performed at the bedside or in the out-patient clinic is needed to better define the correct interpretation of TE individual data. Indeed, these frequent or rare diseases may be occasionally encountered in patients with hepatitis (B, C, auto immune), HIV infection, NASH, iron overload or alcohol-related disorders.

Our observation underscores the inherent challenges to pathologic diagnosis or even the limited assessment of fibrosis or cirrhosis using a surrogate biochemical or indirect physical measurements; no alternative alone can or should be translated literally into the more complex designation of *cirrhosis*, and neither should the term *fibrosis* be used synonymously with *cirrhosis* [5]. So far there is now clinical evidence that TE does not measure fibrosis exclusively (for review see 2).

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doi:10.1016/j.jhep.2008.11.003

Interpreting liver stiffness in the cirrhotic range: What are we measuring?

To the Editor:

We thank Dr. Bioulac-Sage and colleagues for their interest in our review. The case of cardiac hepatopathy they report emphasizes the fact that interpretation of the results of transient elastography (TE) and other non-invasive tests should always be done by expert clinicians according to the clinical context. Indeed, although the clinical and biological features of this patient could be consistent with the diagnosis of cirrhosis related to NASH, the patient had a past history of severe cardiac disease (myocardial infarction, triple coronary bypasses, and an implantable cardioverter–defibrillator). No information regarding physical examination or ultrasonography is provided by the authors. The presence of enlarged liver and/or hepato-jugular reflux on physical examination together with dilated hepatic veins and inferior vena cava, suggestive of chronic heart failure, would have helped to anticipate the findings of liver biopsy. Although we believe that combining TE with serum non-invasive markers such as Fibrotest increases diagnostic accuracy, we emphasize that so far, it has only been studied in patients with hepatitis C [1]. The discrepancy between the two TE results is surprising within such a short period of time (2 months): "quality criteria" (interquartile range (IQR) and success rate) for interpretation of TE results are not provided by the authors but measurements are said to be satisfactory. Although TE has been shown to be a reproducible technique, longitudinal data are still lacking. Preliminary results regarding liver stiffness dynamics in a control group of untreated patients with chronic hepatitis C suggest that liver stiffness values are stable over time [2]. Thus, this discrepancy remains difficult to explain.

As stressed by the authors, it is likely that perisinuosidal fibrosis contributed to the increased liver stiffness values. As suggested by morphometric studies [3], liver stiffness seems to accurately reflect the amount of liver fibrosis whatever its location and influence on liver architecture. Consistent with this finding, marked perisinusoidal fibrosis was reported in 10 out of 45 patients

without cirrhosis but with increased liver stiffness values suggestive of cirrhosis (>14.6 kPa) [4]. Also, a good correlation between liver stiffness values and the degree of perisinusoidal fibrosis has been shown in a recent study in alcoholic patients in whom perisinusoidal fibrosis is common [5]. However, in multivariate analysis, liver stiffness values were only correlated with Metavir Fibrosis score. Apart from fibrosis, other parameters such as necro-inflammatory activity may also influence liver stiffness measurements [6], but the reported patient had normal transaminase levels and no inflammation on liver biopsy. Finally, the influence of architectural disturbances such as sinusoidal dilatation on liver stiffness measurements remains unknown. More data are awaited on that specific issue in patients with sinusoidal and vascular diseases.

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doi:10.1016/j.jhep.2008.11.004