


Advancing the access to cardiovascular diagnosis and treatment among women with cardiovascular disease: a joint British Cardiovascular Societies' consensus document

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ABSTRACT

Despite significant progress in cardiovascular pharmacotherapy and interventional strategies, cardiovascular disease (CVD), in particular ischaemic heart disease, remains the leading cause of morbidity and mortality among women in the UK and worldwide. Women are underdiagnosed, undertreated and under-represented in clinical trials directed at management strategies for CVD, making their results less applicable to this subset. Women have additional sex-specific risk factors that put them at higher risk of future cardiovascular events. Psychosocial risk factors, socioeconomic deprivation and environmental factors have an augmented impact on women's cardiovascular health, highlighting the need for a holistic approach to care that considers risk factors specifically related to female biology alongside the traditional risk factors. Importantly, in the UK, even in the context of a National Health Service, there exist significant regional variations in age-standardised mortality rates among patients with CVD. Given most CVDs are preventable, concerted efforts are necessary to address the unmet needs and ensure parity of care for women with CVD. The present consensus document, put together by the British Cardiovascular Society (BCS)'s affiliated societies, specifically portrays the current status on the sex-related differences in the diagnosis and treatment of each of the major CVD areas and proposes strategies to overcome the barriers in accessing diagnoses and treatments among women. This document aims at raising awareness of the scale of the current problem and hopes to stimulate a multifaceted approach to address sex disparities and enable future comprehensive sex- and gender-based research through collaboration across different affiliated societies within the BCS.

INTRODUCTION

Globally, cardiovascular disease (CVD) is the leading cause of death in women.¹ However, the misconception that it is a 'man's disease' underlines that CVD in women has contributed to its

under-recognition and undertreatment.² Over 3.6 million women in the UK are currently affected by ischaemic heart disease, which kills one in 14 women.³ Approximately 30% of the total 81765 myocardial infarctions (MI) registered in the UK between 2022 and 2023 occurred in women. A discrepancy between women and men in the proportion of patients admitted with higher-risk ST-segment elevation myocardial infarction (STEMI) not receiving reperfusion treatment has been reported, and this phenomenon occurs more frequently in older women. The possible reasons might include delayed access to the emergency care compared with men secondary to lack of awareness, underestimated risk and social barriers. In addition, a lower proportion of women admitted with lower-risk non-ST segment elevation myocardial infarction (NSTEMI) receive angiography within 72 hours compared with men.⁴ In addition, women are under-represented in cardiovascular clinical research, meaning that many treatments are mainly investigated in men and then applied to women, with the expectation that sex-based differences in physiology and pharmacokinetics will have little impact.⁵ Even where prognostic cardiovascular therapies are well established, women are frequently under-referred for treatment which leads to poorer outcomes (figure 1).⁶

This consensus document put together by representatives from each of the leading UK cardiovascular affiliated societies including the Primary Care Cardiovascular Society, nursing and patient affiliated societies, outlines the key sex-specific differences across the CVD spectrum and the recommendations for addressing cardiovascular health inequalities among women in the UK (figure 2). The terms sex and gender are closely related to each other, and they are often identified as the same erroneously. According to the WHO, sex refers to the biological characteristics that define humans as female or male such as chromosomes, hormones and reproductive



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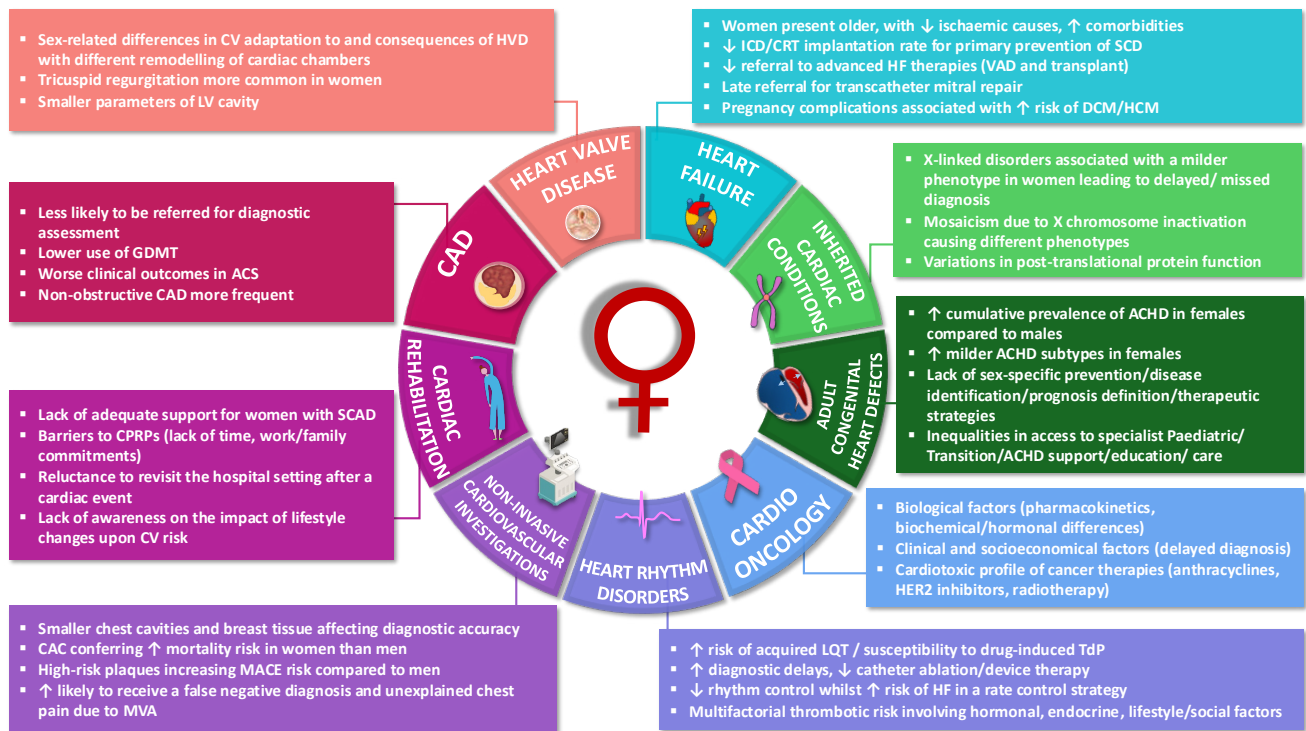


Figure 1 Main factors contributing to sex differences across different subareas of cardiovascular disease. ACHD, adult congenital heart defects; ACS, acute coronary syndrome; CAC, coronary artery calcium; CAD, coronary artery disease; CPRPs, cardiovascular prevention and rehabilitation programmes; CRT, cardiac resynchronisation therapy; CV, cardiovascular; DCM, dilated cardiomyopathy; GDMT, guideline-directed medical therapy; HCM, hypertrophic cardiomyopathy; HER2, human epidermal growth factor receptor 2; HF, heart failure; HVD, heart valve disease; ICD, implantable cardiac defibrillator; LQT, long QT; LV, left ventricular; MACE, major adverse cardiovascular events; MVA, microvascular angina; SCAD, spontaneous coronary artery dissection; SCD, sudden cardiac death; TdP, torsades de pointes; VAD, ventricular assist device.

organs, while gender refers to the socially constructed roles comprehensive of norms and behaviours associated, as well as relationships.⁷ The main purpose of this document is to advance the access to diagnosis and treatment among women suffering from CVD with provision of disease-specific action points to be implemented by all affiliated societies, mostly referring to a sex-based approach since the pathophysiological differences in CVD are driven by biological sex and the associated reproductive organs/hormones (tables 1–3). However, we recognise that a gender-based perspective would be helpful to explore and call attention to acquired risk factors secondary to personal choices, hormonal fluctuation and social/physical relationships. Gender is a wider concept which can vary across different cultures and over time. We hope this document will help improve the care of women with CVD worldwide.

TRADITIONAL CARDIOVASCULAR RISK FACTORS IN WOMEN

The leading modifiable cardiovascular risk factors such as hypertension, smoking, diabetes, obesity and dyslipidaemia together account for approximately 50% of preventable cardiovascular deaths. Hypertension (30% and 38%) and smoking (36% and 17%) alone account for most of these deaths in both men and women, respectively. Data from the 2012–2017 Health Survey for England showed that significantly more women have no ‘traditional’ cardiovascular risk factors compared with men (36% vs 29%), although the difference is blunted in the older age group and in the most deprived areas of England. The prevalence of hypertension was lower in women than men (24% vs 27%), with a similar trend in smoking (15% vs 19%), diabetes

(6% vs 9%) and dyslipidaemia (48% vs 52%). However, obesity was more common in women (30% vs 28%) who were less likely to be treated for dyslipidaemia and to achieve therapeutic targets. Control of hypertension and diabetes was similar between sexes.⁸ Systolic blood pressure (BP) was lower in women than men until the age of 60 when it equalises.⁹ Oestrogens have a major role in BP regulation in premenopausal women, modulating non-genomic pathways as well as expression of vasoconstrictors. Given that the diagnosis of hypertension relies on crossing a sex-independent threshold ($\geq 140/90$ mmHg), this may explain the lower prevalence of hypertension in younger women.¹⁰

Conversely, smoking is more deleterious in younger women than men, with a 25% greater excess risk of coronary artery disease (CAD).¹¹ Diabetes is also a significantly more potent risk factor for CVD in women, with a 50% higher relative risk for fatal CAD associated with diabetes in women.¹² Possible hypotheses explaining this difference include a higher body mass index, systemic inflammation and worse glycaemic control at the time of diabetes diagnosis in women compared with men.¹³ Similarly, the Framingham study showed that the excess risk of CVD from obesity was significantly higher in women than in men.¹⁴ These data suggest that the cardiovascular burden, based on the incidence of risk factors, should be interpreted within a sex-specific context.¹⁵ In addition, an important difference in cardiovascular risk factor susceptibility across races has been acknowledged. Behavioural, environmental and social factors affect cardiovascular health and risk in women.¹⁶

