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GUIDELINES

Update of the French Society of Cardiology recommendations on indications for Doppler echocardiography published in 1999

Mise à jour des Recommandations de la Société Française de Cardiologie concernant les indications de l'échocardiographie Doppler publiées en 1999

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In order for recommendations to be constructed, different experts must be brought together to review the literature and it is only after several revision stages that a consensus document is produced. Despite this long process, one of the main limitations remains the lack of unequivocal studies to enable evidence-based recommendations to be proposed. In order to construct unambiguous recommendations it is essential that these are based on studies examining the utility (diagnostic, prognostic, therapeutic) of echocardiography data comparing a strategy which includes these findings with one which does not. In general terms, medical imaging particularly lacks this type of study [1]. In writing these recommendations, we are aware of these limitations, together with our inability to produce very regular updates (because of the constant changes in medical technology and information).

Our aim above all is to guide the practitioner by placing particular emphasis on obtaining the parameters necessary for a good quality investigation. These recommendations by no means claim to be a "gold standard": we have not attempted here to offer statements representing what is "right", to say what a cardiologist is or is not entitled to do.

This update is also designed to review the information published in 1999 [2] on hypertension, organic valve disease, prosthetic valves, chronic ischaemic heart disease and acute coronary syndromes, coronary risk stratification before non-cardiac surgery. We felt it was important to describe and extent the initial document on the subject of obtaining parameters during the investigation by describing wherever possible the methodology, interpretation and utility of the parameter together with the specific problems it raises. A review of the essential parameters to be collected (the "minimum" data set) and optional parameters is provided in tabular form.

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HYPERTENSION

Several cohort studies have shown left ventricular hypertrophy (LVH) is a powerful risk factor for cardiac and cerebrovascular events in hypertensive patients even after adjusting for conventional risk factors (blood pressure value, sex, age, smoking, diabetes, dyslipidaemia) [1, 2]. Echocardiography can be used to diagnose LVH with far greater specificity and sensitivity than ECG [3]. Some risk data however suggest that echocardiographical and ECG LVH provides an additional predictive value [4].

Echocardiography remains the routine method for measuring left ventricular mass (LVM) and the only one which has been validated anatomically [5]. MRI, which is more expensive, less widely available and cannot be used in routine practice, is considered to be the most accurate method [6].

Measurement and classification technique for left ventricular geometry

Echocardiographical measurement of LVM in M-mode has been validated anatomically. It must be performed following precise recommendations [7], using the left parasternal or, failing that, subcostal view. The use of harmonic imaging overestimates wall thicknesses [8]. If it is not possible to align the M-mode beam correctly, thickness and diameter measurements may be performed on a left parasternal long axis image frozen in end diastole [9]. LVM is calculated from the diastolic diameter and LV diastolic thicknesses: it is essential to calculate an average of several measurements (minimum 2 ideally 3).

Studies on normal subjects have shown that LVM partly depends on sex and on body shape [10]. In order to be interpreted, the LVM result is therefore expressed as an index (divided) by the patient body shape index. In general, body surface area is used although we must be aware that the definition of the LVH threshold is still blurred illustrating the difficulty of defining a pathological threshold for a continuous variable: 111 g/m^2 to 134 g/m^2 in men and 100 g/m^2 to 125 g/m^2 in women depending on the publication [11]. The recent ASE recommendations [9] propose a value of 115 g/m^2 in men and 95 g/m^2 in women. This approach underestimates the prevalence of LVH in obese people and a height indexation^{2,7} is recommended (hypertrophy threshold around 50 g/m^{2,7} in both sexes) [12] (table 1).

Although the measurement technique is robust the reproducibility of LVM measurements is not perfect. Comparisons of two successive investigations in the same patient (between-test reproducibility) or reading the same M-mode tracing on two separate occasions may result in a difference range of 60 g in the LVM calculation (standard deviation 30 g) [13]. In other words, a change of at least 18-20% in LVM is required to be considered to reflect a true difference: these values can be reduced to 10 to 13% in the hands of experienced users with good quality instruments [14].

Several studies have shown that calculating LVM alone is inadequate and that it is useful to describe left ventricular geometry to better stratify risk [15, 16]. Calculating certain additional parameters (h/r or relative wall thickness defined as the ratio of "sum of diastolic thicknesses/left ventricular diastolic diameter") can define four geometrical shapes [17] of "increasing" severity according to a detailed international classification: normal geometry (no LVH and h/r≤0.44), concentric remodeling (no LVH but h/r>0.44), eccentric LVH (LVH and h/r≤0.44), and concentric LVH (LVH and h/r>0.44). The recent ASE recommendations [9] propose a threshold of 0.42 for the h/r ratio. This classification must be used routinely and shown in all reports on hypertensive patients (table 1).

Other measurements

Measurement of the endocardial fractional shortening (eFS) or left ventricular ejection fraction (LVEF) raises no specific questions in terms of interpretation. In particular, a low LVEF can reflect impaired intrinsic contractility but may also occur secondary to raised afterload with no impairment of intrinsic contractility. Calculation of end-systolic stress (ESS) which is obtained from arterial blood pressure and left ventricular parameters obtained in M-mode (equation 1), can identify these situations [18]. An inverse linear relationship exists between the eFS and ESS and it is therefore possible in a given patient to obtain a theoretical normal value for the eFS from calculating ESS: in the presence of a high ESS a relatively low LVEF (or eFS) value should be interpreted as normal.

Equation 1: Calculation of meridional end-systolic stress (mESS)

$$mESS = (0.334 \times SBP \times LVIDs)/(PWTs \times (1+PWTs/LVIDs)) \text{ in } 10^3 \text{ dyn/cm}^2$$

LVIDs: end-systolic left ventricular internal diameter; PWTs: end-systolic posterior wall thickness; SBP: systolic blood pressure.

Studying the midwall fractional shortening (mFS) can also identify unrecognised contractility disorders (equation 2) [19, 20]. This involves identifying shortening of the myocardial layers in midwall which are very rich in circumferential fibres and which therefore contribute to circumferential shortening (unlike the subendocardial layers which are rich particularly in longitudinal fibres). This calculation appears complicated but is based on routine parameters [20] (cf. table 2). It has been shown to be of prognostic value [21]. The inclusion of ESS is also recommended in difficult situations, particularly when the mFS appears to be reduced.

Equation 2: Calculation of midwall fractional shortening (mFS)

$$mFS\ (\%) = [((LVIDd+(PWTd/2)+(IVSd/2))-((LVIDd+(IVSd/2)+(PWTd/2))^3-(LVIDd^3-LVIDs^3))^{1/3})/(LVIDd+(PWTd/2)+(IVSd/2))]*100$$

IVSd: interventricular septum at end diastole; LVIDd: end-diastolic left ventricular internal diameter; LVIDs: end-systolic left ventricular internal diameter; PWTd: posterior wall thickness at end diastole.

Filling pressures are assessed from conventional parameters: generally reverse transmitral flow (Em<Am) reflects normal or only slightly increased filling pressures [22]. In LVH, however, this rule varies and this type of flow may be compatible with any situation, including a large rise in left ventricular filling pressures [23]. In most situations, therefore (Em>Am or Em<Am with LVH), other indices are required to assess filling pressures. Studies conducted in various populations have shown that examination of the mitral annulus by pulsed tissue

Table 1 Hypertension: obligatory parameters to be recorded.

Measurements	Calculations	Technical comments	Value
LVIDd, LVIDs		Parasternal M-mode; failing that sub-costal M-mode; failing that parasternal 2D	LV dilated if: LVIDd >31 mm/m^2(men) LVIDd >32 mm/m^2(women)
IVSd, PWTd		Parasternal M-mode; failing that sub-costal M-mode; failing that parasternal 2D	PWTd >11 mm generally indicates LVH
	(IVSd+PWTd)/ LVIDd		Eccentric if <0.42-0.44; Concentric if ≥0.42-0.44 Prognostic value
	LV mass/m^2	Penn or ASE equations	Unusable in obese people Thresholds: men: 111-134 g/m^2 ; women:100-125 g/m^2 Prognostic value
	eFS		Normal: 27-45% (if concentric geometry, eFS normal >40%) If eFS <27%, take ESS into account in interpreting
Aorta		High parasternal for the tubular	Small tubular dilatation possible in hypertensive
Left atrium		Diameter (Parasternal M-mode); Area (4 chambers vue); Volume (4 and 2 chambers vue).	No apparent dilatation (literature ambiguous)
Em, Am		Pulsed Doppler, between annulus and tip of valve	If Em<Am and no LVH; suggests normal LV filling pressures For other situations, see table 2
VmaxTR		Failing this, record pulmonary regurgitation.	SPAP threshold = 40 mmHg from 50 years old

Am: transmitral flow A wave; ASE: American Society of Echocardiography; ESS: end-systolic stress; LVIDd: end-diastolic left ventricular internal diameter; LVIDs: end-systolic left ventricular internal diameter; Em: transmitral flow E wave; eFS: endocardial fractional shortening; LVH: left ventricular hypertrophy; SPAP: systolic pulmonary artery pressure; PWTd: posterior wall thickness at end diastole; IVSd: interventricular septum at end diastole; LV: left ventricle; Vmax TR: maximum tricuspid regurgitation velocity: 2D: 2 dimensional

Doppler [24], and/or measurement of colour M-mode propagation velocity [25], or even studying pulmonary venous flow by pulsed Doppler [26] can provide a qualitative evaluation of left ventricular filling pressures.

The initial part of the aorta must be measured at the level of the sinus of Valsalva and at tubular level as a small dilatation of the initial tubular aorta has been described in hypertensives [27]; left atrial dilatation is found variably in the literature in hypertensives [28].

Measurement of pulmonary pressures (tricuspid or pulmonary regurgitation flow) is essential to assess the pulmonary consequences of hypertensive heart disease (systolic and/or diastolic left ventricular dysfunction). Being hypertensive shifts the limit of normality for pulmonary pressures upwards [29].

Dynamic obstruction may be seen in a hypertensive patient with LVH, generally concentric (small cavity, thick walls). Typically, these are patients who have become symptomatic (dyspnea, chest pain) after vasodilators, or fluid depletion (diuretics) [30, 31].

Utility of Doppler echocardiography in the hypertensive

Measurement of LVM and assessment of left ventricular geometry contribute to risk prediction in the hypertensive.

Many studies have shown 1) the adverse prognostic role of LVH [1, 2], 2) the increased risk associated with concentric geometry in patients with LVH [32], 3) the increased risk in patients with concentric remodelling (normal mass, increased h/r) compared to people with a strictly normal left ventricle [15, 16]. Doppler echocardiography may therefore form part of the initial assessment of any hypertensive patient, particularly to obtain baseline left ventricular geometry.

The presence of LVH or even concentric remodelling in the low risk hypertensive should lead to pharmacological treatment of the hypertension [33]. Concentric remodelling ultimately resulting in treatment being started was found in 13% of a population of hypertensive patients left without treatment after strict application of international recommendations (particularly not taking account of echocardiography findings) [34].

Conversely, some studies have shown that regression of LVH is beneficial [35], whereas progression is associated with poor prognosis [36]. These results however do not support repeated LVM measurements in the follow-up of a hypertensive patient as the changes in LVM in response to treatment are usually within the limits of reproducibility of the measurement [37].

Apart from assessment of cardiovascular risk Doppler echocardiography findings are also extremely useful in the symptomatic hypertensive patient: identification of associated heart disease, raised left ventricular filling pressures (Em<3 cm/s has a poor prognostic value) [38], pulmonary arterial hypertension, left intraventricular dynamic obstruction on treatment, etc.

Finally, Doppler echocardiography findings can guide treatment in some situations: the presence of normal left

ventricular geometry in mild hypertension can predict good blood pressure control by lifestyle and dietetic measures alone [39]. The choice of treatment for isolated LVH and/or left ventricular dilatation will be different.

Recommendations from the learned societies

The main published recommendations on the management of hypertension (HT) are still vague about the role of echocardiography. This lack of detail may be explained by 1) the absence of pragmatic studies comparing management strategies which do or do not incorporate echocardiography findings and 2) the financial implications if the investigation were to become used systematically, even in targeted cases.

The American findings from the Joint National Committee [40] recall the need to identify target organ damage and the possibility of using clinical and laboratory findings and other tests to do this… although no further details are given.

European recommendations [33] are more precise and stress the role of possible LVH in assessing overall cardiovascular risk: they suggest up to 50% of hypertensives may be underestimated, being considered to be at low or moderate risk after a standard assessment becoming high risk when echocardiography or carotid findings are incorporated. These tests are therefore recommended particularly when a decision is being taken not to treat or doubt about the need to treat exists [34].

The British Hypertension Society [41] states that echocardiography is one of the assessment tools without giving any specific guidance. The recommendations stress the role of target organ damage, for example, in guiding the decision to treat mild hypertension (SBP 140-159 mmHg and/or DBP 90-99 mmHg).

Echocardiography is not recommended routinely in all patients in the Canadian recommendations [42] and must not be used to investigate for regression of LVH on treatment. These minimalist recommendations appear however to have been very widely disputed and because of the increasing amount of data showing the prognostic role of LVH, the next recommendations should provide more specific details about the place of echocardiography.

The French Higher Health Authority [43] stresses the role of target organ damage (LVH), requiring drug treatment for SBP of 140-179 mmHg or DBP 90-109 mmHg. Echocardiography is not however recommended systematically but can be performed in specific situations. The real place of echocardiography in the initial assessment will need to be described in the future, particularly from medico-economic studies.

All the recommendations therefore stress the prognostic value of LVH and its consequences in terms of management…, but very few take the further step of clearly describing the place of echocardiography.

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Table 2 Hypertension: optional parameters to be recorded.

Measurements	Calculations	Technical comments	Value
SBP		During the investigation (automatic measurement)	
PWTs			
	LV mass/height^{2,7}	Height in "meters"	Prognostic value Thresholds 51 g/m^{2,7} Suitable in both sexes and in all body types (obese ++)
	mFS	Calculated from the LVIDd, LVIDs, IVSd, PWTd	Normal values 15-23% Prognostic value
	End systolic stress	Requires SBP, PWTs, LVIDs	Aid to interpreting eFS <27% Aid to interpreting mFS <15%
Ea		Septal and lateral site	Prognostic value
Vp		Color M-mode	
Ap duration		Pulsed Doppler	
	Em/Ea Em/Vp Duration (Ap-Am)	Necessary to assess LV filling pressures When Em>Am When Em<Am and LVH	Filling pressure is raised if: Em/Ea>15 Em/Vp>2 Duration (Ap – Am) >20ms
Testing for coarctation		Continuous wave Doppler, suprasternal	
LV outflow tract obstruction		Continuous wave Doppler, apex	Consider in patients who have become symptomatic on treatment

Am: transmitral flow A wave; Ap: pulmonary venous flow A wave; Em: transmitral flow E wave; Ea: pulsed tissue Doppler E wave at the mitral annulus; eFS: endocardial fractional shortening; IVSd: interventricular septum at end diastole; LVIDd: end-diastolic left ventricular internal diameter; LVIDs: end-systolic left ventricular internal diameter; mFS: midwall fractional shortening; LVH: left ventricular hypertrophy; PWTd: posterior wall thickness at end diastole; PWTs: end-systolic posterior wall thickness; SBP: systolic blood pressure; Vp: color M-mode flow propagation velocity.

Consensus indications for Doppler-echocardiography in the initial assessment of a hypertensive patient

Class I
– Transthoracic Doppler echocardiography in the initial assessment of repercussions of hypertension associated with known or suspected heart disease.
– Transthoracic Doppler echocardiography in the initial assessment of repercussions of hypertension associated with unexplained cardiac symptoms.
– Transthoracic Doppler echocardiography in the initial assessment of repercussions of hypertension associated with severe hypertension (high risk) even if the ECG is normal.
– Transthoracic Doppler echocardiography in the initial assessment of an ECG abnormality: LVH, LBBB, repolarisation disorders.

Class II
Transthoracic Doppler echocardiography in the initial assessment of repercussions of hypertension particularly if the results may alter management (for example low or medium risk level, guiding a decision not to treat).

Class III
Transoesophageal echocardiography or stress echocardiography assessment of uncomplicated hypertension.

Consensus indications for transthoracic Doppler echocardiography in the follow-up of a hypertensive patient

Class I
– Annual follow-up of treated hypertension with systolic left ventricular dysfunction on initial Doppler echocardiography.
– Follow-up of left ventricular function every 2 to 3 years in treated hypertension associated with LVH on initial Doppler echocardiography.
– Evaluation of treatment-resistant hypertension even with a normal ECG.

Class II
2 to 3 years follow-up of treated hypertension associated with left ventricular diastolic dysfunction on initial Doppler echocardiography.

Class III
Evaluation of regression of LVH on treatment.

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ORGANIC VALVE DISEASES

Introduction

Doppler echocardiography has become the reference method to describe valvular anatomy, establish the diagnosis of dysfunction and quantify precisely the mechanism responsible for dysfunction by assessing the repercussions on dimensions and function of the cardiac cavities and to assess the resultant haemodynamic disturbances.

Quantification relies on the coherence of a number of parameters which when taken in isolation may be a source of error. Assessment of regurgitation which is traditionally more difficult than stenosis, has benefited from the use of the proximal isovelocity surface area method (PISA).

