Ultrasound in Medicine & Biology 000 (2024) 1–17

Contents lists available at ScienceDirect

Ultrasound in Medicine & Biology

journal homepage: www.elsevier.com/locate/ultrasmedbio

Review Article

WFUMB Guideline/Guidance on Liver Multiparametric Ultrasound: Part 1. Update to 2018 Guidelines on Liver Ultrasound Elastography

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ARTICLE INFO

Keywords:
Shear wave elastography
Vibration-controlled transient elastography
Acoustic radiation force impulse
Cirrhosis
Portal hypertension
Non-alcoholic fatty liver disease
Non-alcoholic steatohepatitis
Metabolic dysfunction-associated steatotic liver disease
Alcohol-related liver disease
Pediatrics

The World Federation for Ultrasound in Medicine and Biology (WFUMB) endorsed the development of this document on multiparametric ultrasound. Part 1 is an update to the WFUMB Liver Elastography Guidelines Update released in 2018 and provides new evidence on the role of ultrasound elastography in chronic liver disease. The recommendations in this update were made and graded using the Oxford classification, including level of evidence (LoE), grade of recommendation (GoR) and proportion of agreement (Oxford Centre for Evidence-Based Medicine [OCEBM] 2009). The guidelines are clinically oriented, and the role of shear wave elastography in both fibrosis staging and prognostication in different etiologies of liver disease is discussed, highlighting advantages and limitations. A comprehensive section is devoted to the assessment of portal hypertension, with specific recommendations for the interpretation of liver and spleen stiffness measurements in this setting.

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https://doi.org/10.1016/j.ultrasmedbio.2024.03.013
Received 24 January 2024; Revised 20 March 2024; Accepted 25 March 2024

Introduction

Shear wave elastography (SWE) has gained an important role in the diagnosis and management of patients with chronic liver disease (CLD). Liver stiffness assessment with SWE has increasingly been used not only for the non-invasive staging of liver fibrosis but also for evaluation of the risk of complications or the clinical outcome of CLD patients. Evidence from the literature has highlighted that SWE can be used to predict the presence of clinically significant portal hypertension (CSPH) and the risk of liver-related events (LREs) in patients with compensated advanced CLD (cACLD) [1,2]. Therefore, SWE can be considered a mature technique for the evaluation of patients with CLD.

New ultrasound (US)-based biomarkers that non-invasively quantify liver fat content are currently available [3–5]. Because of the steatotic liver disease "epidemic," their use in assessing the presence and severity of hepatic steatosis is attractive.

In 2021, the World Federation for Ultrasound in Medicine and Biology (WFUMB) released a position paper on liver fat quantification providing expert opinion [6]. Since then, several other studies have been published. However, confounding factors that may affect the US estimation of liver fat are inadequately understood, and a protocol for the acquisition of these parameters that mitigate the differences in values between observers or between algorithms from different manufacturers is lacking.

Therefore, the WFUMB leadership has promoted the development of a document on multiparametric US that includes both new evidence on the role of SWE in CLD and available data on the quantitative US evaluation of liver fat content.

The availability of US-based biomarkers for the evaluation of liver inflammation is of great interest, and research on their value and applicability in clinical practice is increasing. However, it is too early to include them in this document because the results obtained so far are still uncertain and the evidence is limited.

The steering committee designated by the WFUMB leadership invited experts from each ultrasound federation; they were chosen for their outstanding contributions in this field. Meetings were held online or in a hybrid mode. Ultrasound companies were not invited and did not participate in the development of the guidelines in any manner. The final consensus on the recommendations was reached during an online meeting.

The document is divided into two parts. The first part is a further update to the WFUMB liver elastography guidelines update released in 2018 [7]. The second part is a guidance on the role of the new ultrasound tools for liver fat quantification.

As in the previous update, for SWE the recommendations were made and graded using the Oxford classification, including level of evidence (LoE), grade of recommendation (GoR) and proportion of agreement (Oxford Centre for Evidence-Based Medicine [OCEBM] 2009). Online meetings were held for voting on the recommendations (for, against and abstain).

For guidance on the US biomarkers for quantification of liver fat content, the recommendations were based on published studies and experts' opinions but were not graded because the body of evidence remained low at the time this document was drafted.

Terminology

Acoustic radiation force impulse (ARFI) is a special ultrasound pulse, often called a push pulse, that applies focused high energy to create tissue compression (strain) and generate shear waves perpendicular to the push pulse. Note: ARFI generates the shear waves, but B-mode imaging tracks and measures the shear waves.

Shear wave elastography (SWE) describes any technique that generates shear waves and measures shear wave speed. This includes VCTE, ARFI techniques and magnetic resonance elastography.

Vibration controlled transient elastography (VCTE) uses an external mechanical push to the skin by means of a controlled vibration that generates shear waves.

Acoustic radiation force impulse shear wave elastography (ARFI-SWE) describes the techniques that use ARFI to generate shear waves in tissues. This includes both point SWE (pSWE) and 2D-SWE.

Steatotic liver disease (SLD). This umbrella term covers a range of diseases manifesting as increased hepatic steatosis (defined as ≥5% of hepatocytes having steatosis on a histological specimen). This covers metabolic dysfunction-associated steatotic liver disease (MASLD), alcohol-related liver disease (ALD), the presence of both risk factors (MetALD) and some less common causes of hepatic steatosis (*e.g.,* genetic disease and drug-induced steatosis).

Non-alcoholic fatty liver disease (NAFLD). This older term describes a condition with increased hepatic steatosis in the absence of an alternative cause such as excessive alcohol consumption and drugs. The diagnosis also requires the exclusion of other chronic liver diseases such as chronic viral hepatitis and autoimmune hepatitis.

Non-alcoholic steatohepatitis (NASH). This subtype of NAFLD is characterized by the presence of hepatic steatosis, lobular inflammation, hepatocyte ballooning and varying degrees of hepatic fibrosis.

Metabolic dysfunction-associated fatty liver disease (MAFLD). This term, proposed by Eslam and colleagues in 2020, replaces the term NAFLD [8]. Apart from describing metabolic dysfunction as the cause of MAFLD, the definition also requires the presence of type 2 diabetes, overweight or obesity or two other metabolic risk factors. In contrast, MAFLD can co-exist with other chronic liver diseases.

Metabolic dysfunction-associated steatotic liver disease (MASLD). After the initial MAFLD proposal, the international community conducted a four-round Delphi process to discuss the nomenclature and definition. In the end, both "alcoholic" and "fatty" were deemed stigmatizing and were removed from the terminology. Unlike the MAFLD definition, the diagnosis of MASLD requires only the presence of one or more metabolic risk factors. Again, MASLD can co-exist with other chronic liver diseases. However, although MAFLD can co-exist with ALD, the MASLD definition places MASLD and ALD into different categories.

Metabolic dysfunction-associated steatohepatitis (MASH). This subtype of MASLD is characterized by the presence of hepatic steatosis, lobular inflammation, hepatocyte ballooning and varying degrees of hepatic fibrosis.

Basic principles and protocol for liver stiffness measurement acquisition

Basics

Elastography allows assessment of the biomechanical properties of the tissue and can be regarded as virtual palpation. Under a stress, stiffer tissues exhibit less axial displacement and a higher speed of transverse displacement, namely, shear wave propagation. Shear waves can be generated by applying a mechanical stress externally to the body or by the push-pulse (ARFI) of the US beam directly into the body. The term *shear wave elastography* refers to the techniques based on both types of stress, that is, VCTE and ARFI-based techniques. With the latter, the assessment of shear wave speed/stiffness is made either at one point (approximately 1 cc) as in pSWE or is made using several ARFI lines where it is possible to obtain quantitative color-coded images of the elasticity, as in 2D-SWE. The basic principles of SWE have been fully described elsewhere [7,9,10].

Protocol for acquisition of liver stiffness measurements

To ensure the best possible estimate of liver stiffness measurement (LSM), a protocol for acquisitions has been recommended in the WFUMB

Table 1
Recommended protocol for the acquisition of reliable liver stiffness measurements

1	Fast for 4 h before the examination.
2	Rest for at least 10 min before the examination.
3	Place in supine or slight left lateral position (not >30°) with the right forearm held behind the head and the arm in maximum abduction (180° from the resting position) to widen the intercostal space.
4	Take measurements with an intercostal approach at the location with the best acoustical window.
5	Adequate B-mode liver imaging, without shadowing caused by the lung or ribs, is a prerequisite for the ARFI-SWE techniques, that is, pSWE and 2D-SWE, as shear waves are tracked with B-mode.
6	Transducer should be perpendicular to the liver capsule.
7	The ROI should be parallel to the liver capsule.
8	Measurement should be taken 15–20 mm below liver capsule to avoid reverberation artifact with pSWE.
9	With 2D-SWE, the size of the ROI should be at least 10 mm.
10	The 2D-SWE field of view can be positioned closer to the liver capsule if reverberation artifacts are avoided; however, the ROI, that is, the measurement box, should be positioned 15–20 mm below the liver capsule.
11	In most US systems, the maximum ARFI push-pulse is at 4–4.5 cm from the transducer, which is the optimal location for obtaining measurements. In most US systems, the ARFI push-pulse is attenuated by 6–7 cm, limiting adequate shear wave generation.
12	Placement of the ROIs must avoid large blood vessels, bile ducts and masses.
13	Measurements should be taken at neutral breathing during a breath-hold.
14	For VCTE, the appropriate probe should be selected based on patient's body habitus.
15	Measurements should be taken in independent images, all obtained in the same location.
16	For each acquisition with 2D-SWE, the coefficient of variation, namely, SD/mean, should be <0.25 for stiffness values between 8.8 and 11.9 kPa and <0.10 for stiffness ≥12.0 kPa.
17	For VCTE, 10 acquisitions should be obtained.
18	For pSWE, 5–10 acquisitions are recommended.
19	For 2D-SWE, 3–5 measurements should be obtained.
29	For all SWE techniques, that is, VCTE and ARFI-SWE, the result should be expressed as the median value of the acquisitions together with the IQR/M.
21	The IQR/M should be used as a measure of the quality of the data set.
22	For kPa measurements, the IQR/M should be ≤30%, and for m/s measurements, it should be ≤15% for an accurate data set.
23	Results can be reported in m/s or in kPa.