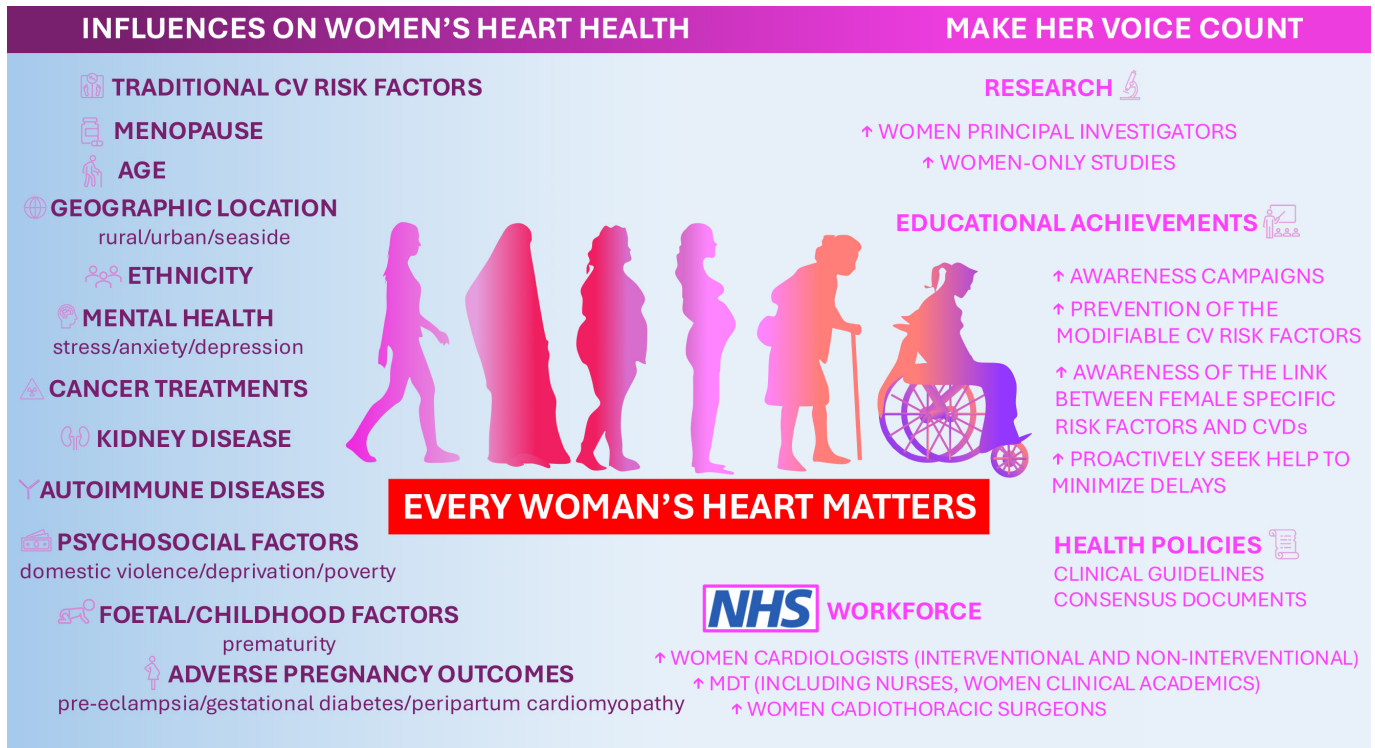


Figure 2 Factors influencing women's heart health and strategies to overcome sex disparities in cardiovascular care. CV, cardiovascular; CVDs, cardiovascular diseases; MDT, multidisciplinary team; NHS, National Health Service.

WOMEN-SPECIFIC RISK FACTORS

Beyond sex differences in conventional cardiovascular risk factors, women-specific risk factors are also important across the lifespan. Sex differences in the hormonal milieu are important underpinning factors. Studies of atherosclerotic CVD indicate differing genetic and epigenetic factors influencing vascular biology in women.¹⁷ The menopause is the most striking example of how hormonal fluctuation impacts on cardiovascular health. Several mechanisms linking oestrogen deficiency and CAD have been proposed, including alteration in fat distribution and heightened BP. Postmenopausal women are also more susceptible to coronary vasomotor disorders due to a higher prevalence of systemic inflammation.

The lower oestrogen levels after menopause also increase the susceptibility to Takotsubo cardiomyopathy in women, constituting 90% of patients presenting with this condition, and around 80% are older than 50 years. The considerable preponderance of postmenopausal women is indicative of a hormonal influence. Oestrogens can attenuate catecholamine-mediated vasoconstriction and decrease the sympathetic response to mental stress in perimenopausal women.¹⁸ Early and late menarche are associated with an increased long-term risk of cardiovascular events, as are infertility and polycystic ovarian syndrome.¹⁹ The combined contraceptive pill, and to a lesser extent depot medroxyprogesterone, also increase the relative risk of cardiovascular events and should be avoided in women at high baseline risk of atherothrombotic events. This is influenced by baseline risk and the timing of therapy, among many other factors requiring personalised risk–benefit evaluation. Women with hypertension are therefore encouraged to use progesterone-only alternatives.²⁰ Post hoc analyses suggest hormone therapy may confer cardiovascular benefits in women commencing hormone therapy within 10 years of menopause at the age of 50–59 years.^{21 22}

Factors during pregnancy, such as pre-eclampsia, gestational diabetes mellitus and peripartum cardiomyopathy, are also associated with wide-ranging cardiovascular risk¹⁹ and these have both immediate and long-term health implications. Among pregnant women, spontaneous coronary artery dissection (SCAD) is responsible for the majority of MI cases. Nonetheless, the scarce enrolment of pregnant women in clinical trials calls for greater representation of this specific subset to better guide the optimal management of women with acute coronary syndrome (ACS).²³

The UK Biobank prospective study explored long-term outcomes of gestational diabetes mellitus in over 220 000 women (1225 with self-reported gestational diabetes) followed up from their first delivery until October 2021. Among parous women, those who developed gestational diabetes had a significantly greater risk of premature all-cause mortality, including CVD death, compared with women with no history of gestational diabetes. Similarly, the risk of incident total and non-fatal CVD and common cardiovascular risk factors (diabetes, hypertension and dyslipidaemia) was significantly greater in women with a history of gestational diabetes compared with those without.²⁴

Historically, adding sex-specific factors to cardiovascular risk models using established cardiovascular risk factors has led to little or no significant improvement in the prediction of cardiovascular events.²⁵ A more recent update of the QRISK calculator for CVD has identified pre-eclampsia and postnatal depression as novel female sex-specific risk factors, highlighting the importance of developing sex-specific risk stratification models.²⁶

CORONARY ARTERY DISEASE IN WOMEN

Although the contemporary precision medicine-based approach allows for better characterisation of CVD, management of CAD in women remains suboptimal. Women presenting with obstructive CAD are older and have more comorbidities than men with

Table 1 Actionable points on disease conditions contributing to CVD

Disease condition	Actionable points
Traditional CV risk factors	<ul style="list-style-type: none"> ⇒ Raise awareness of the suboptimal control of some of the traditional CV risk factors in women to proactively identify any untreated risk factor in the early stage. ⇒ Promote awareness campaigns among premenopausal women to proactively seek support to address modifiable CV risk factors.
Women-specific risk factors	<ul style="list-style-type: none"> ⇒ Raise awareness among public and clinicians about the link between female-specific risk factors and CVD. ⇒ Determine how to integrate reproductive life course events into personalised CV care to improve risk prediction for women. ⇒ Investigation of specific subsets such as pregnant, pre- or post-menopausal women through dedicated study protocols in collaboration with other specialties such as obstetrics and/or gynaecologists.
Coronary artery disease	<ul style="list-style-type: none"> ⇒ Increase awareness among public and clinicians that CAD is the leading cause of mortality for women. ⇒ Avoid delays in access to care in the setting of ACS. ⇒ Provide a complete diagnostic work-up in case of non-obstructive coronary arteries (MINOCA, ANOCA, INOCA which occur more frequently in women) to investigate the underlying mechanism and direct medical therapy. ⇒ Proactively enrol female patients with CAD in research studies and undertake women-only studies.
Valvular heart disease	<ul style="list-style-type: none"> ⇒ Raise awareness among clinicians and patients of the sex differences in valve and ventricular parameters in the context of valvular heart disease. ⇒ Proactively enrol female patients in heart valve disease research studies. ⇒ Ensure women have timely access to investigations including echocardiography and valve interventions. ⇒ Identify and address barriers to appropriate referral for valve interventions. ⇒ Device-specific considerations to ensure appropriate intervention to reduce prosthesis mismatch and complications in women.
Heart failure	<ul style="list-style-type: none"> ⇒ Raise awareness that women with HFrEF are less likely to receive GDMT, referral for ICD, CRTD and heart transplant compared with men. ⇒ Ensure women receive GDMT and access to device therapy. ⇒ Undertake research to identify optimal dosing of GDMT for women. ⇒ Ensure representation of women in HF research studies.
Inherited cardiac conditions	<ul style="list-style-type: none"> ⇒ Identify sex-specific thresholds for diagnosis of DCM and HCM. ⇒ Conduct detailed research to address modifiable sex differences in outcomes for ICCs in women. ⇒ Ensure equitable access to specialist cardiac care, genetic testing and family screening. ⇒ Define and ensure training in ICCs which include understanding sex differences and disparities in ICCs. ⇒ Address workforce challenges in ICCs.
Adult congenital heart defects	<ul style="list-style-type: none"> ⇒ Prioritisation of research in this area leading to future sex-specific interventions, treatment and care models. ⇒ Audited sex-specific outcome data in ACHD. ⇒ Harmonisation and standardisation of exposure to and training in ACHD Cardiology and Cardiac Obstetrics/Maternal medicine which include understanding sex differences and disparities in ACHD. ⇒ Address the workforce challenges in ACHD to optimise sex-specific personalised prevention, disease identification, prognosis definition and individualised therapeutic strategies.
Heart rhythm disorders	<ul style="list-style-type: none"> ⇒ Despite the primarily female demographic of postural orthostatic tachycardia syndrome and dysautonomia syndromes, the evidence base is poor and requires significant research and collaborative efforts. ⇒ Further investment in addressing inequalities of arrhythmia management in women requires a multifaceted approach, including promoting sex- and gender-based analysis in research and expanding the use of quality improvement programme.

