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POSITION PAPER

The right heart in patients with cancer. A scientific statement of the Heart Failure Association (HFA) of the ESC and the ESC Council of Cardio-Oncology

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Introduction

Cardio-oncology guidelines, position statements and clinical trials have focused on the cardiotoxic effects of cancer therapies upon the left ventricle.[1,2] However, cancer therapies, including chemotherapy, targeted therapies, immunotherapies and radiation therapy (RT) may also have detrimental effects on the right heart (RH). These effects may manifest as right ventricular (RV) dysfunction, pulmonary hypertension (PH), pericardial disease and valvular abnormalities. The prevalence and clinical significance of RH involvement in patients treated with cancer therapies are being increasingly recognized,[3] as these conditions can significantly impact treatment decisions, prognosis, and overall patient outcomes.[4]

In this scientific statement, we aim to discuss the impact of cancer therapies on the RH and explore the importance of RH assessment in patients with cancer. We discuss the prevalence and the mechanisms of RV dysfunction in cancer patients, the challenges and limitations of traditional imaging modalities, and the role of multimodality imaging in evaluating the RH. The integration of RH assessment into cardio-oncology practice will aid healthcare professionals to proactively manage cardiovascular (CV) complications, optimize cancer treatment strategies, and potentially improve clinical outcomes.

Right ventricular dysfunction in cancer patients

Right ventricular dysfunction may pre-exist in cancer patients at diagnosis or it may be exacerbated by or may develop de novo due to cardiotoxic cancer therapy (anti-neoplastic medications and RT) or due to direct CV complications of the cancer itself (e.g. PH, venous thromboembolism [VTE] and pulmonary embolism [PE], primary or metastatic cardiac tumours, pericardial involvement, or endocarditis). Carcinoid heart disease (CHD) and amyloidosis are special entities where RH involvement is crucial (*Figure 1*). There is no universal definition of cancer therapy-related RV toxicity (CTR-RVT). Based on the current literature, the recent universal definition of heart failure[5] and the 2022 European Society of Cardiology (ESC) guidelines on cardio-oncology,[2] CTR-RVT can be divided into asymptomatic RV dysfunction and symptomatic RV failure, analogous to the concept of cancer therapy-related cardiac dysfunction (CTRCD) for the left ventricle (LV). In this document, we propose the following definitions (*Table 1*).

Asymptomatic RV dysfunction refers to the presence of impaired RV function without overt clinical symptoms or signs, where a subclinical alteration in RV performance can be detected by transthoracic echocardiography (TTE). It can be defined as new relative decline in RV free wall longitudinal strain (RV FWLS) by >15% from the baseline value without significant change of the standard two-dimensional (2D)-echocardiographically assessed parameters (tricuspid annular plane systolic excursion [TAPSE], RV fractional area change [FAC], RV myocardial performance index [MPI], RV S′ or three-dimensional [3D]-assessed RV ejection fraction [RVEF]).

Symptomatic RV failure can be defined as a condition where symptoms and/or signs of RV failure, such as dyspnoea, exercise intolerance, peripheral oedema, are caused by a structural and/or functional abnormality of the RH.

Although there are some data based on RV systolic function assessment by RVEF either by 3D echocardiography[6–9] or by cardiac magnetic resonance (CMR),[10–14] this parameter is not included in the definition of asymptomatic RV dysfunction since 3D echocardiography and CMR are not widely available for the surveillance of asymptomatic cancer patients. Clearly, more work is needed to validate these criteria for the diagnosis of RV cardiotoxicity.

Right ventricular dysfunction induced by systemic cancer therapy

Several cancer therapies may cause RV dysfunction through a number of different mechanisms including direct myocardial toxicity, myocarditis, myocardial ischaemia and/or increased afterload (systemic or pulmonary). Several studies have reported RV dysfunction in cancer patients and survivors, but the number of patients in these studies is relatively small and the methods and cut-off values vary significantly. Most of the studies focus on anthracycline-induced cardiotoxicity,[6,7,9,10,15–27] some evaluate breast cancer patients receiving trastuzumab with or without anthracyclines,[11,28–36] and only a few address the impact of complex chemotherapeutic regimens.[37,38] Reports of immune checkpoint inhibitor-induced RV dysfunction[39] or myocarditis involving the RV apart from the LV[40] or restricted to the RV[41,42] have remained rare.

Results on the effect of chemotherapy on RV diameters and volumes have been conflicting with some studies supporting RV remodelling,[7,10,11,23] while others do not identify significant changes.[9,15,19,29,33,43] This is similar to the results concerning RV systolic function. While some studies do not report significant changes,[15–17,28,29,33,37] others reveal consistent decreases in RV FAC,[9,19,22,23,25,26] TAPSE[9,19,26,43] and RVEF.[7,11,43] On the contrary, RV longitudinal strain (RVLS) was decreased by chemotherapy in almost all studies[6,7,9,16–23,25,27,29,35] with the most consistent results for RV FWLS. The timing of RV dysfunction compared to LV dysfunction is another topic of debate; most[19,22,29,34,35] but not all studies have shown concurrent biventricular insult. Importantly, RV dysfunction expressed by the change in RVLS seems to predict LV cardiotoxicity in some trials.[9,10,23,37] Lastly, the effect of anthracyclines on the RV seems to be dose-dependent similar to LV toxicity.[9,17,21,25] The effects of anti-cancer therapies known to also cause CTRCD, including vascular endothelial growth factor (VEGF) inhibitors, epidermal growth factor receptor (EGFR) inhibitors, Bruton tyrosine kinase inhibitors and proteasome inhibitors, on RV function specifically, remain unknown.

Right ventricular dysfunction induced by radiation therapy

The effects of RT on the RH chambers have been insufficiently investigated.[3,44,45] It is well known that the anatomical position

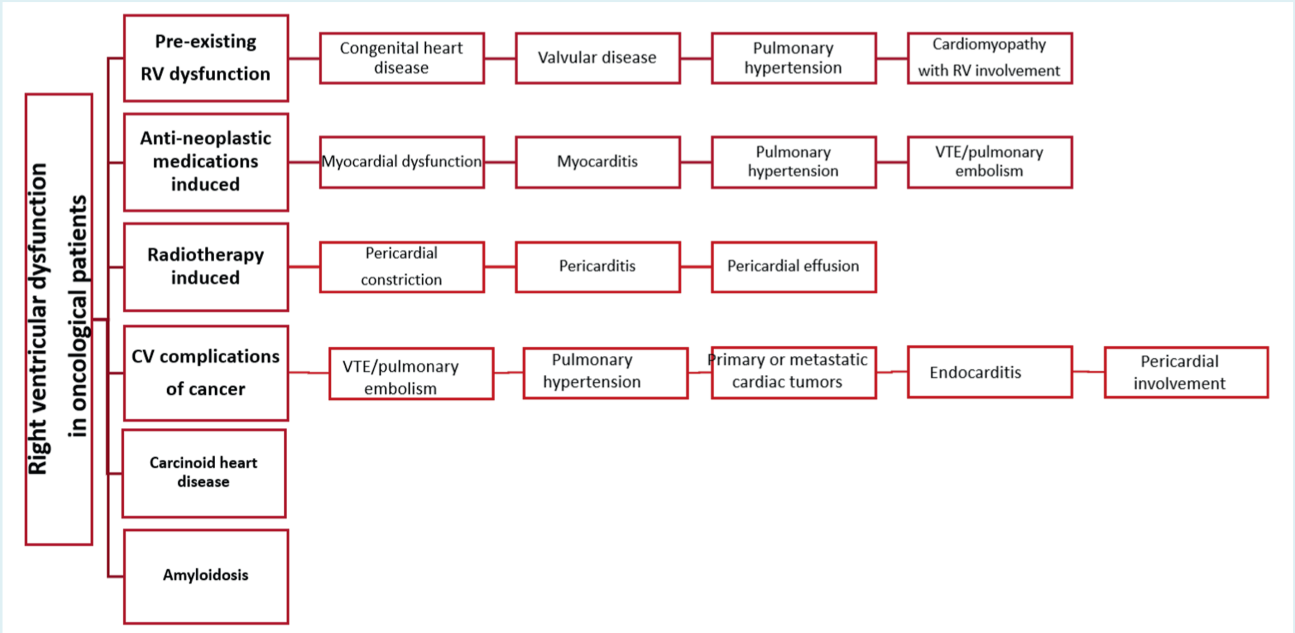

Figure 1 Overview of possible causes of right ventricular (RV) dysfunction in patients with cancer. CV, cardiovascular; VTE, venous thromboembolism.