All the quantification methods, including assessment of a stenosed orifice by the surgeon (which does not strictly represent the functional in vivo orifice) have sources of error, particularly in certain situations highlighting the importance of comparing echocardiography results with clinical findings; and in difficult cases (asymptomatic patients, inconsistency between symptoms and the resting assessment, low output etc.) it is useful to perform a dynamic test (pharmacological or usually physical exercise).

The indications for transoesophageal echocardiography (TOE) have reduced in parallel to improvement in transthoracic imaging and are now reserved for well defined, specific situations.

Finally, a range of investigations (coronary artery CT scan or coronary angiography in particular), are still necessary in a large number of patients prior to surgery.

Assessment of valve diseases: Methods of measurement

Orifice stenoses

Doppler echocardiography can be used to diagnose valvular stenosis, to describe the extent of cusp damage (fibrosis,

calcification), to assess the restriction (tethering?) of the valve apparatus and to assess left ventricular function.

Aetiology and mechanism

Examination of the aortic valve is designed to assess the level of valve calcification and to identify a bicuspid valve. The diagnosis of a bicuspid aortic valve, although difficult in diastole in the presence of a raphe or valve calcifications is based on demonstrating 2 asymmetrical sigmoid cusps in a short axis parasternal view.

Examination of the mitral valve must be exhaustive in order to guide any possible therapeutic procedure. Different scores have been proposed, incorporating cusp thickness and mobility, presence of calcifications, commissural areas and thickening of the subvalvular apparatus. In particular, the importance of valvular calcifications and the state of the subvalvular apparatus are anatomical factors that predict the success of percutaneous commissurotomy.

Quantification

The quantification methods depend partly on the studied orifice (tables 1-4):

- Regardless of valve orifice, Doppler measurement of the mean transvalvular pressure gradient correlates well with the mean gradient found on catheterisation when the measurements are performed simultaneously [1, 2]. It is always important to correctly align the transvalvular flow and the ultrasound beam as a significant angle between these two is liable to lead to gross under-estimation of velocity and pressure gradient. This is a critical point particularly for aortic stenoses — placing the probe in different positions minimizes the angle between the jet and the ultrasound beam. An adequate Doppler signal is generally obtained from the apical position. However, in some patients, despite an apical Doppler envelope of acceptable quality, higher velocities may be are recorded in the right parasternal or even suprasternal or xiphoid views.
- Measurement of the functional surface area by the continuity equation is reliable for both mitral and aortic orifices [3, 4]. To optimise indications for treatment, particularly for the aortic orifice, body surface area needs to be taken into account. This method requires coupling of two-dimensional echocardiography and both Doppler modes (pulsed and continuous). Apart from technical problems related to errors in measuring the diameter of the left ventricular outflow tract or incorrect positioning of the pulsed Doppler sample, the method may be inappropriate in the presence of an accelerated flow in the outflow tract, as well as in patients with mitral regurgitation or atrial fibrillation with rapid ventricular response. Although estimating valve surface area is less dependent on haemodynamic conditions than estimation of the gradient, some authors dispute its validity in patients with low cardiac output [4 bis]. The measurement of the radius of the Doppler colour convergence zone [5] in 2D or TM mode, has been recently proposed for the mitral orifice as alternative to the continuity principle. However, this method is not frequently used in routine practice.
- Measurement of the time required for the pressure across the mitral valve during diastole to fall to one-half of its initial value (pressure half time) can be use for the quantification of mitral stenosis [6]. This is straightforward to perform although its interpretation must take into account technical causes of error (slope too short or non-linear, atrial arrhythmias, etc.) and factors not related to the mitral obstruction, primarily the features of left ventricular filling or left atrial emptying. Concomitant aortic regurgitation can also influence results.
- Direct measurement of the surface area of the stenosed orifice by planimetry in two- or even three-dimensional echocardiography is considered by many authors to be the reference quantification method for mitral stenoses [7]. It can also be used in *selected* cases for aortic stenoses, especially in multiplane transoesophageal echocardiography. Reliability depends on operator experience, quality of instrument, observation conditions and ability to determine the outline of the stenosed orifice (problems with irregular orifices and particularly with extensive calcification etc.).
- Other quantification indices (valve resistance, etc.) have not been shown to be superior to the calculation of surface area and are not used routinely.

Table 1 Aortic stenosis: obligatory parameters to record.

Parameters	Threshold	Comments	Value
Maximal aortic velocity	Severe AS >4 m/s	Asymptomatic patients	Prognostic value
Mean LV-Aorta gradient (rest)	Severe AS: >40 mmHg (ACC/AHA) Severe AS: >50 mmHg (ESC; SFC)	Several views (right parasternal ++)	Depends on output. Risk of underestimation if all views are not analyzed.
Aortic surface area continuity equation	Severe AS <1 cm^2 (ACC/AHA, ESC) Severe AS <0.5 cm^2/m^2 BSA (SFC) Severe AS <0.6 cm^2/m^2 BSA (ACC/AHA, ESC)	BSA indexation debatable: of use for extreme body morphotypes	Less dependent on output. Invalid in outflow tract obstruction

AS: aortic stenosis; BSA: body surface area; LV: left ventricle

Table 2 Aortic stenosis: optional parameters.

Parameters	Threshold	Comments	Value
Exercise	↗ Mean gradient >18 mmHg	Asymptomatic patients	Prognostic value to be confirmed (only 1 study)
Low dose dobutamine	Mean gradient >30 mmHg Surface area <1.2 cm^2	Patients with calcified valve, LVEF <40% and mean basal gradient LV- aorta <30 mmHg	Diagnostic and prognostic value
Surface area by planimetry	Severe AS <1 cm^2 (ACC/AHA, ESC) Severe AS <0.5 cm^2/m^2 BSA (SFC) Severe AS <0.6 cm^2/m^2 BSA (ACC/AHA, ESC)	Transthoracic or transoesophageal view	Limited to moderately calcified orifices when continuity equation invalid
Patency index	Severe AS <25%	Simple	Relatively non-specific
Valve resistance	Severe AS >300 dynes.s.cm^{-5}		Poor

LVEF: left ventricular ejection fraction; AS: aortic stenosis; BSA: body surface area; LV: left ventricle

Table 3 Mitral stenosis: obligatory parameters to record.

Parameters	Threshold	Comment	Value
Mean LA-LV gradient (mmHg) (rest)	Moderate MS: <5 mmHg Severe MS: >10 mmHg	Calculate mean measurements (AF ++)	Mediocre when performed alone (depends on output)
2D surface area	Severe MS <1.5 cm^2 (<1 cm^2/m^2 BSA)	Measure the smaller orifice (funnel tip)	Anatomical surface area Gold standard
PHT surface area	Severe MS <1.5 cm^2 (<1 cm^2/m^2 BSA)	Easy, quick	Poor when performed alone Multiple causes of error

2D: two-dimensional; AF: atrial fibrillation; LA: left atrium; PHT: pressure half time; MS: mitral stenosis; LV: left ventricle
[the mean mitral gradient is only an argument supporting the degree of stenosis but cannot be a unique quantification factor]

Table 4 Mitral stenosis: optional parameters to record.

Parameters	Threshold	Comment	Value
Mean LA-LV gradient on exercise	>15 mmHg	Difficult if rapid heart rate	Useful when basal evaluation difficult. Threshold value poorly documented prognostically
Systolic PAP on exercise	>60 mmHg		Threshold value poorly documented prognostically
Continuity surface area	Severe MS <1.5 cm^2 (<1 cm^2/m^2 BSA)	Pulmonary view rarely used Functional surface < anatomical surface area	Many causes of error
3D surface area	Severe MS <1.5 cm^2 (<1 cm^2/m^2 BSA)	Appears to be more reproducible than 2D with inexperienced observers	Same limitations as 2D (echogenicity) Specific equipment
PISA method (M-mode or 2D)	Severe MS <1.5 cm^2 (<1 cm^2/m^2 BSA)	Angle generally between 100° and 130°	Precise angle determination aleatory

PAP: pulmonary artery pressure; 2D: two-dimensional; 3D: three-dimensional; LA: left atrium; MS: mitral stenosis; LV: left ventricle

Valve regurgitation

Doppler echocardiography provides a positive diagnosis of valve regurgitation regardless of site, with a sensitivity and specificity of close to 100%. It describes the aetiology, mechanism [8] and severity of valve regurgitation [9].

Aetiology and mechanism

As regard to the aortic valve, echocardiography provides morphological information on the number of valves and valve texture, describes the size of the aortic annulus and detects lesions susceptible to change the surgical strategy or complicate surgery (dilatation of the ascending aorta, annulus calcifications extending towards the aorto-mitral trigone etc.).

At the mitral level, the functional classification proposed by Carpentier is widely used: (type 1: normal valve movements; type 2: valve prolapse; type 3: restricted valve movements). In patients with prolapse, a segmental analysis must also be performed. This analysis provides information on the 8 segments of the mitral valve: 2 commissures, 3 segments for the posterior valve (P1, P2, P3) and 3 for the anterior valve (A1, A2, A3). The use of such common classifications improves the collaboration between cardiologist and surgeon and clarifies the indications for treatment as regard to the type of surgery considered, i.e. mitral valvuloplasty or valve replacement.

At a tricuspid level, Doppler echocardiography can detect large functional regurgitations with repercussion on the right cavities which can be surgically resolved simultaneously with left heart lesions. It also describes the mechanisms of rare organic valve damage (table 5).

Quantification

As for stenoses, quantification of regurgitations [9] is based on a comparison of all the direct and indirect *criteria* [10-15] reviewed below (tables 6-11). Indirect criteria are represented by the consequences of the regurgitation on the cardiac cavities reflecting both the importance of the regurgitated volume and the chronicity of the valvular lesion, but also by haemodynamic repercussions (left ventricular filling pressures, pulmonary artery pressure) The mechanism of the regurgitation is sometimes helpful for quantifying the severity (severe valve damage, ruptures of the mitral valve papillary muscle or cordae, and aortic sigmoid eversion are generally associated with major regurgitations). Direct criteria are recorded by Doppler. The study of the jet extension by colour Doppler is a source of errors and has now become obsolete. Semi-quantitative and quantitative methods are widely used. Examining the proximal isovelocity surface area offers major advantages as it provides an estimation of the effective regurgitant orifice and the regurgitated volume (speed, simplicity, validity in the presence of associated valve disease or arrhythmias). However, methodological limitations (particularly interaction with adjacent structures) must not be underestimated. Overall, the number and complementarity of available indices make ultrasound the reference quantification method in routine practice.

In clinical practice, the investigation always begins with two-dimensional echocardiography which can suggest *from the outset* severe regurgitation in the presence of a major defect of valve closure or conversely, minor regurgitation when the valve anatomy is normal. This is followed by a careful assessment of the regurgitant jet by colour Doppler, increasing the section planes. This allows a rapid diagnosis of minimal regurgitation. Further quantification is generally not needed in these minimal regurgitations. In other cases a quantitative methods are required. Analysis of the proximal isovelocity surface area is the preferred method. Results are then compared with semi-quantitative parameters. The repercussion on the cardiac cavities and pulmonary artery pressure are examined. In difficult cases transoesophageal echocardiography is performed although transthoracic approach is usually sufficient.

Table 5 Doppler parameters used to quantify tricuspid regurgitation: threshold values.

Parameters	Mild tricuspid regurgitation	Severe tricuspid regurgitation
Semi-quantitative		
. vena contracta diameter		>7 mm
. systolic reversal of SHVF	absent	present
Quantitative		
. effective regurgitant orifice area incompetence	<20 mm^2	>40 mm^2

SHVF: suprahepatic venous flow

Table 6 Aortic regurgitation: obligatory parameters to record.

Parameters	Comment	Value
Pressure half Time	Apical view usually Right parasternal view (Pedoff) in some cases of prolapse	Reflects LVEDP: Poor specificity for regurgitant volume Index of haemodynamic tolerance
Isthmus speed	Minimum wall filter	Good in adults
VC Diameter	TTE or TOE	Good for central jets
ERO and RV (PISA)	Apical or parasternal view (prolapse ++)	Inferior compared with mitral regurgitation (technical difficulties for radius determination).
+ Systematic left ventricular diameters		

ERO: effective regurgitant orifice area; PISA: proximal isovelocity surface area; LVEDP: left ventricular end-diastolic pressure; SRO: surface area of regurgitant orifice; VC: vena contracta; RV: regurgitant volume

Table 7 Aortic regurgitation: optional parameters to record.

Parameters	Comment
Regurgitant fraction	Long, tedious Causes of error (mitral flow rate ++)
Aortic output	Limited in LV dysfunction LV, AF…

AF: atrial fibrillation; LV: left ventricular

Table 8 Doppler parameters used to quantify aortic regurgitation: threshold values.

Parameters	Moderate aortic regurgitation	Severe aortic regurgitation
Semi-quantitative		
. vena contracta diameter	<3 mm	>6 mm
. aortic isthmus end-diastolic velocity	absent or <10 cm/s	>20 cm/s
. aortic output	<6 L/min	>10 L/min
. pressure half time	>500 ms	<300 ms
Quantitative		
. effective regurgitant orifice area	<10 mm^2	>30 mm^2
. regurgitated volume/beat	<30 ml	>60 ml
. regurgitation fraction	<30%	>50%

Table 9 Mitral regurgitation: obligatory parameters to record.

Parameters	Comment	Value
ERO and RV (PISA)	Limits: Multiple jets (Barlow) Confined jets (commissural prolapse)	Current gold standard Probably overestimates regurgitations in MV prolapse Independent of load conditions
Vena contracta diameter	Preferably left parasternal view Use of zoom	limitations (echogenicity, multiple jets, eccentric jets)
+ indirect criteria systematically (cardiac cavities, pulmonary pressures, etc.)		

ERO: effective regurgitant orifice area; PISA: proximal isovelocity surface area; RV: regurgitated volume.

Table 10 Mitral regurgitation: optional parameters to record.

Parameters	Comment	Value
Regurgitant fraction	Volume ejected at aortic annulus Total volume in 2D echo (Simpson)	Longer and more complex than PISA Useful when PISA invalid or equivocal
Mitral VTI/Aortic VTI	Simpler than calculating regurgitation fraction	Limitations (associated valve disease, atrial fibrillation…)

VTI: velocity time integral; PISA: proximal isovelocity surface area

Table 11 Doppler parameters used to quantify organic, non-ischaemic mitral regurgitation: threshold values.

Parameters	Mild mitral regurgitation	Severe mitral regurgitation
Semi-quantitative		
. vena contracta diameter	<3 mm	>7 mm
. Mitral VTI/Aortic VTI	<1	>1.4
. Pan- or mid-systolic reversal of PVF	absent	present
Quantitative		
. effective regurgitant orifice area	<20 mm^2	>40 mm^2
. regurgitant volume/beat	<30 ml	>60 ml
. regurgitation fraction	<30%	>50%

VTI: velocity time integral; PVF: pulmonary venous flow.

Follow-up of organic left heart valve disease: transthoracic Doppler echocardiography

The surgical indications, which largely depend on the echocardiographical findings, are described in the recently published recommendations from the valve group of the Société Française de Cardiologie (SFC) [16], the European Society of Cardiology (ESC) [17], and the American Heart Association/American College of Cardiology (AHA/ACC) [17 bis].

Aortic valve diseases

Moderate valve disease with no repercussions on the cardiac cavities or left ventricular function, which do not produce symptoms and are clinically stable do not require close regular monitoring. In other cases the methods and frequency of Doppler echocardiography monitoring depend on the valve disease in question.

Aortic stenosis

The natural history of degenerative aortic stenosis is relatively well known: the aortic surface area has been shown to fall by an average of 0.1 to 0.2 cm^2 per year, although considerable inter-individual differences exist. Asymptomatic aortic stenosis has a good prognosis with a very low risk of sudden death [17, 18]. Different predictors of poor outcome in asymptomatic patients have been identified: a peak aortic jet velocity >4m/s, rapid increase in velocity >0.3 m/s per year, the presence of extensive valve calcifications and finally an abnormal response to exercise testing [16-19]. There are few data suggesting the prognostic value of exercise echocardiography and its precise role remains to be established. The prognostic value of severe left ventricular hypertrophy (wall thickness >15 mm) or a very high mean gradient (>75 mmHg) is still subject of debate. Echocardiography has become the standard method for assessing the severity of aortic stenosis. The two main criteria in favor of severe aortic stenosis are a mean gradient >50 mmHg, and an aortic valve area <1 cm^2 (or indexed aortic valve area <0.6 or 0.5 cm^2/m^2 in patients with either unusually small or large body surface areas). It should be emphasized that all of these measurements have potential inaccuracies and must be considered in combination with flow rate, ventricular function and functional status for clinical decision making [16]. Annual Doppler echocardiography helps to distinguish rapid progressors (reduction in aortic surface area of more than 0.1 cm^2/year) from patients in whom the stenosis progresses slowly (reduction in aortic surface area of less than 0.1 cm^2/year). It is also used to monitor patients with well tolerated severe aortic stenosis.