ACHD, adult congenital heart defects; ACS, acute coronary syndrome; ANOCA, angina with non-obstructive coronary arteries; CAD, coronary artery disease; CRTD, cardiac resynchronisation therapy with defibrillator; CV, cardiovascular; CVD, cardiovascular disease; DCM, dilated cardiomyopathy; GDMT, guideline-directed medical therapy; HCM, hypertrophic cardiomyopathy; HF, heart failure; HFrEF, heart failure with reduced ejection fraction; ICCs, inherited cardiac conditions; ICD, implantable cardiac defibrillator; INOCA, ischaemia with non-obstructive coronary arteries; MINOCA, myocardial infarction with non-obstructive coronary arteries.

the same condition, and are less likely to be referred for diagnostic assessment in both the acute and chronic setting,²⁷ despite reporting a higher angina burden.²⁸ A link between ageing and sex differences in atherosclerotic plaque composition has been confirmed. A large optimal coherence tomography study including patients stratified by age (<58, 58–68 and >68 years) showed that women are characterised by less pronounced and

severe CAD than men, but the sex difference was less evident in the oldest group.²⁹ Similarly, the PROSPECT study showed that sex differences in the plaque extent and composition were detected in patients aged <65 years but not in older patients, mainly explained by accelerated atherosclerosis progression in older women compared with older men.³⁰ In premenopausal women, oestrogens protect vascular structures and stabilise the

Table 2 Actionable points on healthcare services with a potential role in primary and secondary prevention of CVD in women

Disease condition	Actionable points
Cardio-oncology	<ul style="list-style-type: none"> ⇒ An individualised multidisciplinary approach to the management of women receiving cancer treatment, with a focus on screening, monitoring and early detection of cardiac toxicity, and also on the inclusion of traditional modifiable CV risk factors into the risk assessment and optimisation of pre-treatment, to improve survival and quality of life. ⇒ Address the under-representation of women in clinical trials of novel cancer immunotherapy treatments. ⇒ Need for registries monitoring cardiac toxicity in general and specifically in women. ⇒ Identify cardioprotective strategies for women undergoing cancer treatment with anthracyclines.
Cardiac rehabilitation	<ul style="list-style-type: none"> ⇒ Need to flexibly deliver individualised CPRPs which can overcome barriers and enhance participation. ⇒ More flexible rehabilitation such as home/virtual/hybrid individual and group-based rehabilitation options. ⇒ To enhance participation and outcomes there needs to be continued focus, from both a clinical and research perspective, on better meeting the needs of women within CPRPs.
Non-invasive CV investigation	<ul style="list-style-type: none"> ⇒ Consider sex stratified population level recommendations for cardiovascular imaging. ⇒ Increase clinician awareness about the strengths and limitations of each diagnostic modality in women with proven or suspected CVD.
Primary care	<ul style="list-style-type: none"> ⇒ A new paradigm is needed, a contractual data driven enabler to ensure that primary and secondary prevention of CVD has a unique focus on women's health, and that colleagues recognise this area of medicine as core work. ⇒ Given the workforce pressures, the workplace and citizens themselves should be part of the solution, empowered to understand the value and purpose of focusing on CVD prevention. ⇒ The digital age should be harnessed in the NHS to 'automate what can be automated' in order to reduce workforce burden and enhance patients' experience. ⇒ To accelerate change, national contracts are required, ICBs being held to account as systems rather than siloed providers, to make women's CVD health an 'everyone's responsibility' approach. ⇒ National adoption of technology is also required rather than ICBs being left to procure at a local level, taking valuable time and energy.

CPRPs, cardiovascular prevention and rehabilitation programmes; CV, cardiovascular; CVD, cardiovascular disease; ICBs, integrated care boards.

fibrous cap inhibiting oxidised low-density lipoproteins which activate metalloprotease expression in macrophages. However, oestrogen is also associated with hyaluronan deposition and CD44 expression, common in plaque erosion, which represents

the dominant pathophysiologic mechanism in young women with ACS.³¹

The use of guideline-directed medical therapy remains lower in women, who are less likely to attain adequate BP, low density

Table 3 Actionable points on patients and nursing perspectives to help clinicians in the decision-making approach to CVD in women

Disease condition	Actionable points
Nursing perspectives	<ul style="list-style-type: none"> ⇒ Nurses serve as staunch advocates for women in several CV domains including promoting awareness, delivering personalised care, rectifying disparities, fostering shared decision-making, advocating for policy reforms and contributing to research and educational endeavours. ⇒ Leverage influence to highlight and address sex biases in healthcare and work towards reducing disparities and enhancing access to care and outcomes for women with CVD.
Patient perspectives	<ul style="list-style-type: none"> ⇒ Call for a holistic woman-centred approach to heart care that listens to and incorporates women's experiences and insights. ⇒ A collaborative campaign involving stakeholders from various sectors, including the media, to raise awareness about the importance of CV health for women. ⇒ Disseminated information should be consistent, evidence-based and tailored to the unique needs of women, ensuring accessibility, inclusivity and cultural sensitivity. ⇒ Highlight heart conditions that predominantly or exclusively affect women. ⇒ Any initiative focusing on women's heart health should prioritise improving the communication skills of healthcare professionals to understand and appropriately respond to women's experiences. ⇒ Co-designed training programmes should be developed for healthcare professionals to become more attuned to women's specific needs, including the nuances of women's cardiac symptoms. ⇒ Women should be empowered to openly discuss their symptoms and worries. ⇒ Ensure women are not only heard but also accurately assessed and treated in a timely manner. ⇒ Every woman should feel that her heart matters too, feeling respected and understood.

CV, cardiovascular; CVD, cardiovascular disease.

lipoproteins and glycated haemoglobin targets.²⁸ Women with MI and obstructive CAD have significantly higher in-hospital and 12-month mortality compared with men, and this can be ascribed to their different risk profile.^{32–33} The significantly worse outcomes in women with ACS compared with men persist even after risk adjustment for age and comorbidities, especially in patients with STEMI.³⁴ In the era of primary percutaneous coronary intervention, women with MI continue to present with cardiogenic shock more frequently than men.³³ However, referral for percutaneous coronary intervention in this setting is still lower in women than in men.³⁵

Despite international guidelines advocating parity in the management of patients with ACS regardless of sex, women are under-represented in clinical trials investigating interventional treatment strategies and are less likely to receive evidence-based therapies such as coronary angiography and revascularisation.^{23–36} Compared with men, women develop more complications following NSTEMI.^{37–38} Sex-specific risk factors are not accounted for in contemporary risk scores for patients presenting with ACS.^{39–40} Women are at greater risk of bleeding complications following ACS and revascularisation compared with men,³⁶ largely due to differences in age and comorbidities that predispose to bleeding.⁴¹ Women with non-ST elevation ACS are more likely to present with comorbidities that are more strongly associated with mortality compared with men, including hypertension, smoking, diabetes and frailty.^{36–39} Therefore, a paradigm shift in the diagnosis and management of CAD in women is needed (table 1). There also needs to be a greater focus on women with angina/ischaemia and non-obstructive coronary arteries (ANOCA/INOCA), which occurs more frequently in women than in men. The prevalence of ANOCA/INOCA is estimated to increase with the widespread use of non-invasive imaging. This subset is less likely to have traditional risk factors, and novel risk factors such as pro-inflammatory markers seem to be involved. Psychosocial stress and the sympathetic nervous system may play a crucial role. A recent study has demonstrated the utility of exercise treadmill testing as a rule-in investigation to confirm coronary microvascular dysfunction in patients with ANOCA/INOCA in a timely and cost-effective manner.⁴² However, invasive coronary vasomotor function testing represents the only diagnostic tool able to systematically investigate all pathophysiological mechanisms underlying ANOCA, guiding the choice of the subsequent patient-tailored medical treatment.^{43–44} Myocardial infarction with non-obstructive coronary arteries (MINOCA) represents a heterogeneous group of conditions that disproportionately affect women, including SCAD, coronary artery spasm and coronary microvascular dysfunction.^{45–46}

HEART VALVE DISEASE IN WOMEN

Heart valve disease equally affects men and women,⁴⁷ however, there are sex-related differences in epidemiology, pathophysiology and clinical presentation.⁴⁸ Women are more often affected by mitral valve disease of degenerative or rheumatic cause,⁴⁷ while men are more often affected by aortic valve disease, with bicuspid aortic valve disease being 3–4 times more frequent in men.⁴⁹ Furthermore, women who have a bicuspid aortic valve are more likely to develop aortic stenosis, while men are more likely to develop aortic regurgitation.⁴⁹ In the case of acquired degenerative calcific aortic stenosis, women have slower haemodynamic progression of the valve disease than men.⁵⁰ Across the UK, transcatheter aortic valve implantation (TAVI) is currently provided by 35 NHS centres and eight private hospitals. In 2022/2023, women constituted 43% of all

TAVI patients with a median age of 82 years whereas the median age of male patients was 81 years.⁵¹ There are sex-related differences in cardiovascular adaptation to and consequences of heart valve disease, with different remodelling of cardiac chambers in women and men.^{47–50–52} For example, in calcific aortic stenosis, women are more likely to have preserved left ventricular (LV) ejection fraction and concentric LV hypertrophy, with consequently smaller LV cavity size and higher filling pressures.⁵⁰ Consequently, women are more likely to develop paradoxical low-flow aortic stenosis and heart failure with preserved ejection fraction (HFpEF). In chronic severe aortic regurgitation, women develop less dilatation of the left ventricle and experience more hospitalisation for HF, urgent aortic valve replacement or death with lower LV indexed volumes.⁵²

The smaller average size of the female heart can affect the clarity of heart valve disease assessment.⁵² This is because smaller ventricles generate lower stroke volumes producing less pronounced physiological signals. However, the noise associated with cardiac imaging is generally independent of chamber size. Accordingly, a less favourable signal to noise ratio is present when evaluating heart valve disease in women and consequent decision making is rendered less clearcut. This issue, combined with the typically male heart-derived reference ranges, results in an inherent bias against timely intervention in women.