Table 1 Cancer therapy-related right ventricular toxicity definitions

Asymptomatic RV dysfunction	New relative decline in RV FWLS by >15% from baseline value without significant change of the standard 2D-echocardiographically assessed parameters (TAPSE, RV FAC, RV MPI, RV S′) or 3D-assessed RVEF.
Symptomatic RV failure	Symptoms and/or signs of RV failure, such as dyspnoea, exercise intolerance, peripheral oedema, caused by a structural and/or functional abnormality of the right heart.

2D, two-dimensional; 3D, three-dimensional; FAC, fractional area change; FWLS, free wall longitudinal strain; MPI, myocardial performance index; RV, right ventricular; RVEF, right ventricular ejection fraction; TAPSE, tricuspid annular plane systolic excursion.

of heart structures, tumour localization and radiation dose exposure dictate which cardiac chamber(s) will be most affected.[46] Even though the RV is anatomically positioned more anteriorly and, therefore potentially, more exposed to superficial radiation targeting the chest wall or breast tissue, the majority of studies have focused on left-sided RT effects.[47,48] Moreover, pericardial disease, especially constriction, can significantly influence presentations and measurements.

Tissue fibrosis related to micro- and macro-vascular injury is the most important cause for cardiac impairment in radiation-induced heart disease. Microvascular injury induces myocardial and pericardial fibrosis, as well as reduced capillary density. These alterations are related to LV and RV remodelling, including diastolic and systolic dysfunction, as well as pericardial effusion and fibrosis that can possibly lead to constriction. Macrovascular injury is related to accelerated coronary artery atherosclerosis.

The effects of RT on RV systolic function depend on several factors including: the location of the cancer, the total dose of the radiation delivered, the mean dose to the heart and specifically to the RH, the combination of RT with antineoplastic drug therapy and the duration of the follow-up. The most consistent data about RV systolic function assessed by echocardiography involve TAPSE, which has been shown to be reduced in patients with left-sided breast cancer receiving RT[49] or RT and hormonal therapy (aromatase inhibitors)[50] or RT and chemotherapy,[44] in patients with haematological malignancies, oesophageal cancer, central lung cancer and thymoma.[45,51] The changes of RV FAC, tricuspid valve (TV) S′ and RVEF are inconsistent.[3,44,49,51]

A growing body of evidence shows that RV LS and particularly RV FWLS, are early markers of RT-induced RV damage.[3,51–53] The combination of RT with chemotherapy has an additive effect on RT-induced RV strain changes.[3,51] A study with stage III non-small cell lung cancer patients revealed the independent predictive value of RV FWLS at baseline and its change after anti-cancer therapy on 6-month all-cause mortality[52] and the researchers proposed a percentage change of RV FWLS of 10.1% as clinically significant. Data for RV strain changes in breast cancer patients from CMR also report a deterioration in RV mechanics soon after completion of RT.[53] Notably, the decline in RV LS appears to be primarily attributed to a reduction in apical strain.

Right ventricular dysfunction in pericardial diseases

Pericardial disease in cancer patients manifesting as RH failure may be due to constrictive pericarditis or pericardial effusion. Pericardial effusions may occur in up to 15% of cancer patients secondary to direct invasion, metastasis, or cancer treatment.[54] Gradual accumulation of fluid in the pericardial sac can compress the thin-walled RV manifesting as acute RH failure.[55] This occurs due to external restriction of diastolic biventricular filling and elevated systemic and pulmonary venous pressures.[56] Diagnosis of RH failure in this context is made initially by echocardiography which may reveal signs of dissociation of intrathoracic and intracardiac pressures and haemodynamic compromise.[57] CMR can accurately assess pericardial thickness, tumour infiltration and inflammation along with ventricular interdependence.[58] Cardiac computed tomography (CCT) can also determine pericardial thickness and visualize multiple tumour planes.[59,60] RH catheterization (RHC), which is the gold-standard diagnostic test, may be needed to make an invasive diagnosis of pericardial constriction, if non-invasive tests are inconclusive.[61] Management of acute pericarditis is in line with the 2015 ESC guidelines for the diagnosis and management of pericardial disease[61] and if secondary to immune checkpoint inhibitors then follow the 2022 ESC guidelines on cardio-oncology.[2] Surgical window may be considered for effusions inaccessible to percutaneous drainage or in cases of recurrent effusion.[2]

Right ventricular dysfunction induced by venous thromboembolism

Cancer and cancer therapies are predisposing factors for the development of VTE.[62] VTE is 4–7 times more frequent in cancer patients[63] and PE is the most critical complication associated with significant morbidity and mortality[64]. The incidence of PE in cancer patients ranges from 0.13% to 8.65%.[65] The higher risk of PE has been associated with central nervous system, pancreatic, upper gastro-intestinal and lung/pleural malignancies, as well as with renal and uterine cancers.[66,67] The highest risk of mortality due to PE is reported in oesophageal cancer patients.[64]

Risk factors for development of VTE in cancer patients can be patient-related (i.e. comorbidities or hereditary coagulation defects), cancer-related (i.e. pancreatic cancer, especially if metastatic), and cancer therapy-related.[2] Clinically, cancer patients with positive computed tomography (CT) scan for PE are older, have a lower body mass index and are less frequently active smokers, have a lower percentage of chest pain or syncope as initial manifestations of PE and are less likely to present with bilateral PE and RH strain at CT compared to non-cancer patients.[68] While PE in-hospital mortality is similar to that of non-cancer patients, 1-year overall survival is lower in cancer patients, especially in patients with metastatic cancer, regardless of exposure to chemotherapy.[68] A positive CT scan for PE (performed for other clinical reasons) defines 'incidental PE'. Its incidence is greater than 3% in cancer patients, especially with prostate, hepatobiliary and pancreatic cancer.

