Review

Identification of cardiac organ damage in arterial hypertension: insights by echocardiography for a comprehensive assessment

Matteo Cameli[a,*], Maria Lembo[b,*], Carlotta Sciaccaluga[a], Francesco Bandera[c], Marco M. Ciccone[d], Antonello D'Andrea[e], Flavio D'Ascenzi[a], Roberta Esposito[b], Vincenzo Evola[f], Riccardo Liga[g], Giulia E. Mandoli[a], Pasquale Palmiero[h], Ciro Santoro[b], Pietro Scicchitano[d], Regina Sorrentino[b], Annapaola Zito[d], Roberto Pedrinelli[g], Sergio Mondillo[a], Anna V. Mattioli[i], Maurizio Galderisi[b], on behalf of Working Groups of Echocardiography and Arterial Hypertension of Italian Society of Cardiology (SIC)

Arterial hypertension, a widespread disease, whose prevalence increases with age, represents a major risk factor for cardiovascular events, causing damage in several organs, including the heart. In this context, echocardiography has a clear and pivotal role, being able to assess cardiac morphology and detect haemodynamic changes induced by this disease. 2018 European Society of Cardiology/European Society of Hypertension guidelines on AH identified main echo parameters such as left ventricular mass, relative wall thickness and left atrial volume, for detecting cardiac organ damage. The present review highlights the advantage of additional echocardiographic parameters such as diastolic measurement and both thoracic and abdominal aortic dimensions. An overlook on aortic valve should also be suggested to detect aortic regurgitation and stenosis, both frequent complications in hypertensive patients. In this kind of comprehensive assessment, the combination of standard and advanced echocardiography (speckle tracking echocardiography and, with a lesser extent, three-dimensional echocardiography) could be considered to improve the diagnostic accuracy, stratify prognosis and address management in arterial hypertension.

Keywords: arterial hypertension, cardiac organ damage, diastolic dysfunction, echocardiography, left ventricular hypertrophy

Abbreviations: AAA, abdominal aorta aneurysm; AH, arterial hypertension; CMR, cardiac magnetic resonance; EDV, end-diastolic volume; GLS, global longitudinal strain; HFpEF, heart failure with preserved ejection fraction; LV, left ventricular; LVEF, left ventricular ejection fraction; LVH, left ventricular hypertrophy; LVM, left ventricular mass; PWV, pulse wave velocity; RWT, relative wall thickness; STE, speckle tracking echocardiography; TDI, tissue doppler imaging

INTRODUCTION

Arterial hypertension (AH) is one of the major contributors to the global burden of disease, showing a high prevalence which progressively increases as the age advances, reaching a value more than 60% in the population aged 60 years or over [1]. It is an important cardiovascular risk factor as it is independently associated with the occurrence of major cardiovascular events, including myocardial infarction, heart failure, peripheral artery disease, ischaemic and haemorrhagic stroke [2,3] and atrial fibrillation as well [4]. Subclinical target organ damage involves heart, brain, kidney, eyes and is considered a marker of preclinical cardiovascular disease [5]. To avoid irreversible organ damage, it is important to promptly diagnose cardiac organ damage and to initiate an early and effective treatment to reduce the progression towards overt involvement. In this view, echocardiography plays a central role in detecting subclinical cardiac remodelling, which develops as a result of pressure overload. These changes include left ventricular (LV) concentric remodelling and hypertrophy (LVH), diastolic dysfunction and left atrial enlargement, all factors predisposing to heart failure. The detrimental effect of AH on aorta and its elastic properties induces a progressive wall stretching and increased arterial stiffness, which is a predictor of both aortic valve and aortic vessel disease.

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[a]Department of Cardiovascular disease, University of Siena, Siena, [b]Department of Advanced Biomedical Sciences, Federico II University Hospital, Naples, [c]Heart Failure Unit, Cardiology University Department, University of Milan, IRCCS Policlinico San Donato, San Donato Milanese, Milan, [d]Cardiovascular Diseases Section, Department of Emergency and Organ Transplantation (DETO), University of Bari, Bari, [e]U.O.D. Diagnostica Cardiologica Integrata, Seconda Università degli Studi, AORN dei Colli-Monaldi, Napoli, [f]Department of Clinical and Experimental Medicine, University of Palermo, Palermo, [g]Department of Surgical, Medical, Molecular Pathology and Critical Area, University of Pisa, Pisa, [h]ASL Brindisi, Cardiology Equipe, District of Brindisi, Brindisi and [i]Department of Surgical, Medical and Dental Morphological Sciences related to Transplant, Oncology and Regenerative Medicine, University of Modena and Reggio Emilia, Modena, Italy

Correspondence to Maurizio Galderisi, MD, FESC, Interdepartmental Laboratory of Cardiac Imaging, Federico II University Hospital, Via Pansini 5, 80131 Naples, Italy. Tel: +39 081 7464749; e-mail: mgalderi@unina.it

[*]Matteo Cameli and Maria Lembo contributed equally to the article.

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The latest European Society of Cardiology/European Society of Hypertension (ESC/ESH) guidelines [6] on AH recommend the use of echocardiography in presence of electrocardiographic abnormalities, suggesting the assessment of standard echo parameters such as LV mass (LVM), relative wall thickness (RWT) and left atrial volume, for the definition of organ damage. The present review aims to underline the possible advantage of using additional parameters, including aortic dimension and cardiac function, obtainable from standard and advanced echocardiography that could provide a wider view of cardiovascular involvement and identify subclinical organ damage in patients affected by AH.