Indications for transthoracic Doppler echocardiography in aortic stenosis

Classe I
– Initial work-up for clinically diagnosed aortic stenosis.
– Investigation of functional signs that might be due to aortic stenosis if the physical examination was not informative.
– Changes in functional signs or physical examination findings in a patient with known aortic stenosis.
– Annual or twice-yearly re-assessment of asymptomatic, severe aortic stenosis.
– Annual re-assessment of moderate aortic stenosis with impaired left ventricular systolic function caused by some other aetiology.
– Twelve-month re-assessment of asymptomatic aortic stenosis initially judged as moderate. Later, annual check-ups in the event of rapid deterioration; every two or three years if deterioration is slower.
– Re-assessment of moderate-to-severe aortic stenosis before intermediate-to-high risk extracardiac surgery (see page 284) or before cardiovascular surgery if the last Doppler echocardiography was performed more than one year previously.

Classe II
– Re-assessment of moderate-to-severe aortic stenosis before intermediate-to-high risk extracardiac surgery (see page 284) or before cardiovascular surgery if the last Doppler echocardiography was performed less than one year previously.

Classe III
– Annual re-assessment of clinically stable, mild or slowly-deteriorating, asymptomatic aortic stenosis, without any significant impact on the heart cavities according to the initial examination.

Aortic regurgitation

The indication for surgery in patients with severe aortic regurgitation is undisputed clear in the presence of symptoms due to the regurgitated volume and its haemodynamic repercussions [16, 17]. The risks of surgery and of prosthetic valve complications in asymptomatic

Consensus indications for transthoracic Doppler echocardiography in aortic regurgitation

Class I
— Initial assessment of clinically diagnosed aortic regurgitation.
— Changes in symptoms or clinical examination in patients previously diagnosed with known aortic regurgitation.
— Annual or bi-annual (if rapid progression) evaluation of ascending aorta if >45 mm, in patients with aortic root aneurysm and bicuspid aortic valve
— Annual or bi-annual (according to severity and evolutivity of left ventricular involvment) evaluation of severe asymptomatic aortic regurgitation medically followed.
— Evaluation of a known moderate to severe AR before intermediate or high-risk extracardiac surgery (see page 284) if the last Doppler echocardiography was performed more than one year previously.
— Reference examination within 3 months after aortic valve conservative surgery or Ross procedure.

Class II
— Annual assessment of moderately severe aortic regurgitation.
— Evaluation of a known moderate to severe AR before intermediate or high-risk extracardiac surgery (see page 284) if the last Doppler echocardiography was performed less than one year previously.

Class III
— Annual re-assessment of minor aortic regurgitation with no left ventricular repercussions and stable clinical parameters.

Consensus indications for transthoracic Doppler echocardiography in mitral stenosis

Class I
— Initial assessment of clinically diagnosed or suspected mitral stenosis.
— Changes in symptoms or clinical examination in patients with known mitral stenosis.
— Assessment of complications during evolution of mitral stenosis.
— *Systematic* reassessment of mitral stenosis, with a frequency depending on the results of the initial evaluation: yearly in severe mitral stenosis to several year intervals in moderate mitral stenosis
— Initial reassessment (first month) of mitral stenosis treated by percutaneous mitral commissurotomy (the frequency of subsequent follow-up is determined by the quality of the initial result).
— Re-assessment of moderately severe to severe mitral stenosis before intermediate or high risk extra-cardiac surgery (see page 284) or before cardiovascular surgery if the last Doppler echocardiography was performed more than one year previously.

Class II
— Re-assessment of moderately severe to severe mitral stenosis before intermediate or high risk extra-cardiac surgery (see page 284) or before cardiovascular surgery if the last Doppler echocardiography was performed less than one year previously.

Class III
— Annual re-assessment of clinically stable, non-tight mitral stenosis.

patients must be balanced against those due to the natural progression of the valve disease. It has been shown that symptoms and/or left ventricular dysfunction develop at an annual rate of approximately 4% in asymptomatic patients with aortic regurgitation [20]. Left ventricular dilatation (left ventricular end-diastolic diameter >70 mm and in particular, left ventricular end-systolic diameter >50 mm or >25 mm/m^2) and/or reduction in LV ejection fraction (<50%) represent arguments for surgery in the presence of severe aortic regurgitation [21]. Changes in left ventricular function parameters have also a prognostic role [20, 21] and some authors have proposed biannual follow-up in patients with left ventricular end-systolic diameters between 50 and 55 mm [20].

The natural history of aortic root aneurysm has been mainly evaluated mostly in patients with Marfan's syndrome or bicuspid aortic valve. The main predictive factors of aortic complications are the aortic diameter, the rate of progression of aortic dilatation and a family history of sudden death or aortic complication.

Mitral valve diseases

Mitral valve diseases must be investigated by Doppler echocardiography in order to assess anatomical changes, severity, left ventricular repercussions (in mitral regurgitation) and pulmonary pressures [9, 16].

Mitral stenosis

Mitral stenosis is considered to be tight if the surface area of the mitral valve is less than 1.5 cm^2 (or 1 cm^2/m^2 body surface area).

Mitral stenosis usually progresses slowly, with a long asymptomatic period. Progression of mitral stenosis is assessed by measuring the mitral valve surface area and the variation of the other parameters (gradient/pulmonary artery pressure/left atrial diameter).

Percutaneous mitral dilatation

Baseline Doppler echocardiography must be performed after percutaneous mitral dilatation, preferably under stable haemodynamic conditions, i.e. more than 48 hours after the procedure. The frequency of subsequent investigations is governed by the quality of the initial result.

Organic mitral regurgitation

The indication for surgery in patients with severe mitral regurgitation is clear in the presence of symptoms due to the regurgitated volume and its haemodynamic repercussions. The approach varies in asymptomatic patients. The development of left ventricular repercussions (left ventricular end-systolic diameter of 45 mm or more, left ventricular ejection fraction <60%) represents an indication for surgery. In asymptomatic low-risk patients with severe regurgitation (regurgitant orifice >40mm^2 and/or regurgitated volume >60 ml/

Consensus indications for transthoracic Doppler echocardiography in organic mitral regurgitation

Class I
— Initial assessment of clinically diagnosed or suspected mitral regurgitation.
— Change in symptoms or clinical examination in patients with previously diagnosed mitral regurgitation.
— Bi-annual or annual follow-up (depending on the severity and progression of the repercussions on left ventricular function, left atrium and pulmonary artery pressure) for severe asymptomatic mitral regurgitation
— Baseline assessment during the 3 months following mitral valvuloplasty.
— Re-assessment of severe or moderately severe mitral regurgitation before intermediate or high risk extra-cardiac surgery (see page 284) or before cardiovascular surgery if the last Doppler echocardiography was performed more than one year previously.

Class II
— Annual assessment of moderately severe mitral regurgitation without repercussions on cavities or left ventricular function on the initial investigation with clinically stable parameters.
— Re-assessment of severe or moderately severe mitral regurgitation before intermediate or high risk extra-cardiac surgery (see page 284) or before cardiovascular surgery if the last Doppler echocardiography was less than one year previously.
— Re-assessment of minor mitral regurgitation with valvular abnormalities and no repercussions on the cavities or left ventricular function on the initial examination, with stable clinical parameters, every 4 to 5 years.

Class III
— Annual re-assessment of moderate mitral regurgitation with no repercussions on the cavities or left ventricular function on the initial assessment, with stable clinical parameters.

beat) and favourable mitral anatomy, the current trend is to operate early and perform valvuloplasty in order to preserve left ventricular function and avoid left atrial dilatation [22]. If uncertainty about the feasibility of valvuloplasty exists, or in the presence of co-morbidities regular follow-up is appropriate. Surgery becomes the rational choice at the occurrence of symptoms, left ventricular dysfunction, pulmonary arterial hypertension or atrial fibrillation [22 bis]. In patients with severe mitral regurgitation managed medically the main risk is related to the insidious development of LV dysfunction. This justifies annual or bi-annual follow-up depending on the initial findings.

Follow-up left heart valve diseases: transoesophageal echocardiography

- Transoesophageal echocardiography is particularly useful for the diagnosis of complications of known valve disease.

Consensus indications for transoesophageal echocardiography in mitral stenosis

Class I
— Severe mitral stenosis when percutaneous dilatation is being considered (minimising the time between TOE and dilatation).
— Mitral stenosis complicated by an embolic event.
— Atrial dysrhythmias when cardioversion is being considered in a non-anticoagulated or poorly anticoagulated patient.

Class II
— Before intermediate or high risk extra-cardiac surgery (see page 284) or major cardiovascular surgery.
— Atrial dysrhythmias when cardioversion is being considered in a well anticoagulated patient.

Class III
— Annual re-assessment of a mild mitral stenosis.

Occasionally it helps determining the operative strategy in patients in whom surgery is indicated. Transoesophageal echocardiography is recommended in patients with a history of embolic events or in those in whom left atrial thrombosis has to be excluded before cardioversion or percutaneous mitral dilatation. The close relationship between the oesophagus and the left atrium [23] and the poor sensitivity of transthoracic echocardiography to detect left atrial thromboses make transoesophageal echocardiography a valuable procedure for assessing cardiac thrombosis. Transoesophageal echocardiography is also frequently used in patients with suspected endocarditis, aortic wall complications or as an adjunct for examining the ascending aorta, particularly in bicuspid aortic valve before surgery. Patients with severe mitral regurgitation in whom the feasibility of conservative surgery remains unclear after transthoracic echocardiography may also benefit from the transesophageal approach.

In aortic stenosis, the technique may be used for the planimetry of the aortic orifice [24] in a minority of patients in whom the transthoracic Doppler echocardiography quantification provided inconsistent results (poor observation conditions, flow acceleration in the outflow tract etc.) and occasionally in selected patients with atheroma of the aortic arch at risk of complications at catheterisation or cardiac surgery. Transoesophageal echocardiography must be avoided in highly symptomatic tight aortic stenosis. The preferred indications for transoesophageal echocardiography in aortic regurgitation are suspected endocarditis and suspected aortic wall complication, particularly in dystrophic aortic regurgitation. The method is also recommended for the assessment of the regurgitation mechanism when transthoracic observation conditions do not permit definitive conclusions.

Follow-up of organic left heart valve diseases: transoesophageal echocardiography

Several publications analyze the haemodynamic behaviour of different left heart valve diseases during pharmacological or physical stress [25-27]. In general terms, Doppler stress echo-

Consensus indications for transoesophageal echocardiography in organic mitral regurgitation

Class I
— Mechanism and quantification of mitral regurgitation inadequately assessed by transthoracic Doppler echocardiography.
— Mechanism and quantification of mitral regurgitation when transthoracic Doppler echocardiography and clinical assessment produce inconsistent results.
— Development of a clinical event (suspected endocarditis, embolic event, etc.).
— Atrial dysrhythmias when cardioversion is being considered in a non-anticoagulated or poorly anticoagulated patient.
— Per-operative assessment of mitral regurgitation in mitral valvuloplasty.

Class II
— Assessment of incompetent mitral valve before surgical correction.
— Assessment of the mechanism of moderate mitral regurgitation.
— Atrial dysrhythmias when cardioversion is being considered in a well anticoagulated patient.

Class III
— Moderate mitral regurgitation with no symptoms or repercussion on the cavities or left ventricular function on transthoracic Doppler echocardiography.

Consensus indications for transoesophageal echocardiography in aortic stenosis

Class I
— Suspected or overt infective endocarditis.

Class II
— Technical inability to correctly assess the severity extent of aortic stenosis by transthoracic Doppler echocardiography.
— Investigation for associated lesions (thoracic aorta, other valve lesions, etc.) before aortic valve replacement.

Class III
— Initial assessment of aortic stenosis that was completely assessed by transthoracic Doppler echocardiography.

cardiography can provide additional information to the baseline assessment of valve diseases and refines indications for treatment, particularly in asymptomatic patients or if symptoms are inconsistent with the baseline assessment. This investigation is particularly useful when the "dynamic" nature of the valve disease needs to be demonstrated (for example, functional mitral regurgitation in heart failure). One specific situation is aortic stenosis with severe left ventricular systolic dysfunction associated with low output and a small gradient (<30 mmHg). The haemodynamic response to dobutamine has diagnostic value distinguishing significant stenoses from moderate stenoses associated with independent left ventricular dysfunction but also prognostic value(assessment of the LV contractile reserve) [26, 27].

Consensus indications for transoesophageal echocardiography in aortic regurgitation

Class I
— Suspected aortic dissection in the presence of a dilated and / or dystrophic aorta.
— Suspected or overt infective endocarditis.
— Mechanism and quantification of aortic regurgitation inadequately assessed by transthoracic Doppler echocardiography.
— Mechanism and quantification of aortic regurgitation when transthoracic Doppler echocardiography and clinical assessment produce inconsistent results.
— Pre-operative assessment of annuloaortic ectasia when transthoracic Doppler echocardiography is inadequate.

Class II
— Large volume aortic regurgitation with poor observation conditions by transthoracic Doppler echocardiography.
— Follow-up of progression of annuloaortic ectasia.

Class III
— Assessment of moderate aortic regurgitation with no symptoms or repercussion on the cavities or left ventricular function on transthoracic Doppler echocardiography.

Consensus indications for stress echocardiography in organic left heart valve diseases

Class I
— Low dose dobutamine Doppler echocardiography in aortic stenosis with low output (and small transvalvular gradient <30 mm Hg with left ventricular dysfunction)
— Exercise Doppler echocardiography to assess the tolerance of mitral stenosis and its repercussions on pulmonary pressure when symptoms are difficult to assess or do not appear to be explained by the valve disease.

Class II
— Exercise Doppler echocardiography to assess the tolerance of organic mitral regurgitation, its severity and its repercussions on pulmonary pressure when symptoms are difficult to assess or do not appear to be explained by the valve disease.

Class III
— Exercise or Dobutamine Doppler echocardiography for routine assessment of organic mitral or aortic valve disease.

Infective endocarditis

In infective endocarditis, an early diagnosis is essential. Doppler echocardiography is currently the investigation with the best performance in visualising vegetations and/or assess valve dysfunction due to the endocarditis.

Consensus indications for transthoracic Doppler echocardiography in endocarditis

Class I
— Assessment of suspected or overt endocarditis.
— Surveillance of suspected or overt endocarditis even when the initial transthoracic Doppler echocardiography is normal (the frequency of subsequent investigations should be established on an individual basis).
— Assessment of complications occuring during the course of the disease (the frequency of subsequent investigations should be established on an individual basis)
— Follow-up of lesions and of haemodynamic repercussions after clinical and laboratory recovery (the frequency of subsequent investigations should be established on an individual basis).

Consensus indications for transoesophageal echocardiography in endocarditis

Class I
— Systematic complement to transthoracic Doppler echocardiography in overt or suspected endocarditis on a native or prosthetic valve.
— Clinical and /or transthoracic echocardiographical suspicion of complications during the course of the disease (abscess, loss of material, embolism etc.), the frequency of subsequent investigations should be established on an individual basis
— Follow-up in cases of strong clinical suspicion even when the initial transthoracic Doppler echocardiography is normal (the frequency of subsequent investigations should be established on an individual basis)
— Control when transthoracic Doppler echocardiography is non-contributory.

Class II
— Systematic control in endocarditis due to particularly virulent organisms (Staphylococci, Enterococci, yeasts, fungi, etc.).
— Control after clinical and laboratory recovery (assessment of lesions and haemodynamic consequences).

Endocarditis on native valves

The diagnostic criteria for infective endocarditis have recently been modified [28] and Doppler echocardiography findings have been included among in the major criteria (vegetations, abscess, valve perforation, detachment of prosthesis, recent regurgitation). The use of the new criteria improves the accuracy of the diagnosis of endocarditis [29]. The sensitivity of transthoracic Doppler echocardiography to detect vegetations is 50 to 60%. Transoesophageal echocardiography has increased sensitivity (90 to 95%). When used together, the specificity of these techniques is close to 100% [30].

Endocarditis on prosthetic valves

The incidence of endocarditis on prosthetic valves is 0.3 to 1.2%/patient year. Its' complications carry a high mortality [30 to 80% in early endocarditis; 20 to 40% in late forms [31-32]], and are similar for mechanical prostheses and bioprostheses.

Early diagnosis is essential. Doppler echocardiography is currently the best investigation to visualise and measure the size of vegetations, [33] and/or assess prosthetic dysfunction due to the endocarditis.

The diagnosis of endocarditis solely by transthoracic Doppler echocardiography is difficult (acoustic shadow of the prosthetic material). This limitation is resolved in part by transoesophageal echocardiography, with a good sensitivity and diagnostic accuracy as reported in the literature [34, 35]. The use of bi-and multi-plane probes undoubtedly improves the sensitivity of the investigation [36]. However, normal transoesophageal echocardiography does not formally exclude the diagnosis of endocarditis on prosthesis and the investigation should be repeated if suspicion persists. The frequency of subsequent transoesophageal examinations during follow-up is to be established considering all clinical and laboratory parameters. Finally, transoesophageal echocardiography is an essential investigation in planning valve surgery and helps the choice of the best operative technique.