Pulmonary embolism is the second most common cause of severe RV dysfunction,[69] and its presence has relevant prognostic implications.[70] RV dysfunction is the direct consequence of acute increase in RV afterload during PE. The adaptation of the RV to its afterload should aim at maintaining the coupling of the RV/pulmonary artery (PA) unit.[71] Because of the intrinsic anatomic and physiological characteristics of the RV, it is unable to properly increase its contractility acutely during PE. Therefore, the RV/PA system uncouples, with an abrupt reduction in RV stroke volume.[72] This process ultimately also causes a reduction in LV stroke volume, due to the interdependence relation between the two ventricles.[73]

Right ventricular dysfunction in PE can be detected by CT scan, echocardiography and biomarkers.[74] CT scan provides information on RV dilatation with significant prognostic value for adverse events for the next 15 days.[75] On the other hand, echocardiography gives information on RV function and can help in the estimation of PA pressures. RV hypokinesis in haemodynamically stable patients (systolic arterial pressure $\geq$90 mmHg) with PE is a predictor of early mortality independently of cancer.[76] Increased levels of biomarkers, such as troponin T and B-type natriuretic peptide (BNP)/N-terminal proBNP (NT-proBNP) are associated with higher risk of mortality in clinically stable patients with PE.[74] However, it is noteworthy that increased troponin levels perform slightly worse in predicting RV dysfunction in oncologic patients with acute PE.[77] The treatment of cancer patients with PE should follow the 2019 ESC guidelines for the diagnosis and management of acute PE.[70]

Patients with active cancer have high (>8% per year) estimated risk for long-term PE recurrence (especially if anticoagulation is discontinued after the first 3 months) and a higher risk of developing post-PE syndrome.[70] Post-PE syndrome is characterized by chronic thrombotic remains in the PA, persistent RV dysfunction, decreased quality of life and/or chronic functional limitations. It includes two specific conditions: chronic thromboembolic disease and chronic thromboembolic pulmonary hypertension (CTEPH). RV failure is one of the most frequent causes of death in CTEPH.[78]

Right ventricular dysfunction induced by pulmonary hypertension

All five groups of PH can be noted in cancer patients.[78,79] Most commonly, PH is secondary to left heart or lung disease (group 2 and group 3 PH, respectively), while pulmonary arterial hypertension (PAH) is much less frequent, induced for instance by cancer therapeutics, such as dasatinib, a BCR-ABL tyrosine kinase inhibitor (TKI). The estimated incidence of dasatinib-induced PAH is <0.5%, and drug discontinuation usually results in functional and haemodynamic improvement.[80] Nonetheless, up to 40% of subjects do not recover and need pulmonary vasodilators.[81] Other BCR-ABL TKIs such as bosutinib, ponatinib, and nilotinib have also been associated with the development of PAH.[82] The pathogenesis

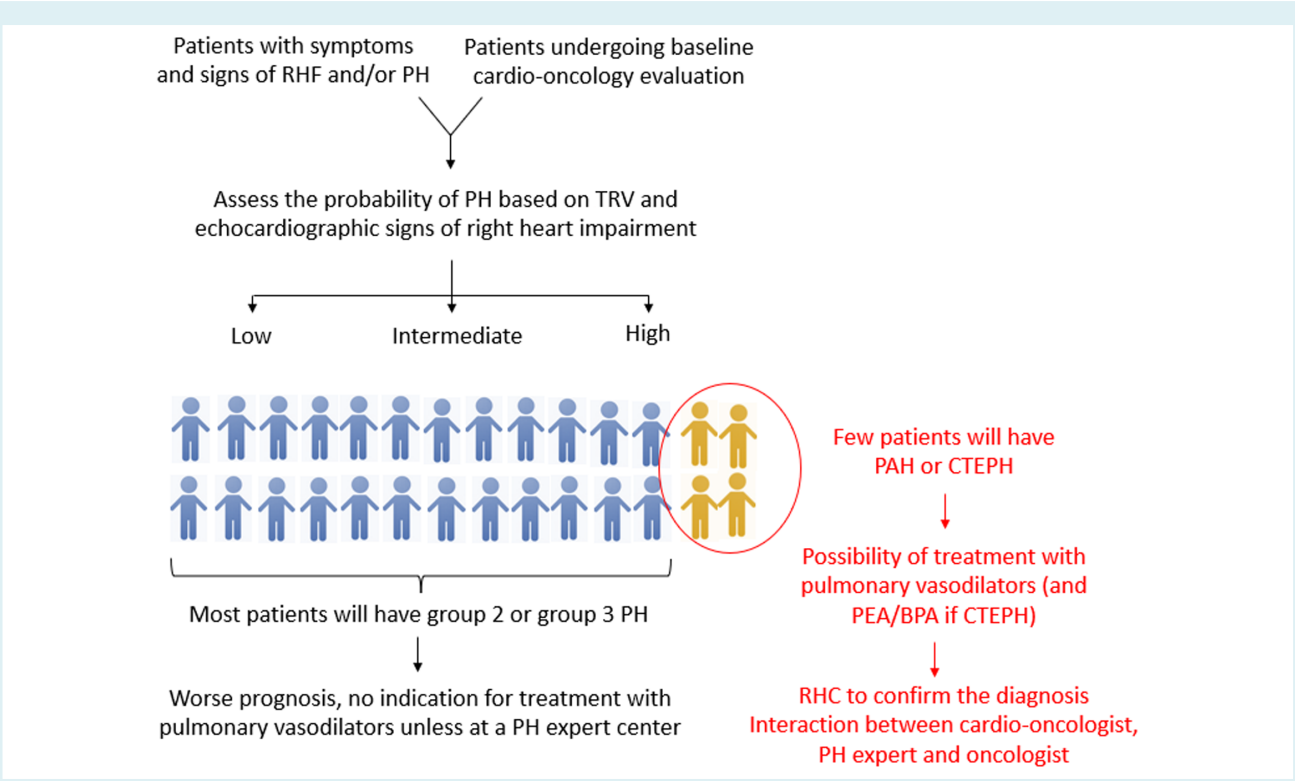

Figure 2 Diagnostic algorithm of pulmonary hypertension (PH) in patients with cancer. BPA, balloon pulmonary angioplasty; CTEPH, chronic thromboembolic pulmonary hypertension; PAH, pulmonary arterial hypertension; PEA, pulmonary endarterectomy; RHC, right heart catheterization; RHF, right heart failure; TRV, tricuspid regurgitation velocity.

of pulmonary arterial toxicity of these TKIs is at least in part secondary to off-target inhibition of protein kinases other than BCR-ABL.[82] Additional mechanisms play a role, such as oxidative stress and individual predisposition.[83] PAH has also been described after exposure to the alkylating agents, cyclophosphamide and mitomycin C, and to the proteasome inhibitor, carfilzomib.[2] It is notable that PAH due to alkylating agents has a distinctive pathobiology resembling pulmonary veno-occlusive disease.[84] PH is reported in up to 30% of patients with myeloproliferative neoplasms[85] and the outcome is worse than in the absence of PH.[86–88] Finally, emboli of cancer cells may activate the coagulation cascade and elicit vascular inflammation, fibrointimal proliferation, vascular occlusion including pulmonary venous occlusive disease and eventually a rare and peculiar form of PH, so-called pulmonary tumour thrombotic microangiopathy.[89]

A diagnostic algorithm of PH in cancer patients is presented in *Figure 2*. Left heart disease[90] and lung disease should be considered first in cancer patients with RH failure or echocardiographic abnormalities indicating PH. Furthermore, it is important not to limit TTE to evaluation of peak TV regurgitation velocity and to search for indirect signs of RH impairment. When PH cannot be convincingly ascribed to left heart or lung disease, there are risk factors for PAH, including the use of anti-tumour drugs with potential pulmonary vascular toxicity, and the echocardiographic probability of PH is high, referral to a PH expert centre and RHC are recommended as per general guidelines on PH[91] (*Figure 2*).

