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


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.

References


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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 longitudinal 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 LVH 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/m² to 134 g/m² in men and 100 g/m² to 125 g/m² in women depending on the publication [11]. The recent ASE recommendations [9] propose a value of 115 g/m² in men and 95 g/m² in women. This approach underestimates the prevalence of LVH in obese people and a height indexation has been validated anatomically. It must be performed following precise recommendations [7].

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 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.

Studying the midwall fractional shortening (mFS) can also identify unrecognised contractility disorders (equation 2)

\[
\text{Equation 1: Calculation of meridional end-systolic stress (mESS)}
\]

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

\[
\text{LVIDs: end-systolic left ventricular internal diameter; PWTs: end-systolic posterior wall thickness; SBP: systolic blood pressure.}
\]

\[
\text{Equation 2: Calculation of midwall fractional shortening (mFS)}
\]

\[
\text{mFS} = \left[\frac{((\text{LVIDd}+(\text{PWTd}/2)+\text{IVSd}/2)-(\text{LVIDd}+(\text{PWTd}/2)+\text{IVSd}/2))^3}{(\text{LVIDd}+(\text{PWTd}/2)+\text{IVSd}/2)^{100}}\right]
\]

\[
\text{LVIDd: end-diastolic left ventricular internal diameter; PWTd: posterior wall thickness at end diastole.}
\]

[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. Filling pressures are assessed from conventional parameters: generally reverse transmural 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.

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Calculations</th>
<th>Technical comments</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVIDd, LVIDs</td>
<td>Parasternal M-mode; failing that sub-costal M-mode; failing that parasternal 2D</td>
<td>LV dilated if: LVIDd &gt;31 mm/m² (men) LVIDd &gt;32 mm/m² (women)</td>
<td></td>
</tr>
<tr>
<td>IVSd, PWTd</td>
<td>Parasternal M-mode; failing that sub-costal M-mode; failing that parasternal 2D</td>
<td>PWTd &gt;11 mm generally indicates LVH</td>
<td></td>
</tr>
<tr>
<td>(IVSd+PWTd)/LVIDd</td>
<td>Penn or ASE equations</td>
<td>Prognostic value</td>
<td></td>
</tr>
<tr>
<td>LV mass/m²</td>
<td>Eccentric if &lt;0.42-0.44; Concentric if ≥0.42-0.44</td>
<td>Prognostic value</td>
<td></td>
</tr>
<tr>
<td>eFS</td>
<td>Normal: 27-45% (if concentric geometry, eFS normal &gt;40%) If eFS &lt;27%, take ESS into account in interpreting</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| 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 dietic 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].

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, 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.

**References**


[9] Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Perlilka PP, Picard MH, Roman MJ, Seward J, Shanevise JS, Solomon SD, Spencer KT, Sutton MS, Stewart WJ; Chamber Quantification Writing Group; American Society of Echocardiography's Guidelines and Standards Committee; European Association of Echocardiography. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr 2005;18:1440-63.


[15] Koren MJ, Devereux RB, Casale PN, Savage DD, Laragh JH. Relation of left ventricular mass and geometry to morbidity and mortality in uncomplicated essential hypertension. Ann Intern Med 1991;114:345-52.


Table 2  Hypertension: optional parameters to be recorded.