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VALVE PROSTHESES, VALVES REPAIR AND HOMOGRAFTS

When damage is incompatible with preserving the valve, the treatment of the valve disease is replacement with a prosthetic valve (approximately 9,000 cases per year in France). Two main types of prostheses exist: mechanical prostheses (which require long term anti-coagulation although their robustness and longevity have been widely demonstrated [1, 2]) and biological prostheses (bioprostheses) which do not require any specific treatment but have a shorter lifespan (average 10 to 15 years) because of risk of degeneration, which occurs faster in people under 40 years [2, 3]. Aortic and mitral valve homograft implantation is not widely used. In addition, whilst mitral valve repair techniques are making rapid advances, aortic valve repair techniques are not widely used in France.

Normal valve prostheses, valve repair and homografts

These display no abnormal clinical or radiological signs of dysfunction.

Initial assessment (2 or 3 months post-operatively)

Prosthetic valves

Each type of prosthesis has specific characteristics, representing a true echocardiographic and Doppler signature, which depend on their haemodynamic profile, position and size. This identity card is essential for patient follow-up.

Baseline investigation

Each recipient of a prosthetic cardiac valve must have a baseline assessment in order to record the haemodynamic constants for the type of prosthesis implanted [4-7]: this investigation should be performed when the patient has returned to a stable haemodynamic situation (correction of anaemia, restoration of normal heart rhythm etc.) i.e. generally within two to three months after the procedure. The first month assessment must not be used as the baseline record.

The following findings are recorded in the baseline report: date of the procedure, type and size of prosthesis implanted, heart rhythm, any associated procedures (bypass, tricuspid annuloplasty, other valve replacement, preservation of the mitral subvalvular apparatus). This report can be attached to the document describing the features of the prosthesis and given to the patient when discharged from the surgical department.

As for a native valve disease, the echocardiographer must assess left ventricular function in M-mode and two-dimensional mode, the size of the different cardiac cavities and the pulmonary pressures when accessible to Doppler mode.

As for any valve disease, the echocardiographic assessment of a prosthetic valve is based on a combination of anatomical findings provided by M-mode and, particularly, two-dimensional imaging and Doppler haemodynamic findings. M-mode and two-dimensional modes are of lower value for the anatomical analysis of mechanical prostheses than for bioprostheses and native valves because of the reverberations caused by the metallic structures: the motion of the discs of bileaflet prostheses can, nevertheless, be clearly analysed in most patients through the quality of current imaging. The morphology of the leaflets of bioprostheses can also be clearly assessed by 2D imaging [8]. The anatomical assessment is still therefore a fundamental initial stage in the echocardiographic investigation of prosthetic valves.

The assessment of prostheses also uses Doppler examination. Regardless of type, any prosthesis causes a degree of obstruction, which varies depending on its haemodynamic profile, position and size: monitoring of the prosthesis and patient follow-up are based on recording the transprosthetic velocities by continuous Doppler.

Colour Doppler mode occasionally allows small para- or intraprosthetic regurgitations from aortic prostheses to be identified [9].

The essential parameters to be recorded to assess *mitral prostheses* (table 1), which are analysed particularly from the apical 4 chamber view are:

- the mean transprosthetic gradient which is usually of 5±3 mmHg, and clearly abnormal when above 10 mmHg. It is relatively independent of size of prosthesis, unlike aortic prostheses but depends mostly on the type of valve (the lowest gradients are seen with bioprostheses and bileaflet prostheses and the highest with ball prostheses), heart rate, rhythm and cardiac output.
- the "pressure half time" (PHT) as in the case of mitral stenosis. This is a simple parameter to monitor prostheses which reflects transprosthetic diastolic filling. Only the uncorrected value of the PHT not adjusted for prosthetic surface area is used. It should be remembered that calculating the prosthetic surface area from the PHT (Hatle equation: mitral area = 220/PHT) has only been validated for mitral stenosis in native valves but not for normal prosthetic valves. Only the PHT value should appear on the Doppler echocardiography reports. PHT values are normally between 70 and 100 msec: in atrial fibrillation it varies greatly depending on the length of diastole. A PHT >150 msec is formally pathological.
- the functional surface area of the prosthesis by the continuity equation, calculated in the same way as the mitral surface area in mitral stenosis can be used if the patient is in sinus rhythm and if no significant concomitant aortic valve regurgitation is present. It varies from 1.6 to 2.8 cm^2.
- the presence of intra or para-prosthetic regurgitations: continuous-wave Doppler must be used, because colour Doppler is severely affected by reverberations from the prosthesis. The procedure follows in reverse to usual order, identifying regurgitations first by continuous Doppler and then localising them with colour Doppler. Intraprosthetic regurgitations are common: 30 to 40% by TTE and in almost 100% of cases by TOE [10-15]. They are located at the junction between the mobile part and the metallic frame of the prosthesis: their topography is therefore characteristic of type of prosthesis. Para-prosthetic regurgitations are relatively common during the immediate post-operative period, are usually minimal and frequently disappear spontaneously over a few weeks.

The main parameters to record for the analysis of *aortic prostheses* (table 2), which are best seen in the apical 5 chamber and right parasternal views by TTE or suprasternally in young people are:

- the maximum and mean gradients: the mean gradient is normally between 7 and 25 mmHg and is usually pathological above 30 mmHg. It is useful to identify opening and closing clicks of the prosthesis for a better recording of the transprosthetic flow. The mean gradient depends on the *type of prosthesis* (lower gradients are found with bioprostheses and bileaflet mechanical prostheses and higher values for ball prostheses), *size (number)* +++ and *cardiac output*.
- the functional or "effective" surface area of the prosthesis by the continuity equation may be calculated in the same way as for native valves if the patient is in sinus rhythm. For outflow tract diameter, the diameter (or number) of the prosthesis can be used. The prosthesis surface area ranges from 0.9 to 2.2 cm^2, depending on the type and calibre of the prosthesis. As for aortic stenosis on native valves it is also recommended that this surface be expressed as an index to body surface area.
- the permeability index (PI), which is similar to the one used for aortic stenosis, is a simple index, independent of cardiac output and does not require measurement of the left ventricular outflow tract diameter. The ratio of velocity time integrals or maximum velocities of the outflow tract and transprosthetic flow can be used. In atrial fibrillation it is essential to take into account the subaortic flow and transprosthetic flow on the same cycle by continuous Doppler. Normal PI values vary depending on the type and diameter of prosthesis: they increase with decreasing physiological obstruction from the prosthesis. For mechanical valves the best index is seen with bileaflet prostheses: 0.41±0.12; for tilting disc prostheses the PI is 0.33±0.06, and for ball prostheses it is 0.29±0.069. The value is even higher for bioprostheses, especially homografts: 0.56±0.10.

- the presence of intra- or para-prosthetic regurgitations which must always be investigated for by continuous, pulsed and colour Doppler. [11-15].

High transprosthetic gradients and low prosthetic surface areas can be seen in the absence of any dysfunction if the prosthesis/patient size match is disproportionate ("mismatch"): the indexed valve area should be >0.9–1 cm^2/m^2. Mismatch is present if it is <0.85 cm^2/m^2 [13] and is severe if the indexed surface area is <0.6 cm^2/m^2. Gradients may also be elevated in high cardiac output. Conversely, with low cardiac output, a low gradient can not exclude a prosthetic dysfunction.

However the finding of a high gradient and low prosthetic surface area with Doppler is not synonymous with stenosis of the prosthesis. "Falsely elevated" gradients can be seen on Doppler. This gradient over-estimation phenomenon on Doppler compared to the invasive haemodynamic gradient is especially frequent with bileaflet mechanical aortic prostheses but is also possible with all types of prosthesis, including bioprostheses [14]. The problem is due to the difference in gradient measurement site between Doppler and haemodynamics and is explained by the pressure recovery phenomenon. With bileaflet prostheses, Doppler records localised high velocity flows in the central orifice of the prosthesis whereas velocities are lower in the lateral orifices [16, 17]. In the absence of a baseline record, interpretation of a high gradient on a mechanical aortic prosthesis is impossible and requires more investigations such as by radio-cinema of the prosthesis and, depending on clinical context, transoesophageal echocardiography.

Apart from these specific problems which are now clearly understood and are associated with the architecture and small size of some prostheses there are considerable in vivo and in vitro data to confirm the reliability of gradient and surface area measurements as follow-up parameters for prosthetic valves [18].

Table 1 Mitral prostheses: essential parameters to record.

Parameter	Normal value	Technical comments	Diagnostic value
Mean gradient	5±3 mmHg Pathological if >10 mmHg	Variable with rate, rhythm, output	+++
Pressure Half Time (PHT)	70-100 msec Pathological if >150 msec	Do not extrapolate for surface area	+++
Regurgitations	— Intraprosthetic — Paraprosthetic	TOE > TTE	++

Table 2 Aortic prostheses: essential parameters to record.

Parameter	Normal value	Technical comments	Diagnostic value
Mean gradient	<30 mmHg	Variable with type and size ++ of the prosthesis	+++
Permeability index	0.23-0.66	Use the same cycle for 2 flows in AF by continuous Doppler	+++
Prosthesis surface area	0.9-2.2 cm^2	If sinus rhythm	++
Indexed prosthesis surface area			
regurgitations	— Intraprosthetic — Paraprosthetic	TTE > TOE	++

The use of systematic post-operative transoesophageal echocardiography

Some publications have described early abnormalities after mitral valve replacement [19]; they included non-obstructive thrombi, fibrin strands, and more rarely obstructive thrombi [20-23]. Physiological regurgitations are not accessible to transthoracic echocardiography which also fails to identify small para-prosthetic regurgitations that are important to record for the patient follow-up in the event of subsequent complications. Some authors have therefore proposed systematic transoesophageal echocardiography during the months following mitral valve replacement [19-23].

Conversely, there is no need to a systematic transoesophageal echocardiography in the post-operative period after isolated aortic valve replacement if not combined with replacement of the ascending aorta.

Mitral and aortic repair, mitral and aortic homografts

Because of the complexity of these procedures a control transoesophageal echocardiography is required in the operating theatre to confirm the quality of functional results and to re-operate in the same procedure if a problem exists in order to ensure an optimal result.

Patients who undergo a mitral or much less frequently aortic repair or homograft insertion (aortic or more rarely mitral) should undergo post-operative transthoracic Dop-

Indications for transthoracic Doppler ultrasonography in the initial assessment (first 3 months) of normal prosthetic valves

Class I
— Post-operative examination before discharge from hospital
— Reference examination within 3 months of surgery in patients considered as normal

Indications for transoesophageal echocardiography in the initial assessment (first 3 months) of normal prosthetic valves

Class I
— Assessment of atrial arrhythmias when cardioversion is being considered in a non-anticoagulated or poorly anticoagulated patient.
— Suspected infectious endocarditis

Class II
— Per-operative transoesophageal echocardiography in mitral valve replacement with conservation of the subvalvular apparatus.
— Assessment after mitral valve replacement with a mechanical or biological prosthesis: investigation for para-prosthetic regurgitations, obstructive and/or non obstructive thrombi and/or strands, identification of physiological regurgitations.
— Assessment after aortic valve replacement combined with ascending aortic surgery or surgery for infectious endocarditis.
— Atrial arrhythmias when cardioversion is being considered in a well anticoagulated patient.

Class III
Assessment after aortic valve replacement when transthoracic Doppler echocardiography is normal.

pler echocardiography during the three months following surgery to precise their valve statement, the existence of any residual regurgitation and to investigate for a left intraventricular obstruction due to post-operative anterior mitral systolic movement following mitral valvuloplasty.

Follow-up beyond the 3rd month

Doppler echocardiography is the only truly reliable follow-up investigation for prosthetic valves both in routine use and in the event of dysfunction. In addition, in the absence of dysfunction, the baseline Doppler haemodynamic assessment of prosthetic valves is highly reproducible. As a result, any changes in follow-up parameters found under similar haemodynamic conditions, even if minimal, must be taken into account in the subsequent follow-up [4].

Mechanical prostheses

Once a baseline record has been obtained during the first 3 post-operative months, Doppler echocardiography follow-up is indicated every 2 years in the absence of any new clinical findings. This record should be compared with the post-operative findings and interpreted according to changes in left ventricular function, heart rhythm and/or treatment.

Indications for Doppler echocardiography in the initial assessment (first 3 months) of mitral and aortic repairs and normal aortic and mitral homografts

Class I
— Per-operative transoesophageal echocardiography to guide the valve repair procedure.
— Per-operative transoesophageal echocardiography during mitral or aortic homograft insertion
— Baseline transthoracic Doppler echocardiography in the 3 months following mitral repair or homograft.

Class II
— Transoesophageal echocardiography if the mechanism and/or quantification of possible residual mitral regurgitation are not well identified by transthoracic Doppler echocardiography.

Indications for transthoracic Doppler echocardiography in the follow-up (beyond 3 months) of normal mechanical prostheses

Class I
— Control every 2 years, in the absence of any new clinical or biological event.
— Control every 3 months during pregnancy because of physiological changes and changes in anti-coagulation modalities.

Class II
— Annual control if patient is unable to correctly follow his/her anti-coagulation treatment.

Indications for transoesophageal echocardiography in the follow-up (beyond 3 months) of normal mechanical prostheses

Class III
— Transoesophageal echocardiography if transthoracic Doppler echocardiography is normal in a clinically stable patient

A three-monthly control of mechanical prostheses is mandatory during pregnancy: this is justified by the increased follow-up required because of changes in haemodynamics and anti-coagulation modalities during pregnancy.

Mechanical prostheses are rarely implanted in the tricuspid position because of their high thrombogenic potential: annual Doppler echocardiography is appropriate in these cases.

Biological prostheses

Follow-up every 2 years is appropriate in the absence of any new clinical or laboratory event: because of the potential degeneration specific to the bioprostheses, and adverse haemodynamic changes after the 5th year of implantation, annual follow-up is required beyond this time [8]. The frequency of controls should be determined

Indications for transthoracic Doppler echocardiography in the follow-up (beyond 3 months) of biological prostheses

Class I
– Control every 2 years, in the absence of any new clinical or biological event.
– Annual control from year 5 in the absence of any new clinical or biological event.
– Annual control in subjects under 40 years.

Class II
– Annual control in elderly patients and for biological prostheses less than 5 years.

Indications for transthoracic Doppler echocardiography in the follow-up (beyond 3 months) of valve repairs and homografts

Class I
– Initial control at the end of the first year in the absence of any new clinical biological event.
– Control every 2 years in the absence of any new, clinical or biological event.

Class II
– Systematic annual control in the absence of any new, clinical or biological event.

Indications for transoesophageal echocardiography in the follow-up (beyond 3 months) of biological prostheses

Class III
– Transoesophageal echocardiography if transthoracic Doppler echocardiography is normal in a clinically stable patient

Indications for transoesophageal echocardiography in the follow-up (beyond 3 months) of valve repairs and homografts

Class III
– Transoesophageal echocardiography if transthoracic Doppler echocardiography is normal in a clinically stable patient

on an individual basis depending on the extent of dysfunction of the bioprosthesis until the time of reoperation.

Annual transthoracic Doppler echocardiography is mandatory from the year of the procedure onwards if the patient is under 40 years.

Valve repairs and homografts

A Doppler echocardiography control every 2 years is indicated for mitral and aortic repairs with no significant residual regurgitations. If a moderate or severe residual mitral or aortic leak is present, the situation is the same as for the follow-up of moderate or severe mitral or aortic regurgitation.

Aortic homografts are mostly indicated in young people and in the treatment of endocarditis. The long-term outcome of the latest generation of homografts is not yet known [24]. The follow-up for aortic homografts can reasonably be based on that of the aortic bioprostheses.

One elective indication for the mitral homografts is inability to perform mitral valvuloplasty [25]. There are still too few cases of these for their follow-up to be defined.

Complications of valve prosthesis, valve repair and homografts

These are suggested by signs of dysfunction (abnormal murmur, arterial embolism, endocarditis, haemolysis).

Thrombo-embolic complications

Obstructive prostheses thrombosis

The incidence of obstructive thromboses in prostheses varies from 0.3 to 1.3 per 100 patient-years in the literature. The main factors responsible for thromboses are inadequate anti-coagulation and the mitral localization. Bioprostheses are not free from this type of complication [26], although thrombosis occurs mainly in mechanical prostheses. Despite recent advances in bio-compatibility and haemodynamic profile all kinds of prostheses are likely to thrombose.

If prosthesis thrombosis is clinically suspected, auscultation may reveal anomalies (occurence of an abnormal murmur, reduction in prosthetic sounds). Radio-cinema of the prosthesis is particularly useful in this situation revealing sometimes blockage or reduced motion of a mobile part. Doppler echocardiography is the investigation of choice to provide a reliable, rapid and inexpensive diagnosis [27, 28].