Right heart masses

A mass in the cardiac chambers can be a tumour (benign or malignant), a thrombus, a vegetation or a normal structure (variant), a device, or an artefact. Primary cardiac tumours are quite rare in the general population.[92] Secondary or metastatic tumours, on the other hand are 20 to 40 times more common,[92] while cardiac involvement has been found in 14.2% of patients with distant metastases.

Sarcomas are the most common cardiac primary malignant neoplasms (64.8%), more prevalent in males. Angiosarcomas are the most frequent tumours in the RH, especially in the right atrium (RA) (80% of cardiac angiosarcomas) followed by lymphomas.[93] Primary benign tumours in the RH include myxoma (5–20% of cases, usually in the RA), lipoma (typically in the RA, rarely can obstruct the TV or the vena cava), papillary fibroelastoma (at the TV or the pulmonary valve, or at endocardial surface of the RA, RV, right atrial appendage, or Eustachian valve), haemangioma (with a predilection for the RA).[94]

Tumours can metastasize to the heart via different pathways: haematogenous spread, lymphatic spread, transvenous extension (via inferior vena cava to RA, e.g. renal cell carcinoma and hepatocellular carcinoma), and direct extension (invading the pericardial sac, such as mesothelioma, lung and oesophageal cancer).[95] The pericardium is the most frequent site of metastasis (64–69%), while the epicardium (25–34%) and myocardium (30%)

follow. Intracavitary metastases are infrequent (3–5%).[95] Any type of tumour can metastasize to the heart, but the probability of cardiac involvement is a function of anatomic considerations, stage of disease, and individual tumour and host biology. Primary lung cancer represents 36% to 39% of cardiac metastases, followed by breast cancer (10–12%) and haematologic malignancies (10–21%). Pleural mesothelioma and melanoma (28–56%) have an unusual predisposition to involve the heart.[93]

Thrombi are the most common intracavitary masses. Thrombi can occur in any of the cardiac chambers but are typically found in the left atrium in patients with atrial fibrillation. RH thrombi are a rare, underdiagnosed and potentially fatal medical emergency, with a mortality between 27% and 100%, if not treated.[96,97] Right atrial thrombi have been described in 7% of autopsies (similar to left atrial thrombi) and in about 10% of patients with PE.[98] Right-sided mechanical valves, indwelling central venous lines, pacemaker leads, ventricular or atrial septal closure devices increase the risk of RA thrombus formation. Morphologically, there are three types of R thrombi, with different prognosis and embolization risk: type A in transit, type B in situ, and type C mobile in situ with a stalk.[99] RV thrombosis may occur in the setting of intracardiac procedures, PE, myocarditis, or RV myocardial infarction. The diagnosis is made basically by TTE and/or transoesophageal echocardiography (TEE), but multimodality imaging is imperative in certain cases.[100] There is no consensus on the treatment of RH thrombosis and management should be individualized.[100–102]

Vegetations in the RH in cancer patients can be attributed to two main conditions: infective endocarditis (IE) and non-bacterial thrombotic endocarditis (NBTE). IE is a quite common entity, especially in patients with colorectal cancer due to *Streptococcus gallolyticus*, previously known as *Streptococcus bovis*, subtype 1.[103,104] Cancer may serve as a port of entry for bacteria,[105] but other factors like immune suppression, hypercoagulability, indwelling catheters, and the need for invasive procedures increase the risk of IE in cancer patients. *Staphylococcus aureus* is the most common pathogen and survival is worse in this specific population.[106,107] IE involves native or prosthetic valves, any intracardiac device and more rarely the endocardium or other intracardiac structures,[108] like embryonic remnants in the RA.[109] TTE and TEE provide valuable clues about the diagnosis but other imaging techniques like CCT and ^{18}F-fluorodeoxyglucose (^{18}F-FDG) positron emission tomography (PET)/CT may be used for the identification of extent and the consequences of IE (abscesses, pseudoaneurysms, fistulae).[110]

Non-bacterial thrombotic endocarditis, also referred to as marantic, thrombotic, verrucous or Libman–Sacks endocarditis, is a type of non-IE, characterized by the presence of sterile vegetations on the heart valves. The vegetations are composed of platelets and fibrin, which are prone to systemic embolization. The exact cause of NBTE is not fully understood; however, it is well-established that the condition is associated with hypercoagulability states. NBTE is commonly associated with malignancies that primarily involve mucin-releasing adenocarcinomas. These malignancies often arise in the lung, ovary, biliary system, pancreas, breast, and stomach.[111] However, haematologic malignancies and bone marrow transplantation have been also associated with NBTE.[112,113] Usually, it occurs in the metastatic setting[114] and it is considered a paraneoplastic phenomenon. However, it can also be reported in patients with autoimmune diseases, such as systemic lupus erythematosus and rheumatoid arthritis.[115]

The clinical consequences of NBTE are primarily related to arterial thromboembolism and secondarily, valvular dysfunction. The most commonly affected valves are the aortic and the mitral valve, while rarely the RH valves[116,117] and the endocardium can be affected.[118] If embolic events do not occur, NBTE is either diagnosed as an incidental imaging finding or on post-mortem examination. TTE is the first diagnostic modality to be used (TEE is the method of choice), while CMR and CCT are often used to confirm the diagnosis of NBTE.[119] NBTE treatment includes anti-tumour therapy for the underlying malignancy and indefinite systemic anticoagulation with unfractionated or low molecular weight heparin,[120] however, the prognosis usually is dismal. In cases of potentially curable cancers or when severe valvular dysfunction or recurrent embolic events persist despite adequate anticoagulation, surgical intervention may be considered as a treatment option.

Right heart involvement in amyloidosis

Right ventricular systolic dysfunction in cardiac amyloidosis (CA) may occur due to (i) direct RV amyloid infiltration, or (ii) secondary due to group 2 post-capillary PH with increased RV afterload in LV failure, restrictive diastolic LV function and/or heart failure with preserved ejection fraction, as well as ventricular interdependence.[121] CA leads to predominantly concentric LV hypertrophy as can be depicted with echocardiography and CMR[122]; biventricular involvement with RV hypertrophy is observed in 66% to 70% of patients.[123]

The prognostic implication of the RV for outcomes in CA was previously underestimated.[124] RV involvement in CA occurs later in the course of LV amyloid deposition and impaired RVLS was shown to be a negative prognostic marker.[124] In a recent study of light-chain (AL) and transthyretin CA patients, RV FWLS <16% was associated with worse 1- and 3-year cumulative survival (81% vs. 31% and 67% vs. 2%, respectively, $p < 0.001$).[125] In CA patients with wild-type transthyretin amyloidosis (ATTRwt), advanced disease was associated with larger RH dimensions and gradual decrease in RV FWLS as a powerful marker of poor prognosis.[126] More than 50% of CA patients present pleural and pericardial effusions. In AL amyloidosis, pleural effusion was correlated well with poor RV function as measured by RV FWLS ($p = 0.007$).[127]

Novel emerging treatment options in CA are expensive and seem most efficient when started at an early stage of the disease and therefore risk stratification in CA appears crucial. In patients with transthyretin amyloid cardiomyopathy, disease-specific treatment with tafamidis delayed further deterioration also of RV LS as compared to treatment-naïve patients.[128]

Moderate-to-severe tricuspid regurgitation (TR) is frequent in CA and was observed in about a quarter of ATTRwt or AL patients.[129] While amyloid valvular mitral and tricuspid infiltration

has been described in autopsy studies, a predominantly functional, secondary mechanism of TR appeared to be present in these patients, and moderate-to-severe TR was associated with reduced RV systolic function.[129] Mortality in patients with ATTRwt is significantly associated with moderate-to-severe TR, whereas in AL, this study observed no correlation between TR and death.[129] Importantly, data for tricuspid intervention in transthyretin amyloidosis (ATTR) is lacking for AL-CA.