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Calculations</th>
<th>Technical comments</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP</td>
<td></td>
<td>During the investigation (automatic measurement)</td>
<td></td>
</tr>
<tr>
<td>PWTs</td>
<td></td>
<td>Height in “meters”</td>
<td>Prognostic value</td>
</tr>
<tr>
<td></td>
<td>LV mass/height²,7</td>
<td>Height in “meters”</td>
<td>Thresholds 51 g/m²,7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Suitable in both sexes and in all body types (obese ++)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>mFS</td>
<td>Calculated from the LVIDd, LVIDs, IVSd, PWTd</td>
<td>Normal values 15-23%</td>
</tr>
<tr>
<td></td>
<td>End systolic stress</td>
<td>Requires SBP, PWTs, LVIDs</td>
<td>Prognostic value</td>
</tr>
<tr>
<td></td>
<td></td>
<td>End systolic stress requires SBP, PWTs, LVIDs</td>
<td>Aid to interpreting eFS &lt;27%</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Aid to interpreting mFS &lt;15%</td>
</tr>
<tr>
<td></td>
<td>Ea</td>
<td>Septal and lateral site</td>
<td>Prognostic value</td>
</tr>
<tr>
<td></td>
<td>Vp</td>
<td>Color M-mode</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ap duration</td>
<td>Pulsed Doppler</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Em/Ea</td>
<td>Necessary to assess LV filling pressures</td>
<td>Filling pressure is raised if:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>When Em&gt;Am</td>
<td>Em/Ea&gt;15</td>
</tr>
<tr>
<td></td>
<td></td>
<td>When Em&lt;Am and LVH</td>
<td>Em/Vp&gt;2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Duration (Ap-Am)</td>
<td>Duration (Ap – Am) &gt;20ms</td>
</tr>
<tr>
<td>Testing for</td>
<td>Continuous wave Doppler,</td>
<td>Consider in patients who have become symptomatic on treatment</td>
<td></td>
</tr>
<tr>
<td>coarctation</td>
<td>suprasternal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV outflow tract</td>
<td>Continuous wave Doppler,</td>
<td></td>
<td></td>
</tr>
<tr>
<td>obstruction</td>
<td>apex</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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**


**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 nonlinear, 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.**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Threshold</th>
<th>Comments</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximal aortic velocity</td>
<td>Severe AS &gt;4 m/s</td>
<td>Asymptomatic patients</td>
<td>Prognostic value</td>
</tr>
<tr>
<td>Mean LV-Aorta gradient (rest)</td>
<td>Severe AS: &gt;40 mmHg (ACC/AHA)</td>
<td>Several views</td>
<td>Depends on output. Risk of...</td>
</tr>
<tr>
<td></td>
<td>Severe AS: &gt;50 mmHg (ESC; SFC)</td>
<td>(right parasternal ++)</td>
<td>if all views are not analyzed.</td>
</tr>
<tr>
<td>Aortic surface area continuity</td>
<td>Severe AS &lt;1 cm² (ACC/AHA, ESC)</td>
<td>BSA indexation debatable: of use for...</td>
<td>Less dependent on output. Invalid</td>
</tr>
<tr>
<td>equation</td>
<td>Severe AS &lt;0.5 cm²/m² BSA (SFC)</td>
<td>for extreme body morphotypes</td>
<td>in outflow tract obstruction</td>
</tr>
<tr>
<td></td>
<td>Severe AS &lt;0.6 cm²/m² BSA (ACC/AHA, ESC)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

AS: aortic stenosis; BSA: body surface area; LV: left ventricle
### Table 2  Aortic stenosis: optional parameters.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Threshold</th>
<th>Comments</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exercise</td>
<td>Mean gradient &gt;18 mmHg</td>
<td>Asymptomatic patients</td>
<td>Prognostic value to be confirmed (only 1 study)</td>
</tr>
<tr>
<td>Low dose dobutamine</td>
<td>Mean gradient &gt;30 mmHg&lt;br&gt;Surface area &lt;1.2 cm²</td>
<td>Patients with calcified valve, LVEF &lt;40% and mean basal gradient LV- aorta &lt;30 mmHg</td>
<td>Diagnostic and prognostic value</td>
</tr>
<tr>
<td>Surface area by planimetry</td>
<td>Severe AS &lt;1 cm² (ACC/AHA, ESC)&lt;br&gt;Severe AS &lt;0.5 cm²/m² BSA (SFC)&lt;br&gt;Severe AS &lt;0.6 cm²/m² BSA (ACC/AHA, ESC)</td>
<td>Transthoracic or transoesophageal view</td>
<td>Limited to moderately calcified orifices when continuity equation invalid</td>
</tr>
<tr>
<td>Patency index</td>
<td>Severe AS &lt;25%</td>
<td>Simple</td>
<td>Relatively non-specific</td>
</tr>
<tr>
<td>Valve resistance</td>
<td>Severe AS &gt;300 dynes.s.cm⁻⁵</td>
<td></td>
<td>Poor</td>
</tr>
</tbody>
</table>