LEFT VENTRICLE

LV is directly affected by systemic AH. Elevated blood pressure (BP) is responsible for increased LV afterload, which implies that the LV must develop a higher pressure to guarantee adequate cardiac output and peripheral organ perfusion. In response to AH, LV wall stress increases, LV walls become thicker and LVM greater, due to interposition of interstitial fibrosis among cardiomyocytes. The echocardiographic assessment is a cornerstone in this kind of evaluation, it corresponding to the detection of LV geometric patterns and diagnosis of LVH.

Left ventricular hypertrophy

Echocardiographic-derived LVH has proven to be a strong predictor of mortality in both the general population and in patients affected by AH [7]. Furthermore, the regression of LVH during antihypertensive treatment is a good predictor of improved prognosis [8–10]. Accordingly, an exhaustive quantification of LVM in hypertensive patients is of paramount importance. As reported in the ASE/EACVI Chamber Quantification recommendations [8,11], LVM can be determined by linear measurements of septal and posterior wall thickness, and of LV internal cavity dimension, all at end-diastole, by using two-dimensional-guided M-mode echocardiography or directly by two-dimensional echocardiography [8]. These approaches imply the assumption of a geometric model and the use of the following formula:

$$\mathrm{LVM} = 0.8 \times 1.04 \times [(\mathrm{IVST} + \mathrm{LVID} + \mathrm{PWT})3 - \mathrm{LVID}^3] + 0.6\,\mathrm{g}$$

where *IVST* is the interventricular septal thickness, *LVID* is LV internal diameter and *PWT* is the infero-lateral wall thickness. LVM determined by this formula has been successfully validated against heart cardiac autopsy [12]. LVM can be calculated also by two-dimensional echocardiography by using area-length formula and the truncated ellipsoid formula [13]. The indexation of LVM is mandatory, because it allows comparisons among patients with different body sizes. In the hypertensive setting, LV mass is usually indexed for BSA or for height raised to allometric powers such as 2.7 [14] or 1.7 [15]. As reported in the ESC/ESH guidelines [6,11], the indexation for height has advantages over indexing to BSA, to avoid the underestimation of the rate of LVH in overweight/obese subjects [16].

The standard echocardiographic approaches to LVM calculation presume a normal LV shape and have several intrinsic technical limitations including the need of a geometric assumption, frequent difficulties in the assessment due to beam orientation (often inducing off-axis views), and inaccuracy in presence of dilated ventricles or asymmetric hypertrophy. For these reasons, reproducibility of M-mode and two-dimensional-derived LVM appears in general to be suboptimal, limiting sometimes the possibility to follow the evolution of LVM over time [17,18]. Conversely, the novel three-dimensional reconstruction of LVM potentially represents a more reliable method for the assessment of LVM since does not need a geometric assumption and allows to obtain its direct calculation, even in patients with abnormal LV shape [19]. Three-dimensional echo-derived LVM has been validated against cardiac magnetic resonance (CMR), that is the gold standard imaging technique for the evaluation of this parameter [20–22] but is less expensive and more widely applicable in the clinical practice than CMR. Some limitations of three-dimensional LVM computation shall be acknowledged: mainly the possible incorrect detection of LV epicardial contours and of LV apex and also the impossibility of obtaining suitable three-dimensional images in patients with basically two-dimensional inadequate imaging. Thus, at the present time, both the feasibility and reproducibility of three-dimensional LVM determination are still suboptimal. Nevertheless, technical advancement in temporal and spatial resolution allows to acquire suitable three-dimensional echo images, with the possibility of amplifying LV apex region (virtual apex), which is the most critical part of the assessment [23].

Table 1 summarizes the cut-off values of abnormalcy of LVM with the different techniques. The knowledge of the cut-off points by using the different imaging techniques should be carefully considered. Notably, the cut-off points of LVM derived from standard echocardiography are prognostically validated, whereas those obtainable by three-dimensional echo are not. CMR-derived LVM and LVH were demonstrated to be prognosticators in AH [24].

The evaluation of differential diagnosis for LVH is important to exclude other possible causes of increased LV parietal walls, such as hypertrophic cardiomyopathy. Regional strain could be helpful in this context. Patients with hypertrophic cardiomyopathy present a more impaired regional longitudinal strain, particularly in apical segments, compared with hypertensive-LVH [23,34]. The assessment of regional strain is also useful for differentiating myocardial effects of AH from infiltrative diseases, as cardiac amyloidosis, being characterized by a regional impairment of longitudinal function which spares the apical segments (apical sparing) [35], and storage cardiomyopathy, such as Anderson Fabry disease [36].

Left ventricular geometry

In the early stages of AH, LV geometry remains generally normal, but as consequence of increased afterload, the shape of the LV is prone to morphological changes [37]. The standard echocardiographic definition of LV geometry presumes the use of LVM and RWT; the latter is commonly determined as the ratio between twice the posterior wall

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TABLE 1. Parameters used for assessment of left ventricular geometry