The aetiological progression of heart valve disease also differs in women compared with men. An important example of this is that aortic stenosis in women follows a more fibro-calcific than calcific pattern.⁵³ This means that an equivalent level of valve dysfunction in women is often associated with a less dramatic anatomical appearance of valve morphology. Last, pregnancy may contribute to an acceleration of valve dysfunction.⁴⁸ In particular, tricuspid regurgitation is more common in women than men, potentially relating to the failure of the tricuspid annulus to fully regress back to normal size following exposure to the physiological stress of pregnancy.

Most landmark studies underpinning guideline recommendations enrolled predominantly men, causing sex disparity in the diagnosis of heart valve disease severity and in the timing of intervention (table 1).⁴⁷ For example, parameters of LV cavity size used for timing of intervention are smaller in women. Indexing dimensions and volumes for body surface area only partially resolve the disparity, because the LV response to valve disease differs.^{47–52} Women are less likely to be referred for heart valve intervention, despite similar mortality.^{47–48–52} More so, late referral of women due to extrapolation from indications from men-dominated cohorts results in worse outcomes.⁴⁷

HEART FAILURE (HF) IN WOMEN

The lifetime risk of HF is similar in women and men, but the prevalence of HFpEF is more common in women than men and increases with age.⁵⁴ Women present at a later age, have less ischaemic aetiology and more comorbidities including hypertension, atrial fibrillation and obesity.⁵⁵ More fundamentally, the normal ejection fraction range is higher in women and the risk of CVD remains increased up to an ejection fraction of 60–65% compared with 50–55% in men. Such physiological differences are not taken into account in guideline recommendations.⁵⁶ Women are less likely to receive evidence-based treatment for heart failure with reduced ejection fraction (HFrEF)⁵⁷ and may require lower doses of guideline-directed medical therapy to achieve optimal clinical effectiveness.⁵⁸ However, the sex-specific subgroup analysis is limited due to under-representation in HF clinical trials.⁵⁹

Sex-specific differences in treatment effectiveness exist for HFpEF. A prespecified subgroup analysis of the PARAGON-HF trial showed a greater reduction in the risk of HF rehospitalisation in women than in men with HF with an ejection fraction >45% treated with sacubitril-valsartan.⁶⁰ Similarly, a greater effect of spironolactone in reducing all-cause mortality in women with HFpEF was shown by a post hoc analysis of the TOPCAT trial.⁶¹ The implantation rate of an implantable cardiac defibrillator for primary prevention of sudden cardiac death is lower in women⁶² and may not incur the same survival benefit as for men.⁶³ Lower implant rates are also seen for cardiac resynchronisation therapy,⁶³ even though there is evidence for increased benefit for women.⁶⁴ Women are less likely to be referred for advanced HF therapies such as ventricular assist device and transplant,⁶⁵ and are referred late for transcatheter mitral repair with worse outcomes.⁶⁶

Women are more likely to present acutely with HFpEF than HFrEF, at an older age and less likely to be admitted to cardiology and receive an echocardiogram. Acute HF registries show lower rates of revascularisation, device therapy and direct current cardioversion in women compared with men.⁶⁷ Moreover, self-care—pivotal in HF management—is hindered due to societal roles, economic factors and limited attendance in rehabilitation programmes, impacting women's ability to prioritise personal health and engage in self-care practices (table 1).⁶⁸

INHERITED CARDIAC CONDITIONS IN WOMEN

Inherited cardiac conditions (ICCs) comprise a heterogeneous group of genetically determined disorders including cardiomyopathies, ion channel disorders, aortopathies, mitochondrial diseases and some neuromuscular conditions. The causes for sex-related differences include:

Inheritance patterns: X-linked and matrilineal diseases are sex-specific ICCs. X-linked disorders such as Fabry and Danon disease are associated with a milder phenotype in women which can lead to delayed or missed diagnoses.^{69 70} Variants in the dystrophin gene cause severe Duchenne and Becker muscular dystrophy in men but not women.⁷¹

Mosaicism: Somatic (more common) or germline (rare) mosaicism occurs when different cell lines exist within an individual due to X chromosome inactivation, causing a wide range of phenotypes in women affecting diagnostic accuracy.

Phenotypic expression: Variations in post-translational protein function between men and women can explain differences in phenotypic expression. For example, ion channel expression in the right ventricular outflow tract explains the higher prevalence of Brugada syndrome in men than women.⁷² The prevalence of dilated cardiomyopathy (DCM) and those requiring transplantation for DCM is higher in men than women.⁷³ Additionally, men have a higher penetrance of truncating titin variants, present at a younger age, exhibit worse systolic dysfunction and higher rates of atrial fibrillation.⁷⁴ Outcomes in women with hypertrophic cardiomyopathy (HCM) are worse than in men, with a later age at diagnosis, worse symptoms, different haemodynamics (greater degree of obstructive physiology and mitral regurgitation, higher E/E' ratio and pulmonary artery systolic pressure), worse exercise performance and greater all-cause mortality.^{75 76}

Risk of sudden death: Biological sex is an important predictor of risk in several conditions and has been incorporated into established risk algorithms—for example, long QT syndrome.⁷⁷ Women with Lamin A/C gene (LMNA)-associated cardiomyopathy also have a 45% lower risk of life-threatening arrhythmias than men.⁷⁸

Pregnancy in women with ICCs appears to be associated with worse outcomes. Peripartum cardiomyopathy develops towards the end of pregnancy or in the months following delivery.⁷⁹ It shares a genetic predisposition similar to DCM.⁸⁰ Pregnancy complications, including hypertensive disorders, are also associated with a greater risk of developing DCM and HCM.⁸¹ The interplay of environmental, societal discrepancies, socio-economic factors, healthcare system biases and disparities in access to care lead to inequitable obstacles in accessing specialised cardiac care, genetic testing and familial screening which is particularly relevant for ICCs.

ADULT CONGENITAL HEART DEFECTS IN WOMEN

Congenital heart defects represent the most frequent human birth defects, occurring in almost 1% of all live newborns.⁸² Sex differences in the worldwide prevalence of adult congenital heart disease (ACHD) are recognised. The cumulative prevalence of congenital heart disease is considerably higher in females, with a greater risk for males to be born with severe subtypes and for females with milder subtypes.⁸³ Despite no difference in mortality, significant sex differences in morbidity have been reported such as an increased risk of pulmonary hypertension for women but a lower risk of infective endocarditis, aortic complications and implantable cardiac defibrillator implantation compared with men.⁸⁴ The aetiology of the different distribution of congenital heart disease among sexes is still under investigation and a deeper understanding of how sex influences the risk of congenital heart disease is warranted.⁸³ Due to advances in fetal diagnosis, intervention, surgery and care in congenital heart disease, there are more adults surviving with congenital heart disease than children. Furthermore, the increase in moderate to complex disease with advancing age and acquired comorbidities describes the shifting landscape of the epidemiology of congenital heart disease. Sex differences and disparities in clinical need, evidence-based care and outcomes are as yet unknown and should be a priority for research.

Understanding the effects of sex on the prevalence of congenital heart disease has a key role in defining personalised prevention, disease identification, prognosis definition and individualised therapeutic strategies.⁸³ Yet there is a paucity of research in this area, leading to inequity, disparities in care and the potential for poorer outcomes. Since the outcomes significantly improved, there is a need to move towards facilitating well-being for individuals with ACHD. Living with ACHD carries several lifestyle implications. However, counselling and support for a fulfilling sexual life or women planning pregnancy are often overlooked, and they should be prioritised in young women suffering from a complex lifelong cardiac condition.⁸⁵ Variability in standardised training and dire workforce challenges in congenital heart disease, and a lack of understanding of sex differences, further compound these issues. The most pressing actionable strategies to address sex differences in congenital heart disease epidemiology, prognosis, treatment, intervention outcomes and sequelae are summarised in table 1.

HEART RHYTHM DISORDERS IN WOMEN

Sex differences in electrocardiography include women having marginally narrower QRS complexes and a slower baseline heart rate. Premenopausal adult women have longer QT intervals corrected for heart rate (QTc) than men of the same age, with consistent findings that the QTc difference between premenopausal women and men of similar age diminishes with increasing heart rate.⁸⁶ Consequently, women are at greater

risk of developing acquired long QT and are more susceptible to drug-induced torsades de pointes (specifically class I and III anti-arrhythmic drug therapy, prolonging ventricular repolarisation).⁸⁶ Sex differences are also observed in arrhythmia symptom severity, with women having fewer clinical symptoms and later onset than men.⁸⁷ This is further compounded with women being less likely to receive appropriate treatment for arrhythmias including catheter ablation, more likely to experience diagnostic delays, and lower utilisation rates of device therapy.⁸⁶

Atrial fibrillation is the most common arrhythmia and is expected to become more prevalent in the coming decades.⁸⁸ However, there are important sex disparities in management. Women are less likely to receive treatment for rhythm control while having a greater risk of HF in a rate control strategy.⁸⁹ Despite female sex being included in the stroke risk CHA2DS2-VASc score, women are less likely to receive oral anticoagulation.⁸⁹ Increased thrombotic risk in women is poorly researched; however, it is likely to be multifactorial, involving hormonal changes post menopause, structural, endocrine and lifestyle/social factors.⁸⁶ Randomised studies show that women benefit from anticoagulant treatment and that their bleeding risk is similar to men. Women should therefore receive equivalent treatment to men. Women are not represented equally in the large randomised studies and sex-related information in most arrhythmia studies is incomplete (table 1).⁹⁰ Recommendations regarding knowledge gaps to address sex disparities in arrhythmia are summarised in online supplemental tables S1 and S2.

CARDIO-ONCOLOGY IN WOMEN

Over the past few decades there has been remarkable improvement in cancer survival owing to advances in screening, diagnosis and efficacy of therapeutics. Nevertheless, there has been, paradoxically, an increase in the relative threat of cardiovascular problems, whether through patients surviving cancer long enough to acquire CVD or the treatment itself adversely affecting the heart. To address these issues, cardio-oncology has emerged as a new interdisciplinary subspecialty. There is emerging, although limited, evidence of disparities in cardio-oncology outcomes in women compared with men. These may relate to biological factors (such as pharmacokinetics, biochemical and hormonal differences, and pregnancy), clinical factors (such as delayed diagnosis) and socioeconomic factors (such as access to screening).