- For mitral prostheses, transthoracic echocardiography often allows the diagnosis to be made from the presence of reduced motion of the mobile part and occasionally from direct visualisation of a thrombus. Colour Doppler is also extremely valuable, showing frequently an abnormal eccentric left ventricular filling jet. Continuous mode Doppler however has the most important role, showing an obstructive haemodynamic profile with elevated early diastolic velocity, elevated mean gradient, prolongation of the PHT, reduction in functional surface area if this can be calculated and appearance of an intraprosthetic leak compared to the baseline trace [5].
  Whilst the positive diagnosis of obstruction is confirmed by transthoracic Doppler echocardiography, the aetiological diagnosis can only be made by transoesophageal echocardiography [27-29]. Thrombosis of the prosthesis must be distinguished from other causes of obstruction such as an endocarditis vegetation, fibrous pannus (these 2 situations are often difficult to distinguish), or blockage of a cusp by the mitral subvalvular apparatus, when it has been preserved [30].
- Thrombosis is a clinically rare situation with aortic prostheses. Diagnosis is based on CW-Doppler which shows a rise of transthoracic gradients, a fall in the permeability

index and a reduction in functional surface area. These abnormalities are of great value if they can be compared with previously normal results, which again highlights the importance of having a baseline investigation. Transoesophageal echocardiography is less effective than in prosthetic mitral valve thrombosis: it can however assess the movement of the mobile components and occasionally directly visualise a thrombus. Radio-cinema of the prosthesis must also be performed if the echocardiography is equivocal.

Non-obstructive prosthesis thrombosis and embolus

The annual risk of embolism in patients with prosthetic valves varies depending on authors [26, 30] from 0.7 to 6 per 100 patient-years: the risk is high for mitral prostheses and when concomitant atrial fibrillation is present. Cerebral or peripheral embolism may occur in the context of obstructive prosthetic dysfunction but may also be seen in cases of non-obstructive thrombosis, intra-atrial thrombosis [29] or even endocarditis (septic embolus).

Non-obstructive thrombosis appears to be particularly common in the immediate post-operative period when it can be screened for by systematic transoesophageal echocardiography. After the post-operative period, in the presence of peripheral embolism in a patient with a normally functioning prosthetic valve at the tranthoracic echocardiogram, only multiplane transoesophageal echocardiography can reveal non-obstructive thrombosis or left intra-atrial thrombus [22, 29-31]. Transoesophageal echocardiography also provides information about the size of the prosthesis thrombus, a factor involved in the choice of treatment [32].

Bioprosthetic failure

More than 30% of bioprostheses require valve replacement 10 to 15 years after implantation.

This time is usually shorter in patients under 40 years and with prostheses implanted in the mitral position [3]. Regardless of the type of dysfunction, stenosis or regurgitation, the diagnosis sometimes suggested by auscultation relies on transthoracic Doppler echocardiography, which quantifies the dysfunction and its haemodynamic repercussions. The investigation is usually sufficient to provide all of the information required for reoperation. Transoesophageal echocardiography is only needed if the results of transthoracic Doppler echocardiography are inconclusive or in specific clinical situations (occurrence of or change of a pre-existing murmur, fever, arterial embolism, congestive heart failure, haemolysis).

Echocardiography generally shows morphological abnormalities of the prosthetic cusps which are thickened and calcified and poorly mobile, occasionally with prolapse or eversion of a cusp. Doppler may show chronic obstruction of the prosthesis (with raised gradients, prolongation of the PHT and reduced prosthetic surface areas) or moderate or occasionally massive intraprosthetic leak in the event of sudden rupture of a cusp.

Indications for transthoracic Doppler echocardiography in thrombo-embolic complications

Class I
— Assessment of suspected or overt thrombosis of a prosthetic valve.
— Control after increasing anti-coagulation treatment or thrombolysis (the frequency of subsequent investigations should be determined on an individual case basis).

Indications for transoesophageal echocardiography in thrombo-embolic complications

Class I
— Assessment in clinically suspected prosthetic valve thrombosis as a systematic complement to transthoracic Doppler echocardiography, even if normal.
— Follow-up of a non-obstructive thrombosis remaining asymptomatic (the frequency of subsequent investigations should be determined on an individual basis)

Indications for transthoracic Doppler echocardiography in structural valve deterioration (svd) of bioprostheses

Class I
— Diagnosis of SVD of bioprostheses suspected from the occurence of or change in a pre-existing murmur, apparition of fever, arterial embolism, congestive heart failure or haemolysis.
— Follow-up of diagnosed SVD of a bioprosthesis (the frequency of subsequent investigations is guided by the quality of the initial results depending on the extent of bio-prosthesis dysfunction and its haemodynamic consequences, until the time of reoperation).

Consensus indications for transoesophageal echocardiography in structural valve deterioration of bioprostheses

Class I
— Assessment if transthoracic Doppler echocardiography does not provide the necessary information for a treatment decision.

Class II
— Assessment if transthoracic Doppler echocardiography does not identify the mechanism of the regurgitation (degeneration, dehiscence of the bioprosthesis and/or endocarditis).

Class III
— Repeat investigation in the absence of a change in clinical situation or haemodynamic parameters assessed by transthoracic Doppler echocardiography.

Dehiscence of a prosthesis

This is a serious complication which is particularly common with repeated procedures which damage the valve annnuli [33, 34]. It affects both bioprostheses and mechanical prostheses without distinction and may or may not be due to infectious endocarditis.

In the aortic position, the diagnosis and quantification of the leak are based on transthoracic Doppler echocardiography. Transoesophageal echocardiography is indicated for suspected endocarditis (examination for aortic wall abscess), in the event of concomitant surgery on the ascending

Consensus indications for transthoracic Doppler echocardiography in dysfunction due to prosthetic dehiscence

Class I
– Clinical or biological (haemolysis) suspicion of prosthetic dehiscence
– Annual follow-up of chronic prosthetic dehiscence in the absence of any new clinical or laboratory findings (particularly indicators of haemolysis).
– Follow-up if clinical or laboratory findings change (the frequency of subsequent investigations should be determined on an individual basis).

Indications for transoesophageal echocardiography in dysfunction due to prosthetic dehiscence

Class I
– Systematic assessment as a complement to transthoracic Doppler echocardiography, particularly to exclude infectious endocarditis.

Class II
– Per-operative assessment during reinsertion or replacement of a prosthesis.

Class III
– Repeat investigation in the absence of a change in clinical situation or haemodynamic parameters assessed by transthoracic Doppler echocardiography .

aorta and if regurgitation increases over time. Some specific complications (aortic root abscess, perivalvular damage,..) are sometimes difficult to asses by transthoracic Doppler echocardiography.

In the mitral position, para-prosthetic regurgitations can be missed by transthoracic Doppler echocardiography because of the acoustic shadow of the prosthesis. Diagnosis and quantification of these para-prosthetic regurgitations are then based on multiplane transoesophageal echocardiography [26, 33, 34].

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CHRONIC ISCHAEMIC HEART DISEASE

Transthoracic Doppler echocardiography (TTE) is the preferred tool for the diagnosis and follow-up of patients with chronic ischaemic heart disease. TTE enables repeated study of global and regional left ventricular systolic function, left ventricular remodelling and its consequences, analysis of left ventricular filling and assessment of pulmonary pressures. The investigation therefore provides considerable useful information to adjust medical treatment, monitor evolution and assess prognosis. Systematic control of these parameters is useful in patients with abnormal left ventricular systolic function even if no clinical changes have occurred. Conversely, clinical symptoms guide the frequency of Doppler echocardiography controls in patients with good left ventricular function.

Resting Doppler echocardiography

Left ventricular geometry and function (table 1)

Left ventricular morphology. Investigation for left intraventricular thrombus (cf acute coronary syndrome chapter)

The global morphology of the left ventricle is assessed by two-dimensional echocardiography. Ventricular diameters are measured systematically, in M-mode with long axis parasternal or subcostal views.

Global left ventricular remodelling can be assessed by measuring volumes (Simpson biplane rule) and by measuring the sphericity indices obtained in systole and diastole (ratio of maximum length of the left ventricle to its width in the apical view) [1-4].

Left ventricular aneurysm is characterised by deformity of the diastolic outline in an akinetic or dyskinetic area with a thin wall. The functional value of the residual normal myocardium must be stated [5].

The investigation should always seek to identify a left intraventricular thrombus. This is particularly common as a sequela to anterior or apical infarction with an apical aneurysm [6] (table 2).

Left ventricular systolic function

The assessment of regional left ventricular function involves describing akinetic or hypokinetic areas in different views, describing their extent, morphological features and segmental wall thickness. A thin (diastolic thickness <6 mm) and dense appearance of akinetic myocardium indicates fibrous scarring and is highly predictive of lack of viability [7].

Estimation of left ventricular ejection fraction (LVEF) by two-dimensional echocardiography is part of the routine investigation. The resting LVEF is the most important prognostic indicator in patients with chronic ischaemic heart disease and plays an important role in treatment decisions, including consideration for cardiac resynchronisation [8, 9].

Semi-quantitative visual assessment of the LVEF has the major limitation of limited reproducibility, particularly for low LVEF. LVEF is best measured by the modified Simpson biplane method using harmonic imaging. This method also has limitations in detecting the left ventricular endocardium or left ventricular dilatation. Other techniques which are not used routinely improve the repeatability and reproducibility of measurements, such as the use of intravenous contrast agents [10] or real time transthoracic three-dimensional echocardiography, which has recently been introduced [11].

Left ventricular filling pressures (table 3) *(cf recommendations on the echocardiographic assessment of cardiomyopathies)*

Evaluation of diastolic function is essential. This enables filling pressures to be measured and provides prognostic information.

Transmitral flow analysis by pulsed Doppler is performed routinely. An E wave deceleration time of <150 msec and/or E/A ratio of >2 support raised left ventricular filling pressures in the presence of left ventricular systolic dysfunction [12-16]. Other parameters need to be recorded if mitral flow is "normal" in appearance: Ea wave measurement by tissue Doppler at the annulus with calculation of the E/Ea ratio; pulmonary vein flow by pulsed Doppler (Ap-Am time); left ventricular flow propagation during early filling by colour M-mode (Vp).

Functional mitral regurgitation

Functional mitral regurgitation is often found in heart failure. It is dynamic in nature and may increase or reduce as a result of changes in left ventricular size, geometry and load conditions. Left ventricular dilatation causes apical and lateral displacement of the papillary muscles, increasing the papillary muscle — valve annulus distance [17]. Increased tension on the cordae is responsible for valve restriction, apical displacement of the closure point and reduced closure surface area resulting in an increase in the surface area between the mitral cusps and the annulus in mid-systole (area under the tent) and the height of the closure point. These geometrical and contractile changes cause imbalance between the traction and closure forces on the mitral valve. During ventricular systole, the mitral valve is subjected to two competing forces: closure forces which are represented by left intraventricular pressure and by systolic contraction of the annulus (reducing the annulus surface area to be covered by the mitral cusps) and the

traction forces which oppose the closure forces by restricting valve movements in systole (and which occur as a result of the force exerted on the mitral cusps by cordae tendineae and by annulus dilatation) [17].

Increase in mitral regurgitation as a result of physical exercise in patients with heart failure adds to restricting exercise capacity and is associated with a poor prognosis [18-20]. These dynamic changes are often accompanied by an increase in pulmonary arterial systolic pressure and are associated with changes in valve geometry and the dynamic nature of asynchronism [21, 22].

Right cavities, pulmonary pressures and right ventricular filling pressures (table 4)

Analysis of the right cavities involves a morphological assessment of the cavities, right ventricle and atrium, pulmonary artery, superior vena cava and suprahepatic veins. Analysis of right ventricular function is particularly useful following a large right ventricular infarction. Right ventricular systolic and diastolic functions are difficult to assess, requiring parameters obtained using two-dimensional echocardiography to be combined with Doppler flow measurements of pulmonary, tricuspid and suprahepatic vein regurgitation flows.

Pulmonary pressures can be measured from the tricuspid regurgitation or pulmonary regurgitation flow (continuous Doppler).

Right atrial pressure (RAP) is defined either empirically (generally 10 mmHg) or from the diameter of the inferior vena cava (IVC) and its respiratory variations.

Transoesophageal echocardiography is not used routinely but is useful to assess ischaemic mitral regurgitation.

Stress echocardiography (table 5)

Dobutamine echocardiography

One of the most widely used indications for this technique is the investigation of myocardial viability by stress, particularly dobutamine stress echocardiography although this is however at the cost of increased ventricular arrhythmias if LVEF is <25% [23]. Several types of response have been described as predicting subsequent functional recovery: viability only in the presence of segmental resting asynergy with increase in contractility at low doses (protocol limited to low dose or load): a biphasic response, representing an improvement in myocardial thickening at low dose (or low load in the exercise test) with secondary deterioration in the same territory in response to high doses of dobutamine or high load in the exercise test: and sustained improvement, defined as an improvement in myocardial thickening at low dose or load maintained at high doses or high loads. Documenting contractile reserve, even though not synonymous with cell viability [24], predicts functional recovery after revascularisation with good accuracy, particularly if extensive (>4 segments) [25] and is also a risk marker for cardiovascular events [26]. The optimum time to reassess left ventricular function after revascularisation and the methods for doing this (echocardiography or other imaging methods) are debated. It appears however that functional (and LVEF) reassessment are indicated beyond the 5th month, improvement being seen up to 14 months after revascularisation in one third of cases [27].

Left ventricular diastolic wall thickness measured by MRI or echocardiography [7] is an excellent predictive indicator for functional myocardial recovery (sensitivity 94% and specificity 48%) after revascularisation. Diastolic thickness <6 mm excludes viability with excellent diagnostic accuracy.

The meta-analysis published by Bax [28] compared the diagnostic performance of different imaging methods. This was based on 32 studies which used dobutamine echocardiography, 53 isotopic studies and 20 studies which used positron emission topography (with 18-fluorodeoxyglucose); 11 studies directly compared dobutamine echocardiography with an isotopic technique. The overall sensitivity of low dose or high dose of dobutamine infusion was 81%, with a specificity of 80% and negative predictive value of 89%; for low dose dobutamine echocardiography, sensitivity was 82%, specificity 79% and the

Table 1 Chronic ischaemic heart disease: main parameters to record *to analyse left ventricular morphology and function*.

Parameters	Thresholds/quantification	Technical comments	Value
Segmental Wall motion	Qualitative or semi quantitative scoring scale (cf table)	16 or 17 segment model (ASE) [1]	Prognostic value of WMSI
	Describe the number of abnormal segments Segmental kinetics score (WMSI) [1]	2 parasternal views, 3 apical view, sub-costal view	
Wall thickness	>6-mm thick: preserved <6-mm: fibrous scaring [7]	Measured in M-mode (difficult)	No reversibility if fibrous scaring (fibrosis)
Left ventricular ejection fraction (LVEF)	Visual analysis (avoid)		Limitation of visual estimation
	Modified Simpson biplane method (disc summation method) [1]	Harmonic imaging Apical 4 and 2 cavity views	Reference method Limitations: — risk of under-estimating volumes — limited to 2 planes (use of real time 3D)

ASE: American Society of Echocardiography; WMSI: Wall motion score index.

Table 2 Chronic ischaemic heart disease: other essential parameters to record.

Parameters	Thresholds/ quantification	Technical comments	Value
Mitral regurgitation (MR)	State presence or absence of MR, even if mild and mechanism	Colour Doppler, apical approach	
	Threshold for significant MR: ERO >20 mm^2 [17]	Quantification by PISA or Doppler volumetric techniques	Adverse prognostic value in the absence of pre-existing valve disease
Investigation for thrombus	State dimensions and features (sessile or pedunculated)	Harmonic imaging Apical views showing the apex	False positives and negatives diagnosis possible
Cardiac output	Low output <2.2L/min/m^2	left ventricular outflow tract output	Haemodynamic consequence
Pericardium	Topography and volume if effusion	Multiple views	Signs of intolerance (cavity collapse, respiratory flow variations)

PISA: proximal isovelocity surface area; ERO: effective regurgitant orifice

Table 3 Chronic ischaemic heart disease: essential parameters to record *to analyse left ventricular filling* [12-16].

Parameters	Thresholds/quantification	Technical comments	Value
Mitral flow (at valve tip)	Type 1: relaxation abnormality (E/A <1 before 50 years old and <0.5 after 50 years old	Filling flow has multi-factorial dependency (age)	If LVH absent suggests filling pressures not raised
	Type 2: pseudo normal	Use other indices (E/Ea, E/Vp, Pulmonary venous flow)	
	Type 3: restrictive E/A >2 and/or EDT <150ms		If LVEF reduced suggests raised filling pressures
Tissue Doppler at annulus	E/Ea <8	Suggests filling pressures not raised	
	E/Ea >15	Suggests filling pressures raised	In post infarction, independent prognostic value (mortality)

E: transmitral E wave; Ea: tissue Doppler E wave at annulus; LVEF: left ventricular ejection fraction; LVH: left ventricular hypertrophy; EDT: E wave deceleration time; Vp: colour M-mode propagation velocity.

negative predictive value 83%. For high dose dobutamine echocardiography (investigation for biphasic response or sustained improvement), the corresponding values were 79, 85 and 90%. For thallium myocardial scintigraphy with reinjection, the values were 88, 50 and 83% respectively and for positron emission tomography, 93%, 58% and 86% respectively. This meta-analysis confirmed that the isotopic techniques are significantly more sensitive than dobutamine echocardiography although the echocardiography method has higher specificity and negative predictive value ($p<0.005$). The impact of documenting viability on indications for revascularisation and prognosis were described in the meta-analysis by Allman [26]. This included 24 studies on a total of 3088 patients who were examined by dobutamine echocardiography (n=7), thallium myocardial scintigraphy (n=6) or positron emission tomography (n=11). LVEF ranged between 25 and 31% and viability was found in 42% of cases; 35% of patients underwent revascularisation with an average follow-up period of 25 months. The mortality rate was significantly lower in the group with viability when revascularisation was performed (mortality rate 3.2%/year compared to 16%/year, $p<0.0001$, reduction in relative risk 79.6%). There was no significant difference in the annual mortality rate in the patient group with no detected viability (6.2 and 7.7%/year respectively). The annual mortality rate in the group which underwent revascularisation was 3.2% with viability compared to 7.7%/year without viability. This rose to 16%/year in the medically treated group with viability compared to 6.2%/year without, highlighting the “loss of

Table 4 Chronic ischaemic heart disease: essential parameters to record *for the right heart*.