Method	Parameter	Thresholds of normalcy		References
Echo linear method	LV mass (g)	Women ≤162	Men ≤224	Lang *et al.* [13] Ilercil *et al.* [25]
	LV mass/BSA (g/m^2)	Women ≤95	Men ≤115	Lang *et al.* [13] Devereux *et al.* [26]
	LV mass/height (g/m)$^{2.7}$	Women ≤45	Men ≤49	Lang *et al.* [13] de Simone *et al.* [27]
	LV mass/height (g/m)$^{1.7}$	Women ≤60	Men ≤81	Chirinos *et al.* [15]
	Relative wall thickness (cm)	Women <0.42	Men <0.42	Lang *et al.* [8] Ganau *et al.* [28]
Echo 2D method	LV mass (g)	Women ≤150	Men ≤200	Lang *et al.* [8] Park *et al.* [29]
	LV mass/BSA (g/m^2)	Women ≤88	Men ≤102	Lang *et al.* [8]
	LV mass/height (g/m)$^{2.7}$	Women ≤47	Men ≤50	de Simone *et al.* [14]
Echo 3D method	LV mass (g)	Women ≤130	Men ≤170	Mizukoshi *et al.* [30]
	LV mass/BSA (g/m^2)	Women ≤80	Men ≤88	Mizukoshi *et al.* [30]
	LV mass/EDV ratio	Women <1.23	Men <1.22	Lembo *et al.* [31]
MRI method	LV mass (g)	Women ≤146	Men ≤186	Maceira *et al.* [32]
	LV mass/BSA (g/m^2)	Women ≤77	Men ≤93	Maceira *et al.* [32]
	LV mass/EDV ratio	Women <1.0	Men <1.12	Rider *et al.* [33]

EDV, end diastolic volume; LV, left ventricular.

thickness and LV diastolic diameter at end-diastole [38]. Adopting these two parameters, four geometrical patterns are described: normal geometry, concentric remodelling, concentric LVH – which consists in uniformly increased LV wall thickness, an increased LVM with normal cavity size [8,39] – and eccentric LVH, characterized by increased LV cavity size and LVM with normal LV wall thickness (Fig. 1). More recently, Khouri *et al.* [39] proposed a new classification of LV geometry patterns, considering also LV dilatation. By using this novel classification, survival was similar between hypertensive patients with eccentric LVH and normal LVM, whereas it was progressively reduced as LV dilatation occurred, achieving the lowest rate in patients with concentric LVH and LV dilatation [40–42].

An accurate evaluation of LV geometry is essential for the patient's risk stratification, to guide antihypertensive therapy and to identify a target organ damage [6]. However, due to the above mentioned limitations of two-dimensional assessment, this approach can be even improved by using LVM/end-diastolic volume (EDV) ratio, a novel index which has been firstly introduced by CMR [31]: higher the ratio greater the thickness to cavity ratio of the LV. This ratio was also found to correlate with myocardial fibrosis and outcome in hypertensive patients [31]. The feasibility of this index has been recently shown by using three-dimensional -echocardiography [31,43]. Three-dimensional echo-derived LVM/EDV ratio was also able to detect a higher rate of LV concentric geometry in comparison with two-dimensional assessment; three-dimensional LV mass/EDV ratio identified also patients with low stroke volume (SV) in the context of LV remodelling due to AH [31]. This aspect is particularly relevant as it might reveal an early functional impairment beyond the information carried by LV geometry alone. Accordingly, the use of three-dimensional echo could be a good compromise between two-dimensional echo and CMR in characterizing subclinical organ damage in AH. Table 1 shows the main parameters and their cut-off points of abnormalcy used for assessment of LV geometry by echo techniques.

Left ventricular function

Currently two-dimensional echocardiographic-derived LV ejection fraction (LVEF) is the most frequently used parameter for the assessment of LV systolic function. Nevertheless, LVEF suffers the limit of geometric assumption, it has poor reproducibility (day-to-day variability of about 10%) [44,45] and therefore it is able to detect LV dysfunction only in clinically overt stages [46]. In addition, LVEF is deeply influenced by load conditions and changes of LV geometry [47,48], both critical points in hypertensive patients [49]. Accordingly, LVEF is poorly accurate for detecting subclinical LV dysfunction in the clinical setting. In the 1980s this concept was firstly highlighted by the calculation of mid-wall fractional shortening, an index which, incorporating half part of the myocardial wall, allows to identify early LV systolic dysfunction in presence of LV concentric geometry, when LVEF is still normal [47]. To date, the assessment of myocardial mechanics can be much more easily performed by using pulsed Tissue Doppler imaging (TDI) or, better, speckle tracking echocardiography (STE), depending on the available level of technology [49]. TDI has demonstrated the diagnostic capability of differentiating between physiological and pathological LVH [50]. STE is angle-independent and very reproducible, being also relatively operator independent. It allows quantifying the different directional components of myocardial deformation such as longitudinal, circumferential and radial strain, and LV twisting as well. Although all these strain components well correlate with LVEF, global longitudinal strain (GLS) appears to be superior because of its largely better feasibility and reproducibility (about 6%) [51]. Moreover, despite being load dependent similarly to LVEF, GLS can be altered independently on changes of LV geometry [52]. Accordingly, a decline in GLS, has been shown to be evident even in presence of LVEF, being useful to identify preclinical stages of LV involvement in AH [53]. GLS has been found to be also associated with both the degree of LV filling pressures and the extent of myocardial fibrosis in uncomplicated hypertensive patients [54–58]. Preliminary experience showed