Breast cancer is by far the most common cancer in women in the UK⁹¹ and the cardiotoxic profile of many cancer therapies is well established. Anthracyclines remain a cornerstone of treatment in breast cancer, and they are associated with a substantial cardiotoxic profile. The emergence of targeted immunotherapy has revolutionised cancer treatment, bringing with it an even higher incidence of adverse cardiovascular consequences, even though the mechanisms and severity are generally less injurious than for anthracyclines.⁹² Trial evidence suggests an increased risk of cancer treatment-related toxicity in women, especially with immunotherapy.⁹³ Radiotherapy, especially localised to the chest, can lead to fibrosis, significantly increasing cardiovascular morbidity and mortality.

Despite burgeoning interest in the field and the development of cardio-oncology guidelines, including risk scores to identify women with vulnerability in this regard, there is a paucity of evidence regarding optimal cardioprotective strategies to reduce the impact of cardiotoxic therapies (table 2).^{94,95}

CARDIOVASCULAR REHABILITATION IN WOMEN

There is a robust evidence base demonstrating the benefits of cardiovascular disease prevention and rehabilitation programmes (CPRPs) for those with CVD.⁹¹ Specifically, CPRPs reduce hospital admissions and cardiovascular mortality in those with CAD, and reduce hospital admissions and improve quality of life in those with HF.⁹¹ One notable gap is in relation to SCAD. This predominantly female population cannot routinely access rehabilitation and often lacks adequate support.⁹⁶

For women, barriers to accessing CPRPs may be personal, logistical or related to programme characteristics.⁹⁷ Women often cite lack of time as a barrier due to the complex interaction of their work and family commitments. Travel/transport/parking and financial costs are also often implicated.⁹⁷ Women report reluctance and anxiety around revisiting the hospital setting following a cardiac event, and research has found they lack awareness on the impact of lifestyle changes on cardiovascular risk.⁹⁷ They are more likely to lack social support and may seek this from a CPRP, yet can find a traditional group-based rehabilitation class format unappealing, particularly where there is a high proportion of male attendees (table 2).⁹⁷

NON-INVASIVE CARDIOVASCULAR INVESTIGATIONS IN WOMEN

Echocardiography

The acquisition of accurate echocardiographic data depends on three key factors: (1) echocardiographer skills; (2) body habitus and mobility to maximise acoustic access; (3) pre-test awareness to inform a rigorous search for relevant findings. The variables influencing these key factors are accentuated in women. Echocardiography necessitates exposing the top half of a patient and this can impact the effort made to seek out the best images, particularly in the apical window. In addition, maximising acquired Doppler gradients requires the best acoustic window and angle. This is even more important in women since their chest cavities are smaller and the margin for error is therefore narrower.

The British Society of Echocardiography minimum dataset outlines the essential echocardiographic data to be achieved for every echo,⁹⁸ but this does not describe the second part of the process of acquiring echo data which is based on a pre-test assessment made by the echocardiographer about the pathology for which they need to search. Echocardiography is a content dynamic test. One size does not fit every patient. We must acknowledge the impact of the patient's biological sex on pre-test pathological awareness and the influence this may have on identifying relevant information.

Cardiovascular magnetic resonance (CMR)

CMR imaging does not involve ionising radiation and may be undertaken with or without contrast media. Ionising radiation exposure enhances the lifetime risk of cancer, and this is a particular concern for younger individuals. CMR involves objective measures of cardiac dimensions, function and pathology. Access to advanced cardiovascular imaging varies by postcode, being much more widely available in London and academic medical centres in England than in regional centres, especially in Scotland, Wales and Northern Ireland.⁹⁹ There are no data to confirm that access to advanced imaging differentiates by sex, but as women are recognised as an underserved group, they may be disproportionately affected. When options are available for diagnostic tests, patient-specific factors should be considered. Women may have preferences on the type of test, such as avoiding invasive coronary angiography (in preference for non-invasive computed

tomography (CT)), or ionising radiation exposure, or multiple visits.

Women are more likely to experience microvascular angina due to small vessel disease.⁴⁴ Microvascular angina is likely to be associated with impaired myocardial perfusion (at rest and/or during stress), therefore stress testing (by exercise, CMR, echocardiography, nuclear) is likely to be abnormal, despite a normal angiogram. For these reasons, men are more likely to receive a true positive diagnosis and women are more likely to receive a false negative diagnosis when following current guidance for investigation of chest pain.¹⁰⁰ Therefore, women are more likely to have unexplained chest pain,¹⁰¹ potentially leading to disparities in healthcare. Appropriate use of CMR and other non-invasive modalities may mitigate this.

Computed tomography coronary angiography (CTCA)

CTCA is an effective modality to investigate stable chest pain, and its use in women as a first-line investigation when compared with functional testing has been shown to result in fewer downstream diagnostic tests along with reduced costs.¹⁰² The strength of CTCA lies in its high sensitivity and negative predictive value. The presence of coronary artery calcium confers an increased mortality risk in women compared with men.¹⁰³ While CTCA provides accurate diagnostic information by identifying obstructive CAD, it can also guide commencement of preventative therapy. CTCA allows assessment of plaque composition and identification of high-risk features such as positive remodelling, low attenuation, spotty calcifications and napkin ring sign. The presence of high-risk plaques has been shown to increase the risk of major adverse cardiovascular events in women compared with men.¹⁰⁴

CT may have a role in assessing for SCAD in proximal and mid coronary vessels. However, the appearances of dissection flaps and intramural haematoma may be challenging to diagnose on a CT scan, particularly when it involves the distal vessels. CT also provides accurate annulus measurements as well as assessment for peripheral access for TAVI patients.¹⁰⁵ The radiation dose to breast tissue needs to be borne in mind. The availability of multidetector CT scans and adjusting scan protocols with ECG modulation can reduce the radiation burden.

Nuclear imaging

Single-photon emission computed tomography (SPECT) myocardial imaging is a widely available imaging modality for investigation of chest pain. The lower LV volume in women and the presence of breast tissue affects the diagnostic accuracy.¹⁰⁶ Additional tools such as CT-based attenuation correction, upright and prone scanning should be used to mitigate the effects of breast tissue. Radiation burden also needs to be considered. Positron emission tomography (PET) myocardial perfusion imaging (MPI) has a higher accuracy than SPECT and is associated with lower radiation dose.¹⁰⁶ Studies have shown that the tracer Flurpiridaz F1-18 is useful in the evaluation of CAD, particularly in women.¹⁰⁷ 18F-Sodium fluoride PET MPI can be used to assess microcalcification in the coronary arteries and also has a role in assessing plaque progression.¹⁰⁸ Fluorodeoxyglucose (FDG) PET imaging plays a role in the diagnostic and prognostic evaluation of sarcoidosis and myocarditis.^{109 110}

PRIMARY CARE MANAGEMENT OF CVD IN WOMEN

Given that the demand on the NHS far exceeds the supply of healthcare professionals, enabling the system to focus on value-based medicine across the whole patient pathway (what we call

‘unified value’) and a greater focus on prevention are both essential. Furthermore, enabling all care professionals to focus on the risk of CVD in women through culture change, education, data and contracts is the key to raising its priority, ensuring the NHS delivers care where needed most.

Importantly, care professionals have a lower perception of CVD risk in women. A study based on primary care has shown that women at high risk of or with established CVD are less likely to be prescribed CVD medication.¹¹¹ The longer-term risks related to the development of diabetes or hypertension during pregnancy are well established, yet the evidence suggests that women experiencing such complications in pregnancy are not routinely followed up.¹¹² There is no doubt that women’s CVD health should be made a priority, but it requires the political will to ignite and enable change (table 2, online supplemental table S3).

NURSING PERSPECTIVE ON THE MANAGEMENT OF CVD IN WOMEN

Uniquely placed to provide holistic support along the entire health pathway, nurses play a crucial role in addressing the distinctive challenges associated with CVD in women. Nurses have responsibility for providing individualised care for women, taking into account women’s unique physiological and psychosocial factors.¹¹³ Nurses are advocates for women, challenging considerable power dynamics to help women navigate complex treatment plans. As the largest workforce within the NHS and working closely with patients, nurses are in a position to undertake regular risk assessment and screening for CVD in women. Nurses need to recognise that women may present with different symptoms or risk factors compared with men and ensure that diagnostic tools and screening guidelines are sensitive to these differences. Nurses also have an important role in providing patient education to enable effective self-care.⁶⁸ By empowering women with context-specific empathetic knowledge gained from real-world experiences, nurses enable women to make informed decisions about their health and seek timely medical assistance if required. It is critical that nurses collaborate with the multidisciplinary team, such as exercise physiologists and physiotherapists, to design and deliver personalised care programmes for women. Nurses have a lot to contribute to research efforts aimed at advancing our understanding of CVD in women and improving outcomes through evidence-based practice.¹¹⁴ They can ensure cardiovascular teams stay updated on the latest research findings, clinical guidelines and best practices in women’s cardiovascular care.

PATIENT PERSPECTIVE ON THE MANAGEMENT OF CVD IN WOMEN

The misconception that heart disease seems to only affect men urgently needs correcting. Myths and unconscious biases within clinical practices and societal perceptions further obscure the reality that heart disease does not discriminate by sex.