ARFI, acoustic radiation force impulse; IQR/M, interquartile range/median; pSWE, point shear wave elastography; ROI, region of interest; SWE, shear wave elastography; US, ultrasound; VCTE, vibration-controlled transient elastography.

2018 update [7]. This protocol is now updated based on the current literature and is reported in Table 1.

For pSWE techniques, studies have indicated that a reliable LSM can be obtained using the median value of only five acquisitions with an interquartile range/median (IQR/M) ≤30% (for measurements in kPa) [11–14]. For 2D-SWE, studies have reported that a minimum of three individual acquisitions is sufficient to compute a reliable LSM [15-17]. With liver biopsy as reference, it has been found that there is no difference in diagnostic accuracy between reporting the mean of five acquisitions and reporting the mean of three acquisitions [18]. However, for beginners, it is preferable to perform 10 acquisitions with pSWE and 5 with 2D-SWE and to decrease the number of acquisitions when the operator's expertise is improved [19].

With real-time 2D-SWE, improved accuracy has been observed when the distribution of the color-coded elasticity signals in the measurement box is homogeneous and consistent. Artifacts can also be identified on the color map and avoided. Several parameters have been proposed to evaluate the homogeneity [15,18,20].

A study in a large series of patients with CLD who underwent liver biopsy reported that, for LSM with 2D-SWE ≥8.8 kPa, the quality criterion for each single LSM is the coefficient of variation (CV), namely, standard deviation (SD)/mean [15]. New criteria were derived to define a reliable 2D-SWE measurement: for each acquisition, the CV should be <0.25 for LSMs between 8.8 and 11.9 kPa and <0.10 for LSMs ≥12.0 kPa. Below 8.8 kPa, the reliability of 2D-SWE measurement was not affected by the CV. The following workflow based on two steps was suggested: (i) define a reliable LSM; (ii) perform three reliable LSMs. When compared with the study by Thiele et al. [18], their reliability criteria were found to be more discriminant, better separating reliable LSMs from those with very poor accuracy that should not be used for the evaluation of liver fibrosis in clinical practice.

An increase in LSMs has been reported after intense physical exercise; therefore, at least 10 min of rest is recommended [21–23]. Of note, LSMs obtained in the left lateral position at 90° are significantly higher than those obtained in the supine position [22].

One study found that artificial intelligence (AI) might significantly improve the accuracy of 2D-SWE; however, this finding lacks further validation [24]. Currently, several manufacturers are using AI to help users in choosing the best area for positioning the region of interest (ROI), that is, the measurement box. AI assistance facilitates the stiffness measurement, but whether it also improves accuracy must still be verified.

In the pediatric population it could be challenging or even impossible to follow all the recommendations for a correct acquisition, particularly the breath-hold and fasting. In infants and young children who cannot follow breath-hold instructions, the Society of Radiologists in Ultrasound (SRU) consensus suggests acquiring a long cineloop when using real-time 2D-SWE, reviewing it and choosing the image with the most stable pattern for the LSM [10]. Performing LSM during shallow free breathing could be acceptable. With use of an ultrafast 2D-SWE technique it has been reported that LSMs are not affected by free breathing [25,26]. However, it must be considered that free breathing can generate movement artifacts that can affect the LSM. Of note, a study performed in adults reported that LSMs obtained in free breathing were consistently 20%–25% lower than those obtained with breath-hold [27]. Likewise, a study performed in children reported that with free breathing, LSMs were systematically lower with respect to those with breath-hold, with a mean difference of −11.1% [28]. Eating might increase liver stiffness. In newborns and infants, the LSM can be performed just before the next meal or at the start of eating. An epigastric approach can be used in some conditions, such as in the setting of liver transplant. A study that compared the epigastric and intercostal approaches to LSMs in children reported that the differences were not significant [26].

Interpretation of LSM results

As already highlighted in previous guidelines, the LSM must be interpreted considering the anamnesis of the patient, the etiology of liver disease and the clinical and laboratory data [7,29,30]. In fact, there are several factors that may lead to an LSM increase independently of liver fibrosis, and these are confounding factors when LSM is used for staging liver fibrosis. These factors have been fully detailed in previous guidelines [7,29,30]. Briefly, they include acute hepatitis, transaminase flares, obstructive cholestasis, infiltrative diseases, congestive heart disease and any other condition that increases the volume of blood in the liver, such as eating, intense physical exercise or holding the breath in deep inspiration. For ALD, ongoing drinking per se does not seem to increase LSM [31]. However, alcohol binges may increase LSM, which commonly decreases after reduction or cessation of alcohol intake [32].

The effect of inflammation on LSM can play an important role, particularly in some etiologies of CLD, such as autoimmune hepatitis, alcohol-related liver disease with alcohol-associated hepatitis and primary sclerosing cholangitis (PSC) [30]. In the latter, the presence of biliary obstructions also contributes to an increase in LSM.

Of note, it has been reported that inflammatory activity on histology significantly affects LSMs made using VCTE but not those made with

2D-SWE in MASLD [33]. Similar findings were observed in a large series of patients with mixed etiologies of CLD [34].

As for the effect of steatosis on LSM, there are conflicting results in the literature for all the SWE techniques. Studies reporting that the presence of severe steatosis led to an overestimation of liver fibrosis were performed with VCTE using only the M probe [35–37]. Other studies performed using the appropriate probe, that is, M or XL depending on the body mass index or skin-to-liver capsule distance, did not confirm these results [33,38,39]. The LSM overestimation found in previous studies might be explained by the fact that, with VCTE, the assessment is made in a fixed area, and therefore, the ROI could be too close to the liver capsule or may even include the subcutaneous tissue in persons with very thick subcutaneous tissue [40]. Of note, the discriminative accuracy of VCTE for significant and advanced fibrosis decreases in patients with a body mass index >30 kg/m^2 regardless of the type of probe used [39,41].

With ARFI-SWE techniques, it is more challenging to obtain a reliable LSM in individuals with liver steatosis because the energy of the US beam is attenuated by the fat. It has been reported that the diagnostic performance of a 2D-SWE technique is affected by the presence of severe steatosis [42,43]. On the contrary, in a study also performed with a 2D-SWE technique and that included 981 patients, using liver biopsy as a reference, it was found that steatosis and BMI did not overestimate fibrosis and did not affect accuracy [44]. In two large cohorts in which a 2D-SWE technique was used, it was reported that the LSM values in patients with no/mild fibrosis were significantly higher in the case of severe steatosis; this effect was not detected in higher stages of liver fibrosis [43,45].

Staging liver fibrosis

Liver histology is the reference standard used to evaluate the accuracy of SWE techniques in staging liver fibrosis. Histopathology uses semiquantitative scoring systems to stage liver fibrosis based on the subjective evaluation of the amount and distribution of fibrous tissue that ultimately leads to architectural distortion of the hepatic lobules, with bridging fibrosis characteristic of severe fibrosis and regenerative nodules characteristic of cirrhosis [46]. Previous research has revealed a close correlation between the amount of liver fibrosis evaluated histologically and the LSM obtained with the SWE techniques [47–51]. Hence, liver stiffness estimation has been accepted as a reliable noninvasive substitute for liver biopsy in several clinical scenarios [7,10,30].

It should, however, be stressed that liver inflammation, congestion, intrahepatic cholestasis, food intake and obesity are well-known confounding factors for LSM, increasing the risk of falsely increased results. The overlap in LSM for individual fibrosis stages prohibits LSM from being directly translated into a specific pathohistological fibrosis stage. In most clinical scenarios of CLD, both intrahepatic inflammation and fibrosis contribute to liver stiffness, and it is challenging to disentangle the exact role of each in determining the LSM even when the clinical context is known, and the laboratory tests, for example, aspartate transaminase (AST)/alanine transaminase (ALT) as surrogates of hepatic injury/inflammation, are available.

Although LSMs provide continuous numerical values, any histologic scoring system is based on categorical scales for fibrosis and inflammation. Therefore, even under the best conditions, an overlap of LSM between consecutive histologic stages of liver fibrosis is unavoidable. Thus, it is more clinically relevant to provide an estimation of the risk of significant/severe fibrosis (≥F2/≥F3≥, that is, when patients are prone to develop liver-related complications) in a clinical and prognostic context rather than rigidly trying to use LSM to classify patients into consecutive histological stages of liver fibrosis.

The Baveno VI consensus on portal hypertension has highlighted that the spectrum of advanced fibrosis (F3–F4) is a continuum in asymptomatic patients, and distinguishing between the two stages is often not possible on clinical grounds [52]. Therefore, the term *cACLD* was proposed and has been widely accepted by hepatologists since then.

Table 2
Interpretation of liver stiffness measurement using VCTE (rule of five)

VCTE-LSM	Interpretation
≤5 kPa	Normal
<10 kPa	Exclude cACLD: Risk of LREs is negligible.
≥10 to <15 kPa	Potential cACLD: Risk of LREs starts to increase. + Platelets ≥150 × 10^9/L: Exclude CSPH.
≥15 to <20 kPa	Confirm cACLD: patients are at clinically relevant risk of LREs. + Platelets ≥150 × 10^9/L: Exclude HRVs.
≥20 to <25 kPa	cACLD with potential CSPH ("gray zone" for CSPH). The ANTICIPATE model[a] can be used to predict the risk of CSPH in patients with viral hepatitis, alcohol-related liver disease and non-obese MASH.
≥25 kPa	Assume CSPH in patients with viral hepatitis, alcohol-related liver disease and non-obese MASH.

cACLD, compensated advanced chronic liver disease; CPSH, clinically significant portal hypertension; HRVs, high-risk varices; LREs, liver-related events; LSM, liver stiffness measurement; MASH, metabolic dysfunction-associated steatohepatitis; VCTE, vibration-controlled transient elastography.
[a] ANTICIPATE model: LSM values between 20 and 25 kPa and platelet count <150 × 10^9/L or LSM values between 15 and 20 kPa and platelet count <110 × 10^9/L have a CSPH risk of at least 60%.