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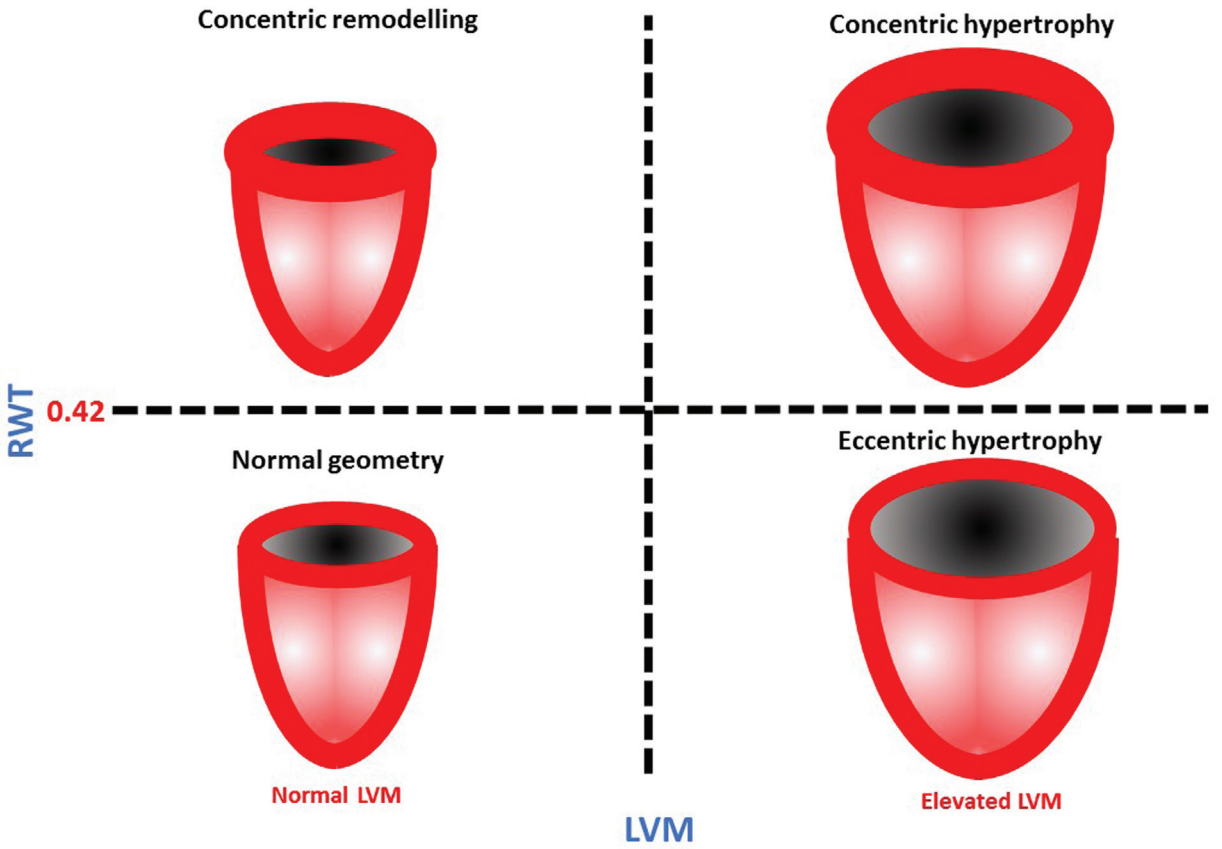


FIGURE 1 Schema showing different left ventricular geometry based on the left ventricular mass index and relative wall thickness. LV, left ventricular; LVM, left ventricular mass; RWT, relative wall thickness.

the potential usefulness of three-dimensional strain rate imaging in AH [59]. However, this technique is not ready yet to be used in the clinical practice, due to its limited availability and feasibility.

DIASTOLIC FUNCTION

In early stages of AH, LV diastolic dysfunction generally occurs long before LVEF is compromised [60]. Persisting elevated BP levels promotes LV diastolic dysfunction through various mechanisms, including increased afterload, myocardial ischemia [61], and myocardial fibrosis, which constitutes the main determinant of diastolic properties' alteration, it being characterized by an altered myocardial relaxation that interferes with normal LV diastolic filling [62].

According to the latest ASE/EACVI recommendations on diastolic function [62], when LVEF is above 50%, and any myocardial disease (e.g. presence of LVH, ischaemic or significant valvular heart disease) is excluded, recommendations suggest the use of four variables to determine the presence of diastolic dysfunction (septal $e' < 7$ or lateral $e' < 10$ cm/s, average $E/e' > 14$, tricuspid regurgitation systolic jet velocity >2.8 m/s, left atrial maximum volume index >34 ml/m^2) (Fig. 2). On the contrary, in presence of myocardial disease, even in presence of normal LVEF, recommendations suggest to apply the same algorithm used for patients with reduced LVEF to estimate LV filling pressures degree (Fig. 3) [62]. Thus, if LVH is present or not, the second or the first algorithm should be used respectively.

Significantly, diastolic dysfunction is strongly related with LV longitudinal systolic dysfunction, and it might occur even before the development of LV concentric geometry [63]. This implies that subtle systolic dysfunction might be responsible for an increase in LV filling pressures, which is a strong predictor of both prognosis and clinical functioning [64]. It has to be stressed that AH generally clusters with other cardiovascular risk factors [64], and diabetes mellitus, impaired renal function, aging and obesity augment the progression of diastolic dysfunction in these patients [65]. Furthermore, diastolic dysfunction tends to improve during antihypertensive treatment [66–68], which makes it a valuable tool to test therapy efficacy, as it is also easily assessed through the echocardiographic exam.