The importance of screening for cervical and breast cancer among women is already well understood; however, the awareness surrounding the significance of CVD lags significantly behind. Women do not always have a positive experience of cardiology care. Many report feeling they are not listened to, with their symptoms attributed to non-cardiac causes.¹¹⁵ It is common for women to be mistakenly reassured that their heart is fine, only to be later diagnosed with CVD. Women may need to be persistent and make repeated visits to medical staff before their heart symptoms are recognised. This issue calls for a

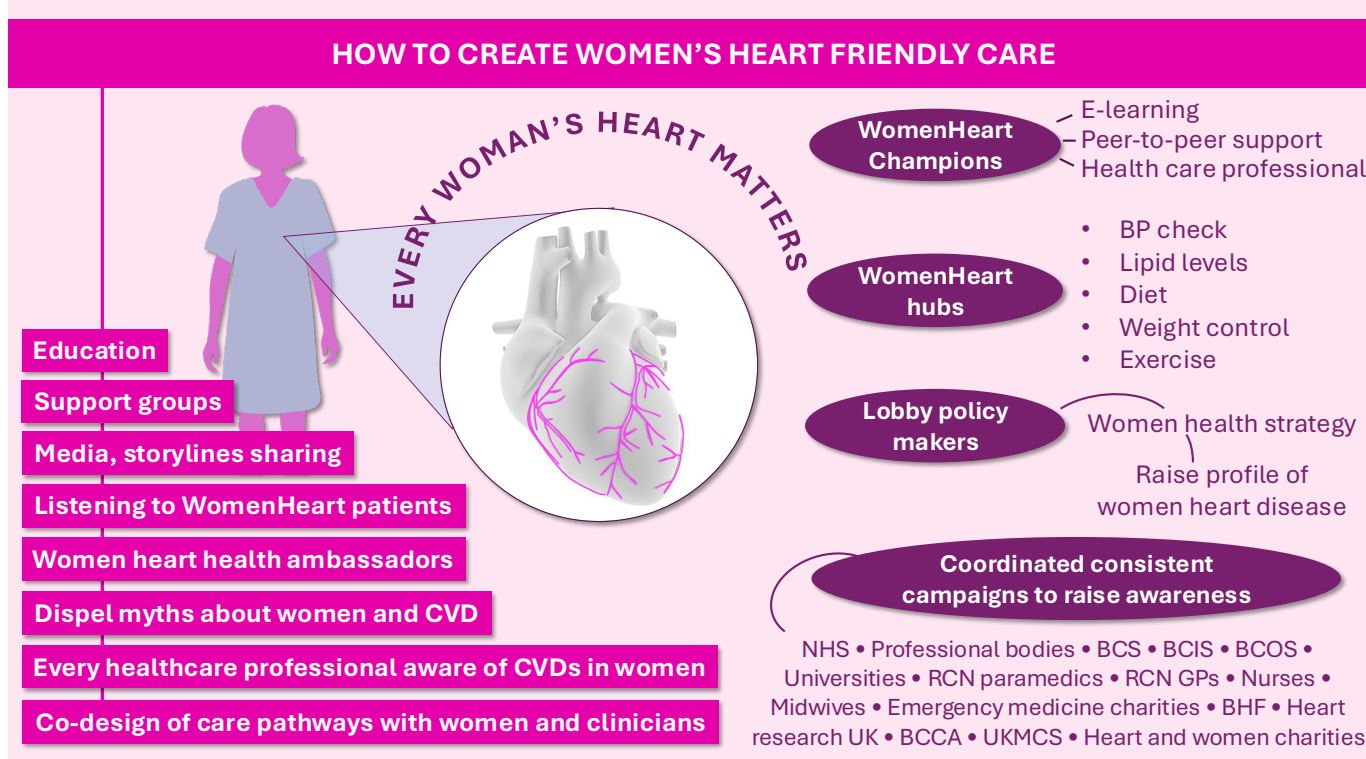


Figure 3 How to create women's heart friendly care. BCCA, British Congenital Cardiac Association; BCIS, British Cardiovascular Intervention Society; BCOS, British Cardio-Oncology Society; BCS, British Cardiovascular Society; BHF, British Heart Foundation; BP, blood pressure; CVD, cardiovascular disease; GPs, general practitioners; NHS, National Health Service; RCN, Royal College of Nursing; UK, United Kingdom; UKMCS, UK Maternal Cardiology Society.

holistic woman-centred approach to heart care that incorporates patients' experiences and insights (see figure 3 and table 3).

CONCLUSION

Despite significant progress in the management of CVD, it remains UK's number one killer for women. Unfortunately, women are underdiagnosed, undertreated and under-represented in all CVD areas. This consensus outlines actionable points provided by each of the affiliated societies to address the sex disparities in everyday care of patients in all settings, aiming at saving many women from losing their lives unnecessarily from preventable conditions in the UK and also worldwide.

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REFERENCES

- Virani SS, Alonso A, Aparicio HJ, *et al.* Heart Disease and Stroke Statistics-2021 Update: a Report from the American Heart Association. *Circulation* 2021;143:e254–743.
- Vogel B, Acevedo M, Appelman Y, *et al.* The Lancet women and cardiovascular disease Commission: reducing the global burden by 2030. *Lancet* 2021;397:2385–438.
- British Heart Foundation. UK factsheet. 2024. Available: <https://www.bhf.org.uk/-/media/files/for-professionals/research/heart-statistics/bhf-cvd-statistics-uk-factsheet.pdf>
- BCS. Management of heart attack: myocardial ischaemia national audit project (minap) with reference to the national audit of percutaneous coronary intervention (napci). 2024 summary report. 2024. Available: <https://www.nicor.org.uk/publications/ncap/heart-attack/2024-2/minap-final-report-2022-23>
- Tobb K, Kocher M, Bullock-Palmer RP. Underrepresentation of women in cardiovascular trials- it is time to shatter this glass ceiling. *Am Heart J Plus* 2022;13:100109.
- Tribouilloy C, Bohbot Y, Rusinaru D, *et al.* Excess Mortality and Undertreatment of Women With Severe Aortic Stenosis. *J Am Heart Assoc* 2021;10:e018816.
- Kaufman MR, Eschliman EL, Karver TS. Differentiating sex and gender in health research to achieve gender equity. *Bull World Health Organ* 2023;101:666–71.
- Pinho-Gomes AC, Peters SAE, Thomson B, *et al.* Sex differences in prevalence, treatment and control of cardiovascular risk factors in England. *Heart* 2021;107:462–7.
- Falascchetti E, Mindell J, Knott C, *et al.* Hypertension management in England: a serial cross-sectional study from 1994 to 2011. *Lancet* 2014;383:1912–9.
- Gerds E, Sudano I, Brouwers S, *et al.* Sex differences in arterial hypertension. *Eur Heart J* 2022;43:4777–88.
- Huxley RR, Woodward M. Cigarette smoking as a risk factor for coronary heart disease in women compared with men: a systematic review and meta-analysis of prospective cohort studies. *Lancet* 2011;378:1297–305.
- Huxley R, Barzi F, Woodward M. Excess risk of fatal coronary heart disease associated with diabetes in men and women: meta-analysis of 37 prospective cohort studies. *BMJ* 2006;332:73–8.
- Broni EK, Ndumele CE, Echouffo-Tcheugui JB, *et al.* The Diabetes-Cardiovascular Connection in Women: Understanding the Known Risks, Outcomes, and Implications for Care. *Curr Diab Rep* 2022;22:11–25.
- Wilson PWF, D'Agostino RB, Sullivan L, *et al.* Overweight and obesity as determinants of cardiovascular risk: the Framingham experience. *Arch Intern Med* 2002;162:1867–72.
- Rajendran A, Minhas AS, Kazzi B, *et al.* Sex-specific differences in cardiovascular risk factors and implications for cardiovascular disease prevention in women. *Atherosclerosis* 2023;384.
- Mehta LS, Velarde GP, Lewey J, *et al.* Cardiovascular Disease Risk Factors in Women: The Impact of Race and Ethnicity: a Scientific Statement From the American Heart Association. *Circulation* 2023;147:1471–87.
- Sakkers TR, Mokry M, Civelek M, *et al.* Sex differences in the genetic and molecular mechanisms of coronary artery disease. *Atherosclerosis* 2023;384.
- Ghadri JR, Wittstein IS, Prasad A, *et al.* International Expert Consensus Document on Takotsubo Syndrome (Part I): Clinical Characteristics, Diagnostic Criteria, and Pathophysiology. *Eur Heart J* 2018;39:2032–46.
- O'Kelly AC, Michos ED, Shufelt CL, *et al.* Pregnancy and Reproductive Risk Factors for Cardiovascular Disease in Women. *Circ Res* 2022;130:652–72.
- Curtis KM, Mohlajee AP, Martins SL, *et al.* Combined oral contraceptive use among women with hypertension: a systematic review. *Contraception* 2006;73:179–88.
- 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;42:967–84.
- Boardman HMP, Hartley L, Eisinga A, *et al.* Hormone therapy for preventing cardiovascular disease in post-menopausal women. *Cochrane Database Syst Rev* 2015;2015:CD002229.
- Byrne RA, Rossello X, Coughlan JJ, *et al.* 2023 ESC Guidelines for the management of acute coronary syndromes. *Eur Heart J* 2023;44:3720–826.
- Michalopoulou M, Piernas C, Jebb SA, *et al.* Association of gestational diabetes with long-term risk of premature mortality, and cardiovascular outcomes and risk factors: a retrospective cohort analysis in the UK Biobank. *Diabetes Obes Metab* 2024;26:2915–24.
- Tschiderer L, Seekircher L, Willeit P, *et al.* Assessment of Cardiovascular Risk in Women: Progress so Far and Progress to Come. *Int J Womens Health* 2023;15:191–212.
- Hippisley-Cox J, Coupland CAC, Bafadhel M, *et al.* Development and validation of a new algorithm for improved cardiovascular risk prediction. *Nat Med* 2024;30:1440–7.
- Gaudino M, Di Franco A, Cao D, *et al.* Sex-Related Outcomes of Medical, Percutaneous, and Surgical Interventions for Coronary Artery Disease: JACC Focus Seminar 3/7. *J Am Coll Cardiol* 2022;79:1407–25.
- Reynolds HR, Cyr DD, Merz CNB, *et al.* Sex Differences in Revascularization, Treatment Goals, and Outcomes of Patients With Chronic Coronary Disease: Insights From the ISCHEMIA Trial. *J Am Heart Assoc* 2024;13:e029850.
- Qian J, Maehara A, Mintz GS, *et al.* Impact of gender and age on in vivo virtual histology-intravascular ultrasound imaging plaque characterization (from the global Virtual Histology Intravascular Ultrasound [VH-IVUS] registry). *Am J Cardiol* 2009;103:1210–4.
- Ruiz-García J, Lerman A, Weisz G, *et al.* Age- and gender-related changes in plaque composition in patients with acute coronary syndrome: the PROSPECT study. *EuroIntervention* 2012;8:929–38.
- Gurgoglione FL, Solinas E, Pflieger B, *et al.* Coronary atherosclerotic plaque phenotype and pathophysiologic mechanisms: Is there an influence of sex? Insights from intracoronary imaging. *Atherosclerosis* 2023;384:117273.
- Lawless M, Appelman Y, Beltrame JF, *et al.* Sex differences in treatment and outcomes amongst myocardial infarction patients presenting with and without obstructive coronary arteries: a prospective multicentre study. *Eur Heart J Open* 2023;3:oead033.
- Kunadian V, Qiu W, Bawamia B, *et al.* Gender comparisons in cardiogenic shock during ST elevation myocardial infarction treated by primary percutaneous coronary intervention. *Am J Cardiol* 2013;112:636–41.