For assessing the severity of liver disease based on LSM using VCTE, the Baveno VI consensus proposed the "rule of five," which was endorsed by the WFUMB 2018 update [7]. The "rule of five" has been further reinforced and expanded in the Baveno VII consensus (Table 2) [1]. For ALD and MASLD, a recent large multicenter study suggested that 8 and 12 kPa by VCTE LSM are better cutoffs for ruling out and ruling in cACLD, instead of 10 and 15 kPa [53].

The literature indicates that although the different elastography techniques exhibit a strong linear correlation with increasing stages of liver fibrosis, LSMs obtained with the ARFI-SWE techniques are lower than those obtained with VCTE. This difference increases at higher stages of liver fibrosis. Moreover, different US systems provide different LSMs in the same individuals; therefore, cutoffs for exact fibrosis staging that mimic the histologic classifications cannot be interchangeably used between US systems, and the same US system should be used for follow-up measurements in the same patient. To this end, the US system used to measure liver stiffness must be indicated in the report.

The SRU consensus has, however, highlighted that evidence from the literature suggests that differences between the different US systems are smaller than the overlap between consecutive stages of liver fibrosis and has suggested the "rule of four" for assessing the severity of liver disease with the ARFI-SWE techniques (Table 3) [10]. Moreover, because of the efforts of the Quantitative Imaging Biomarkers Alliance (QIBA) committee of the Radiological Society of North America (RSNA), the differences in values obtained with ARFI-SWE techniques from different manufacturers are mitigated.

The panel agrees that the "rule of four" for ARFI-SWE techniques may be considered for evaluating the risk of advanced disease, and it can be used independently from the etiology of liver disease when the presence of confounding factors on LSMs can confidently be ruled out. However, it must be underscored that the independence of this rule from the etiology of liver disease still requires validation.

Metabolic dysfunction-associated steatotic liver disease/nonalcoholic fatty liver disease

Metabolic dysfunction-associated steatotic liver disease (MASLD, previously classified as non-alcoholic fatty liver disease [NAFLD]) is currently the leading cause of CLD worldwide. Recently, it has been proven that almost the totality of patients with NAFLD meet the criteria proposed to define MASLD [54,55]. Its prevalence is currently estimated to

Table 3
Interpretation of liver stiffness measurement obtained using ARFI-SWE techniques (rule of four)

ARFI-SWE LSM	Interpretation
≤5 kPa (1.3 m/s)	High probability of being normal
<9 kPa (1.7 m/s)	In the absence of other known clinical signs, rules out cACLD. If there are known clinical signs, further testing may be needed for confirmation.
9–13 kPa (1.7–2.1 m/s)	Suggestive of cACLD but further testing is required for confirmation.
>13 kPa (2.1 m/s)	Rules in cACLD
>17 kPa (2.4 m/s)	Suggestive of CSPH
>21 kPa (2.6 m/s)	High probability of CSPH

Reproduced (modified), with permission, from Barr et al. [10].
ARFI, acoustic radiation force impulse; cACLD, compensated advanced chronic liver disease; CSPH, clinically significant portal hypertension; LSM, liver stiffness measurement.

be at least 30% in adults and 10% in children and adolescents [56–58]. However, the disease burden is >60% in people who are overweight/obese or who have type 2 diabetes mellitus [59–61]. Furthermore, liver fibrosis can develop in MASLD as a consequence of steatohepatitis (MASH).

Given the high burden of MASLD worldwide, non-invasive tests (NITs) are key to the diagnosis and establishment of the severity of the disease, the prediction of prognosis and the monitoring of disease progression or improvement (either spontaneous or resulting from treatment). Currently, the diagnosis of MASLD is based mostly on B-mode liver US imaging, with liver elastography playing an important role as a biomarker for measuring the severity of hepatic fibrosis.

Once MASLD is diagnosed, the key question to be answered from a liver perspective regards the presence and severity of fibrosis, which is the major factor associated with the occurrence of liver outcomes [62]. Moreover, the presence of advanced fibrosis or cirrhosis necessitates initiation of screening for portal hypertension and surveillance for hepatocellular carcinoma (HCC). Sequential algorithms using a two-step approach have been proposed for the detection of advanced fibrosis. At the primary care level, simple inexpensive and widely available blood-based tests, such as the FIB-4 (AST, ALT, platelet count and age) allow to rule out, with acceptable accuracy, the presence of advanced fibrosis and to identify patients requiring further specialist hepatology assessment with more specific NITs [30].

Fibrosis staging

In patients referred for specialist assessment, data support the use of LSM. Values <8 kPa with VCTE reliably exclude advanced liver fibrosis, while values of LSM ≥ 8 kPa should be considered as suggestive of fibrotic MASLD and should prompt further testing (*e.g.,* liver biopsy). In a recent individual patient meta-analysis [63] including 37 studies and 5735 patients with histologically proven MASLD (30% had advanced fibrosis), the use of a sequential combination of FIB-4 (cutoffs: <1.3 and ≥2.67) followed by VCTE LSM (cutoffs: <8.0 and ≥10.0 kPa) to rule out or rule in advanced fibrosis had a sensitivity and specificity (95% confidence interval [CI]) of 66% (63%–68%) and 86% (84%–87%). In 33% of cases, liver biopsy was needed to achieve a final diagnosis. The use of FIB-4 (cutoffs: <1.3 and ≥3.48) followed by LSM (cutoffs: <8.0 and ≥20.0 kPa) to rule out advanced fibrosis or rule in cirrhosis had a sensitivity of 38% (37%–39%) and specificity of 90% (89%–91%); in this case, 19% required liver biopsy to achieve a definite diagnosis.

Interestingly, LSM using VCTE can be used in combination with the controlled attenuation parameter (CAP) and with AST, in the so-called FAST score to identify patients with at-risk MASH (NAFLD activity score ≥4 points and fibrosis stage ≥2), who should be considered for pharmacologic treatment when available, with an accuracy >80% for this diagnosis.

The use of SWE techniques to stage fibrosis in MASLD/NAFLD has been addressed in a recent meta-analysis of the LITMUS consortium [64], including 53 VCTE studies (11,701 patients), 12 pSWE studies (1312 patients) and 4 2D-SWE studies (502 patients); in all cases, liver histology was used as reference standard. Summary area under the curve (sAUC) for the diagnosis of significant fibrosis, advanced fibrosis and cirrhosis is outlined in Table 4. As shown, pSWE had a very high discriminative value in the reported studies and was the only SWE method meeting a sensitivity and specificity of at least 80% to diagnose advanced fibrosis [64]. However, it must be highlighted that VCTE was the technique used in most of the studies that were included in the meta-analysis.

The best cutoff value to rule-in advanced fibrosis with VCTE was 12 kPa [65].

Recommendation 1. SWE can be used to rule out (< 8 kPa) and rule in (>12–15 kPa) advanced liver fibrosis in patients with MASLD (LoE 1a, GoR A). Broad consensus (11/0/1, 92%).

Prognosis

Several studies on the prognostic value of VCTE LSMs in histologically proven NAFLD/MASLD are available and have been the subject of a recent individual participant data meta-analysis in 2518 patients from 25 studies [66]. During the follow-up (median time = 57 mo), 5.8% of patients developed the composite endpoint (all-cause mortality, HCC, liver transplantation or decompensation of cirrhosis). The time-dependent AUCs at 5 y were 0.72 (95% CI: 0.62–0.81) for histology and 0.76 (0.70–0.83) for VCTE LSM, confirming that LSM can be considered as an alternative to histology for prognostic aims. The higher the value of LSM, the higher is the risk of liver outcomes. The ANTICIPATE-NASH model, which is based on VCTE LSM, platelet count and BMI, has been proposed and validated to assess the risk of CSPH [67–69].

Longitudinal changes in LSM provide insight into the progression or regression of liver disease. European Association for the Study of the Liver (EASL) clinical practice guidelines suggested then to repeat measurement of LSM at 1- to 3-y intervals according to the clinical scenario [30]. Recent American Gastroenterological Association (AGA) clinical practice guidelines also suggest that patients with NAFLD/MASLD and

Table 4
Performance of SWE techniques in staging fibrosis in MASLD/NAFLD in the meta-analysis of the LITMUS consortium [64]

Fibrosis stage	VCTE	pSWE	2D-SWE
Significant fibrosis	[37; 2763][a] 0.83 (3.8–10.2)[b]	[9; 805][a] 0.86 (4.2–9.8)[b]	[4; 488][a] 0.75 (8.3–11.6)[b]
Advanced fibrosis	[44; 4219][a] 0.85 (6.8–12.9)[b]	[11; 1209][a] 0.89 (5.4–53.9)[b]	[4; 488][a] 0.72 (9.3–13.1)
Cirrhosis	[22; 337][a] 0.89 (6.9–19.9)[b]	[8; 759][a] 0.90 (5.6–19.4)[b]	[4; 372][a] 0.88 (14.4–15.7)[b]

AUC, area under the curve; pSWE, point shear wave elastography; SWE, shear wave elastography; VCTE, vibration-controlled transient elastography.
[a] Number of studies; number of patients.
[b] AUC (cutoff range, kPa).

NITs suggestive of advanced hepatic fibrosis or cirrhosis should be monitored with serial LSMs [65].

Data regarding pSWE and 2D-SWE in the setting of prognostic stratification and longitudinal assessment are currently limited.