In CA, PH is highly prevalent. In patients with CA who underwent CMR and RHC, the rate of isolated post-capillary PH was predominant with a prevalence of 55% but combined pre-capillary and post-capillary PH was also present in 29% of patients.[130] Increased transpulmonary gradient and reduced RVEF were strongly associated with major adverse CV events.[130]

Atrial fibrillation is encountered in about 50% of patients with CA, with a 10-fold risk in ATTR patients. Echocardiographically, worse LV and RV function, higher PA pressures, and higher rates of mitral regurgitation were observed in CA patients with atrial fibrillation.[131]

Importantly, in AL amyloidosis, RV function deterioration can also be caused directly by treatment, especially with proteasome inhibitors, e.g. bortezomib, leading to PH, thromboembolism and heart failure, as outlined in the current ESC guidelines.[2]

Finally, although the RV clearly plays an important role in the prognosis of patients with CA, more data are needed, and the role of risk stratification in upcoming therapies should be further elucidated.

Right heart involvement in carcinoid heart disease and neuroendocrine tumours

The prevalence of carcinoid syndrome among neuroendocrine tumours (NET) patients currently ranges between 19% and 35% and CHD is present in 20–50% of these patients, leading to reduced survival at 3 years of 31% compared to 69% in patients without CHD.[132] Secretion of vasoactive substances by the tumour, including serotonin or its metabolite 5-hydroxyindolacetic acid (5-HIAA) into the systemic circulation, can cause plaque-like fibrous deposits on right-sided heart valves and endocardial RV surfaces.

Tricuspid and or pulmonary valve leaflet thickening with regurgitation and/or stenosis can cause RV volume overload and RV failure.[133] Elevation in natriuretic peptide levels is an early marker and can direct further management efforts.[134] Cardiac imaging of the RV function and valves are key to determine timing of surgery and management of CHD.[133] Valve replacement surgery is recommended by the 2022 ESC guidelines on cardio-oncology in symptomatic patients with severe carcinoid tricuspid or pulmonary valvular heart disease if NET-related survival is expected ≥12 months.[2] In patients with progressive RV dysfunction/dilatation, according to guidelines, valve intervention should be considered also in asymptomatic patients with severe carcinoid tricuspid or pulmonary valvular heart disease if NET-related survival is expected ≥12 months.[2]

Although no randomized controlled trials are available, valvular surgery along with modern medical as well as surgical NET treatments, has been shown to improve long-term prognosis in patients with severe carcinoid valve disease. Surgical valve strategies remain controversial. Importantly, RV size was reduced after combined bioprosthetic tricuspid (TVR) and pulmonary valve replacement (PVR), whereas interventions without PVR did not significantly affect RV size postoperatively in a recent cohort.[135] Survival in concomitant TVR and PVR was significantly higher in comparison to interventions without PVR.[135] Bioprosthetic valve degeneration can accelerate in persistent serotonin elevation with recurrence of RV failure.[136,137] For the new transcatheter valve interventions for the treatment of TR, including TricValve, EVOQUE and transcatheter edge-to-edge repair of the TV, no specific data have been collected in cancer patients to date. However, a multidisciplinary team approach considering eligibility criteria and cancer prognosis, similar to that generally proposed for the treatment of TR, seems warranted.[138,139] In summary, in CHD, the RV function due to right valvular and direct involvement remains a major outcome variable.

Right heart assessment in cancer patients

How to assess right heart structure and function

The assessment of RH size and shape can be done using echocardiography, CMR and CCT (*Figures 3* and *4*), while for RH masses nuclear imaging techniques and hybrid imaging may also be of value. TTE is the most used non-invasive modality to assess RH structure and function in daily practice (*Figure 5*).[140] Conventional RV assessment includes RV linear dimensions and at least one of the following RV systolic function parameters: FAC, TAPSE, pulsed tissue Doppler systolic velocity of the lateral tricuspid annulus (S') and MPI calculated with pulsed tissue Doppler of the lateral tricuspid annulus.[141] In recent years, the integration of 3D echocardiography and speckle tracking deformation imaging has shown good feasibility and reproducibility to analyze RV structure and function. RVLS measurements have demonstrated clinical utility to stratify the prognosis and address management in patients with heart failure, PE, PAH, and congenital heart diseases.[142] A RV focused 4-chamber apical view is suggested by EACVI/ASE[143] to analyse RVLS and nowadays, peak systolic values of RV FWLS are advised to be considered part of the RV TTE evaluation.[143] RV 3D TTE assessment overcomes some of the 2D TTE limitations related with RV complex morphology and in laboratories with appropriate experience 3D RV volumes and 3D RVEF compared well with CMR.[140,144] Normal values of right chamber size and RV function[43,140] assessed by echocardiography are presented in *Table 2*.

Transthoracic echocardiography also provides detailed information about the morphology, size, location, extent, and haemodynamic consequences of RH masses. It allows for the assessment of mass mobility, attachment to cardiac structures, and the presence of associated complications such as valvular dysfunction or

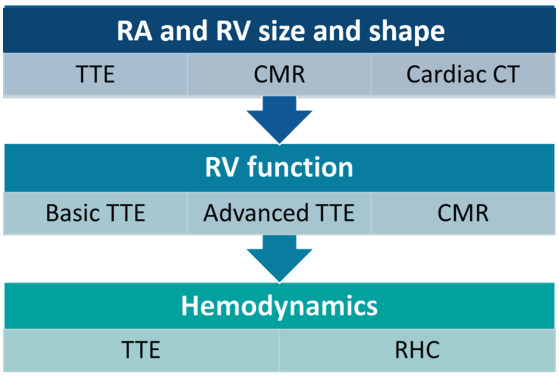

Figure 3 Imaging modalities for the assessment of right heart morphology and right ventricular (RV) function in cancer patients.