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

### Table 3  Mitral stenosis: obligatory parameters to record.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Threshold</th>
<th>Comment</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean LA-LV gradient (mmHg) (rest)</td>
<td>Moderate MS: &lt;5 mmHg&lt;br&gt;Severe MS: &gt;10 mmHg</td>
<td>Calculate mean measurements (AF ++)&lt;br&gt;Mediocre when performed alone (depends on output)</td>
<td></td>
</tr>
<tr>
<td>2D surface area</td>
<td>Severe MS &lt;1.5 cm²&lt;br&gt; &lt;1 cm²/m² BSA</td>
<td>Measure the smaller orifice (funnel tip)&lt;br&gt;Anatomical surface area&lt;br&gt;Gold standard</td>
<td></td>
</tr>
<tr>
<td>PHT surface area</td>
<td>Severe MS &lt;1.5 cm²&lt;br&gt; &lt;1 cm²/m² BSA</td>
<td>Easy, quick&lt;br&gt;Poor when performed alone&lt;br&gt;Multiple causes of error</td>
<td></td>
</tr>
</tbody>
</table>

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.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Threshold</th>
<th>Comment</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean LA-LV gradient on exercise</td>
<td>&gt;15 mmHg</td>
<td>Difficult if rapid heart rate&lt;br&gt;Useful when basal evaluation difficult. Threshold value poorly documented prognostically</td>
<td></td>
</tr>
<tr>
<td>Systolic PAP on exercise</td>
<td>&gt;60 mmHg</td>
<td>Threshold value poorly documented prognostically</td>
<td></td>
</tr>
<tr>
<td>Continuity surface area</td>
<td>Severe MS &lt;1.5 cm²&lt;br&gt; &lt;1 cm²/m² BSA</td>
<td>Pulmonary view rarely used Functional surface&lt;br&gt; &lt; anatomical surface area</td>
<td>Many causes of error&lt;br&gt;Same limitations as 2D (echogenicity) Specific equipment</td>
</tr>
<tr>
<td>3D surface area</td>
<td>Severe MS &lt;1.5 cm²&lt;br&gt; &lt;1 cm²/m² BSA</td>
<td>Appears to be more reproducible than 2D with inexperienced observers</td>
<td></td>
</tr>
<tr>
<td>PISA method (M-mode or 2D)</td>
<td>Severe MS &lt;1.5 cm²&lt;br&gt; &lt;1 cm²/m² BSA</td>
<td>Angle generally between 100° and 130°&lt;br&gt;Precise angle determination aleatory</td>
<td></td>
</tr>
</tbody>
</table>

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 transeosophageal echocardiography is performed although transthoracic approach is usually sufficient.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Mild tricuspid regurgitation</th>
<th>Severe tricuspid regurgitation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Semi-quantitative</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>. vena contracta diameter</td>
<td>absent</td>
<td>&gt;7 mm</td>
</tr>
<tr>
<td>. systolic reversal of SHVF</td>
<td></td>
<td>present</td>
</tr>
<tr>
<td><strong>Quantitative</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>. effective regurgitant orifice area incompetence</td>
<td>&lt;20 mm²</td>
<td>&gt;40 mm²</td>
</tr>
</tbody>
</table>

SHVF: suprahepatic venous flow

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

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.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regurgitant fraction</td>
<td>Long, tedious</td>
</tr>
<tr>
<td></td>
<td>Causes of error (mitral flow rate ++)</td>
</tr>
<tr>
<td>Aortic output</td>
<td>Limited in LV dysfunction LV, AF...</td>
</tr>
</tbody>
</table>