Recommendation 2. LSM using SWE should be used to stratify the risk of liver-related events and mortality (LoE 1a, GoR A). Strong consensus (12/0/0, 100%).
Recommendation 3. Yearly repetition of LSM is suggested in patients with compensated advanced chronic liver disease, who represent the main risk group for developing CSPH or decompensation (LoE 3, GoR C). Strong consensus (12/0/0, 100%).

Alcohol-related liver disease

Alcohol-related liver disease represents a substantial and growing worldwide health crisis, affecting millions annually. ALD causes at least 25% of global cirrhosis deaths, up to 50% in Europe, and is associated with a 14- and 16-y loss in life expectancy for men and women, respectively [70,71]. Early diagnosis and prognosis are critical as 60%–75% of patients with ALD cirrhosis are currently diagnosed at the time of decompensation, which is much later than for all other CLD etiologies [72,73]. At the time of decompensation, median survival is 3–5 y, and treatment options are limited [72]. In parallel, there are ample possibilities for detection of ALD in high-risk cohorts, as patients with alcohol use disorder (AUD) or prolonged, excessive alcohol intake frequently visit primary and secondary healthcare [74].

Consequently, precise diagnostic and prognostic tests are important tools to ensure timely AUD treatment to patients with ALD at high risk of progressing to liver-related complications [75,76].

Impact of ongoing alcohol use on liver stiffness

Excess use of alcohol may cause steatohepatitis and alcohol-associated hepatitis. Hepatic inflammation is a known cause of increased liver stiffness across elastography techniques, in ALD as in other etiologies [77]. Specifically for ALD, ongoing drinking in itself does not seem to cause false-positive LSMs, as seen in a biopsy-controlled study investigating VCTE and 2D-SWE in outpatients with moderate to high alcohol consumption [78]. A number of studies have investigated VCTE during detoxification in patients with heavy drinking [79–88]. Overall, these studies find that elevated liver enzymes correlate with elevated VCTE LSM and that resolution of hepatic inflammation evidenced by AST normalization is paralleled by reduced liver stiffness after a period of 1–8 wk. One biopsy-controlled study found that ALD patients with advanced fibrosis decreased from an average VCTE LSM of 21.5 kPa at hospitalization to 11.4 kPa after 2 mo of abstinence. In parallel, AST decreased from 70 U/L (IQR: 49–102) to 30 U/L (IQR: 21–49) [87]. Another study found steeply increasing LSM in ALD patients with advanced fibrosis when AST levels exceeded 70 U/L [82].

In contrast, several studies find that low LSMs can be used to exclude the presence of advanced fibrosis regardless of ongoing drinking or concomitant alcohol-associated steatohepatitis [86,87,89].

Finally, an individual patient data meta-analysis revealed that optimal LSM cutoffs for VCTE to stage fibrosis differed according to AST and bilirubin levels, with the highest cutoffs at AST >75 U/L and bilirubin >16 μmol/L (0.94 mg/dL) [77].

Recommendation 4. In patients who drink alcohol in excess and have elevated liver stiffness, LSM should be repeated after at least 4 wk of abstinence if there are concurrent signs of inflammatory activity in the form of aspartate transaminase >70 U/L and/or elevated bilirubin (LoE 2a, GoR B). Strong consensus (11/0/0, 100%).

Use of SWE to diagnose advanced fibrosis in alcohol-related liver disease

One individual patient data meta-analysis and several multicenter studies have been published on the diagnostic accuracy of VCTE for alcohol-related liver fibrosis [53,77,87]. The meta-analysis revealed a discriminative accuracy of AUC = 0.92 for advanced fibrosis and cirrhosis, from 10 studies comprising 1026 patients [77]. The cutoffs derived by optimizing the Youden index did not reach a sensitivity or specificity >90% to rule out or rule in fibrosis. The summary cutoff for advanced fibrosis was 12.1 kPa. A 2021 multicenter, biopsy-controlled study validated and modified Baveno VI-suggested cutoffs of 10 and 15 kPa in 946 ALD patients from 10 centers [53]. The authors concluded that a cutoff value of 10 kPa ruled out advanced fibrosis in ALD with a sensitivity of 87%, increasing to 94% when using a cutoff of 8 kPa. Similarly, 12 kPa ruled in advanced fibrosis in ALD with a specificity of 89%, and 15 kPa with a specificity of 92%. These findings are backed up by a recent biopsy-controlled, multicenter study in 259 ALD patients recruited from addiction units, where 10 kPa ruled out advanced fibrosis when detoxification started at a sensitivity of 96%, and 87% after 2 mo of abstinence [87]. For ruling in advanced fibrosis, 12 kPa had a specificity of 92% at detoxification, increasing to 96% after 2 mo of abstinence.

Three ALD studies have investigated the use of pSWE with VTQ (Acuson, Siemens) to diagnose fibrosis in ALD [90–92]. A study in 83 ALD patients with a 20% prevalence of advanced fibrosis found a sensitivity and specificity of 82% and 79%, respectively, at a cutoff of 1.84 m/s (10.2 kPa) for advanced fibrosis (AUC 0.86). A cutoff of 1.94 m/s (11.3 kPa) diagnosed cirrhosis with a sensitivity of 92% and specificity of 82% [90]. Another study in 112 patients, 25% with advanced fibrosis, found similar discriminatory accuracy, but lower cutoff values: 1.40 m/s (5.9 kPa) for advanced fibrosis (sensitivity 84%, specificity 82%) and 1.65 m/s (8.2 kPa) for cirrhosis (sensitivity 89%, specificity 84%) [91]. The most recent study included 251 patients of whom 70% had advanced fibrosis and 38% had decompensated cirrhosis, most with concomitant alcohol-associated hepatitis [92]. They reported liver stiffness values of 1.47–1.66 m/s (6.5–8.3 kPa) for severe fibrosis and >1.66 m/s (>8.3 kPa) for cirrhosis. These findings contrast with the rule-out ability of the recently proposed "rule of four," suggesting that pSWE or 2D-SWE <9 kPa (1.7 m/s) rules out advanced fibrosis. The findings do, however, support the proposal that ARFI-SWE LSM 9–13 kPa (1.7–2.1 m/s) is suggestive of advanced fibrosis and that a value ≥13 kPa (2.1 m/s) rules in advanced fibrosis.

For 2D-SWE, the results obtained using the US Aixplorer system (SuperSonic Imagine) in a single-center cohort were published in two articles [78,89]. The cohort comprised 289 ALD patients with a 23% prevalence of advanced fibrosis. AUCs of 0.88, 0.97 and 0.97 for the diagnosis of significant fibrosis (≥F2), advanced fibrosis (≥F3) and cirrhosis were reported. A cutoff of 16.4 kPa yielded a sensitivity and specificity of 90% and 96% for advanced fibrosis in per-protocol analyses [89].

Recommendation 5. VCTE can be used for liver fibrosis assessment in patients with ALD. Values <8 kPa rule out advanced liver fibrosis, and values >12–15 kPa rule in advanced fibrosis (LoE 1b, GoR B). Strong consensus (11/0/0, 100%).
Recommendation 6. pSWE and 2D-SWE may be used for diagnostic purposes in patients with ALD, as their accuracy is comparable to that of VCTE. In the absence of validated cutoffs, the "rule of 4" may be considered (LoE 2b, GoR D). Strong consensus (11/0/0, 100%).

Use of SWE to predict liver-related events and monitor liver disease progression or regression in alcohol-related liver disease

Three large cohort studies have evaluated the prognostic ability of VCTE to predict LREs and decompensation during a median follow-up of

4–5 y in ALD patients without decompensated cirrhosis at baseline [93–95]. One systematic review published before those three studies found the heterogeneity was too high to perform a meta-analysis, and reported VCTE cutoff values of 20 and 25 kPa used in two published abstracts [96]. Overall, the cohort studies find that 10 kPa is a good cutoff to rule out the risk for decompensation or LREs, with 3%–5% of events occurring during 5 y of follow-up. The risk substantially increases for ALD patients with baseline VCTE LSM ≥15 kPa, although with varying estimates. One study reported an event rate of 54% during a median follow-up of 4.1 y for a broadly defined outcome of LREs [93]. Another study predicted a 25% 5-y risk of death, decompensation or HCC in ALD patients with a VCTE LSM of 15 kPa, increasing to 50% for ALD patients with a VCTE LSM of 30 kPa [95].

More prognostic studies have been conducted in mixed etiologies, where ALD patients constitute up to half of the cohort, but more often below 20% [97–99]. These studies confirm that VCTE LSM according to the Baveno VII "rule of five" (10–15–20–25 kPa) predicts increasingly higher risk of decompensation and liver-related death.

Although many use liver stiffness to monitor patients for progression of disease, data on monitoring are limited currently, and no single etiology study has been published for ALD. One mixed-etiology study found that a decrease in VCTE LSM of 20%, or to <20 kPa, translated into a clinically significant improvement in prognosis [100]. However, this study included primarily HCV patients. Therefore, it is not currently possible to make recommendations for monitoring ALD improvement or worsening using VCTE.

Elastography is not recommended for prognostication in patients with decompensated disease, although one study in mostly ALD cirrhosis patients found that VCTE LSM after a first episode of variceal bleeding predicted further decompensation with an AUC of 0.93, significantly better than the MELD-Na score (AUC 0.78), with a sensitivity of 90% at a cutoff of 38 kPa [101].

Two studies investigated the prognostic accuracy of 2D-SWE [93,102]. In a single-center study of 462 patients, the *C*-statistic was 0.87 for prediction of LREs during 4.1 y of follow-up, with rates of events of 5% for 2D-SWE LSM <10 kPa, 15% for 10.0–16.4 kPa and 64% for >16.4 kPa. A multicenter study with 23% ALD patients found that the patients with a MELD score <10 and 2D-SWE LSM <20 kPa had a 2-y mortality of 1.5%, increasing to 12% at MELD <10 and 2D-SWE ≥20 kPa or MELD ≥10 and 2D-SWE <20 kPa, and to 39% at MELD ≥10 and 2D-SWE≥20 kPa [102].