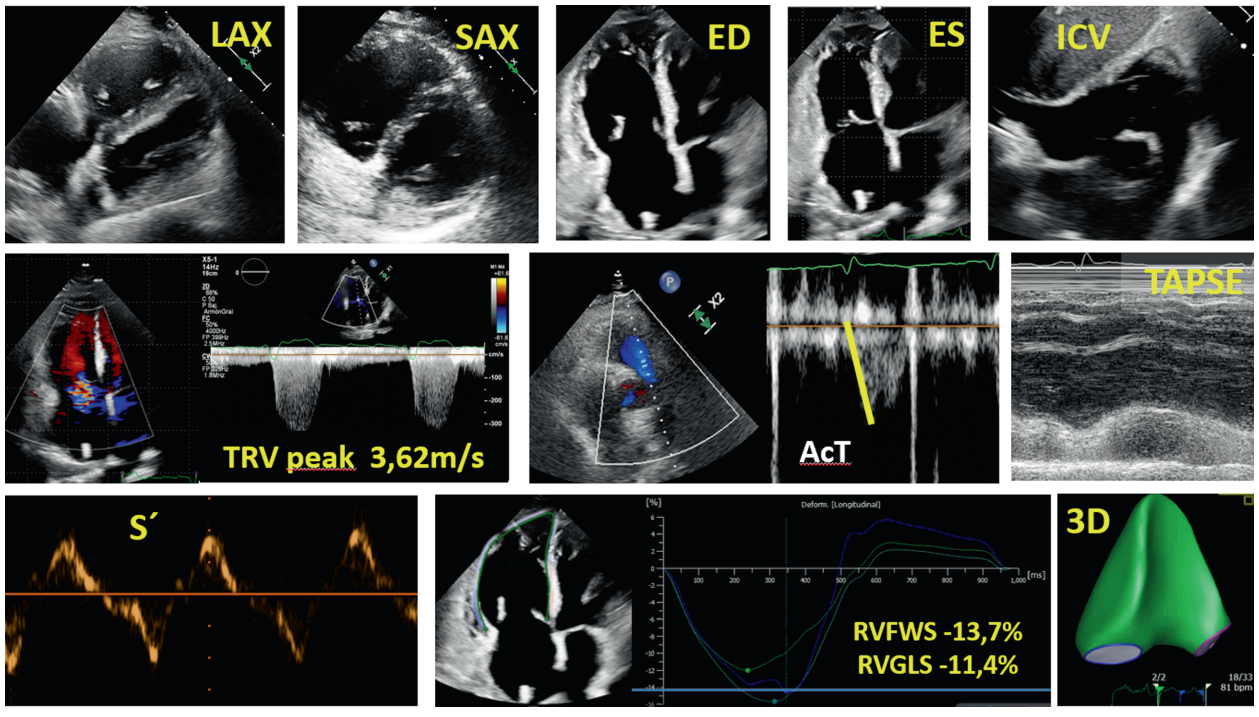

Figure 4 Standard views and measurements for the assessment of right heart chambers by transthoracic echocardiography (TTE). 3D, three-dimensional; AcT, acceleration time; CMR, cardiac magnetic resonance; ED, end-diastole; ES, end-systole; ICV, inferior caval vein; LAX, long-axis; CT, computed tomography; RA, right atrial; RHC, right heart catheterization; RVFWS, right ventricular free wall strain; RVGLS, right ventricular global longitudinal strain; SAX, short-axis; TAPSE, tricuspid annular plane systolic excursion; TRV, tricuspid regurgitation velocity.

obstruction. The use of ultrasound-enhancing agents (contrast agents) allows better definition of intracavitary masses and it can assess the vascularity of a cardiac mass aiding in the differential diagnosis of a mass from a thrombus. However, images of the RH can be suboptimal in up to 15% of patients and artefacts may further complicate the diagnosis. Moreover, tissue characterization is not possible and extracardiac manifestations may be missed due to the narrow field of view. TEE overcomes some of the aforementioned limitations (e.g. poor acoustic windows) and can visualize more efficiently the RA, superior and inferior vena cava, providing a surgical anatomical view in several cases, especially if real-time 3D echocardiography is applied.[93]

Nevertheless, TTE has several limitations due to the limited acoustic window in some patients and CMR is considered the gold standard technique to assess RV anatomy and function. In patients with cancer, according to 2022 ESC guidelines on cardio-oncology, CMR is recommended in some clinical scenarios like cardiac AL amyloidosis and should be considered for the assessment of cardiac function when echocardiography is unavailable or non-diagnostic[2] and in patients at high risk of RV dysfunction. Normal values of RV

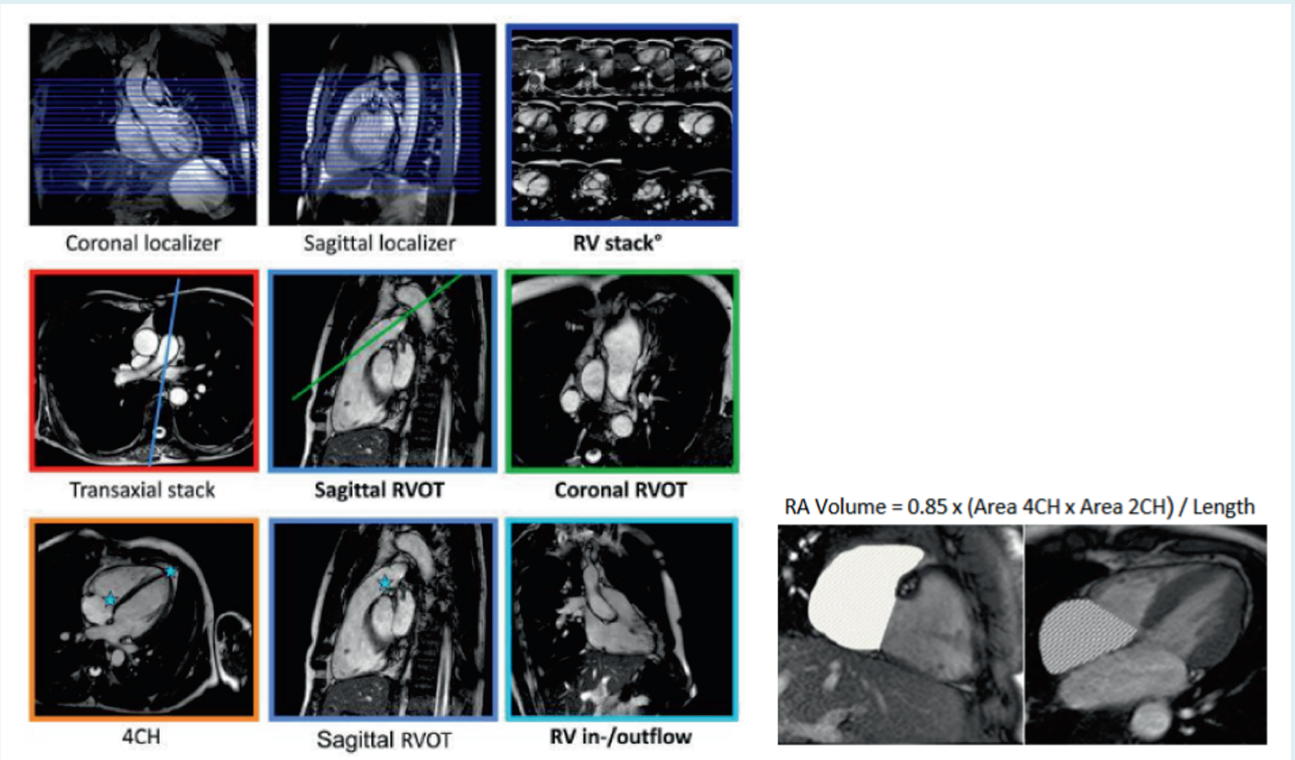

Figure 5 Standard views of the right heart by cardiac magnetic resonance. 2CH, 2-chamber; 4CH, 4-chamber; RA, right atrial; RV, right ventricular; RVOT, right ventricular outflow tract.