The role of hypertensive-induced diastolic dysfunction in heart failure

AH is one of the main risk factor for the development of heart failure, in particular in presence of preserved LVEF (HFpEF). Traditionally, HFpEF pathophysiology was explained by several conditions that induced an increased LV work due to high afterload, while emerging models have highlighted the role of systemic proinflammatory changes determined by different comorbidities, including AH [69,70]. In fact, one of the first signs of an increased LV afterload corresponds to diastolic dysfunction. When this

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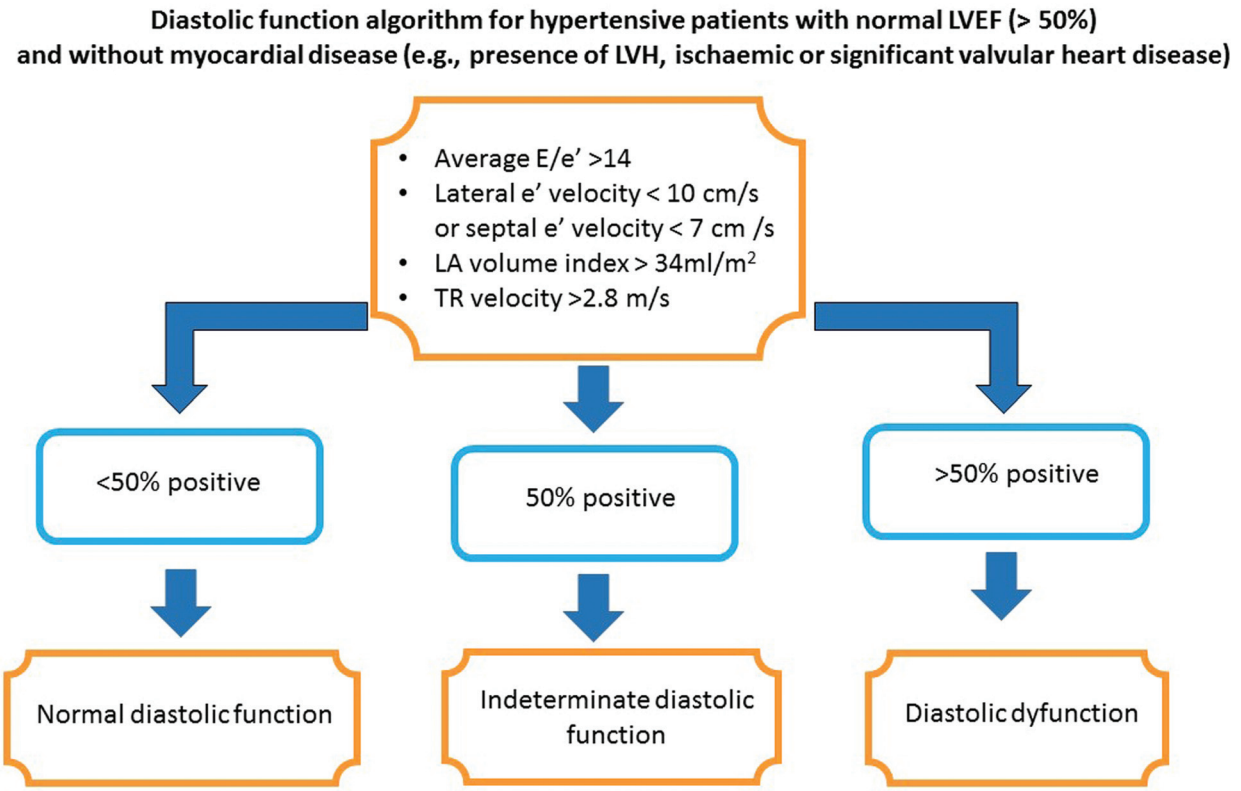


FIGURE 2 Diagnostic algorithm for diastolic dysfunction in hypertensive patients with normal left ventricular ejection fraction and absence of myocardial disease. LA, left atrium; LV, left ventricular; LVEF, left ventricular ejection fraction; LVH, left ventricular hypertrophy; TR, tricuspid regurgitation.