Recommendation 7. VCTE LSM may be used to predict liver-related events in patients with ALD and compensated disease (LoE 2b, GoR C). Strong consensus (11/0/0, 100%).

Viral hepatitis

Fibrosis staging

Liver stiffness measurement by US elastography has been extensively evaluated in patients with chronic viral hepatitis. In a meta-analysis of 19 studies in patients with chronic hepatitis B, LSM had summary AUCs of 0.82 for significant fibrosis and 0.91 for cirrhosis [103].

In chronic hepatitis B, the presence of significant liver fibrosis is an indication for antiviral therapy regardless of the serum ALT level [104,105].

For chronic hepatitis C, the current direct-acting antivirals are highly efficacious and well-tolerated and can result in sustained virological response (SVR) in over 95% of patients. Therefore, direct-acting antivirals should be given regardless of fibrosis stage. However, it remains useful to assess fibrosis in patients with chronic hepatitis C. According to current guidelines, in patients with advanced fibrosis and cirrhosis, surveillance for HCC must continue because the risk of HCC is reduced but not abolished [106].

The use of LSM to monitor treatment response is controversial. In chronic viral hepatitis, LSM is driven by not only liver fibrosis but also hepatic inflammation. Most patients with acute viral hepatitis or acute exacerbation of chronic hepatitis B can have LSM interpreted as being in the cirrhotic range [107,108]. Confounding of LSM is also well reported in patients with moderate degrees of ALT elevation [109]. For this reason, a reduction in LSM during and after antiviral therapy, particularly in the early phase of treatment, largely represents a reduction in hepatic inflammation rather than genuine fibrosis improvement [110].

Although a reduction in LSM cannot reliably reflect fibrosis improvement and regression of cirrhosis, current data suggest that the Baveno VI criteria are sufficient to spare patients from upper gastrointestinal endoscopy for surveillance of varices. The Baveno VI criteria state that patients with LSM <20 kPa by VCTE and a normal platelet count ≥150 × 10^9/L have a <5% risk of high-risk varices [1]. The criteria have been validated in patients on antiviral therapy for hepatitis B-related cirrhosis [111]. The addition of spleen stiffness measurement to LSM according to the Baveno VII consensus avoided more endoscopies than the Baveno VI criteria alone with a comparable missed rate. In another study of hepatitis C-related cirrhosis, achieving an LSM <12 kPa and normal platelet count after SVR excluded CSPH with a sensitivity of 99.2% [112]. In such patients, only 1.3% developed hepatic decompensation in 3 y. Recently, the Baveno VI criteria have also been validated in patients with HCC [113].

Prognostication

The assessment of liver fibrosis is important throughout the patient journey in patients with chronic viral hepatitis (Table 5). The degree of liver fibrosis correlates well with the risk of developing LREs [114]. Obviously, cirrhotic complications can develop only after progression to cirrhosis, and cirrhosis remains the most important risk factor for HCC in patients with chronic viral hepatitis [115].

Current guidelines recommend HCC surveillance in patients with cirrhosis and patients with chronic hepatitis B and high-risk features [116,117]. Several HCC risk scores have been proposed to aid selection of patients for HCC surveillance, among which some incorporated LSM into the model [118,119]. Regression of cirrhosis is well recognized in most patients with chronic hepatitis B with complete viral suppression and chronic hepatitis C with SVR [120,121]. However, the risk of HCC in such patients remains higher than that in patients who have never had cirrhosis. Currently, there are insufficient data to recommend cessation of HCC surveillance after treatment for chronic viral hepatitis in patients with cACLD before antiviral therapy.

Recommendation 8. Ultrasound elastography is useful to exclude significant fibrosis and diagnose cirrhosis in patients with chronic hepatitis B and C (LoE 1a, GoR B). Strong consensus (11/0/0, 100%).
Recommendation 9. In patients with treated (suppressed) hepatitis B and cured (*i.e.,* SVR) hepatitis C, the Baveno VI criteria are useful in predicting high-risk varices and clinically significant portal hypertension. Patients with a VCTE LSM <20 kPa and platelet count ≥150 × 10^9/L may be spared from endoscopic surveillance for varices even if they had cirrhosis (ACLD) prior to antiviral treatment (LoE 2b, GoR A). Strong consensus (11/0/0, 100%).
Recommendation 10. HCC surveillance should continue despite decreased LSM in patients with advanced liver disease before antiviral treatment (LoE 2b, GoR A). Strong consensus (11/0/0, 100%).

Table 5
Role of liver stiffness measurement in patients with chronic viral hepatitis

Role	Description
Prognostication	There is a strong association with future risk of cirrhotic complications and hepatocellular carcinoma.
Treatment decision	Antiviral therapy is indicated in patients with chronic hepatitis B and significant liver fibrosis.
Follow-up	Patients with hepatitis C virus who do not have advanced chronic liver disease can be discharged from the hepatology clinic after achieving a sustained virological response. Patients with advanced chronic liver disease but fulfilling the Baveno VI criteria can be spared from endoscopic surveillance for varices.

Cholestatic liver disease, autoimmune hepatitis and rare liver diseases

Primary biliary cholangitis

The prognosis of primary biliary cholangitis (PBC) depends largely on the extent of liver fibrosis [122,123]. LSM with VCTE not only correlates with fibrosis presence, as shown in a meta-analysis [124], but is also an important indicator for prognosis. Corpechot et al.'s international multicenter study involving 3985 patients found that each additional kilopascal in LSM increases the hazard ratio by 1.040 (1.026 −1.054), establishing LSM as an independent prognostic factor for PBC [125]. Thresholds of 8 and 15 kPa effectively categorize patients into low-, medium- and high-risk groups.

In managing PBC, treatment success is typically gauged through laboratory values and symptoms reduction [126]. However, evaluating the effectiveness of treatment remains complex, with estimates often overly optimistic in about 20% of cases [127]. Recent findings from the Corpechot group suggest that LSM values exceeding 10 kPa could signal the necessity for second-line treatments in PBC patients [128].

Although other SWE techniques such as 2D-SWE also reveal a correlation with liver fibrosis in PBC and overlap syndromes [129,130], the diversity in technology across different manufacturers and the low level of evidence preclude a universal recommendation for these methods.

Primary sclerosing cholangitis

Primary sclerosing cholangitis (PSC), marked by progressive inflammation and fibrosis of bile ducts, presents challenges in monitoring and staging because of its heterogeneous manifestation and fluctuating progression.

Liver stiffness measurement has exhibited a correlation with fibrosis stages in PSC patients [131]. A key study by Corpechot et al. [132], involving 73 biopsy-controlled patients, identified LSM cutoffs for fibrosis stages ≥F1, ≥F2, ≥F3 and F4 of 7.4, 8.6, 9.6 and 14.4 kPa, respectively. This research underscored the prognostic significance of both initial LSM values and their longitudinal changes in PSC. Interestingly, simpler markers, such as spleen length assessed by B-mode US, may offer comparable predictive accuracy [133]. A recent systematic review on prognostic markers highlighted the need for further prospective studies comparing LSM methods with established approaches such as the Mayo Risk Score [134].

Similar to PBC, ARFI-SWE techniques reveal correlations with liver fibrosis in PSC; however, their utility is constrained by variable cutoff values and limited evidence [135]. Recently, Roccarina et al. [136] reported that in a large cohort of 152 patients, LSM using pSWE was highly accurate in detecting any grade of fibrosis in PSC. Additionally, they proposed a cutoff value of 11.2 kPa, suggesting that beyond this threshold, spleen stiffness measurement (SSM) should be added to better stratify patients for the need for screening for esophageal varices.

It is crucial to recognize that inflammatory activity and cholestasis can have an impact on LSM accuracy in PSC patients. Therefore, interpretation of LSM results should be approached with caution and after ruling out significant acute biliary obstructions.

Other cholestatic liver diseases

Liver stiffness measurement has also been assessed in other cholestatic conditions, such as cystic fibrosis-related liver disease (CFLD) [137]. Because of the low prevalence of these diseases, comprehensive data on the incidence of advanced stages and the diagnostic and prognostic utility of LSM remain limited. Nevertheless, significantly elevated LSM values typically indicate advanced disease, warranting further diagnostic evaluation in affected patients (see also the section on Pediatrics).

Autoimmune hepatitis

Autoimmune hepatitis, a chronic inflammatory liver disease of unknown etiology [138], is marked by inflammatory flares that can occur with or without symptoms, potentially leading to liver fibrosis and cirrhosis. Although liver biopsy remains crucial for diagnosing autoimmune liver diseases, LSM methods provide valuable supplementary information on disease severity. A study of 90 patients undergoing long-term immunosuppressive therapy revealed that liver SWE correlated with active hepatitis and fibrosis presence [139]. The study identified an optimal cutoff for cirrhosis detection using 2D-SWE at 16.1 kPa, with an AUC of 0.93. Incorporation of spleen SWE could enhance diagnostic accuracy, as it is less affected by inflammatory activity. Similar findings were reported for liver elastography using VCTE and pSWE in a smaller study by Paranaguá-Vezozzo et al. [140].

Rare liver diseases

Numerous studies have explored the diagnostic and prognostic capabilities of SWE techniques across a range of rare acute and chronic liver disorders. For instance, in patients with hepatic manifestations of Wilson's disease, LSM has exhibited a correlation with clinical algorithms [141] and degree of liver fibrosis [142]. A decrease in LSM values during treatment is indicative of a stable course in Wilson's disease [142]. Similarly, studies on patient cohorts with conditions such as hemochromatosis [143] and α-1-antitrypsin deficiency [144] have verified the association between heightened LSM values and advanced parenchymal damage.