Table 2 Normal values of echocardiographically assessed right chamber size and right ventricular function

Normal values of right chamber size		Normal values of RV function	
Parameter	**Normal values**	**Parameter**	**Normal values**
RV basal diameter (mm)	<42	TAPSE (mm)	>17
RV mid diameter (mm)	<36	TV S′ (cm/s)	>9.5
RVOT PLAX diameter (mm)	<31	TDI MPI	>0.54
RVOT proximal diameter (mm)	<36	RV FAC (%)	>35
RVOT distal diameter (mm)	<28	2D RV global longitudinal strain (%)	<−15.5
RAVi (ml/m^2)	<33 (M) <28 (F)	2D RV free wall strain (%)	<− 20
3D echo RV volumes		3D echo RV parameters	
RVEDVi (ml/m^2)	<88 (M) <75 (F)	RVEF (%)	>45
RVESVi (ml/m^2)	<45 (M) <37 (F)	3D RV global longitudinal strain (%)	<−7.3
		3D RV free wall strain (%)	<−10.1

2D, two-dimensional; 3D, three-dimensional; F, female; FAC, fractional area change; M, male; MPI, myocardial performance index; PLAX, parasternal long axis; RA, right atrial; RAVi, right atrial volume index; RV, right ventricular; RVEDVi, right ventricular end-diastolic volume index; RVEF, right ventricular ejection fraction; RVESVi, right ventricular end-systolic volume index; RVOT, right ventricular outflow track; TAPSE, tricuspid annular plane systolic excursion; TDI, tissue Doppler imaging; TV, tricuspid valve.

and right atrial parameters assessed by CMR in men and women are presented in *Table 3*.[145] CMR is also particularly useful in the evaluation of RH masses. It provides detailed information about tissue composition, vascularity, and invasion of surrounding structures. It can differentiate between benign and malignant tumours,[122] solid and cystic masses, identify haemorrhage or necrosis within the lesion, and assess the presence of associated pericardial or mediastinal involvement and local infiltration. CMR has higher sensitivity and specificity than TTE or TEE in detection of intraventricular thrombus, especially if posteriorly located in the RV.

Table 3 Normal values of right ventricular and right atrial parameters assessed by cardiac magnetic resonance in men and women[145]

	Men			Women		
	20–29 years	**30–39 years**	**40–49 years**	**20–29 years**	**30–39 years**	**40–49 years**
EDV (ml)	177 (127–227)	171 (121–221)	166 (116–216)	142 (100–184)	136 (94–178)	130 (87–172)
ESV (ml)	68 (38–98)	64 (34–94)	59 (29–89)	55 (29–82)	51 (25,77)	46 (20–72)
SV (ml)	108 (74–143)	108 (74–142)	107 (73–141)	87 (61–112)	85 (59–111)	84 (58–109)
EF (%)	61 (48–74)	63 (50–76)	65 (52–77)	61 (49–73)	63 (51–75)	65 (53–77)
Mass (g)	70 (42–99)	69 (40–97)	67 (39–95)	54 (33–74)	51 (31–72)	49 (28–70)
Indexed to BSA						
EDV/BSA (ml/m^2)	91 (68–114)	88 (65–111)	85 (62–108)	84 (65–102)	80 (61–98)	76 (57–94)
ESV/BSA (ml/m^2)	35 (21–50)	33 (18–47)	30 (16–45)	32 (20–45)	30 (17–43)	27 (14–40)
SV/BSA (ml/m^2)	56 (40–72)	55 (39–71)	55 (39–71)	51 (39–63)	50 (38–62)	49 (37–61)
Mass/BSA (g/m^2)	36 (23–50)	35 (22–49)	34 (21–48)	32 (22–42)	30 (20–40)	29 (19–39)
	50–59 years	**60–69 years**	**70–79 years**	**50–59 years**	**60–69 years**	**70–79 years**
EDV (ml)	160 (111–210)	155 (105–205)	150 (100–200)	124 (81–166)	117 (75–160)	111 (69–153)
ESV (ml)	55 (25–85)	50 (20–80)	46 (16,76)	42 (15–68)	37 (11–63)	32 (6–58)
SV (ml)	106 (72–140)	105 (71–139)	104 (70–138)	82 (56–108)	80 (55–106)	79 (53–105)
EF (%)	66 (53–79)	68 (55–81)	70 (57–83)	67 (55–79)	69 (57–81)	71 (59–83)
Mass (g)	65 (37–94)	63 (35–92)	62 (33–90)	47 (26–68)	45 (24–66)	43 (22–63)
Indexed to BSA						
EDV/BSA (ml/m^2)	82 (59–105)	79 (56–101)	75 (52–98)	72 (53–90)	68 (49–86)	64 (45–82)
ESV/BSA (ml/m^2)	28 (13–42)	25 (11–40)	23 (8–37)	24 (11–37)	21 (8–34)	19 (6–32)
SV/BSA (ml/m^2)	54 (38–70)	53 (37–69)	52 (36–69)	48 (36–60)	46 (34–58)	45 (33–57)
Mass/BSA (g/m^2)	33 (20–46)	32 (19–45)	31 (18–44)	27 (17–37)	26 (16–36)	24 (14–35)

RA volumes	**Men/Women**
4CH RA area (cm^2)	14–30
2CH RA area (cm^2)	14–30
4CH RA area/BSA (cm^2/m^2)	8–16
2CH RA area/BSA (cm^2/m^2)	7–17
RA volume (Bi-plane) (ml)	37–169
RA volume/BSA (ml/m^2)	18–90

2CH, 2-chamber; 4CH, 4-chamber; BSA, body surface area; EDV, end-diastolic volume; EF, ejection fraction; ESV, end-systolic volume; RA, right atrial; SV, stroke volume.

However, it may miss small, highly mobile masses and it is inferior to echocardiography in detecting mass calcification. In daily practice, its use is limited by availability and the lack of relevant expertise in certain centres.[146]

Cardiac CT is another valuable imaging modality for the assessment of RH chambers in cancer patients.[147] It allows for the evaluation of RV and RA dimensions and volumes, and it also enables the assessment of RV and RA myocardial perfusion.[93] Normal values for CT measured parameters for the assessment of the RV are included in *Table 4*.[148] CT imaging can provide valuable information about the anatomical characteristics, tissue composition, and extent of RH masses. It allows for the assessment of mass enhancement patterns, tumour vascularity and calcification (superior to CMR), and potential invasion into adjacent structures. CCT can efficiently discriminate between benign and malignant cardiac lesions[149] and it can also help in the evaluation of extracardiac metastases and aid in staging of the disease. Furthermore, CCT is the imaging modality of choice for masses adjacent to prosthetic valves. In addition, CCT can provide valuable information on the pulmonary vasculature and help to identify any associated abnormalities or emboli.

Table 4 Normal values for computed tomography-measured parameters for the assessment of the right ventricle[148]

CT-measured parameters for RV assessment	**Normal values (mean ± SD)**
Structural	
EDV (ml)	199 ± 34 (M) 163 ± 28 (F)
ESV (ml)	86 ± 40 (M) 66 ± 28 (F)
EDV/BDA (ml/m^2)	96 ± 15 (M) 88 ± 12 (F)
ESV/BSA (ml/m^2)	42 ± 19 (M) 36 ± 15 (F)
Functional	
EF (%)	58 ± 16 (M) 61 ± 14 (F)
SV (ml)	112 ± 23 (M) 97 ± 18 (F)
SV/BSA (ml/m^2)	54 ± 13 (M) 53 ± 10 (F)

BSA, body surface area; CT, computed tomography; EDV, end-diastolic volume; EF, ejection fraction; ESV, end-systolic volume; F, female; M, male; RV, right ventricular; SC, stroke volume; SD, standard deviation.

Nuclear imaging techniques, such as PET or single-photon emission computed tomography (SPECT), can aid in the differential diagnosis of RH masses. PET offers functional and metabolic information that complements the anatomical imaging provided by other modalities, such as echocardiography, CMR and CT. PET utilizes radiotracers labelled with positron-emitting isotopes, such as ^{18}F-FDG, which is commonly used in oncology. Malignant tumours, such as primary cardiac sarcomas or metastatic lesions, often exhibit increased glucose utilization compared to benign lesions or normal cardiac tissue. PET can also contribute to the identification of occult metastases and to the assessment of treatment response.