AF: atrial fibrillation; LV: left ventricular

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

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Moderate aortic regurgitation</th>
<th>Severe aortic regurgitation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Semi-quantitative</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>vena contracta diameter</td>
<td>&lt;3 mm</td>
<td>&gt;6 mm</td>
</tr>
<tr>
<td>aortic isthmus end-diastolic velocity</td>
<td>absent or &lt;10 cm/s</td>
<td>&gt;20 cm/s</td>
</tr>
<tr>
<td>aortic output</td>
<td>&lt;6 L/min</td>
<td>&gt;10 L/min</td>
</tr>
<tr>
<td>pressure half time</td>
<td>&gt;500 ms</td>
<td>&lt;300 ms</td>
</tr>
<tr>
<td><strong>Quantitative</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>effective regurgitant orifice area</td>
<td>&lt;10 mm²</td>
<td>&gt;30 mm²</td>
</tr>
<tr>
<td>regurgitated volume/beat</td>
<td>&lt;30 ml</td>
<td>&gt;60 ml</td>
</tr>
<tr>
<td>regurgitation fraction</td>
<td>&lt;30%</td>
<td>&gt;50%</td>
</tr>
</tbody>
</table>

### Table 9  Mitral regurgitation: obligatory parameters to record.

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

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

### Table 10  Mitral regurgitation: optional parameters to record.

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

VTI: velocity time integral; PISA: proximal isovelocity surface area
The risks of surgery and

Mild mitral regurgitation


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

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Mild mitral regurgitation</th>
<th>Severe mitral regurgitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Semi-quantitative</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vena contracta diameter</td>
<td>&lt;3 mm</td>
<td>&gt;7 mm</td>
</tr>
<tr>
<td>Mitral VTI/Aortic VTI</td>
<td>&lt;1</td>
<td>1.4</td>
</tr>
<tr>
<td>Pan- or mid-systolic reversal of PVF</td>
<td>absent</td>
<td>present</td>
</tr>
<tr>
<td>Quantitative</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Effective regurgitant orifice area</td>
<td>&lt;20 mm²</td>
<td>&gt;40 mm²</td>
</tr>
<tr>
<td>Regurgitant volume/beat</td>
<td>&lt;30 ml</td>
<td>&gt;60 ml</td>
</tr>
<tr>
<td>Regurgitation fraction</td>
<td>&lt;30%</td>
<td>&gt;50%</td>
</tr>
</tbody>
</table>

VTL: 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 cm² 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 >4 m/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 cm² (or indexed aortic valve area <0.6 or 0.5 cm²/m² 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 cm²/year) from patients in whom the stenosis progresses slowly (reduction in aortic surface area of less than 0.1 cm²/year). It is also used to monitor patients with well tolerated severe aortic stenosis.

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
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/m²) 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].
Consensus indications for transthoracic Doppler echocardiography in organic mitral regurgitation

<table>
<thead>
<tr>
<th>Class</th>
<th>Indications</th>
</tr>
</thead>
</table>
| Class I | - Initial assessment of clinically diagnosed or suspected mitral regurgitation.  
- Change in symptoms or clinical examination in patients with previously diagnosed mitral regurgitation.  
- Biannual 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.  

Consensus indications for transoesophageal echocardiography in mitral stenosis

<table>
<thead>
<tr>
<th>Class</th>
<th>Indications</th>
</tr>
</thead>
</table>
| 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 transoesophageal 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 left heart valve diseases: transoesophageal echocardiography

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

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-

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.
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 ventricu-

**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.
- 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 II**
- 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 III**
- Initial assessment of aortic stenosis that was completely assessed by transthoracic Doppler echocardiography.

**Consensus indications for stress echocardiography in organic left heart valve diseases**

**Class I**
- 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 occurring 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 (clinical and laboratory recovery: the frequency of subsequent investigations should be established on an individual basis).

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).

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).

References


Endocarditis on native valves

The diagnostic criteria for infective endocarditis have recently been modified [28] and Doppler echocardiography findings have been included among 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.


**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 intraprothetic 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 (Hatile 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 cm².
- 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. Intraprothetic 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. Para-prosthetic regurgitations are relatively common during the immediate post-operative period, are usually minimal and frequently disappear spontaneously over a few weeks.
- 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 cm², 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 cm²/m². Mismatch is present if it is <0.85 cm²/m² [13] and is severe if the indexed surface area is <0.6 cm²/m². 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 [14] and 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.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal value</th>
<th>Technical comments</th>
<th>Diagnostic value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean gradient</td>
<td>5±3 mmHg</td>
<td>Variable with rate, rhythm, output</td>
<td>+++</td>
</tr>
<tr>
<td></td>
<td>Pathological if &gt;10 mmHg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pressure Half Time (PHT)</td>
<td>70-100 msec Pathological if &gt;150 msec</td>
<td>Do not extrapolate for surface area</td>
<td>+++</td>
</tr>
<tr>
<td>Regurgitations</td>
<td>– Intraprosthetic – Paraprosthetic</td>
<td>TOE &gt; TTE</td>
<td>++</td>
</tr>
</tbody>
</table>