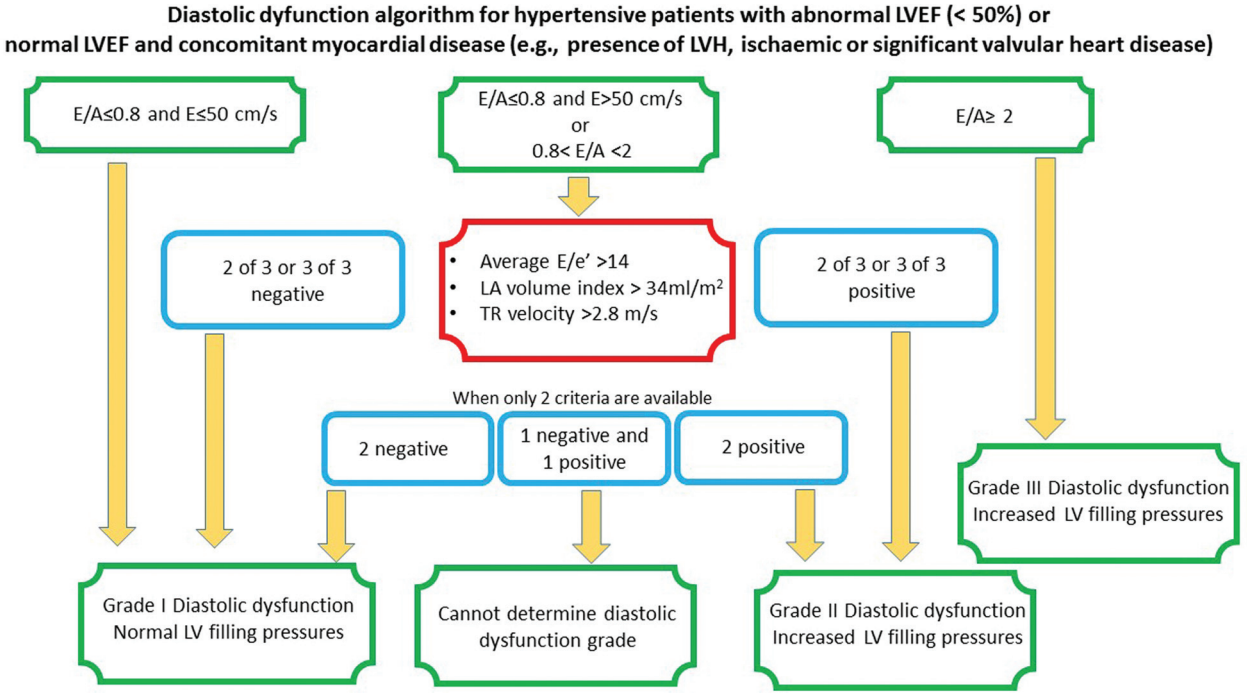


FIGURE 3 Diagnostic algorithm for diastolic dysfunction in patients with reduced left ventricular ejection fraction or normal left ventricular ejection fraction and concomitant myocardial disease. LA, left atrium; LV, left ventricular; LVEF, left ventricular ejection fraction; LVH, left ventricular hypertrophy; TR, tricuspid regurgitation.

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pressure overload is sustained over time, diastolic function appears more impaired, LV remodelling becomes progressively decompensated, and HFpEF ensues [71,72].

LEFT ATRIUM

Left atrium is the other cardiac chamber affected by the pressure overload that accompanies AH. In fact, when diastolic dysfunction has developed, the contribute of left atrium to LV filling becomes essential, therefore left atrial pressure tends to progressively increase [73], which then leads to a gradual left atrial dilatation. It has been proven that left atrial enlargement is proportional to the severity of diastolic dysfunction and to the duration of the haemodynamic overload [74], representing the memory of chronic increase of left atrial pressure in AH. As a matter of fact, an increase of left atrial size frequently occurs in hypertensive patients, and besides diastolic dysfunction, it appears to correlate also with obesity, older age and particularly with a clear-cut LVH [75]. LV geometry and mechanics are important factors influencing left atrial afterload.

While 2018 ESC/ESH guidelines propose to identify left atrial dilation computing left atrial size as left atrial volume indexed to height powered to 2 [6,76], the current ASE/EACVI echocardiographic recommendations on diastolic function suggest that left atrial size should be assessed by measuring left atrial volume indexed for BSA, a measure that is highly validated and commonly used in the clinical practice [11,62]. Left atrial antero-posterior diameter, measurable in the parasternal long-axis view, preferably using the two-dimensional mode, despite largely applied in the past, does not accurately represent the size of this chamber which is tridimensional. Left atrial volume should be preferably assessed through the biplane disk summation technique, as it is characterized by fewer geometric assumptions, rather than through the area-length method [11]. In both cases, left atrial endocardial borders are traced in the apical four-chamber and two-chamber views. Three-dimensional -echocardiographic assessment of left atrial volume has been shown promising results, it being more accurate in comparison with CMR [75]. Also three-dimensional echo-derived left atrial phasic volumes have been demonstrated to correlate with hypertensive organ damage (a left atrial active SV index of 5.9 ml/m^2 predicted end-organ damage with a sensitivity of 82% and a specificity of 92%) [77] and with the severity of hypertensive retinopathy [78]. However, these experiences are preliminary and three-dimensional echo of left atrial volume should not be considered in the routine assessment of AH.

STE could also help to assess left atrial function. A reduced left atrial strain was found in patients with suboptimal control of BP [79], it occurring before the onset of clear-cut left atrial dilatation [80] and independently on LV longitudinal dysfunction [81]. In addition, when AH is complicated by paroxysmal atrial fibrillation, STE-derived left atrial reservoir, conduit and pump function are early impaired [82].

AORTA

Current ESC/ESH guidelines on AH put emphasis on the importance of evaluating arterial stiffness [6,83] but not examining the aortic size at both ascending and abdominal level. Arterial stiffness can be used to define asymptomatic organ damage in AH and is a substrate for the development of resistant hypertension. It refers to the elastic properties of the aorta, which affect vessel dimension, pressure and blood flow across every cardiac cycle [83]. It is also known to be a predictor of adverse cardiovascular outcomes [84]. ESC/ESH guidelines propose the carotid–femoral pulse wave velocity (PWV) as the gold standard to assess arterial stiffness [6,85]: the stiffer the arteries, the higher the PWV. Therefore, correctly diagnosing and treating patients at the beginning of the disease could prevent this further complication by limiting the aortic damage [86,87]. However, this tool is not currently used in the clinical practice.

Although aorta is greatly affected by chronically elevated BP, it is not frequently evaluated in the routine echocardiographic work up of hypertensive patients. Nevetherless, several studies demonstrated that AH accelerates the ageing dependent enlargement of thoracic aorta and particularly affects ascending aorta and aortic arch. This process can induce a progressive dilatation and loss of shape of sinotubular junction, causing aortic regurgitation [88]. Functional classification of aortic root abnormalities responsible for aortic regurgitation provides information for surgical management. This information can be useful for targeting the optimal time and strategy for aortic valve-sparing surgery in ascending aorta aneurysms [89]. Hypertensive-induced ascending aorta dilation is also associated with both increased LVM and arterial stiffness [90,91]. It is conceivable that AH could lead to a progressively increased mechanical stress on the aortic wall, which in turn induces elastin fragmentation, and finally aortic dilatation [91]. Although a certain degree of controversy exists about the effect of AH on the aortic root [92–94], recent studies seem to demonstrate that elevation in DBP influences the aortic root dilation, also in relation with the AH disease duration [95].