Parameters	Thresholds/ quantification	Technical comments	Value
RV kinetics and function	RV wall kinetics RV dilatation (RV/LV diameter ratio >0.6)	Parasternal (short axis) apical (4C) and subcostal views	
Pulmonary ejection flow	Pulmonary output Acceleration time Calculation of resistances [31]		
Pulmonary regurgitation Doppler flow	PHT and Vmin/Vmax of PR PAPm and PAPd	Coupled Doppler and Pedoff probe	PHT (PR) <150 ms and/or Vmin/Vmax <0.5 supports raised RVEDP and prognostic value
Tricuspid regurgitation Doppler flow	TRVmax TR morphology (raised RAP) PAPs Pulmonary resistances	Multiple views and Pedoff probe	Vmax >2.5 m/s (take account age, body surface area)
Inferior vena cava	Diameter and respiratory variations	Subcostal 2D and TM mode	IVC collapse <50% in favor of raised RAP

LV: left ventricle; RV: right ventricle; TR: tricuspid regurgitation; PR: pulmonary regurgitation; PAPs: systolic pulmonary arterial pressure; PAPm: mean pulmonary arterial pressure; PAPd: diastolic pulmonary arterial pressure; PHT: pressure half time; RAP: right atrial pressure; RVEDP: RV end-diastolic pressure; Vmin: minimal velocity; Vmax: maximum velocity; ICV: inferior vena cava.

Table 5 Chronic ischaemic heart disease: optional parameters to record for the left heart.

Parameters	Thresholds/ quantification	Technical comments	Value
Left ventricular morphology			
Sphericity index [4]	L/l ratio [4] LV at end-diastole and endsystole divided by the volume of a sphere of identical diameter to the long axis of the LV [4]	Repeated assessment for remodelling (treatment evaluation protocols)	The lower this ratio the closer the LV appears as a sphere. (normal L/l ratio = 2)
Left ventricular opacification with contrast agent [10]	Harmonic imaging Specific settings for the contrast agent Apical 4 and 2 cavity views	Precise measurement of volumes and LVEF (treatment evaluation protocols)	Improved detection of the endocardium and measurement of volumes and LVEF [10]
Stress echocardiography			
Dobutamine echocardiography	WMSI at different steps LVEF at different steps E/Ea PAPs	Investigation for myocardial viability involving at least 4 segments	Excellent predictive value for functional recovery (NYHA, LVEF) and prognosis No use for mitral regurgitation
Exercise echocardiography	Mitral regurgitation (ERO), RV E/Ea PAPs Area under the tent	Standard protocol, semi-seated on bicycle Zoom on PISA TR (Vmax)	Prognostic value (resting ERO >20mm^2 and Δ exercise ERO >13 mm^2 [20]

E: transmitral E wave; Ea: tissue Doppler E wave at annulus; LVEF: left ventricular ejection fraction; L: length of LV in 4C apical view; l: LV width in 4C apical view; PAPs: systolic pulmonary arterial pressure; PISA: proximal isovelocity surface area; ERO: effective regurgitant orifice; RV: regurgitated volume; TR: tricuspid regurgitation; WMSI: wall motion score index

Consensus indications for Doppler-echocardiography in the diagnosis and follow-up of chronic ischaemic heart disease

Class I
— Assessment of new symptoms or cardiovascular physical signs in a patient with known coronary artery disease.
— Assessment of recovery of left ventricular function after revascularisation
— Repeated echocardiographical assessment of left ventricular function when results are useful to guide treatment.

Class II
— Follow-up in the absence of signs of progression in treated coronary artery disease patients in order to screen for deterioration in left ventricular function.

Class III
— Systematic repeated echocardiography in the absence of a change in clinical state in patients without left ventricular systolic dysfunction.

Consensus indications for exercise or pharmacological stress Doppler-echocardiography in the follow-up of chronic ischaemic heart disease

Class I
— Assessment for changes in symptoms suggesting progression of coronary artery lesions when the exercise test is:
- impossible to perform (orthopaedic problem, arterial disease, elderly patients, etc.);
- uninterpretable (left ventricular hypertrophy, left bundle branch block, etc.);
- negative submaximal and therefore inconclusive;
- equivocal (ST segment depression limited in time and amplitude).

— Detection of restenosis after coronary angioplasty when the exercise test is:
- impossible to perform (orthopaedic problem, arterial disease, elderly patients, etc.);
- uninterpretable (left ventricular hypertrophy, left bundle branch block, etc.);
- negative submaximal and therefore inconclusive;
- equivocal (ST segment depression limited in time and amplitude).

— Identification of the topography and/or extension of myocardial ischaemia to decide on possible myocardial revascularisation.
— Investigation of myocardial viability in the presence of reduced left ventricular systolic function to guide the decision for revascularisation.
— Coronary risk stratification before non-cardiac surgery in intermediate or high risk patients.

Class II
— Exercise echocardiography to estimate the volume and tolerability (pulmonary pressures) of moderate resting mitral regurgitation.
— Dobutamine echocardiography to investigate for myocardial viability in the presence of reduced left ventricular systolic dysfunction to consider the indication for cardiac resynchronisation.

Class III
— Follow-up of a stable coronary artery disease patient able to perform a maximum exercise test.
— Repeated investigation in a stable coronary artery disease patient without other cardiovascular symptoms.

chance" when a decision is taken not to revascularise in the presence of viability; this decision may be taken as a result of coronary anatomy and/or risk factors or co-morbidities.

Exercise echocardiography

Exercise echocardiography has only been used to a limited extent in the investigation of ischaemic left ventricular systolic dysfunction. The two-dimensional imaging acquisition protocol is classical (resting, low load, high load, recovery, for the 2 parasternal and 3 apical views) with additional Doppler acquisitions of the proximal isovelocity surface area of the mitral regurgitation jet (zoom on PISA), the transmitral flow by pulsed Doppler at the annulus and tricuspid regurgitation for measurements of pulmonary artery systolic pressure in the different stages of the exercise test. The degree of mitral regurgitation under baseline conditions does not correlate at all with the changes in mitral regurgitation during exercise, which vary greatly from one patient to the other [18]. An increase in the volume of mitral regurgitation on exercise is seen in almost 80% of cases. A large increase (increase in the effective regurgitant orifice area or ERO >13 mm^2) is seen in almost 30% of cases. When mitral regurgitation is severe at

rest (ERO ≥20 mm^2), the incidence rises to 40% and when the leak is moderate it is 25% [26, 28, 29]. The large increase in regurgitated volume is usually accompanied by a significant rise in systolic pulmonary artery pressure and a fall in cardiac output [18]. Increased left ventricular asynchronism occurs in 20-30% of patients and is accompanied by an increase in the severity of mitral regurgitation [21, 30].

The dynamic nature of functional mitral regurgitation has prognostic implications, [18-20], the validated threshold being an increase of ≥13 mm^2 in the ERO during the exercise test. If the increase in ERO exceeds this threshold the relative risk of death is increased by a factor of 5 during a 3 year follow-up period. Analysis of dynamic changes in ERO may inform the effectiveness of medical treatments and guide therapy.

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ACUTE CORONARY SYNDROMES

The acute coronary syndromes (ACS) are a group of various clinical situations ranging from unstable angina to ST segment elevation with myocardial infarction. Echocardiography provides important information in the diagnostic and prognostic assessment of ACS. The versatility of echocardiography instruments into coronary care units and the development of portable echocardiography instruments make this technique the non-invasive imaging method of choice for emergency use, which is readily available and easy to repeat at the patient's bedside.

Conditions for performing the investigation

High quality echocardiography imaging including second harmonic imaging is required in order to obtain a reliable assessment of myocardial wall motion and thickening.

Transthoracic echocardiography (TTE) is sufficient in the great majority of cases to provide diagnostic information and screen for complications. Transoesophageal echocardiography (TEE) is rarely indicated and is used above all if a complication is suspected but is not sufficiently assessed by transthoracic echocardiography. Transoesophageal echocardiography requires a trained operator.

Diagnostic utility

Echocardiography may be a valuable aid if the diagnosis is uncertain, particularly when there is a high index of clinical suspicion of ACS with a normal or non-contributory ECG [1-3]. Echocardiography is generally not useful diagnostically in ACS with ST segment elevation and must not delay the institution of reperfusion techniques.

The development of portable echocardiography instruments allows the diagnosis to be confirmed rapidly if necessary by finding typical regional wall motion abnormalities and to screen for some complications (pericardial effusion, LV thrombosis, right ventricular extension of inferior wall myocardial infarction, mechanical complications) [4].

The parameters to be recorded are predominantly regional wall motion abnormalities. These must be examined systematically in all views. Their topography closely reflects the coronary vascular distribution in the absence of some anatomical variations [5] (figures 1 and 2). The presence of akinesia or severe hypokinesia in a vascular territory is a very sensitive marker of ischaemia [6]. The investigation must be performed using the ASE 16 segment (or 17 segment) classification [7].

The thickness of walls with wall motion abnormalities must be reported. Normal wall thickness argues in favour of an ACS being recent.

Echocardiography is highly sensitive (90-95%), to diagnose ischaemia and infarction in patients with a high probability of ACS [1-3]. It also has a high negative predictive value (approximately 95%). Diagnostic false negatives nevertheless occur in non-Q wave infarction. If ACS is suspected and the ECG is non-contributory the sensitivity of echocardiography to diagnose ischaemia is highest if the investigation is performed during pain, particularly if the wall motion abnormalities regress after the ischaemia is treated.

The positive predictive value of echocardiography to diagnose infarction is lower depending on the likelihood of the diagnosis of ACS being present. Echocardiography must not be used in isolation to diagnose myocardial infarction. Segmental wall motion abnormalities are not specific for infarction or acute ischaemia and may represent post-ischaemic myocardial dysfunction (stunning), infarction scar or non-ischaemic segmental abnormalities such as acute myocarditis.

Echocardiographical assessment of myocardial infarction (ACS with ST segment elevation)

All patients with an acute infarction (with or without ST segment elevation) should have an echocardiogram performed during the hospital phase. In some patients the investigation is performed on an emergency basis for diagnostic purposes, if an infarction is complicated by cardiogenic shock or if a mechanical complication is suspected. In uncomplicated infarction, echocardiography is generally performed after reperfusion treatments. Echocardiography must be performed immediately if a patient develops sudden haemodynamic deterioration. Patients with extensive wall motion abnormalities on the initial echocardiogram are at higher risk of developing complications. Repeated echocardiographic follow-up is indicated in these patients.

Parameters to be recorded [7]

Segmental wall motion

Echocardiography should describe the severity and extent of segmental wall motion abnormalities reporting the number of abnormal segments in the different views, results of examining for segmental abnormalities in the different territories or for signs of previous infarction with wall thinning (table 1). The severity of the wall motion involvement must be reported for each segment, taking into account myocardial thickness: akinesia (no wall thickening or endocardial movement), hypokinesia (reduced wall thickening and endocardial movement), or dyskinesia (paradoxical systolic movement associated with absence of wall thickening).

The ischaemic area may be over-estimated as adjacent segments may experience stretching, stunning or load modifications effects.

Left ventricular morphology

Left ventricular dimensions are usually measured in M-mode in parasternal views. Left ventricular volumes (particularly the end-systolic volume) are measured when the left ventricular ejection fraction (LVEF) is measured using the Simpson method in apical views.

Particular attention should be paid to geometrical changes of expansion of the infarcted area, remodelling or the establishment of an early left ventricular aneurysm.

Left ventricular systolic function

Left ventricular systolic function is assessed by measuring the LVEF using the Simpson biplane method. A segmental wall motion score index (WMSI) is calculated from the sum of the abnormal myocardial segmental kinetics scores as the ratio to

the number of segments analysed also provides an estimate of global systolic function.

Diastolic function and left ventricular filling pressures

The main parameters to record are transmitral flow by pulsed Doppler, allowing measurement of the E and A wave and the E/A ratio, E wave deceleration time (DT) and measurement of the Ea wave by tissue Doppler at the mitral annulus, together with calculation of the E/Ea ratio. If mitral flow is "normal" in appearance, other parameters must be recorded such as analysis of pulmonary venous flow or lef ventricular flow propagation during early filling (Vp) by colour M-mode during two-dimensional guidance. (table 2)

Left atrial size should be measured in an apical view (left atrial surface area or volume as demonstrated in epidemiological studies).

Right cavities, pulmonary pressures and right ventricular filling

The size of the right cavities and right ventricular wall kinetics must be assessed. Right ventricular injury should be examined in inferior wall myocardial infarctions. Pulmonary pressures can be calculated from tricuspid and pulmonary regurgitant regurgitation flows. The diameter of the inferior vena cava (IVC) and its respiratory variations must be measured.

Complications of myocardial infarction

Acute mitral regurgitation

Mitral regurgitation (MR) due to papillary muscle rupture is a rare but serious complication of infarction generally resulting clinically in cardiogenic shock. The posterior papillary muscle is the most commonly affected. Papillary muscle rupture is responsible for an eccentric MR jet due to segmental prolapse which is occasionally difficult to assess by TTE colour Doppler and is associated with a hyperkinetic left ventricle. TEE is often required to confirm the diagnosis, frequently enabling the ruptured papillary muscle to be visualised, particularly in a transgastric view.

The prevalence of MR outside of possible papillary muscle rupture ranges from 11% to 59% [8]. The main mechanism is incomplete closure of the mitral valve with restriction of one or both valves, often associated with widespread wall motion abnormalities which may or may not involve the papillary muscles. It is associated with a poor prognosis and increased risk of mortality and heart failure independently of other prognostic factors [9]. It is often clinically silent and must be examined systematically by echocardiography. Quantification of the severity of mitral regurgitation is of prognostic importance. Significant mitral regurgitation is defined by a surface area of the effective regurgitant orifice (ERO) of >20 mm^2 [9].

Septal rupture

The clinical presentation of septal rupture is similar to that of papillary muscles ruptures. The echocardiographical diagnosis is based on the finding of a septal discontinuity with more or less well defined outlines, associated with an abnormal left to right shunt between the ventricular cavities on colour 2 dimensional Doppler. If a large shunt is present, the left ventricle is hyperkinetic, the left-to-right shunt Doppler flow is at low velocity and the pulmonary pressures are raised. The site of rupture depends on the topography of the myocardial infarction. Interventricular communications from anterior infarctions are best assessed using apical views. Interventricular communication from posterior wall myocardial infarctions, which are occasionally associated with a right ventricular myocardial infarction are best assessed by a short axis parasternal and sub-costal view. TEE is only indicated in patients in whom transthoracic echocardiography investigation is insufficient.

Left ventricular wall rupture

This is the most serious complication of infarction and generally results in rapidly fatal tamponade. The echocardiographical diagnosis generally only involves subacute ruptures. Direct visualisation of the rupture is difficult and a Doppler flow is rarely seen. The most highly suggestive echocardiographical sign is the presence of a smaller or larger pericardial effusion which is heterogeneous in appearance and generally circumferential [10].

Right ventricular myocardial wall infarction

Right ventricular extension occurs in 30 to 50% of cases of inferior infarction [11]. Systematic ultrasound examination is appropriate because of the poor specificity of clinical and electrocardiographical signs and because of its prognostic impact. The most common echocardiographical signs are segmental wall motion or global right ventricular abnormalities and right ventricular dilatation and dysfunction. Right Doppler flow recording (pulmonary regurgitation) is useful for diagnosis and allows right ventricular filling pressures to be measured [12]. (table 3)

Left ventricular remodelling, left ventricular aneurysm and thrombus

Wall expansion complicates extensive transmural infarctions and results in thinning of the wall which may be complicated by rupture or early left ventricular aneurysm formation. Repeated echocardiography is useful to identify expansion of the involved left ventricular myocardium. Left ventricular aneurysm is seen on two-dimensional echocardiography as a dyskinetic or akinetic thinner wall with deformity of the diastolic volume. Aneurysm predisposes to intraventricular thrombus formation. Some echocardiographical features of the thrombus (pedunculated and mobile) are associated with increased risk of arterial embolism [13] (table 3).

True left ventricular aneurysm must be distinguished from false ventricular aneurysm secondary to myocardial rupture into partitioned myocardium. The false aneurysm has a narrow neck, an acute angle of attachment with the adjacent myocardium and is characterized by a bi-directional Doppler flow.

Pericardial effusion

A pericardial effusion may be seen with transmural myocardial wall infarction, independently of myocardial rupture. Echocardiography describes its size, distribution, tolerance and progression.

Prognostic impact

Echocardiography provides prognostic information from an assessment of the extent of the wall motion abnormalities, left ventricular systolic and diastolic performance indices and in screening for complications.