### Table 2 Aortic prostheses: essential parameters to record.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal value</th>
<th>Technical comments</th>
<th>Diagnostic value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean gradient</td>
<td>&lt;30 mmHg</td>
<td>Variable with type and size ++ of the prosthesis</td>
<td>+++</td>
</tr>
<tr>
<td>Permeability index</td>
<td>0.23-0.66</td>
<td>Use the same cycle for 2 flows in AF by continuous Doppler</td>
<td>+++</td>
</tr>
<tr>
<td>Prosthesis surface area</td>
<td>0.9-2.2 cm²</td>
<td>If sinus rhythm</td>
<td>++</td>
</tr>
<tr>
<td>Indexed prosthesis surface area</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Regurgitations</td>
<td>– Intraprosthetic – Paraprosthetic</td>
<td>TTE &gt; TOE</td>
<td>++</td>
</tr>
</tbody>
</table>

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-
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.

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.

**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
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 (occurrence 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

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

Class I
- Transoesophageal echocardiography if transthoracic Doppler echocardiography is normal in a clinically stable patient

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

Class III
- Annual control from year 5 in the absence of any new, clinical or biological event.

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

Class I
- Control every 2 years, in the absence of any new clinical or biological event.

Class II
- Initial control at the end of the first year in the absence of any new clinical biological event.

Class III
- Annual control from year 5 in the absence of any new, clinical or biological event.
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 transthoracic 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

### 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) |

### Consensus indications for transthoracic Doppler echocardiography in structural valve deterioration of bioprostheses

| Class I | — Diagnosis of SVD of bioprostheses suspected from the occurrence 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. |
| — 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 annuli [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
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).

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.

Class IV
- Repeat investigation in the absence of a change in clinical situation or haemodynamic parameters assessed by transthoracic Doppler echocardiography, particularly to exclude infectious endocarditis.

References


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 akinesic 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 suprarehepatic 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 suprarehepatic 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

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

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

ASE: American Society of Echocardiography; WMSI: Wall motion score index.
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 2 Chronic ischaemic heart disease: other essential parameters to record.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Thresholds/quantification</th>
<th>Technical comments</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral regurgitation (MR)</td>
<td>State presence or absence of MR, even if mild and mechanism</td>
<td>Colour Doppler, apical approach</td>
<td>Adverse prognostic value in the absence of pre-existing valve disease</td>
</tr>
<tr>
<td></td>
<td>Threshold for significant MR: ERO &gt;20 mm² [17]</td>
<td>Quantification by PISA or Doppler volumetric techniques</td>
<td></td>
</tr>
<tr>
<td>Investigation for thrombus</td>
<td>State dimensions and features (sessile or pedunculated)</td>
<td>Harmonic imaging Apical views showing the apex left ventricular outflow tract output</td>
<td>False positives and negatives diagnosis possible</td>
</tr>
<tr>
<td>Cardiac output</td>
<td>Low output &lt;2.2L/min/m²</td>
<td></td>
<td>Haemodynamic consequence</td>
</tr>
<tr>
<td>Pericardium</td>
<td>Topography and volume if effusion</td>
<td>Multiple views</td>
<td>Signs of intolerance (cavity collapse, respiratory flow variations)</td>
</tr>
</tbody>
</table>

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].

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

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.