In cases of vascular pathologies such as sinusoidal obstruction syndrome (SOS), elevated LSM readings, whether assessed by VCTE or pSWE, are indicative of a more severe course of the disease [145–147]. Notably, LSM increases often precede the clinical symptoms of SOS [147–149]. Current research is focusing on using LSM to identify patients at risk early, allowing for timely preventative treatments [149].

In non-cirrhotic PH causing porto-sinusoidal vascular disorder (PSVD), there is a marked increase in spleen stiffness, whereas the increase in liver stiffness is lower than that observed in cirrhotic PH [150]. Indeed, it has been reported that a VCTE LSM <10 kPa in patients with signs of PH is highly suggestive of PSVD [151]. In a study in a small cohort, the spleen/liver stiffness ratio was proposed for the diagnosis of PSVD [152].

Recommendation 11. VCTE LSM is useful in excluding advanced fibrosis and diagnosing cirrhosis in patients with PBC. Cutoff values of 8 and 15 kPa can be used to distinguish low-, medium- and high-risk groups for liver-related events (LoE 1b, GoR B). Strong consensus (11/0/0, 100%).
Recommendation 12. In the absence of biliary obstruction, elevated VCTE LSM values are suggestive of advanced parenchymal damage in a variety of rare chronic liver diseases including PSC, treated autoimmune hepatitis and storage disorders. In the absence of validated cutoffs, a VCTE LSM of 8 kPa is suggested (LoE 2b, GoR C). Strong consensus (12/0/0, 100%).

Pediatrics

The normative value of LSM in children is generally ≤5.0 kPa with all the SWE techniques [153]. A slight age and transducer dependency has been reported [7]; however, these differences are not clinically relevant when the values are within the normal range. The number of studies that have confirmed the usefulness of the SWE techniques for the evaluation of liver disease in the pediatric population has substantially increased since the previous guideline update [7]. Most studies have been performed in small cohorts or cohorts with mixed etiologies of chronic liver disease. Currently, similar to the adult population, MASLD is the most common chronic liver disease in children [154].

It must be highlighted that some etiologies of chronic liver disease, such as CFLD, biliary atresia (BA) and Fontan-associated liver disease (FALD), among others, are mostly specific to the pediatric population. In these cases, in addition to liver fibrosis and depending on the etiology of the underlying liver disease, there is a complex and variable interplay of other factors that may lead to an increase in LSM. These factors are mainly congestion, obstructive cholestasis and liver inflammation, which are "confounding" factors when using liver stiffness as a biomarker of fibrosis. Therefore, thresholds for fibrosis staging are variable between studies, particularly because cohorts with mixed etiologies of liver disease were evaluated [155–158].

Nonetheless, taking into consideration all the factors contributing to LSM, the use of the SWE techniques is helpful for diagnosis, follow-up and evaluation of the clinical outcome in the pediatric population. For the follow-up, the SRU consensus has suggested using the percentage of LSM change over time referred to the individual baseline LSM [10]. Because there is variability between manufacturers and techniques, the same US equipment and the same transducer should preferably be used in follow-up studies of individual pediatric patients, for consistency. As there is a 10% variability in measurement a clinically significant change should be greater than 10%.

The prevalence of MASLD in children and adolescents has steadily increased in the last two decades [154]. A recent meta-analysis assessed the diagnostic performance of LSM in detecting liver fibrosis in pediatric patients with MASLD. Seven studies with a total of 436 children were included in the analysis. The prevalence of fibrosis was similar to that observed in studies including adults. However, it must be highlighted that the prevalence of fibrosis in this meta-analysis should not be considered representative of the broader population, as these were children in a hospital setting and biopsies were performed on clinical grounds. Therefore, the prevalence of liver fibrosis was likely estimated too high. The AUC revealed a diagnostic performance >0.90 in differentiating the stages of liver fibrosis [159].

A recent meta-analysis, including 11 studies with 1307 children for the diagnosis of BA and 9 studies with 327 children for the follow-up post-Kasai procedure, has confirmed that SWE is useful in differentiating BA from other infantile cholestatic diseases [160]. The mean LSM was significantly higher in the BA group than in the non-BA group (overall standardized mean difference: 2.30 kPa). The AUC was 0.91 with a pooled sensitivity and specificity of 83% and 79%, respectively. In post-Kasai procedure pediatric patients, the mean LSM was significantly higher in patients with varices than in those without (overall standardized mean difference: 1.38 kPa).

It has been suggested that SWE can be used for detecting CFLD [161–172]. The published studies used the criteria suggested by a best practice guidance as reference standard [173], and some of them included both adults and children. A meta-analysis, including six studies with both adults and pediatric data for a total of 605 patients, has reported that the cutoff of LSM by VCTE for the diagnosis of CFLD was ≥5.95 kPa with a sensitivity, specificity and AUC of 55%, 87% and 0.76, respectively [137]. By adding an AST-to-platelet ratio index (APRI) cutoff ≥0.329, the positive predictive and negative predictive values were 92% and 87%, respectively, with a diagnostic odds ratio of 74.9. Comparable results were obtained in a study that used histology as the reference [174]. It has been reported that the change in LSM over time is useful for evaluating the progression of CFLD [175–177].

Liver fibrosis is always present in Fontan patients by adolescence, and the degree of fibrosis increases over time [178]. However, the assessment of liver fibrosis is a challenge in FALD because congestion increases LSM, and the use of LSM thresholds derived from children with other (mixed) etiologies of chronic liver disease will likely overestimate the fibrosis stage in FALD patients. In fact, it has been reported that the LSM values in Fontan patients with mild fibrosis may be much higher than the traditional LSM threshold for liver cirrhosis [179]. For follow-up and longitudinal monitoring of Fontan patients, the SRU consensus has suggested assessing the change in LSM over time [10]. However, as of today, a validated risk-strategy approach is lacking.

Few studies with a limited number of children have been published regarding the use of SWE for the evaluation of fibrosis in pediatric patients after liver transplant, and VCTE was the technique most frequently used [180]. In the largest series available to date, comprising 94 children, the AUC of LSM by VCTE for detecting significant fibrosis was suboptimal (0.71; 95% CI: 0.57–0.85) [181]. No validated LSM cutoffs for identifying children at risk for fibrosis after liver transplant are available [180].

The "rule of four" suggested for adults cannot be applied to the pediatric population. On the basis of the current literature, a value ≤5 kPa can rule out fibrosis whereas a value ≥15 kPa can rule in advanced liver disease except for children with BA or CFLD or for Fontan patients [153].

Recommendation 13. SWE is helpful for the diagnosis and follow-up of chronic liver disease in the pediatric population, as well as for evaluation of the clinical outcomes. However, specific cutoffs for fibrosis staging cannot be recommended for pediatric patients because of the heterogeneity between published studies (LoE 2a, GoR B). Strong consensus (12/0/0, 100%).

Portal hypertension

Clinically significant portal hypertension

Patients with cACLD (for a definition, see the section Staging of Liver Fibrosis) constitute the target population for CSPH screening [1]. Notably, patients with clinical signs of decompensated liver cirrhosis (dACLD) have CSPH by definition and therefore do not need to undergo (non-invasive) screening for CSPH. Treatment with non-selective beta blockers (NSBBs), ideally carvedilol, is indicated in cACLD patients with CSPH [1,182]. Given the large body of evidence available for VCTE LSM to correlate well with portal hypertension severity and to predict the presence of CSPH and the risk of LREs, Baveno VII has recommended the VCTE LSM "rule of 5" [1] (5–10–15–20–25 kPa) that allows clinicians in daily clinical practice to rule out and rule in cACLD and estimate the risk for CSPH and LREs for their patients. A VCTE LSM <10 kPa rules out cACLD and indicates a negligible risk of LREs in patients with MASLD [68]. In a large-scale study including a total of 3317 MASLD patients, the observed LREs rate at 3 y in those 1837 patients with a VCTE LSM <10 kPa was only 0.1% [68]. However, it must be highlighted that the risk of LREs is higher in patients with ALD. In a study of 462 patients, 3% (9/303) of those with a VCTE LSM <10 kPa developed LREs during an average follow up of 4 y [2].

A pragmatic "rule of 4" (5–9–13–17 kPa) was suggested for ARFI-SWE LSMs to support clinical risk stratification, but supporting evidence is not as strong as for VCTE LSMs [10].

Three HVPG-controlled studies investigated the value of VCTE LSMs in predicting CSPH specifically in patients with ALD etiology: A smaller study including 48 patients with ALD reported a VCTE LSM cutoff at 34.9 kPa to rule in CSPH (specificity 88%, negative predictive value [NPV] 64%) [183].

The largest study including 227 ALD patients suggested a VCTE LSM cutoff at 19 kPa (positive predictive value [PPV] 84.1%, NPV 86.2%) [184]. A more recent study (118 ALD patients) found a VCTE LSM cutoff >30.6 kPa for ruling in CSPH (specificity 94%) [86].

The Baveno VII criteria postulate that a VCTE LSM >20 kPa is suggestive of portal hypertension if associated with thrombocytopenia (platelet count [PLT] $<150 \times 10^9$/L) and that a VCTE LSM ≥25 kPa rules in CSPH in ALD, viral hepatitis B (HBV) and C (HCV) and non-obese MASLD [1]. However, these cutoffs have not been sufficiently validated for other liver disease etiologies and for obese MASLD. High BMI, which is commonly found in MASLD patients, is confounding the correlation of VCTE LSM with CSPH (*i.e.,* with hepatic venous pressure gradient) [67]. Thus, the combination of VCTE LSM, PLT and BMI has been proposed [67] and validated [68,69] as a diagnostic and prognostic tool (ANTICIPATE-NASH model) for predicting the risk of CSPH in patients with MASLD.