According to these findings, the aortic diameters of hypertensive patients should be determined at different levels with two-dimensional echocardiography from the parasternal long-axis view. According to the latest ASE/EACVI recommendations [8], the aortic annulus should be measured using the inner edge-to-inner edge, whereas it should be preferred the leading edge-to-leading edge convention for measuring the aortic root and the ascending aorta. Notably, the ascending aorta distensibility appears to be a noninvasive predictor of outcome and might therefore be helpful for guiding the optimal antihypertensive treatment [96]. Recently, TDI has been used to estimate the motion of the aortic wall [97]; the velocity values, expressing the aortic elasticity, were found to be lower in hypertensive patients than in the normal population of the same age [98]. In particular, the anterior wall motion velocity of the ascending aorta has been proven to be a predictor of LV geometry and function [99].

Also the abdominal aorta should be explored by ultrasound in hypertensive patients. AH is one of the main risk factor for the development of abdominal aorta aneurysm (AAA). The prognostic role of abdominal aorta evaluation has been recently investigated in patients awaiting for endovascular repair of AAA [100], the dilation of abdominal aorta being independently associated with long-term mortality in this cohort of patients [101]. These findings highlight the possible screening power of abdominal aorta

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ultrasound assessment, which could be even performed by using hand-held echocardiography [101]. Diameter measurements should be performed in the plane perpendicular to the arterial axis, to avoid any overestimation of the actual diameter.

AORTIC VALVE: AORTIC REGURGITATION AND PARADOXICAL LOW FLOW LOW GRADIENT AORTIC STENOSIS

AH may induce a mechanical damage on the aortic valve, causing abnormally high stress on aortic leaflets, turbulent flow and endothelial injury, and subsequent progression towards alteration in aortic valve morphology, causing both aortic regurgitation and stenosis [102]. Therefore, the evaluation of aortic valve morphology and function represent a valuable point in echo providing information. Aortic valve regurgitation may be due to accelerated AH-induced valve deterioration or it could be functional. In this second circumstance, functional aortic regurgitation is mainly associated to tethering of the leaflets, it depending on the sinotubular junction/annulus mismatch, as a consequence of ascending aorta dilatation [89,103]. AH and aortic stenosis are often concomitant diseases, particularly in the elderly patients [103–105]. Accordingly, the echocardiographic assessment of aortic valve area and gradients is extremely useful for detecting and monitoring the progression of aortic stenosis in AH [106]. A particular type of aortic stenosis, the paradoxical low flow low gradient aortic stenosis, has been typically described in hypertensive patients. It is characterized by a mismatch between aortic valve area and mean pressure gradient: aortic valve area is severely reduced in presence of low mean pressure gradient. The diagnosis of paradoxical low flow low gradient aortic stenosis is performed when valve area less than 1 cm^2 with a peak velocity less than 4 m/s, a mean pressure gradient less than 40 mmHg and SV index less than 35 ml/m^2 despite normal LVEF. LV features of low flow low gradient aortic stenosis include LV concentric geometry and small LV volume [106]. It is conceivable that the hypertensive heart, characterized by deep remodelling with the presence of LV small diameters, LV concentric geometry, high ventricular wall stiffness and reduced LV SV, could not be prone to bear the impact of aortic stenosis. Thus, paradoxical low flow low gradient aortic stenosis is associated with poor prognosis [107]. In some cases it is often difficult to perform a differential diagnosis between paradoxical low flow low gradient aortic stenosis and pseudosevere aortic stenosis. Evaluation of calcification degree with Agatston calcium score by multislice computed tomography may be helpful in this setting and can resolve the diagnosis [106]. Based on the above mentioned evidences, a thorough echocardiographic assessment in patients affected by AH should not overlook valve evaluation.

ECHOCARDIOGRAPHIC PARAMETERS HAVING A PROGNOSTIC IMPACT IN ARTERIAL HYPERTENSION

An appropriate diagnosis of AH and correct assessment of cardiovascular risk are essential to initiate antihypertensive treatment. Echocardiography is a very helpful tool in this context, not only for the evaluation of organ damage, but also for defining the prognostic profile of a given hypertensive patient. LVH and LV geometric pattern provide important prognostic information [7]. LV concentric hypertrophy is associated with an increased rate of mortality and cardiovascular events, even after adjusting for other cardiovascular risk factors including LVM, and showing the greatest mortality risk in patients with suspected coronary artery disease [7,108,109]. Also increased left atrial volume is a prognostic indicator of cardiovascular morbidity and mortality, and a left atrial volume greater than 34 ml/m^2 was associated with poor prognosis including death, heart failure, atrial fibrillation and ischaemic stroke [110,111]. The most consistent evidence regards left atrial volume rather than left atrial area and left atrial antero-posterior diameter [112–114]. Left atrial dilation is also expression of diastolic dysfunction and increased LV filing pressures. Accordingly, diastolic dysfunction and in particular *E/e′* ratio have been shown to be strong predictors of heart failure and cardiovascular events, independently on several confounders including LVM [62,63]. Table 2 shows the main echocardiographic parameters predicting poor

TABLE 2. Echocardiographic parameterspredicting poor prognosis in patients affected by arterial hypertension, their cut-off values of normalcy and references