Hybrid imaging, which combines two or more imaging modalities, has emerged as a valuable tool, especially in the differential diagnosis of RH masses. One of the commonly utilized hybrid imaging modalities is PET/CT. In the evaluation of RH masses, PET/CT offers a synergistic approach that enables simultaneous assessment of metabolic activity and precise localization of the lesion.[149,150] Another hybrid imaging modality that has shown promise in the evaluation of RH masses is magnetic resonance imaging (MRI)/PET, which combines the excellent soft tissue characterization capability of MRI with the functional (metabolic) information provided by PET.[151]

The indications for RHC and exercise/stress test in patients with cancer are the same as for the general population. RHC is the gold standard for diagnosis of PH.[91] It is necessary to confirm or exclude CTEPH or PAH, especially in patients undergoing treatment with PAH-inducing drugs, so that if PAH is demonstrated, alternative oncological treatments are considered. It is noteworthy, however, that the majority of patients with active cancer have PH due to left heart failure or parenchymal lung disease and, in these cases, RHC is not routinely performed. Particularly for survivors of radiotherapy, RHC can help in differentiating post-radiotherapy constrictive pericarditis. Congenital heart disease may warrant RHC to be performed when non-invasive evaluation leaves uncertainty about the diagnosis, but RHC is mainly reserved for resolution of specific anatomical and physiological questions, or for intervention.[152] Myocardial biopsy remains the gold standard for diagnosing myocarditis and infiltrative/storage diseases including amyloidosis,[153] and in these cases RHC may be needed. Lastly, no specific data exist for serial measurements by RHC in cancer patients to assess treatments efficacy.

Standardization of the methodology used to quantify RV function in patients with cancer is a prerequisite for its routine clinical application and for improvement of research practices. This scientific statement has been designed to standardize RV assessment in patients with cancer treated with cardiotoxic drugs. *Table 5* summarizes RV function parameters that should be reported routinely in patients with cancer.

When and how often to assess right ventricular function

Table 6 presents the suggested timepoints and the imaging modalities for RV assessment in cancer patients. According to guidelines, a comprehensive assessment of biventricular function is recommended at baseline and at the end of cancer treatment in all patients referred for TTE.[2] During cancer treatment, RV function monitoring is especially important in patients with pre-existing RV dysfunction and those at presumably high risk of RV damage (i.e. patients scheduled to receive anthracycline chemotherapy, HER2-targeted therapies, VEGF inhibitors, BCR-ABL TKI, EGFR inhibitors, CAR-T cell and TIL therapy, multiple myeloma therapies, RAF and MEK inhibitors, immune checkpoint inhibitors, or thoracic RT).[154] Although the current evidence is scarce, the assessment of RV FWLS is advised to be part of the TTE monitoring in patients at risk of heart failure and/or PH.[145]

Treatment options

Treatment decisions in cancer patients with RV dysfunction should consider disease and CV symptom burden, cancer prognosis, patients' preferences, and treatment requirements to minimize cancer treatment interruptions.[2] For asymptomatic RV dysfunction in patients with normal LV ejection fraction and LV GLS, more data are needed to provide guidance on treatment options. The prevention and management of cancer therapy-related symptomatic

Table 5 Transthoracic echocardiographic parameters to assess right ventricular function in cancer patients

TTE RV-focused views required	Must have	Want to have	Nice to have
RV-RA dimensions[a]	X	X	
TAPSE	X		
TAPSE/PSAP	X		
S'	X		
FAC or MPI (TDI)	High-risk patients[b]		
TRV	X	Absence of TR	
RVOT-AT			
RV FWLS	New CV symptoms High-risk patients[b]	All patients	
3D RV volumes and EF	High-risk patients[b]	Symptomatic patients	All patients

3D, three dimensional; CV, cardiovascular; EF, ejection fraction; FAC, fractional area change; FWLS, free wall longitudinal strain; MPI, myocardial performance index; PSAP, pulmonary systolic arterial pressure; RA, right atrial; RV, right ventricular; RVOT-AT, right ventricular outflow track-acceleration time; TAPSE, tricuspid annular plane systolic excursion; TDI, tissue Doppler imaging; TR, tricuspid regurgitation; TTE, transthoracic echocardiography; TRV, tricuspid regurgitation velocity.

Table 6 Imaging modalities and timepoints for right ventricular assessment in cancer patients

	Must have	Want to have	Nice to have
ECG	All patients before cardiotoxic drugs		
TTE	Baseline: RV function assessment in all patients with cancer referred for TTE evaluation. Follow-up: RV function assessment in all patients with cancer at risk of HF/LVD and/or PH, referred for TTE surveillance[a].		Follow-up: all patients with cancer referred for TTE surveillance
CMR	Baseline: AL-cardiac amyloidosis. Follow-up: symptomatic patients at risk of RV dysfunction when TTE is non-diagnostic.	High-risk patients: patients with pre-existing RV impairment or pre-existing abnormal RV pressure/volume overloading[b]	
RHC	Confirm PH diagnosis Constrictive pericarditis	Complex congenital heart diseases	

AL, light chain; CMR, cardiac magnetic resonance; ECG, electrocardiogram; HF, heart failure; LVD, left ventricular dysfunction; PH, pulmonary hypertension; RHC, right heart catheterization; RV, right ventricular; TTE, transthoracic echocardiography.
[a]Anthracycline chemotherapy, HER2-targeted therapies, vascular endothelial growth factor inhibitors, BCR-ABL tyrosine kinase inhibitors, epidermal growth factor receptor inhibitors, CAR-T cell and TIL therapies, multiple myeloma therapies, RAF and MEK inhibitors, immune checkpoint inhibitors, radiotherapy.
[b]High-risk patients: patients with pre-existing RV dysfunction or pre-existing abnormal RV pressure/volume overloading including: HF with reduced ejection fraction with biventricular dysfunction, HF with preserved ejection fraction with secondary pulmonary arterial hypertension and RV dysfunction, PH with RV dysfunction, cardiomyopathy with RV involvement (arrhythmogenic RV cardiomyopathy, hypertrophic cardiomyopathy, restrictive cardiomyopathy, dilated cardiomyopathy), complex congenital heart diseases, complex valvular heart diseases with RV dilatation/dysfunction.

RV failure should generally follow published ESC guidelines and position statements.[148,155,156]

Gaps in knowledge

The prevalence of RV dysfunction is unknown for most of the cancer drug classes with the majority of data restricted to anthracyclines and trastuzumab. Individuals with pre-existing RV dysfunction or abnormal RV haemodynamic loading (e.g. PH, congenital heart disease, arrhythmogenic RV cardiomyopathy) probably have a higher risk of new or worsening RV dysfunction and CTR-RVT. There is lack of specific biomarkers for the identification of RV dysfunction; troponin and NT-proBNP are 'pan-cardiac' markers, non-specific for the RV. Furthermore, there is lack of evidence to determine the prognostic impact of RV cardiotoxicity on LV cardiotoxicity development and on patients' survival. Consequently, systematic assessment of RV function is needed in all cancer patients being screened for cardiotoxicity, as part of the routine cardiological echocardiographic assessment. This will facilitate the identification of the cancer medications with potential cardiotoxic effects on the RV, will reveal thresholds for clinically significant changes and will identify the high-risk patients for RV dysfunction. Currently, it is unclear whether current heart failure drugs have a favourable impact on the prevention and treatment of RV dysfunction. Frequency of surveillance for RH health remains to be determined and should be a focus of further research.

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