ARFI-SWE techniques (pSWE and 2D-SWE) can rule out and rule in CSPH as well, but fewer data are available, and the cutoffs proposed in the literature vary. On the basis of the available evidence and considering that sufficiently powered head-to-head validation studies are not available, the SRU consensus [10] proposed a vendor-neutral "rule of 4" kPa, with cutoffs at 5, 9, 13 and 17 kPa. According to this rule, in cACLD patients with chronic viral hepatitis or MASLD, ARFI-SWE LSM values >17 kPa (>2.4 m/s) suggest CSPH.

Most data on using ARFI-SWE for the evaluation of CSPH are available for 2D-SWE specific to the Aixplorer US system (SuperSonic Imagine), and for this system published cutoffs for ruling out CSPH range from <13.5 to <16.0 kPa [185–189], with better performance when combined with the criterion of normal platelet count ($\geq 150 \times 10^9$/L). In turn, 2D-SWE stiffness values ranging from >25.8 to >38.0 kPa [187,188] indicated a high likelihood of CSPH. One individual patient data meta-analysis including 328 patients with mixed etiology of liver disease (53% with ALD) from five studies investigated the use of 2D-SWE to diagnose CSPH [186]. This study reported an LSM <14 kPa cutoff to rule out CSPH (sensitivity 91%) and >32 kPa to rule in CSPH (specificity 89%).

Recommendation 14. A simple "rule of 5" for VCTE LSM and "rule of 4" kPa for ARFI-SWE techniques can be used to identify patients with cACLD at risk for LREs (LoE 1a for VCTE, LoE 2b for ARFI-SWE, GoR A). Strong consensus (12/0/0, 100%).
Recommendation 15. A VCTE LSM <10 kPa indicates a low 5-y risk for LREs. (LoE 1b, GoR A). Strong consensus (12/0/0, 100%).
Recommendation 16. VCTE LSMs of 15–20 kPa suggest CSPH in patients with PLT $<110 \times 10^9$/L (LoE 2c, GoR A). Strong consensus (12/0/0, 100%).
Recommendation 17. A VCTE LSM ≥20–25 kPa suggests CSPH if PLT is $<150 \times 10^9$/L (LoE 2a, GoR A). Strong consensus (12/0/0, 100%).
Recommendation 18. A VCTE LSM >25 kPa rules in CSPH in patients with ALD, HBV/HCV and non-obese MASLD (LoE 1b, GoR A). Strong consensus (12/0/0, 100%).
Recommendation 19. An ARFI-SWE LSM <9 kPa indicates a low short-term risk of LREs (LoE 2a, GoR A). Strong consensus (12/0/0, 100%).
Recommendation 20. An ARFI-SWE LSM >17 kPa suggests CSPH, especially in patients with PLT $<150 \times 10^9$/L (LoE 3b, GoR B). Strong consensus (12/0/0, 100%).
Recommendation 21. An ARFI-SWE LSM >21 kPa indicates a high risk of CSPH and LREs (LoE 4, GoR C). Strong consensus (12/0/0, 100%).

Screening for high-risk varices

Medical therapy is indicated in all patients with varices as they have by definition CSPH; thus, the detection of high-risk varices (high-risk varices [HRVs] = medium to large varices or any varices with red-spot signs) is of clinical relevance. The term *varices needing treatment* (VNT) should thus no longer be used to avoid the misunderstanding that there could be varices that do not require medical treatment. Patients with a VCTE LSM <20 kPa and PLT $>150 \times 10^9$/L (Baveno VI criteria) may not need to undergo screening endoscopy. The majority of studies on diagnosing CSPH or HRV using the Baveno VI and VII criteria included patients with cirrrhosis of mixed etiology, with ALD constituting 20%–30% [190–192]. A meta-analysis reported that the Baveno VI criteria resulted in no HRV missed in the subgroup of ALD cirrhosis patients [193]. Different cutoffs for different ARFI-SWE (pSWE and 2D-SWE) techniques have been proposed to rule out and rule in HRV: Patients with LSM values by pSWE <12 kPa and PLT $> 150 \times 10^9$/L (BAVElastPQ criteria, proposed by a monocentric study including 195 patients) may not need to undergo screening endoscopy [194]. Another study including 76 patients (36.8% ALD, 30.3% MASLD, 14.5% viral hepatitis) reported that LSM values by 2D-SWE <19.3 kPa and normal platelet count (PLT $\geq 150 \times 10^9$/L) ruled out HRV (sensitivity 100%) [185].

Recommendation 22. Screening endoscopy can be avoided in cACLD patients with VCTE LSMs <20 kPa and PLT $\geq 150 \times 10^9$/L (LoE 1a, GoR A). Strong consensus (12/0/0, 100%).

Prediction of liver-related events

Liver stiffness measurement by VCTE has been consistently reported to predict LREs (hepatic decompensation, HCC, liver-related mortality) in patients with chronic liver diseases in large meta-analyses [195,196]. Baseline LSM has also been reported to predict liver-related outcomes (liver-related complications, liver transplantation or death) in a large international multicenter cohort of 3985 patients with PBC [197].

Two studies focusing on MASLD patients with cACLD reported similar results [198,199]. For instance, a baseline LSM ≥21 kPa was independently associated with a higher risk of hepatic decompensation (HR: 3.71; 95% CI: 1.89–6.78, p<0.001) in a large multicenter study [199] in 1039 patients with a median follow-up of 3 y, whereas an LSM ≥30.7 kPa predicted LREs (adjusted HR = 10.13) in another study in 1398 MASLD patients enrolled in randomized placebo-controlled trials with 16 mo of follow-up [198]. By contrast, baseline LSM did not predict cardiovascular events or extrahepatic cancers [199,200]. Results on the value of LSM for prediction of overall mortality have been conflicting [199-202].

Finally, repeated LSMs seem to be superior to one-time measurement. For instance, in 533 MASLD patients with cACLD, an increase in LSMs of 20% over a median interval of 37 mo was independently associated with hepatic decompensation, HCC, overall mortality and liver-related mortality [199]. In another retrospective single-center cohort study, including 2508 patients with CLD (non-ACLD, 66%; cACLD, 30%; decompensated ACLD, 4%) followed for a median of 71 mo, an increase in LSMs of 20% at any time (but at least 180 d apart) was associated with a 50% increase in hepatic decompensation and liver-related mortality [203]. Further prospective studies are needed to assess the impact of dynamic changes in LSM on long-term outcomes.

Data on ARFI-SWE LSM (by 2D-SWE or pSWE) to predict LREs are limited. In the largest study to date (1827 ACLD patients, 1490 compensated and 337 decompensated, with a median follow-up of 33 mo), LSM by 2D-SWE ≥20 kPa combined with MELD ≥10 could stratify the risk of mortality and first/further decompensation [102].

A reduction in VCTE LSM to <12 kPa combined with a normal(ized) PLT ($\geq 150 \times 10^9$/L) indicates resolution of CSPH after cure from hepatitis C (HCV-SVR) [1]. A reduction in VCTE LSM to <20 kPa combined

with a normal(ized) PLT ($\geq 150 \times 10^9$/L) rules out HRV in patients after cured HCV and in suppressed hepatitis B [204].

Weight losses >10% and lifestyle modifications have resulted in significant reduction in LSM (and HVPG) in patients with MASLD [205,206]; however, more data are needed to confirm whether sufficient weight loss promotes resolution of CSPH.

Very high values of LSM and SSM have been reported in patients with Budd–Chiari syndrome; after hepatic and portal venous flow is improved by placement of TIPS, there is a decrease in both LSM and SSM, and this can help in assessing shunt patency [207].

More data on the prognostic value and clinically relevant magnitude of absolute/relative change in SWE LSMs is needed.

Recommendation 23. A stable decrease in VCTE LSM below 20 kPa after removal/suppression of the primary etiologic factor[(a)] indicates a significantly decreased risk of LRE-driven portal hypertension that becomes negligible below 10 kPa (LoE 1b, GoR A). Strong consensus (12/0/0, 100%).

[a] Removal/suppression of the primary etiologic factor is defined as sustained abstinence from alcohol abuse in patients with alcohol-related liver disease, SVR in patients with hepatitis C and sustained suppression of virologic replication in patients with hepatitis B.

Role of spleen stiffness measurement in assessment of portal hypertension

The splenic vein drains into the hepatic portal vein; thus, in patients with cirrhosis and portal hypertension, the increased pressure in the splenic vein results in splenic congestion (congestive component of splenomegaly). Additionally, patients with CSPH develop hyperdynamic circulation with increased splanchnic/splenic blood flow (inflow component of splenomegaly). Thus, CSPH is characterized by splenomegaly, hypersplenism (including thrombocytopenia) and increased spleen stiffness. A number of studies have reported a correlation between SSM by SWE and the presence of CSPH or esophageal varices [185,187–189,208–217]. Notably, results on the value of SSM have not only been obtained in different populations with various liver disease etiologies but also with a range of different techniques including pSWE and 2D-SWE, as well as VCTE with different probes (50 and 100 Hz).

In a meta-analysis examining data obtained with the Aixplorer (SuperSonic Imagine) system, SSM by 2D-SWE (two studies) had a pooled AUC, sensitivity and specificity of 0.88, 0.62 and 0.95 for the detection of CSPH, respectively, compared with an AUC of 0.84 for LSM (four studies) [218]. In another systematic review and meta-analysis focusing on varices requiring treatment, SSM had a pooled sensitivity and specificity of 0.91 and 0.79 by pSWE (nine studies) and 0.89 and 0.72 by 2D-SWE (five studies), respectively [219]. Finally, a meta-analysis including VCTE, pSWE and 2D-SWE revealed pooled AUCs of 0.90 for SSM to detect esophageal varices and 0.81 for high-risk varices [220].