Parameter	Thresholds of normalcy	References
LVM	LVM/height$^{2.7}$ ($g/m^{2.7}$) ≤50 men, ≤47 women LVM/BSA (g/m^2) ≤115 men, ≤95 women	Levy *et al.* [7] Ghali [109] Armstrong [115] de Simone *et al.* [14] Krumholz *et al.* [108] Gosse *et al.* [116] Verdecchia *et al.* [41]
RWT	<0.42	Ghali *et al.* [109] Gaasch *et al.* [37]
LA size	LA volume index <34 ml/m^2 LA AP diameter (cm) <4.0 men, <3.8 women	Abhayaratna *et al.* [117] Verdecchia *et al.* [113] Gardin *et al.* [114]
Diastolic function	First or second algorithm according to recommendations septal $e' \geq 7$ or lateral $e' \geq 10$ cm/s, average $E/e' < 14$, tricuspid regurgitation systolic jet velocity <2.8 m/s, LA volume index <34 ml/m^2	Aljaroudi *et al.* [118] Bella *et al.* [119] Dini *et al.* [63] Oh *et al.* [72]

2D, two-dimensional; AP, antero posterior; LA, Left atrial; LVM, Left ventricular mass; RWT, relative wall thickness.

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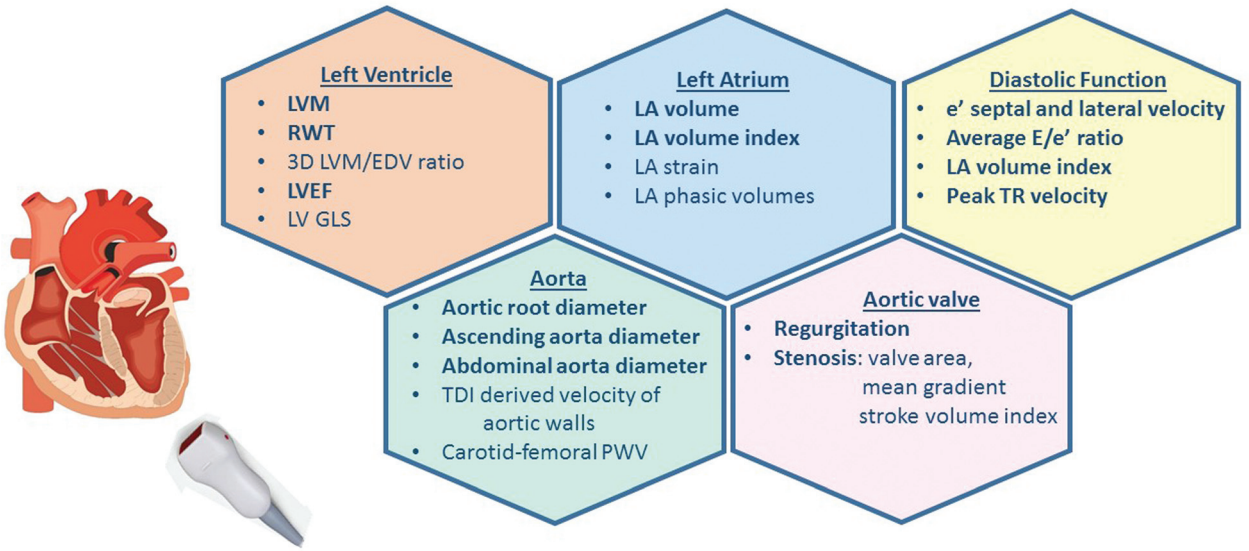


FIGURE 4 Picture showing the echo parameters useful in the evaluation of the hypertensive patient. In bold letters the parameters that should always be assessed in hypertensive patients. EDV, end-diastolic volume; GLS, global longitudinal strain; LA, left atrium; LV, left ventricular; LVEF, left ventricular ejection fraction; LVM, left ventricular mass; PWV, pulsed wave velocity; RWT, relative wall thickness; TDI, tissue Doppler imaging; TR, tricuspid regurgitation.

prognosis in patients affected by AH. On the contrary, evidence on sensitivity and specificity of those parameters in predicting cardiovascular events is lacking in the hypertensive setting.

CONCLUSION

The role of echocardiography in the thorough assessment of the hypertensive patient is very useful, as it allows the measurement of several parameters that correlate with organ damage. Figure 4 summarizes the echocardiographic parameters that should be evaluated in hypertensive patients. In addition to LVH and left atrial enlargement identified from current ESC/ESH guidelines on AH to detect cardiac injury, multiple echocardiographic parameters, such as GLS, left atrial strain and diastolic evaluation could identify an early heart impairment. The evaluation of LV and left atrial function, rather than the simple measure of their dimensions, has given promising results in early detection of cardiac dysfunction, which might help identifying patients that can benefit from a more aggressive treatment and a closer follow-up. Also the evaluation of diastolic dysfunction is extremely important, because it can occur before the development of LV geometry changes. Moreover, in a complete overview on AH-induced cardiac impairment the assessment of aortic dimension and aortic valve function should not be overlooked. In this context, the combination of standard and advanced echocardiographic techniques should be carefully considered to diagnose subclinical cardiac organ damage, stratify prognosis and address management at the best.

In this view, based on a preliminary clinical assessment, the echocardiographic evaluation could gather the maximum of relevant information on heart and aorta by influencing patients' treatment, and also establishing correct timing of follow-up. Accordingly, under well defined circumstances, echocardiography could present even a valuable cost/effectiveness ratio in hypertensive patients.

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Conflicts of interest

There are no conflicts of interest.

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