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

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Thresholds/quantification</th>
<th>Technical comments</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RV kinetics and function</td>
<td>RV wall kinetics</td>
<td>Parasternal (short axis) apical (4C) and subcostal views</td>
<td></td>
</tr>
<tr>
<td>Pulmonary ejection flow</td>
<td>RV dilatation (RV/LV diameter ratio &gt;0.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonary regurgitation Doppler flow</td>
<td>Pulmonary output Acceleration time Calculation of resistances [31]</td>
<td>Closed Doppler and Pedoff probe</td>
<td></td>
</tr>
<tr>
<td>Tricuspid regurgitation Doppler flow</td>
<td>PHT and Vmin/Vmax of PR PAPm and PAPd</td>
<td>Coupled Doppler and Pedoff probe</td>
<td></td>
</tr>
<tr>
<td>Inferior vena cava</td>
<td>Diameter and respiratory variations</td>
<td>Multiple views and Pedoff probe</td>
<td></td>
</tr>
</tbody>
</table>

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; IVC: inferior vena cava.

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

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Thresholds/quantification</th>
<th>Technical comments</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Left ventricular morphology</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sphericity index [4]</td>
<td>L/l ratio [4]</td>
<td>Repeated assessment for remodelling (treatment evaluation protocols)</td>
<td>The lower this ratio the closer the LV appears as a sphere. (normal L/l ratio = 2)</td>
</tr>
<tr>
<td>Left ventricular opacification with contrast agent [10]</td>
<td>Harmonic imaging Specific settings for the contrast agent</td>
<td>Precise measurement of volumes and LVEF (treatment evaluation protocols)</td>
<td>Improved detection of the endocardium and measurement of volumes and LVEF [10]</td>
</tr>
<tr>
<td>Stress echocardiography</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dobutamine echocardiography</td>
<td>WMSI at different steps E/Ea PAPs</td>
<td>Investigation for myocardial viability involving at least 4 segments</td>
<td>Excellent predictive value for functional recovery (NYHA, LVEF) and prognosis No use for mitral regurgitation</td>
</tr>
<tr>
<td>Exercise echocardiography</td>
<td>Mitral regurgitation (ERO), RV E/Ea PAPs Area under the tent</td>
<td>Standard protocol, semi-seated on bicycle Zoom on PISA TR (Vmax)</td>
<td>Prognostic value (resting ERO &gt;20mm² and Δ exercise ERO &gt;13 mm² [20])</td>
</tr>
</tbody>
</table>

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.

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 mm²) is seen in almost 30% of cases. When mitral regurgitation is severe at
The dynamic nature of functional mitral regurgitation has prognostic implications, [18-20], the validated threshold being an increase of ≥13 mm² 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.

REFERENCES

[1] Lang R M, Bierig M, Devereux RB, et al. Recommendations for Chamber Quantification: A Report from the American Society of Echocardiography’s Guidelines and Standards Committee and the Chamber Quantification Writing Group, Developed in Concert with the European Association of Echocardiography, a Branch of the European Society of Cardiology. JASE 2005;18:1440-63.


[30] Ennezat PV, Marechaux S, Le Tourneau T et al. Myocardial asynchronism is a determinant of changes in functional mitral regur-
gitation severity during dynamic exercise in patients with chronic heart failure due to severe left ventricular systolic dysfunction. Eur Heart J. 2006;27:679-83.


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 ana-
lysis of pulmonary venous flow or left ventricular flow propaga-
tion during early filling (Vp) by colour M-mode during two-
dimensional Doppler. If a large shunt is present, the left ven-
tricular systolic and diastolic performance indices and
right ventricular filling 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

Mitrval 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 mus-
cles. It is associated with a poor prognosis and increased risk
of mortality and heart failure independently of other prognos-
tic factors [9]. It is often clinically silent and must be exami-
ned 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 mm² [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 ven-
tricle 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

Right ventricular myocardial infarction

Right ventricular extension occurs in 30 to 50% of cases of infe-
rior 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 seg-
mental wall motion or global right ventricular abnormalities
and right ventricular dilatation and dysfunction. Right Doppler
flow recording (pulmonary regurgitation) is useful for diagno-
sis and allows right ventricular filling pressures to be measured
[12].

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 diast-
tolic volume. Aneurysm predisposes to intraventricular throm-
bus formation. Some echocardiographical features of the
thrombus (pedunculated and mobile) are associated with
increased risk of arterial embolism [13].

True left ventricular aneurysm must be distinguished from
false ventricular aneurysm secondary to myocardial rupture
into partitioned myocardium. The false aneurysm has a nar-
row 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. Echocar-
diography 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.