- 34 Burgess SN, Juergens CP, Nguyen TL, *et al.* Comparison of Late Cardiac Death and Myocardial Infarction Rates in Women Vs Men With ST-Elevation Myocardial Infarction. *Am J Cardiol* 2020;128:120–6.
- 35 Sambola A, Del Blanco BG, Kunadian V, *et al.* Sex-based Differences in Percutaneous Coronary Intervention Outcomes in Patients with Ischaemic Heart Disease. *Eur Cardiol* 2023;18:e06.
- 36 Nadarajah R, Ludman P, Laroche C, *et al.* Sex-specific presentation, care, and clinical events in individuals admitted with NSTEMI: the ACVC-EAPCI EORP NSTEMI registry of the European Society of Cardiology. *Eur Heart J Acute Cardiovasc Care* 2024;13:36–45.
- 37 Tan YC, Sinclair H, Ghoorah K, *et al.* Gender differences in outcomes in patients with acute coronary syndrome in the current era: a review. *Eur Heart J Acute Cardiovasc Care* 2016;5:51–60.
- 38 Kunadian V, Qiu W, Lagerqvist B, *et al.* Gender Differences in Outcomes and Predictors of All-Cause Mortality After Percutaneous Coronary Intervention (Data from United Kingdom and Sweden). *Am J Cardiol* 2017;119:210–6.
- 39 Jackson J, Alkhalil M, Ratcovich H, *et al.* Evidence base for the management of women with non-ST elevation acute coronary syndrome. *Heart* 2022;108:1682–9.
- 40 Balasubramanian RN, Mills GB, Wilkinson C, *et al.* Role and relevance of risk stratification models in the modern-day management of non-ST elevation acute coronary syndromes. *Heart* 2023;109:504–10.
- 41 Vogel B, Baber U, Cohen DJ, *et al.* Sex Differences Among Patients With High Risk Receiving Ticagrelor With or Without Aspirin After Percutaneous Coronary Intervention: a Subgroup Analysis of the TWILIGHT Randomized Clinical Trial. *JAMA Cardiol* 2021;6:1032–41.
- 42 Sinha A, Dutta U, Demir OM, *et al.* Rethinking False Positive Exercise Electrocardiographic Stress Tests by Assessing Coronary Microvascular Function. *J Am Coll Cardiol* 2024;83:291–9.
- 43 Sinha A, Rahman H, Douiri A, *et al.* ChaMP-CMD: a Phenotype-Blinded, Randomized Controlled, Cross-Over Trial. *Circulation* 2024;149:36–47.
- 44 Ford TJ, Stanley B, Good R, *et al.* Stratified Medical Therapy Using Invasive Coronary Function Testing in Angina: the CorMicA Trial. *J Am Coll Cardiol* 2018;72:2841–55.
- 45 Pizzi C, Xhyheri B, Costa GM, *et al.* Nonobstructive Versus Obstructive Coronary Artery Disease in Acute Coronary Syndrome: a Meta-Analysis. *J Am Heart Assoc* 2016;5:e004185.
- 46 Safdar B, Spatz ES, Dreyer RP, *et al.* Presentation, Clinical Profile, and Prognosis of Young Patients With Myocardial Infarction With Nonobstructive Coronary Arteries (MINOCA): results From the VIRGO Study. *J Am Heart Assoc* 2018;7:e009174.
- 47 DesJardin JT, Chikwe J, Hahn RT, *et al.* Sex Differences and Similarities in Valvular Heart Disease. *Circ Res* 2022;130:455–73.
- 48 Hahn RT, Clavel MA, Mascherbauer J, *et al.* Sex-Related Factors in Valvular Heart Disease: JACC Focus Seminar 5/7. *J Am Coll Cardiol* 2022;79:1506–18.
- 49 Kong WKF, Bax JJ, Michelena HI, *et al.* Sex differences in bicuspid aortic valve disease. *Prog Cardiovasc Dis* 2020;63:452–6.
- 50 Hariri EH, El Halabi J, Kassis N, *et al.* Sex Differences in the Progression and Long-Term Outcomes of Native Mild to Moderate Aortic Stenosis. *JACC Cardiovasc Imaging* 2024;17:1–12.
- 51 National Cardiac Audit Programme BCIS. Transcatheter aortic valve implantation (tavi) registry. 2024 summary report (2022/23 data). 2024. Available: <https://www.nicor.org.uk/publications/ncap/uk-transcatheter-aortic-valve-implantation/2024-8/tavi-final-report-2022-23>
- 52 Akintoye E, Saijo Y, Braghieri L, *et al.* Impact of Age and Sex on Left Ventricular Remodeling in Patients With Aortic Regurgitation. *J Am Coll Cardiol* 2023;81:1474–87.
- 53 Jander N, Minners J. Towards a gender-specific and morphology-specific assessment of aortic valve stenosis severity. *Heart* 2024;110:543–4.
- 54 Dewan P, Rorth R, Raparelli V, *et al.* Sex-Related Differences in Heart Failure With Preserved Ejection Fraction. *Circ Heart Fail* 2019;12:e006539.
- 55 Stolfo D, Uijl A, Vedin O, *et al.* Sex-Based Differences in Heart Failure Across the Ejection Fraction Spectrum: Phenotyping, and Prognostic and Therapeutic Implications. *JACC Heart Fail* 2019;7:505–15.
- 56 Stewart S, Playford D, Scalia GM, *et al.* Ejection fraction and mortality: a nationwide register-based cohort study of 499 153 women and men. *Eur J Heart Fail* 2021;23:406–16.
- 57 Lainščak M, Milinković I, Polovina M, *et al.* Sex- and age-related differences in the management and outcomes of chronic heart failure: an analysis of patients from the ESC HFA EORP Heart Failure Long-Term Registry. *Eur J Heart Fail* 2020;22:92–102.
- 58 Santema BT, Ouwerkerk W, Tromp J, *et al.* Identifying optimal doses of heart failure medications in men compared with women: a prospective, observational, cohort study. *Lancet* 2019;394:1254–63.
- 59 Solomon SD, Vaduganathan M, L. Claggett B, *et al.* Sacubitril/Valsartan Across the Spectrum of Ejection Fraction in Heart Failure. *Circulation* 2020;141:352–61.
- 60 McMurray JJV, Jackson AM, Lam CSP, *et al.* Effects of Sacubitril-Valsartan Versus Valsartan in Women Compared With Men With Heart Failure and Preserved Ejection Fraction: Insights From PARAGON-HF. *Circulation* 2020;141:338–51.
- 61 Pitt B, Pfeffer MA, Assmann SF, *et al.* Spironolactone for heart failure with preserved ejection fraction. *N Engl J Med* 2014;370:1383–92.
- 62 Curtis LH, Al-Khatib SM, Shea AM, *et al.* Sex differences in the use of implantable cardioverter-defibrillators for primary and secondary prevention of sudden cardiac death. *JAMA* 2007;298:1517–24.
- 63 Chatterjee NA, Borgquist R, Chang Y, *et al.* Increasing sex differences in the use of cardiac resynchronization therapy with or without implantable cardioverter-defibrillator. *Eur Heart J* 2017;38:1485–94.
- 64 Beela AS, Duchenne J, Petrescu A, *et al.* Sex-specific difference in outcome after cardiac resynchronization therapy. *Eur Heart J Cardiovasc Imaging* 2019;20:504–11.
- 65 MacGowan GA, McDiarmid A, Jansen K, *et al.* Gender differences in the assessment, decision-making and outcomes for ventricular assist devices and heart transplantation: an analysis from a UK transplant center. *Clin Transplant* 2022;36:e14666:20220418.
- 66 Kosmidou I, Lindenfeld J, Abraham WT, *et al.* Sex-Specific Outcomes of Transcatheter Mitral-Valve Repair and Medical Therapy for Mitral Regurgitation in Heart Failure. *JACC Heart Fail* 2021;9:674–83.
- 67 Follath F, Yilmaz MB, Delgado JF, *et al.* Clinical presentation, management and outcomes in the Acute Heart Failure Global Survey of Standard Treatment (ALARMS-HF). *Intensive Care Med* 2011;37:619–26.
- 68 Jaarsma T, Hill L, Bayes-Genis A, *et al.* Self-care of heart failure patients: practical management recommendations from the Heart Failure Association of the European Society of Cardiology. *Eur J Heart Fail* 2021;23:157–74.
- 69 Adalsteinsdottir B, Palsson R, Desnick RJ, *et al.* Fabry Disease in Families With Hypertrophic Cardiomyopathy: Clinical Manifestations in the Classic and Later-Onset Phenotypes. *Circ Cardiovasc Genet* 2017;10:e001639.
- 70 Lotan D, Salazar-Mendiguchia J, Mogensen J, *et al.* Clinical Profile of Cardiac Involvement in Danon Disease: a Multicenter European Registry. *Circ Genom Precis Med* 2020;13:e003117.
- 71 Lim KRQ, Sheri N, Nguyen Q, *et al.* Cardiac Involvement in Dystrophin-Deficient Females: current Understanding and Implications for the Treatment of Dystrophinopathies. *Genes (Basel)* 2020;11:765:11.
- 72 Gaita F, Cerrato N, Giustetto C, *et al.* Asymptomatic Patients With Brugada ECG Pattern: Long-Term Prognosis From a Large Prospective Study. *Circulation* 2023;148:1543–55.
- 73 Seidelmann SB, Laur O, Hwa J, *et al.* Familial dilated cardiomyopathy diagnosis is commonly overlooked at the time of transplant listing. *J Heart Lung Transplant* 2016;35:474–80.
- 74 Akhtar MM, Lorenzini M, Cicerchia M, *et al.* Clinical Phenotypes and Prognosis of Dilated Cardiomyopathy Caused by Truncating Variants in the *TTN* Gene. *Circ Heart Fail* 2020;13:e006832.
- 75 Liu G, Su L, Lang M. A systematic review and meta-analysis of sex differences in clinical outcomes of hypertrophic cardiomyopathy. *Front Cardiovasc Med* 2023;10:1252266.
- 76 Lorenzini M, Anastasiou Z, O'Mahony C, *et al.* Mortality Among Referral Patients With Hypertrophic Cardiomyopathy vs the General European Population. *JAMA Cardiol* 2020;5:73–80.
- 77 Zareba W, Moss AJ, Locati EH, *et al.* Modulating effects of age and gender on the clinical course of long QT syndrome by genotype. *J Am Coll Cardiol* 2003;42:103–9.
- 78 Rootwelt-Norberg C, Lie ØH, Chivulescu M, *et al.* Sex differences in disease progression and arrhythmic risk in patients with arrhythmogenic cardiomyopathy. *Europace* 2021;23:1084–91.
- 79 Iorgoveanu C, Zaghloul A, Ashwath M. Peripartum cardiomyopathy: a review. *Heart Fail Rev* 2021;26:1287–96.
- 80 Ware JS, Li J, Mazaika E, *et al.* Shared Genetic Predisposition in Peripartum and Dilated Cardiomyopathies. *N Engl J Med* 2016;374:233–41.
- 81 Oliver-Williams C, Stevens D, Payne RA, *et al.* Association between hypertensive disorders of pregnancy and later risk of cardiovascular outcomes. *BMC Med* 2022;20:19.
- 82 Tsao CW, Aday AW, Almarazooq ZI, *et al.* Heart Disease and Stroke Statistics-2022 Update: a Report From the American Heart Association. *Circulation* 2022;145:e153–639.
- 83 Pugnali F, Felici A, Corno AF, *et al.* Gender differences in congenital heart defects: a narrative review. *Transl Pediatr* 2023;12:1753–64.
- 84 Verheugt CL, Uiterwaal CSPM, van der Velde ET, *et al.* Gender and outcome in adult congenital heart disease. *Circulation* 2008;118:26–32.
- 85 Swan L, Windram J, Burchill L, *et al.* Sexual Health and Well-Being in Adults With Congenital Heart Disease: a International Society of Adult Congenital Heart Disease Statement. *JACC Adv* 2023;2:100716.
- 86 Linde C, Bongioni MG, Birgersdotter-Green U, *et al.* Sex differences in cardiac arrhythmia: a consensus document of the European Heart Rhythm Association, endorsed by the Heart Rhythm Society and Asia Pacific Heart Rhythm Society. *Europace* 2018;20:1565.
- 87 Prajapati C, Koivumäki J, Pekkanen-Mattila M, *et al.* Sex differences in heart: from basics to clinics. *Eur J Med Res* 2022;27:241.
- 88 Kurokawa J, Kodama M, Clancy CE, *et al.* Sex hormonal regulation of cardiac ion channels in drug-induced QT syndromes. *Pharmacol Ther* 2016;168:23–8.
- 89 Saleh K, Haldar S. Atrial fibrillation: a contemporary update. *Clin Med (Lond)* 2023;23:437–41.