Table 1 Diagnosis and initial assessment of acute coronary syndromes: obligatory parameters to record *for analysis of left ventricular morphology and function*.

Parameters	Thresholds/quantification	Technical comments	Value
Segmental wall motion	Qualitative or semi-quantitative scoring scale (cf table 4)	16 or 17 segment model (ASE) [7]	Sensitive parameter for ischaemia Limited specificity
	Describing the number of abnormal segments	Large number of views: 2 parasternal views, 3 apical views, subcostal views	Investigation for segmental abnormalities in separate territories or a sequella of old infarction with wall thickening
		Use analysis of wall thickness rather than analysis of displacement	
Regional wall motion	Coronary vascular territories	16 or 17-segment model (ASE) [7]	Topographical value
Left ventricular ejection fraction (LVEF)	Visual analysis (avoid)		Limitations of visual estimation
	Modified biplane Simpson method (disc summation method)	Harmonic imaging Apical 4 and 2-cavity views	Reference method Limitations: — risk of under-estimating volumes — limited to 2 planes (utility of real time 3D) — possible rapid change in LVEF post-infarction

ASE: American Society of Echocardiography; LVEF: left ventricular ejection fraction.

Table 2 Diagnosis and initial assessment of acute coronary syndromes: obligatory parameters to record *for analysis of left ventricular filling pressures*.

Parameters	Thresholds/quantification	Technical comments	Value
Mitral flow (at valve tip)	Type 1: relaxation abnormality (E/A <1 before 50 years old and <0.5 after 50 years old	Filling flow has multi-factorial dependency (age, load conditions)	Suggestive of filling pressures not raised if no LVH
	Type 2: pseudo normal	Use other indices (E/Ea, E/Vp, Pulmonary venous flow)	
	Type 3: restrictive (E/A >2 or EDT <130-150ms)		Suggestive of filling pressures if LVEF low EDT ≤150ms has poor prognostic value [16]
Tissue Doppler at annulus	E/Ea <8	Suggestive of filling pressures not raised	
	E/Ea >15	Suggestive of raised filling pressures	Independent prognostic value for post-infarction death [18]

E: transmitral E wave; Ea: tissue Doppler E wave at annulus; LVEF: left ventricular ejection fraction; LVH: left ventricular hypertrophy; EDT: E wave deceleration time; Vp: Propagation velocity on colour M-mode.

Table 3 Diagnosis and initial assessment of acute coronary syndromes: other obligatory parameters to record.

Parameters	Thresholds/quantification	Technical comments	Value
		Others	
Mitral regurgitation (MR)	State presence or absence of MR, even mild and mechanism	Colour Doppler, apical view	
	Threshold for significant MR: ERO>20 mm^2 [9]	Quantification by PISA or Doppler volumetric techniques	Adverse prognostic value in the absence of pre-existing valve disease
Investigation for thrombus	State size and features (sessile or pedunculated)	Harmonic imaging Apical views showing the apex	Possible diagnostic false positives and negatives
Extension to RV	RV wall motion abnormalities RV dilatation (ratio RV/LV diameter >0.6)	Parasternal (short axis), apical (4C and right 2C) and subcostal views	
	Doppler pulmonary regurgitation Doppler flow (PHT <150 ms, V_{min}/V_{max} <0.5)	Couple Doppler and Pedoff probe	Excellent diagnostic and prognostic value
	Inferior vena cava	Subcostal M-mode with respiratory trace	Dimensions determined by blood volume and RAP
Pericardium	Topography and volume of effusion	Multiple views	Signs of intolerance (cavity collapse, respiratory flow variations)

PHT: pressure half time; RAP: right atrial pressure; Vmin: minimum pulmonary regurgitation velocity; Vmax: maximum pulmonary regurgitation velocity; ERO: effective regurgitant orifice area.

Table 4 Segmental wall motion score index (WMSI).

0 **Hyperkinetic:** Increased systolic wall thickening and endocardial movement
1 **Normal:** Normal systolic wall thickening and endocardial movement
2 **Hypokinetic** Reduced systolic wall thickening and endocardial movement
3 **Akinetic** No systolic endocardial wall thickening or movement
4 **Dyskinetic** Paradoxical endocardial movement in systole with no systolic wall thickening

Table 5 Multifactorial prognostic approach to coronary artery syndromes: [20].

	Low risk	Intermediate risk	High risk
LVEF	40%	30-39%	<30%
Transmitral flow	Abnormal relaxation	Normal profile or abnormal relaxation	Restrictive flow
Mitral regurgitation	Absent or mild	Moderate	Severe
E/Ea	<8	8-15	>15
PAPs	<45 mm Hg	46-60 mm Hg	>60 mm Hg

E: transmitral E wave; Ea: E wave by tissue Doppler annulus; LVEF: left ventricular ejection fraction; PAPs: systolic pulmonary arterial pressure.

As wall motion abnormalities may improve after reperfusion treatment, prognostic echocardiography can be performed immediately before discharge from hospital.

Several echocardiographical systolic and diastolic left ventricular function parameters are of established prognostic value. A raised segmental wall motion index (WMSI >2) (table 4) or reduced left ventricular ejection fraction (LVEF <40%) are associated with a high risk of death, heart failure and ventricular arrhythmias [14]. End-systolic volume has a greater prognostic value than LVEF [15]. Several diastolic function indices predict late left ventricular dilatation and death, such as a shortened E wave deceleration time (<130-150 msec), restrictive mitral flow, an E/Ea ratio >15 by tissue Doppler on the mitral annulus and left atrial dilatation [16-19]. The presence of, even slight, mitral regurgitation, left ventricular remodelling [9], or left ventricular aneurysm detected with repeated echocardiography also has a poor prognosis.

A prognostic approach combining these different factors has recently been proposed [20] (table 5).

Other prognostic indices can be obtained with techniques which are not used routinely. These include assessment of cardiac cycle-dependent variation of integrated backscatter

Table 6 Acute coronary syndromes: optional parameters to record.

Parameters	Thresholds/quantification	Technical comments	Value
Segmental wall motion score index (WMSI)	Different segmental scales	Accuracy depends on number of analysable segments	Reflects global left ventricular function Prognostic index
		Increasing WMSI [1] Normal WMSI =1	WMSI >2 predictive of post infarction LV dilatation [1]
		Decreasing WMSI used in the TRACE study [14] 9 segments; WMSI normal =2 (WMSI ≤1.2 equals LVEF ≤35%).	Prognostic value of repeated measurement of WMSI [14]
Colour M-mode on mitral flow	E/Vp≥1.5 predictive of in-hospital heart failure Useful to identify pseudonormal mitral flow	Propagation velocity (Vp) measured on the first aliasing line (45 cm/s), from the mitral annulus to 4 cm into the LV cavity in early diastole	Limited reproducibility of measurement
LA volume	Biplane area-length method Normal value 22±6 mL/m^2		Adverse prognostic value if LA volume >32 mL/m^2 [19]
Stress echocardiography (dobutamine > effort) (*dipyridamole not used in France in this situation*)	Low dose WMSI (5-15mcg/Kg/min)) for contractile reserve and peak WMSI for ischaemia (and biphasic response)	Possible from day 3 post MI if no complications and beta blockers omitted on the same day	No threshold value Importance of topography and extent of induced or modified segmental asynergy

E: transmitral E wave; LVEF: left ventricular ejection fraction; Vp: Propagation velocity in colour M-mode; WMSI: wall motion score index.

Table 7 Acute coronary syndromes: utility of new techniques, optional parameters to be recorded.

Parameters	Thresholds/quantification	Technical comments	Value
Myocardial contrast echocardiography [21]	Assessment of the area at risk Identification of "no reflow" phenomenon Evaluation of viable myocardium	Intra-coronary contrast echocardiography validated, little used Intravenous contrast echocardiography not used routinely	Prognostic value from identifying the "no-reflow" phenomenon
Transthoracic coronary Doppler [22]	Parameters suggestive of "no reflow": — Diastolic flow deceleration time <185 msec — Reverse systolic flow	Recording LAD coronary flow by high frequency transthoracic Doppler	Prognostic value from identifying the "no-reflow" phenomenon
Cardiac cycle-dependant variation of integrated backscatter			Non-invasive identification of reperfusion Predictive of left ventricular remodelling

LAD: left anterior descending artery

signal or the presence of a "no reflow" effect on myocardial contrast echocardiography [21] or in transthoracic coronary Doppler flow analysis [22].

Stress echocardiography

Acute coronary syndromes with ST-segment elevation

Widespread use of primary angioplasty to treat the acute coronary syndrome with ST segment elevation has changed the investigation strategy for these patients. Dobutamine echocardiography which was initially proposed to guide the management of these patients is now prognostic useful and quantifies the amount of stunned myocardium liable to recover in the weeks following recanalisation of the target coronary artery [23].

Transoesophageal echocardiography (mostly dobutamine) is well tolerated and can be performed as early as the 3rd day post MI (omitting the beta blocker on the day of the investigation) in the absence of complications during the acute phase [24]. This identifies areas of stun-

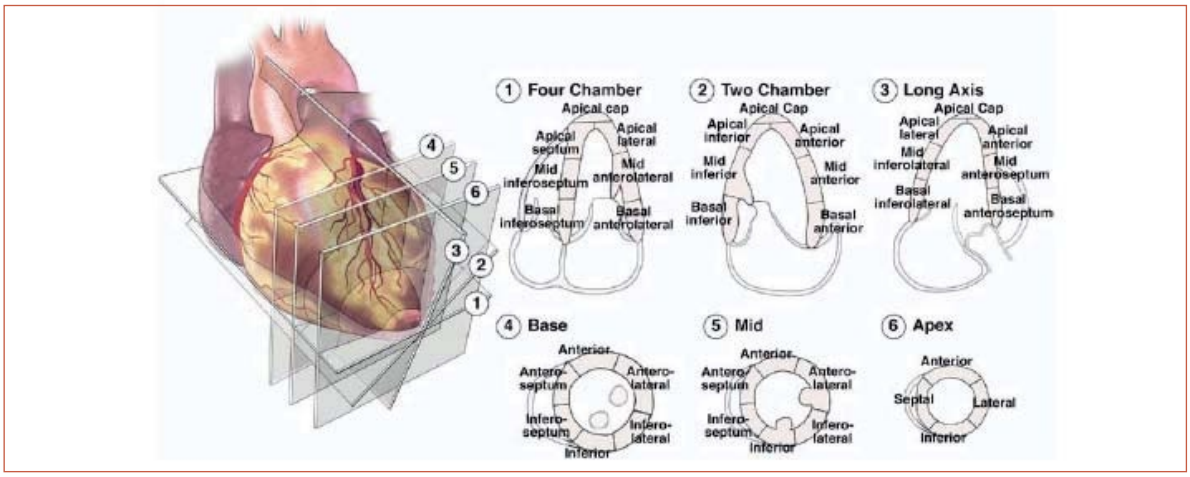

Figure 1. 16-segment classification (ASE) (adapted from 7).

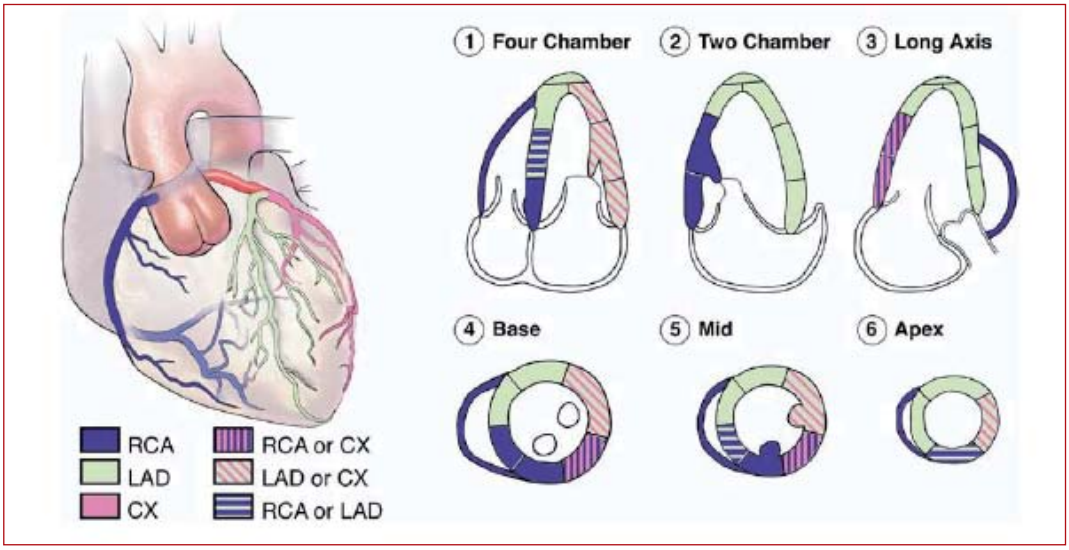

Figure 2. Coronary vascular territories (adapted from 7).

Consensus indications for Doppler-echocardiography in the diagnosis and initial evaluation of acute coronary syndromes (ACS)
Class I — Diagnosis of myocardial infarction or suspected ACS if diagnosis from classical criteria uncertain (ECG, cardiac enzymes) including troponin. — ACS and cardiogenic shock* — Suspected mechanical complications of myocardial infarction* — Investigation for right ventricular extension in the case of suggestive clinical signs in patients with an inferior myocardial infarction. **Class II** — Assessment of the extension of segmental abnormalities and left ventricular function during ischaemia. **Class III** — Diagnosis of myocardial infarction when the diagnosis is clear from classical criteria (ECG, cardiac enzymes)

* TEE indicated if TTE assessment inadequate.

Consensus indications for Doppler-echocardiography in the early assessment and follow-up of acute coronary syndromes (ACS)

Class I
— Initial assessment of the consequences of the infarction and screening for early complications of myocardial infarction
— Pre-hospital discharge prognostic assessment for widespread myocardial infarction
— Repeated echocardiography assessment in the event of clinical changes suspicious of a complication of myocardial infarction
— Repeated echocardiographical assessment of left ventricular function when the results are useful to guide treatment.
— Early assessment for the presence of residual ischaemia which can be triggered by stress echocardiography*
— Early assessment of myocardial viability when results are useful to guide possible revascularisation

Class II
— Assessment of left ventricular dysfunction 2 to 3 months after the acute episode
— Assessment of left ventricular function recovery after revascularisation

Class III
— Systematic repeating of echocardiography beyond month 3 in the absence of a change in clinical state

* dobutamine stress echocardiography or exercise echocardiography

ned myocardium by the infarction but viable with contractive reserve. It also detects residual ischaemia in the infarcted or distant area (detecting multiple vessel disease) [25-26]. Patients with contractile reserve have significant increase in LVEF and a significant decrease of segmental wall motion score index and end-systolic and end-diastolic volumes [27].

The presence of myocardial viability is a powerful predictor of the risk of cardiovascular complications. In the numerous available publications [24,25,28], this test has an excellent negative predictive value of around 90%, relative risk varying between 2 and 5 depending on whether the end point used is death ± recurrence of myocardial infarction ± recurrence of myocardial ischaemia.

Dobutamine echocardiography compares favourably with myocardial scintigraphy post-infarction: it has better specificity although at the cost of poorer sensitivity [29].

Acute coronary syndromes without ST segment elevation

Very little information is currently available on the use of pharmacological stress echocardiography in this context [30, 31]. Lin [31], demonstrated that the feasibility and utility of ECG was limited (ECG uninterpretable in 25% of patients and of those who completed the exercise test, 38% did not achieve the target heart rate) in a population of ACS patients assessed after monitoring for 48 hours and followed up for an average of 29 months. The presence of ischaemia on echocardiography predicted events (death, infarction and subsequent revascularisation) in contrast to ischaemia on ECG.

In the absence of electrical or enzymatic evidence of myocardial ischaemia or myocyte necrosis a negative stress echocardiogram should be reassuring and could avoid unnecessary hospitalisations of patients at very low risk of cardiovascular events.

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INDICATIONS FOR ECHOCARDIOGRAPHY IN CORONARY RISK STRATIFICATION BEFORE NON-CARDIAC SURGERY

Cardiac complications are responsible for the main adverse events following major surgery and detecting patients at high risk of a per- or post-operative coronary event would seem extremely useful. It is important before the procedure to investigate for these as thoroughly as possible and thereafter to monitor the patient at close intervals per and post-operatively. Coronary risk was initially assessed in patients who had undergone vascular surgery [1-4] because of the high prevalence of coronary artery lesions in this population. There are many risk factors, involving clinical, electrocardiographical and historical findings and also the type of surgical procedure planned. This makes stratification difficult and requires a successive stepwise assessment to be used.