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

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.

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

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.
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 remodeling [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
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-

### Table 6  Acute coronary syndromes: optional parameters to record.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Thresholds/quantification</th>
<th>Technical comments</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Segmental wall motion score index (WMSI)</td>
<td>Different segmental scales</td>
<td>Accuracy depends on number of analysable segments</td>
<td>Reflects global left ventricular function</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Increasing WMSI [1]</td>
<td>Prognostic index</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Normal WMSI = 1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Decreasing WMSI used in the TRACE study [14] 9 segments; WMSI normal = 2</td>
<td>Prognostic value of repeated measurement of WMSI [14]</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(WMSI ≤ 1.2 equals LVEF ≤ 35%).</td>
<td></td>
</tr>
<tr>
<td>Colour M-mode on mitral flow</td>
<td>E/Vp≥1.5 predictive of in-hospital heart failure</td>
<td>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</td>
<td>Limited reproducibility of measurement</td>
</tr>
<tr>
<td></td>
<td>Useful to identify pseudonormal mitral flow</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LA volume</td>
<td>Biplane area-length method</td>
<td>Normal value 22±6 mL/m²</td>
<td>Adverse prognostic value if LA volume &gt;32 mL/m² [19]</td>
</tr>
<tr>
<td>Stress echocardiography (dobutamine &gt; effort) (dipyridamole not used in France in this situation)</td>
<td>Low dose WMSI (5-15mcg/Kg/min)) for contractile reserve and peak WMSI for ischaemia (and biphasic response)</td>
<td>Possible from day 3 post MI if no complications and beta blockers omitted on the same day</td>
<td>No threshold value</td>
</tr>
</tbody>
</table>
| 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.

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

LAD: left anterior descending artery
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

Contraction reserve (CR) is useful to detect viable myocardium by the infarction but viable with contractile 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.

References

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 dobutamine-atropine stress echocardiography early in unstable angina. Eur Heart J 2000:1063-71.
rate at which ischaemia develops (ischaemic threshold <70% of MPHR) 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.

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 endarterectomy
  - Head and neck surgery
  - Orthopaedic surgery
  - Thoracic or intraperitoneal surgery
  - Prostate surgery
- Low risk (<1%)
  - Endoscopies
  - Superficial surgery
  - Cataract surgery
  - Breast surgery.

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 Kertai 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 perioperative 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>
<thead>
<tr>
<th>Stage 1</th>
<th>Clinical risk assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>Intermediate</td>
</tr>
<tr>
<td>Intermediate</td>
<td>High</td>
</tr>
<tr>
<td></td>
<td>Postpone procedure + full cardiological assessment</td>
</tr>
<tr>
<td>Stage 2</td>
<td>Functional capacity (clinical evaluation +/- exercise test)</td>
</tr>
<tr>
<td>Good</td>
<td>Intermediate clinical risk (stage 1)</td>
</tr>
<tr>
<td>Poor</td>
<td>Low clinical risk (stage 1)</td>
</tr>
<tr>
<td>Low clinical risk (stage 1)</td>
<td>Intermediate clinical risk (stage 1)</td>
</tr>
<tr>
<td>Surgery</td>
<td>Low clinical risk (stage 1)</td>
</tr>
<tr>
<td>Stage 3</td>
<td>Level of risk of the intended surgical procedure</td>
</tr>
<tr>
<td>Low</td>
<td>Intermediate</td>
</tr>
<tr>
<td>Intermediate</td>
<td>2 options</td>
</tr>
<tr>
<td>High</td>
<td>Functional test</td>
</tr>
<tr>
<td>Surgery</td>
<td>+ beta blockers</td>
</tr>
<tr>
<td>Functional test</td>
<td>Early and/or extensive ischaemia</td>
</tr>
<tr>
<td>Negative test or limited ischaemia</td>
<td>Consider coronary angiography, or surgery on beta blockers.</td>
</tr>
</tbody>
</table>

Consider coronary angiography, or surgery on beta blockers.
Table 4  Echocardiographical parameters to be recorded during stress echo (exercise or dobutamine).

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

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-
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.

References