Spleen stiffness measurement is rarely performed alone because the first step should be the identification of cACLD by LSM. Therefore, in most (if not all) scenarios, SSM should be interpreted together with LSM. The Baveno VII consensus suggested that CSPH can be ruled out in patients with a LSM ≤15 kPa and normal platelet count ($\geq 150 \times 10^9$/L) and ruled in if the LSM is ≥25 kPa (at least in patients with HBV/HCV, ALD and non-obese MASLD) [221]. However, many patients (up to 40%–60%) with cACLD cannot be classified for their CSPH risk by LSM alone (*i.e.,* they remain in the gray zone because of an LSM of 15.1–24.9 kPa) and/or platelet count $<150 \times 10^9$/L [209,222,223]. In this context, the addition of SSM to LSM and PLT can reduce the proportion of patients in the CSPH gray zone and improve the prediction of CSPH or future risk of hepatic decompensation.

Table 6
Combined Baveno VII criteria and SSM for the detection of clinically significant portal hypertension

	Baveno VII–SSM single-cutoff model	Baveno VII–SSM dual-cutoff model
Rule out CSPH if	≥2 of the following criteria: LSM <15 kPa Platelet count $\geq 150 \times 10^9$/L SSM ≤40 kPa	≥2 of the following criteria: LSM <15 kPa Platelet count $\geq 150 \times 10^9$/L SSM <21 kPa
Rule in CSPH if	≥2 of the following criteria: LSM ≥25 kPa Platelet count $<150 \times 10^9$/L SSM >40 kPa	≥2 of the following criteria: LSM ≥25 kPa Platelet count $<150 \times 10^9$/L SSM >50 kPa

CSPH, clinically significant portal hypertension; LSM, liver stiffness measurement; SSM, spleen stiffness measurement.

A recent systematic review and individual patient data meta-analysis examined this concept of combined Baveno VII and SSM and included data of 1245 adult patients from 17 studies [224]. For patients undergoing VCTE (600 patients), the assessment was based on the presence of at least two of three criteria by LSM, platelet count and SSM at either a single SSM cutoff at 40 kPa or dual SSM cutoffs (SSM <21 kPa or >50 kPa) model (Table 6). In the Baveno VII SSM single cutoff model, the sensitivity, specificity and positive and negative predictive values for CSPH were 0.93, 0.86, 0.92 and 0.85, respectively, and 9% of the patients were classified in the gray zone (compared with 48% of patients classified in the gray zone by the Baveno VII criteria alone). In the Baveno VII SSM dual-cutoff model, the sensitivity, specificity and positive and negative predictive values for CSPH were 1.00, 0.89, 0.94 and 0.98, respectively, and 32% of the patients were classified in the gray zone. Two-dimensional SWE appeared to perform similarly well though the data were restricted to 225 patients, and data were insufficient to evaluate pSWE. As such, in patients with parenchymal liver disease (*i.e.,* in the absence of signs of non-cirrhotic portal hypertension), it seems unnecessary to perform SSM when the LSM is ≤15 kPa and the platelet count is $\geq 150 \times 10^9$/L because SSM would not have an impact on clinical decision making.

One randomized controlled trial [225] randomized 548 patients (1:1) to undergo upper gastrointestinal endoscopy to screen for varices or to undergo LSM and SSM by VCTE, with endoscopy performed only in patients with an LSM ≥12.5 kPa and/or SSM ≥41.3 kPa. The study achieved non-inferiority for the detection of both any varices (18.6% in the VCTE arm vs. 24.5% in the endoscopy arm) and varices requiring treatment (4.0% vs. 5.8%). At a mean follow-up of 41 mo, the incidence of acute variceal hemorrhage was again similar in the VCTE arm (4.4%) and endoscopy arm (4.0%) [226].

Spleen stiffness measurement seems to be of value in monitoring dynamic changes in portal hypertension. In one study, reduction of SSM by pSWE [227] reflected decreases in portal pressure (*i.e.,* measured by HVPG) on carvedilol therapy in 106 patients with high-risk varices, and importantly, SSM reduction predicted hemodynamic response to carvedilol.

However, several issues of SSM need to be addressed: in the first studies on VCTE SSM, the VCTE probes introduced for LSM that operate at a frequency of 50 Hz with a stiffness ceiling at 75 kPa were used. Currently, a dedicated VCTE SSM probe operating at a frequency of 100 Hz is available, however it is not well known if the values may differ. Thus, future studies should define the optimal cutoffs for the VCTE SSM obtained with the 100 Hz probe. Second, SSM is technically challenging, especially in individuals with normal spleen size and/or with obesity. Finally, reliability criteria for SSM results have not been established, which seems particularly relevant for the 2D-SWE and pSWE techniques.

Recommendation 24. SSM should be assessed and interpreted together with LSM, and it is useful to assess the risk of CSPH, varices and future variceal hemorrhage (LoE 2b, GoR B). Strong consensus (12/0/0, 100%).
Recommendation 25. SSM may be considered when the LSM is ≥10 kPa or clinical/radiologic features suggestive of CSPH are present (LoE 3b, GoR B). Strong consensus (12/0/0, 100%).
Recommendation 26. A VCTE SSM <21 kPa rules out CSPH in patients who also have a VCTE LSM ≤15 kPa and/or PLT ≥150 × 10^9/L (LoE 2a, GoR B). Strong consensus (15/0/0, 100%).
Recommendation 27. A VCTE SSM >40 kPa rules in CSPH in patients who also have a VCTE LSM ≥25 kPa and/or PLT <150 × 10^9/L (LoE 2a, GoR B). Strong consensus (12/0/0, 100%).

Conflict of interest

G.F. has received a speaker honorarium from Canon Medical Systems, Fujifilm Healthcare, Mindray Medical Imaging, Philips Ultrasound and Siemens Healthineers. She has served on advisory boards for Philips Healthcare and Siemens Healthineers, and her university has received ultrasound equipment grants and unrestricted research grants from Canon Medical Systems, Esaote SpA, Fujifilm Medical Systems and Philips Ultrasound. She receives royalties from Elsevier. R.G.B. has received a speaker honorarium from Canon Medical systems, Philips Ultrasound, Siemens Healthineers, Mindray, Samsung Ultrasound and Hologic Ultrasound. He has received research grants from Philips Ultrasound, Canon Ultrasound, Canon MRI, Samsung, Siemens Healthineers, Hologic and Mindray, and equipment grants from Canon Medical Systems, Philips Ultrasound, Mindray, Samsung Ultrasound and Siemens Healthineers. He is on the advisory board of Lantheus Medical. He receives royalties from Thieme and Elsevier. A.B. is consultant for Boehringer–Ingelheim and has received speaker honoraria from GE Healthcare and Hologic Ultrasound. She has received research ultrasound equipment support from GE Healthcare. I.S. has received speaker fees from Siemens Healthineers, General Electric, Samsung Ultrasound and Canon Medical Systems. V.W.S.W. served as a consultant or advisory board member for AbbVie, Boehringer Ingelheim, Echosens, Gilead Sciences, Intercept, Inventiva, Novo Nordisk, Pfizer, Sagimet Biosciences, TARGET PharmaSolutions and Visirna, and as a speaker for Abbott, AbbVie, Gilead Sciences, Novo Nordisk and Unilab. He has received a research grant from Gilead Sciences and is a co-founder of Illuminatio Medical Technology. T.R. received grant support from Abbvie, Boehringer Ingelheim, Gilead, Intercept/Advanz Pharma, MSD, Myr Pharmaceuticals, Philips Healthcare, Pliant, Siemens and W. L. Gore & Associates; speaking honoraria from Abbvie, Gilead, Intercept/Advanz Pharma, Roche, MSD and W. L. Gore & Associates; a consulting/advisory board fee from Abbvie, Astra Zeneca, Bayer, Boehringer Ingelheim, Gilead, Intercept/Advanz Pharma, MSD, Resolution Therapeutics and Siemens; and travel support from Abbvie, Boehringer Ingelheim, Dr. Falk Pharma, Gilead and Roche. T.K. has received speaker honoraria from Echosens, Falk Foundation and Jazz Pharmaceuticals, and has served on advisory boards for Echosens and his university has received ultrasound equipment and unrestricted research grants from Canon Medical Systems and Echosens. M.T. has received a speaker honorarium from Siemens Healthcare, Echosens, Norgine, Madrigal, Takeda and Tillotts Pharma and an advisory fee from Boehringer Ingelheim, AstraZeneca and GSK; she is a co-founder and board member of Evido and is a board member for Alcohol & Society (non-governmental organization). ACC has received speaker honorarium from Novo Nordisk. O.T.A. has served as a consultant or adviser to Novo Nordisk, Resonance Health, Norgine and Sun Pharma; he has received research ultrasound equipment support from Canon Medical Systems. L.C. has received consulting fees from Boston Pharmaceutical, Echosens, Gilead, GSK, Madrigal, MSD, Novo Nordisk, Pfizer, Sagimet and Siemens Healthineers, and speaker fees from Echosens, Gilead, Inventiva, Madrigal and Novo Nordisk. C.F.D. has received a speaker honorarium from AbbVie, Bracco, Falk Foundation, Fujifilm Healthcare, GE Healthcare, Janssen, Mindray Medical Imaging, Olympus, Pentax, Siemens Healthcare and Sonoscape. H.I. has received research grants from Canon Medical Systems and GE Healthcare and research ultrasound equipment support from Canon Medical Systems. D.H.L. has received research grants and a speaker honorarium from Canon Medical Systems. W.K. has received a speaker honorarium from Novo Nordisk, Eaisi and Roche. C.P.O. has been a consultant or participant in clinical trials for NovoNordisk, Pfizer, Inventiva, Astra-Zeneca and Boehringer Ingelheim. S.K.S. declares no conflicts of interest.

Acknowledgments

The authors thank WFUMB secretary Lynne Rudd for her invaluable and continuous support during the development of this document.

Data availability statement

These guidelines were produced by a panel of experts and are based on evidence from the literature. No unpublished research data of the authors were used.

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