.1093/humupd/dmy039.
30. Hayes SN, Kim ESH, Saw J, et al; American Heart Association Council on Peripheral Vascular Disease; Council on Clinical Cardiology; Council on Cardiovascular and Stroke Nursing; Council on Genomic and Precision Medicine; and Stroke Council. Spontaneous coronary artery dissection: Current state of the science: A scientific statement from the American Heart Association. Circulation 2018;137(19):e523–57. doi: 10.1161/CIR.0000000000000564.
31. Formoso G, Perrone E, Maltoni S, et al. Short-term and long-term effects of tibolone in postmenopausal women. Cochrane Database Syst Rev 2016;10(10):CD008536. doi: 10.1002/14651858.CD008536.pub3.
32. Australasian Menopause Society. Sexual difficulties in the menopause. Australasian Menopause Society Limited, 2021. Available at https://menopause.org.au/hp/information-sheets/sexual-difficulties-in-the-menopause [Accessed 12 October 2025].
33. Shufelt CL, Manson JE. Menopausal hormone therapy and cardiovascular disease: The role of formulation, dose, and route of delivery. J Clin Endocrinol Metab 2021;106(5):1245–54. doi: 10.1210/clinem/dgab042.
34. Salpeter SR, Walsh JME, Ormiston TM, Greyber E, Buckley NS, Salpeter EE. Meta-analysis: Effect of hormone-replacement therapy on components of the metabolic syndrome in postmenopausal women. Diabetes Obes Metab 2006;8(5):538–54. doi: 10.1111/j.1463-1326.2005.00545.x.
35. Walsh BW, Schiff I, Rosner B, Greenberg L, Ravnikar V, Sacks FM. Effects of postmenopausal estrogen replacement on the concentrations and metabolism of plasma lipoproteins. N Engl J Med 1991;325(17):1196–204. doi: 10.1056/NEJM199110243251702.
36. Hodis HN, Mack WJ, Henderson VW, et al; ELITE Research Group. Vascular effects of early versus late postmenopausal treatment with estradiol. N Engl J Med 2016;374(13):1221–31. doi: 10.1056/NEJMoa1505241.
37. Laing A, Thomas L, Hillard T, Panay N, Briggs P. Exploring the potential for a set of UK hormone replacement therapy eligibility guidelines: A suggested proposal on the topic of venous thromboembolism. Post Reprod Health 2024;30(1):39–54. doi: 10.1177/20533691231223682.
38. Melehan K. Menopause and sleep | Information sheet. Australasian Menopause Society Limited, 2025. Available at https://hub.menopause.org.au/Play?pld=d9e361ea-9e11-4f11-81b1-4219222e266f [Accessed 23 September 2025].
39. Philbois SV, Facioli TP, De Lucca I, et al. What do we know about the role of menopause in cardiovascular autonomic regulation in hypertensive women? Menopause 2024;31(5):408–14. doi: 10.1097/GME.0000000000002348.
40. The Royal Australian College of General Practitioners (RACGP). Guidelines for preventive activities in general practice (Red Book). 10th edn. RACGP, 2024. Available at www.racgp.org.au/redbook [Accessed 8 October 2025].
41. Feingold KR. Guidelines for the management of high blood cholesterol. In: Feingold K, Ahmed S,

Anawalt B, editors. Endotext. MDText.com, Inc, 2025. Available at www.ncbi.nlm.nih.gov/books/NBK305897 [Accessed 12 October 2025].

42. Heart Foundation. Practical guide to pharmacological lipid management. Heart Foundation, 2024. Available at www.heartfoundation.org.au/heart-health-check-toolkit/pharmacological-lipid [Accessed 15 June 2026].
43. Queensland Health. Section 4 – Management of diagnosed conditions. Hypertension. Chronic Conditions Manual. The State of Queensland (Queensland Health), 2024. Available at www.ccm.health.qld.gov.au/management-of-diagnosed-conditions/hypertension [Accessed 12 October 2025].
44. The Royal Australian College of General Practitioners (RACGP). Management of type 2 diabetes: A handbook for general practice. RACGP, 2024. Available at www.racgp.org.au/clinical-resources/clinical-guidelines/key-racgp-guidelines/view-all-racgp-guidelines/management-of-type-2-diabetes/introduction [Accessed 8 October 2025].
45. Ellender CM, Vakulin A, Stocks N, Chai-Coetzer CL. Management of obstructive sleep apnoea in primary care. Aust J Gen Pract 2024;53(6):363–69. doi: 10.31128/AJGP-08-23-6933.