Clinical risk markers have been established from large series and are proposed in the North American recommendations (table 1) [5]. In order to facilitate clinical risk assessment, some authors have proposed a simplified score which is currently used by most teams (table 2). Clinical markers are not however sufficient as the surgical procedure also influences the incidence of cardiovascular complications (table 1). The first, clinical and electrocardiographical, stage is used to detect patients at lower risk (patients with one or more minor risk factors but no intermediate or major risk factors). If functional capacity is good in this group (clinical evaluation and if uncertain, exercise test), surgery can be proposed with no other investigations (table 3). If the clinical assessment detects factors suggesting high clinical risk the surgical procedure should be postponed and a full cardiological assessment, possibly combined with coronary angiography, should be obtained. Patients at **intermediary risk from the clinical findings** in practice represent a large proportion (69% of patients in a recent general surgical series) [6]. The management of this group is still being debated and there is no single definition of the group. One definition often used is the presence or 1 or 2 intermediary clinical risk factors. Functional capacity should be assessed first line with an exercise test which is useful prognostically but its feasibility is often limited in an elderly population with poor functional capacity [7, 8]. If satisfactory and if the procedure is not high risk, surgery may then be considered (possibly under beta blocker cover, see below). If functional capacity is poor or unassessable or if the surgery is intermediary or at high risk, a functional test is appropriate (table 3). This is the situation in which stress echocardiography (most often with dobutamine) has a role as it is easy to perform and very widely studied in this type of population.

Published studies on dobutamine echocardiography mostly involve **vascular surgery** with an assessment of immediate pre-operative and late prognosis. All of these indicate that the investigation has excellent negative predictive value (97 to 100%) [1-4]. Development of a wall motion abnormality during the test is however an independent predictive factor for **per-operative** ischaemic events. The positive predictive value of the investigation for serious events (death or infarction) is

Table 1 Pre-operative clinical assessment.

1/ Clinical risk factors

— Major (high clinical risk)
- Acute or recent myocardial infarction (with residual ischaemia), unstable or severe angina
- Decompensated heart failure
- Severe arrhythmias: High degree AVB, symptomatic ventricular arrhythmia, supra-ventricular arrhythmia with rapid heart rate
- Severe heart valve disease.

— Intermediate (intermediate clinical risk)
- Stable angina
- Past history of myocardial infarction (clinical history or ECG Q wave).
- Stable heart failure
- Diabetes (particularly insulin-dependent)
- Renal insufficiency.

— Minor (low clinical risk)
- High age (over 70 years old)
- Abnormal ECG (LVH, LBBB, ST abnormalities).
- Non-sinus rhythm (generally AF)
- Reduced functional capacity
- Past history of cerebrovascular accident
- Poorly controlled hypertension.

2/ Type of surgery

— High cardiac risk (>5%)
- Surgical emergencies, particularly in the elderly
- Aortic or major vascular surgery
- Peripheral vascular surgery
- Long procedures with bleeding risk

— Intermediate risk (1 to 5%)
- Carotid endarteriectomy
- Head and neck surgery
- Orthopaedic surgery
- Thoracic or intraperitoneal surgery
- Prostate surgery

— Low risk (<1%)
- Endoscopies
- Superficial surgery
- Cataract surgery
- Breast surgery.

however relatively low (38% in the larger series) [9]. The heart rate at which ischaemia develops (ischaemic threshold <70% of MPHR) is an independent predictive factor for post-operative events and can increase the positive predictive value of the investigation to 53%. All patients who had a serious event (MI or death) were in this group [9].

The prognostic value of stress echocardiography **distant** to vascular surgery was confirmed by the same authors (10] in a series of 316 patients followed up for 19±11 months. Two factors emerged as predictive of late cardiac events: a past history of MI (RR=3.8) and the extent of ischaemia in the pre-operative dobutamine echocardiogram (RR=6.5 when the number of ischaemic segments was 3 or more).

Table 2 Simplified clinical risk score before non-cardiac surgery (from ref. 21).

Clinical risk markers:
Age ≥70 years
Current angina
Past history of myocardial infarction
Heart failure
Treatment for ventricular arrhythmia
Past history of ischaemic cerebral event
Diabetes
Renal insufficiency
Risk assessment:
Low clinical risk: no marker
Intermediate clinical risk: 1 or 2 markers
High clinical risk: 3 or more markers

These findings on the prognostic value of pre-operative stress echocardiography have been confirmed before **major non-vascular surgery** [6]. A dobutamine stress echocardiography was performed in 530 patients unable to undergo an exercise test prior to major non-vascular surgery in order to stratify **per-operative** cardiac risk. This study confirmed that the investigation had an excellent negative predictive value, all of the cardiac events occurring in patients who had myocardial ischaemia (NPV=100%). The positive predictive value of the test was low (15%), although increased to 43% for a heart rate ischaemic threshold of below 60% of MPHR, confirming the results of Poldermans and co-workers [9] before vascular surgery. There is at present no published work on the prognostic value of stress echocardiography distant to major non-vascular surgery.

Stress echocardiography and myocardial scintigraphy have a similar diagnostic performance in screening for coronary artery disease [11]. Many studies have been conducted in pre-operative coronary risk assessment, mostly in candidates for vascular surgery [12-14].

Meta-analyses of these studies have concluded either that the two techniques performed similarly [15,16] or, more recently, in a meta-analysis published by Kertaï et al. on 8119 patients [17] that stress echocardiography was statistically significantly superior to myocardial scintigraphy, in particular with a better positive predictive value for the development of per-operative cardiac complications. Stress echocardiography is therefore useful to assess and stratify pre-operative coronary risk.

The role of stress echocardiography should be incorporated into the perioperative **therapeutic strategy** for these patients.

Beta blockers are the cornerstone of treatment, producing a significant reduction in the incidence of cardiac events both per-operatively and late after the surgery. The first study, published in 1999 [18], was a randomised prospective study of the effect of a beta blocker (bisoprolol) versus placebo on peroperative morbidity and mortality in **major vascular surgery** in high risk patients with ischaemia in a pre-operative stress echo. One hundred and seventy-three of 846 patients with one or more risk factors had ischaemia in the stress echo. Fifty-nine ultimately received bisoprolol and 53 the placebo

Table 3 Risk stratification algorithm before non-urgent, non-cardiac surgery.

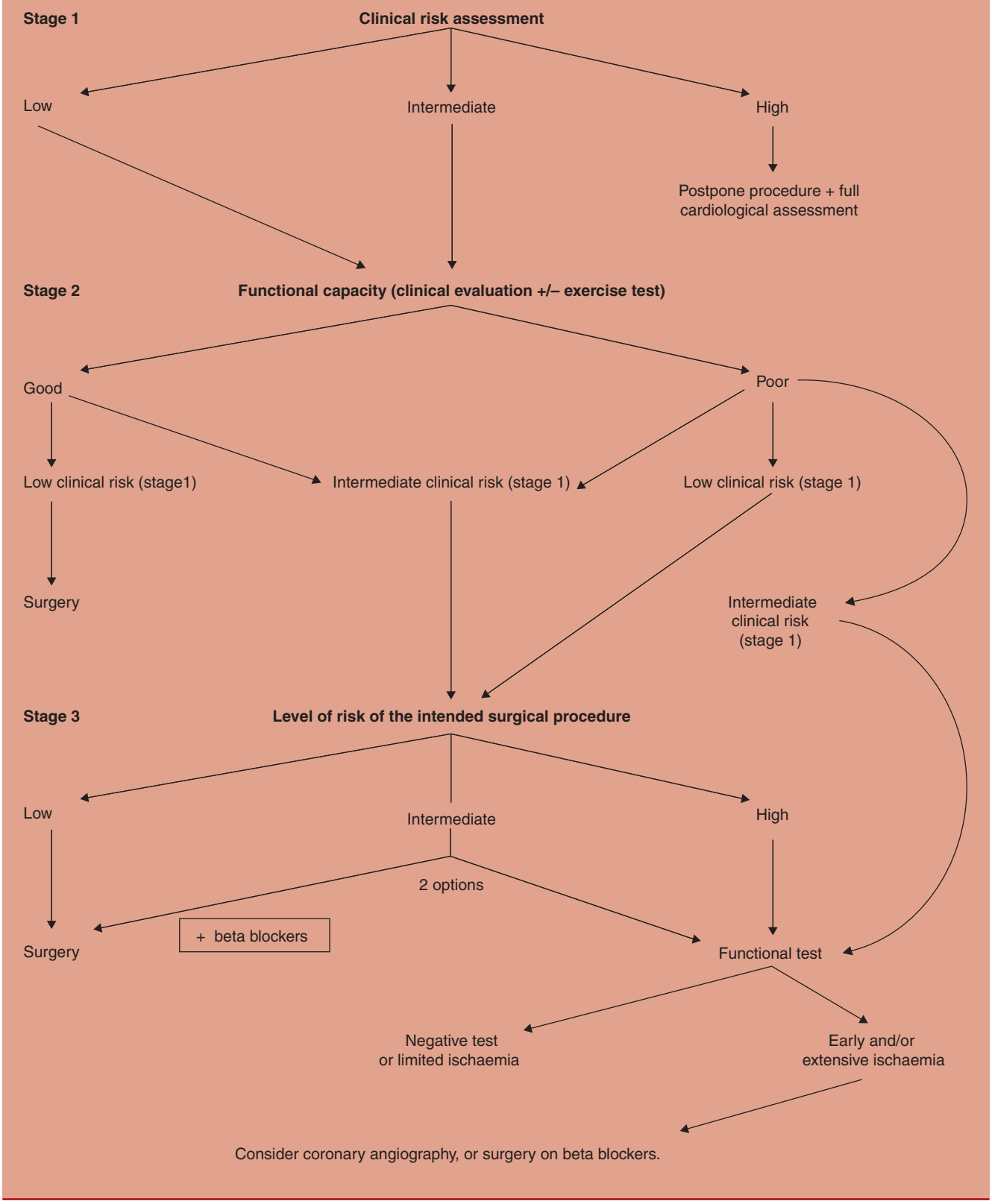

Table 4 Echocardiographical parameters to be recorded during stress echo (exercise or dobutamine).

Parameter	Threshold/quantification	Technical comment	Indication	Prognostic value
Wall motion abnormality	— New abnormality — Or deterioration vs. rest — Affecting at least 1 segment	Interpretation difficulty: inferobasal segment or postero-basal IVS	systematic	— Excellent NPV — Low PPV (for vasc. or non-vascular surgery)
Extent of ischaemia	Number of ischaemic segments	Difficulty when resting WM abnormality is present	systematic	— Probably reduced protective role of beta blockers — Number >3 predictive of increased post-operative risk (vasc)
Ischaemic threshold	— HR at which ischaemia develops — <60% MPHR (non-vasc. surgery) — <70% MPHR (vasc. surgery)	Stop beta blocker 48h before investigation	systematic	— Per-operative prognostic value — No post-operative prognostic value

NPV: negative predictive value, PPV: positive predictive value, HR: heart rate, MPHR: maximum predicted heart rate, IVS: interventricular septum, vasc: vascular.

(61 patients were excluded as they were already taking a beta blocker or had early severe ischaemia). Two patients in the beta blocker group (3.4%) and 18 patients in the placebo group (34%, p<0.001) developed the combined criterion of either cardiac death or non-fatal MI. The risk of peri-operative morbidity-mortality was therefore reduced by a factor of 10 by taking beta blocker treatment. The same patient population was followed up for an average of 2 years after surgery and the benefit of bisoprolol treatment was found to be maintained long term (12% cardiac events with bisoprolol compared to 32% on placebo, p=0.025) [19]. The benefits of beta blocker treatment (pre-operative IV Atenolol) had already been shown [20] in patients with coronary artery disease or at risk of coronary artery disease without documented ischaemia who were undergoing **non-cardiac surgery** (vascular in 40% of cases). The benefit in this situation was a significant reduction in total mortality due to a reduction in cardiac deaths in the beta blocker group which was seen within 6-8 months after surgery and was maintained for 2 years. Work derived from the bisoprolol study showed the limited utility of dobutamine echocardiography in patients who had only one or two clinical risk factors (table 2) and pre-operative beta blocker treatment [21]. The serious event rate in this group was low (0.8%) and the test did not provide significant prognostic information. On the other hand, patients with widespread ischaemia had a high event rate (36%). In order to provide more information about these findings the DECREASE II trial published by the same group in 2006 was a randomised prospective trial of the role of dobutamine echocardiography compared to systematic beta blocker treatment in 770 patients with 1 or 2 clinical risk factors, awaiting vascular surgery [22]. The patients were randomised between dobutamine echocardiography and systematic beta blocker. Revascularisation was considered in patients in the stress echo group who had widespread ischaemia (involving at least 5 segments). Beta blocker therapy was started if limited ischaemia was found, in order to avoid the HR exceeding the ischaemic threshold. Finally, patients with a negative test underwent surgery with no additional treatment. This study showed that no strategy was superior to any other for death or myocardial infarction (1.8 versus 2.3%). The authors concluded that systematic beta blocker therapy could be used to substitute for a functional investigation strategy in this patient group, particularly as in their study, performing stress echocardiography delayed surgery by approximately 3 weeks. Several comments may be made about this conclusion. Firstly the investigation delay varies greatly between centres: this must be kept short in order not to expose the patient to excessive risk. It is also important to consider the beta blocker treatment protocol which began orally before surgery but which notably was then switched to intravenous, with hourly monitoring of the HR in order that it did not exceed 60 to 65/min. This was undoubtedly a crucial factor in obtaining the observed efficacy although requires very close interval monitoring. Finally, it is important to remember that the finding of ischaemia on stress echocardiography reduces the patient's medium term prognosis, independently of surgery. This must be considered in the debate.

The second treatment which appears to be effective in reducing the coronary risk is **statins.** An initial retrospective study [23], in 2816 patients undergoing vascular surgery showed statins to impact in hospital mortality. Fewer people who were taking a statin died from a cardiovascular cause than controls (8% versus 25%, p<0.001), with a relative risk of operative mortality of 0.22 in patients taking the statin compared to those who were not. The same authors demonstrated the statins to be safe, with no increase in the risk of myopathy [24]. Their potential utility was confirmed by another group [25] in a retrospective study in patients undergoing non-cardiac surgery. This study showed a significant reduction in total hospital mortality in the patient group receiving a statin, which was greater in patients at high cardiac risk. Prospective, randomised studies are now required to validate the utility of statin treatment in reducing perioperative coronary risk.

Coronary revascularisation prior to surgery has not yet been shown to be useful in patients with coronary artery disease. In the CARP study [26], 510 patients at increased cardiovascular risk (clinical risk factors and/or ischaemia demonstrated on scintigraphy) with documented coronary artery disease were randomised into 2 groups: one group revascularised before surgery and one group which was not revascularised. The patients were awaiting vascular surgery (33% for abdominal aortic aneurysm, 67% for lower limb arterial disease). Coronary revascularisation was performed on a non-randomised basis by bypass in 41% of cases and by angio-

Consensus indications for transthoracic Doppler echocardiography in coronary risk stratification before non-cardiac surgery

Class I
In known ischaemic heart disease without recent echocardiographical assessment (less than one year) or associated with recent deterioration in clinical state.

Class II
In suspected heart disease as a pre-operative investigation regardless of intended surgery.

Class III
In uncomplicated stable ischaemic heart disease.

Consensus indications for stress echocardiography in coronary risk stratification before non-cardiac surgery *

Class I
— Assessment of patients at intermediate risk from clinical and ECG findings when the exercise test is:
impossible to perform (poor functional capacity, arterial disease, age, etc…)
uninterpretable: pre-excitation, pacemaker, LBBB, LVH
submaximal and therefore non-contributory
equivocal
— Assessment of a coronary artery disease patient with recent change in symptoms.
— Assessment of a known coronary artery disease patient who has not had a functional test within one year.
— Assessment of a patient who has undergone coronary angioplasty within 8 months with no functional test since that time.

Class II
— First line for intermediary risk after analysis of clinical and electrocardiographical data.
— Assessment of a patient with documented coronary artery lesions on recent coronary angiography, treated medically

Class III
— Systematic assessment of asymptomatic low risk people.

*: exercise echocardiography if the patient can perform exercise, if not: dobutamine stress echocardiography

plasty in 59% of cases. Clinical features, beta blocker and statin use were identical in the two groups. The mortality rate after a mean follow-up period of 2.7 years was 22% in the revascularised group and 23% in the non-revascularised group (p=0.92). The MI rate diagnosed from a rise in troponin in the 30 days following surgery was 12% in the revascularised group and 14% in the non-revascularised group (p=0.37). This study therefore appears to indicate that coronary revascularisation before major vascular surgery is not beneficial in the short or long term in stable coronary artery disease patients. This result, however, should be interpreted with caution as this was a study on an average population size with probable recruitment bias: only 62% of the patients had had scintigraphy and only 74% were actually at increased cardiovascular risk. Back and co-workers [27] had already shown that recent coronary revascularisation (<5 years) by bypass or angioplasty had no effect on the long term survival (5 years) in patients at high cardiovascular risk awaiting vascular surgery. This criterion however was only a secondary end point for the study, limiting its value.

Conclusion: Stress echocardiography is a useful examination to stratify coronary risk in patients with clinical risk factors before surgery and has immediate and late prognostic implications. It must include both the positive diagnosis of myocardial ischaemia (development of WM abnormality) and also its extent and the HR threshold at which the ischaemia occurs (table 4). Beta blocker (and possibly statin) treatment is useful in reducing morbidity and mortality before vascular surgery. This must be widely used and can avoid the need for functional tests when the procedure is to be performed shortly and the patient is not at high risk. Coronary revascularisation before surgery has not however been proven to be effective and therefore should be considered on an individual case basis.

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