- 90 Subramanya V, Claxton JS, Lutsey PL, *et al.* Sex differences in treatment strategy and adverse outcomes among patients 75 and older with atrial fibrillation in the MarketScan database. *BMC Cardiovasc Disord* 2021;21:598.
- 91 Lopes LR, Zekavati A, Syrris P, *et al.* Genetic complexity in hypertrophic cardiomyopathy revealed by high-throughput sequencing. *J Med Genet* 2013;50:228–39.
- 92 Lyon AR, López-Fernández T, Couch LS, *et al.* 2022 ESC Guidelines on cardio-oncology developed in collaboration with the European Hematology Association (EHA), the European Society for Therapeutic Radiology and Oncology (ESTRO) and the International Cardio-Oncology Society (IC-OS). *Eur Heart J* 2022;43:4229–361.
- 93 Finkelman BS, Putt M, Wang T, *et al.* Arginine-Nitric Oxide Metabolites and Cardiac Dysfunction in Patients With Breast Cancer. *J Am Coll Cardiol* 2017;70:152–62.
- 94 Unger JM, Vaidya R, Albain KS, *et al.* Sex Differences in Risk of Severe Adverse Events in Patients Receiving Immunotherapy, Targeted Therapy, or Chemotherapy in Cancer Clinical Trials. *J Clin Oncol* 2022;40:1474–86.
- 95 Thavendiranathan P, Houbois C, Marwick TH, *et al.* Statins to prevent early cardiac dysfunction in cancer patients at increased cardiotoxicity risk receiving anthracyclines. *Eur Heart J Cardiovasc Pharmacother* 2023;9:515–25.
- 96 Neuback L, McHale S, Ross M, *et al.* Spontaneous coronary artery dissection: a systematic review of physical and psychosocial recovery following discharge from hospital. *Eur J Cardiovasc Nurs* 2022;21:665–76.
- 97 Vidal-Almela S, Czajkowski B, Prince SA, *et al.* Lessons learned from community- and home-based physical activity programs: a narrative review of factors influencing women's participation in cardiac rehabilitation. *Eur J Prev Cardiol* 2021;28:761–78.
- 98 Robinson S, Rana B, Oxborough D, *et al.* A practical guideline for performing a comprehensive transthoracic echocardiogram in adults: the British Society of Echocardiography minimum dataset. *Echo Res Pract* 2020;7:G59–93.
- 99 Keenan NG, Captur G, McCann GP, *et al.* Regional variation in cardiovascular magnetic resonance service delivery across the UK. *Heart* 2021;107:1974–9.
- 100 Berry C, Kramer CM, Kunadian V, *et al.* Great Debate: computed tomography coronary angiography should be the initial diagnostic test in suspected angina. *Eur Heart J* 2023;44:2366–75.
- 101 Mangion K, Adamson PD, Williams MC, *et al.* Sex associations and computed tomography coronary angiography-guided management in patients with stable chest pain. *Eur Heart J* 2020;41:1337–45.
- 102 Lubbers M, Coenen A, Bruning T, *et al.* Sex Differences in the Performance of Cardiac Computed Tomography Compared With Functional Testing in Evaluating Stable Chest Pain: subanalysis of the Multicenter, Randomized CRESCENT Trial (Calcium Imaging and Selective CT Angiography in Comparison to Functional Testing for Suspected Coronary Artery Disease). *Circ Cardiovasc Imaging* 2017;10:e005295.
- 103 Shaw LJ, Min JK, Nasir K, *et al.* Sex differences in calcified plaque and long-term cardiovascular mortality: observations from the CAC Consortium. *Eur Heart J* 2018;39:3727–35.
- 104 Douglas PS, Hoffmann U, Patel MR, *et al.* Outcomes of anatomical versus functional testing for coronary artery disease. *N Engl J Med* 2015;372:1291–300.
- 105 Blanke P, Weir-McCall JR, Achenbach S, *et al.* Computed tomography imaging in the context of transcatheter aortic valve implantation (TAVI) / transcatheter aortic valve replacement (TAVR): an expert consensus document of the Society of Cardiovascular Computed Tomography. *J Cardiovasc Comput Tomogr* 2019;13:1–20.
- 106 Mikail N, Rossi A, Bengs S, *et al.* Imaging of heart disease in women: review and case presentation. *Eur J Nucl Med Mol Imaging* 2022;50:130–59.
- 107 Maddahi J, Agostini D, Bateman TM, *et al.* Flurpiridaz F-18 PET Myocardial Perfusion Imaging in Patients With Suspected Coronary Artery Disease. *J Am Coll Cardiol* 2023;82:1598–610.
- 108 Singh SB, Ng SJ, Lau HC, *et al.* Emerging PET Tracers in Cardiac Molecular Imaging. *Cardiol Ther* 2023;12:85–99.
- 109 Wicks EC, Menezes LJ, Barnes A, *et al.* Diagnostic accuracy and prognostic value of simultaneous hybrid 18F-fluorodeoxyglucose positron emission tomography/magnetic resonance imaging in cardiac sarcoidosis. *Eur Heart J Cardiovasc Imaging* 2018;19:757–67.
- 110 Cheng RK, Kittleson MM, Beavers CJ, *et al.* Diagnosis and Management of Cardiac Sarcoidosis: a Scientific Statement From the American Heart Association. *Circulation* 2024;149:e1197–216.
- 111 Zhao M, Woodward M, Vaartjes I, *et al.* Sex Differences in Cardiovascular Medication Prescription in Primary Care: a Systematic Review and Meta-Analysis. *J Am Heart Assoc* 2020;9:e014742.
- 112 Kramer CK, Campbell S, Retnakaran R. Gestational diabetes and the risk of cardiovascular disease in women: a systematic review and meta-analysis. *Diabetologia* 2019;62:905–14.
- 113 ProMedical. Supporting women's healthcare: 20 ways nurses can empower their female patients and colleagues. 2023. Available: <https://www.promedical.co.uk/blog/supporting-womens-healthcare/>
- 114 Farquharson B, Austin R, Bernhardt L, *et al.* The life cycle of the lesser-spotted cardiac nurse researcher. *Br J Cardiac Nurs* 2021;16:1–9.
- 115 Gulati M, Khan N, George M, *et al.* Ischemia with no obstructive coronary artery disease (INOCA): a patient self-report quality of life survey from INOCA international. *Int J Cardiol* 2023;371:28–39.