Position Paper

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Revising consensus in portal hypertension: Report of the Baveno V consensus workshop on methodology of diagnosis and therapy in portal hypertension

Roberto de Franchis*, On behalf of the Baveno V Faculty [1]

Department of Medical Sciences, University of Milan, Head, Gastroenterology 3 Unit, IRCCS Ca' Granda Ospedale Maggiore Policlinico Foundation, Milan, Italy

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Introduction

Portal hypertension is associated with the most severe complications of cirrhosis, including ascites, hepatic encephalopathy, and bleeding from gastro-esophageal varices. Despite the progress achieved over the last decades, the 6-week mortality associated with variceal bleeding is still in the order of 10–20%. Awareness of the difficulty inherent to the evaluation of diagnostic tools and the design and conduct of good clinical trials for the treatment of portal hypertension has led to the organization, since 1986, of a series of consensus meetings. The first one was organized by Andrew Burroughs in Groningen, The Netherlands [1]. After Groningen, other meetings followed, in Baveno in 1990 (Baveno I) [2] and in 1995 (Baveno II) [3,4], in Milan in 1992 [5], in Reston, USA, in 1996 [6], in Stresa in 2000 (Baveno III) [7,8], again in Baveno in 2005 (Baveno IV) [9,10] and in Atlanta in 2007 [11].

The aims of these meetings were to develop definitions of key events in portal hypertension and variceal bleeding, to review the existing evidence on the natural history, the diagnosis and the therapeutic modalities of portal hypertension and to issue evidence-based recommendations for the conduct of clinical trials and the management of patients. All these meetings were successful and produced consensus statements on some important points, although some issues remained unsettled.

To continue the work of the previous meetings, a Baveno V workshop was held on May 21–22, 2010. The workshop was attended by many of the experts responsible for most of the major achievements of the last years in this field. Many of them had attended the previous meetings as well.

The main fields of discussion of the Baveno V workshop were the same as in Baveno I–IV, i.e. the definitions of key events concerning the bleeding episode and the therapeutic options in patients with portal hypertension. For each of these topics, a series of consensus statements were discussed and agreed upon. As in Baveno IV, whenever applicable, the level of existing evidence was evaluated and the recommendations were ranked according to the Oxford System [12] (i.e.: level of evidence from 1 = highest to 5 = lowest; grade of recommendation from A = strongest, to D = weakest).

The presentations given during the workshop are reported '*in extenso*' in the Baveno V proceedings [13]. A summary of the most important conclusions is reported here.

Definition of key events regarding the bleeding episode

Definitions and criteria to evaluate failure to control bleeding and failure to prevent re-bleeding were introduced at Baveno II [3,4] and reviewed at Baveno III [7,8]. Since it was found that some of them were rather difficult to apply and did not adequately reflect the situation in clinical practice, new definitions and criteria were proposed at Baveno IV [9,10].

The Baveno IV criteria are reported below:

Baveno IV definitions and criteria for failure to control bleeding

(1) The time frame for the acute bleeding episode should be 120 h (5 days).
(2) Failure signifies the need to change therapy: one criterion defines failure, whichever occurs first:
   a. Fresh hematemesis $\geqslant$2 h after the start of a specific drug treatment or therapeutic endoscopy. In the minority of patients who have a naso-gastric tube in place, aspiration of greater than 100 ml of fresh blood represents failure.
   b. 3 g drop in Hb ($\approx$9% drop in Ht) if no transfusion is administered.
   c. Death
   d. Adjusted blood transfusion requirement index (ABRI, see below) $\geqslant$0.75 at any time point. (The threshold of ABRI defining failure requires validation).

Adjusted blood requirement index (ABRI)

$$\text{ABRI} = \frac{\text{Blood Units transfused}}{[\text{final Ht} - \text{initial Ht}) + 0.1]}$$

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* Address: Gastroenterology 3 Unit, IRCCS Ca' Granda Ospedale Maggiore Policlinico Foundation, Milan, Padiglione Beretta Est, Via F Sforza 35, 20122 Milan, Italy. Tel.: +39 02 5503 5331/2; fax: +39 02 5503 5271.
E-mail address: roberto.defranchis@unimi.it.
[1] The members of Baveno V Faculty given before references.

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- Ht (or Hb) is measured at least every:
  6 h for the first 2 days
  12 h for days 3–5
- The transfusion target should be an haematocrit of 24% or a haemoglobin of 8 g/dl.

Baveno IV definitions and criteria for failure of secondary prophylaxis

Failure to prevent re-bleeding is defined as a single episode of clinically significant re-bleeding from portal hypertensive sources.

Clinically significant re-bleeding:

(a) Hematemesis/melaena. In the minority of patients who have a naso-gastric tube in place, aspiration of greater than 100 ml of fresh blood represents failure, plus

(b) Adjusted Blood Requirement Index (ABRI) $\geqslant$0.5 (The threshold of ABRI defining failure requires validation), or

(c) Decrease 3 g of Hb if no transfusion is given.

After Baveno IV, the diagnostic performance of the Baveno II–III and Baveno IV criteria was evaluated by analysing the population of a study of the use of recombinant factor VII in acute variceal bleeding [14]. The conclusions of the study were as follows: Baveno IV criteria have a rather high accuracy; ABRI in its current definition does not add to the accuracy of the other Baveno IV criteria; the best timing for measurement of hematocrit and the ideal cut off value of ABRI score should be further investigated.

As a consequence, at Baveno V the Baveno IV consensus statements were modified as follows:

Baveno V definitions and criteria for failure to control bleeding

- The time frame for the acute bleeding episode should be 120 h (5 days).
- Failure is defined as death or need to change therapy defined by one of the following criteria: (2b;B)
- Fresh hematemesis or NG aspiration of $\geqslant$100 ml of fresh blood $\geqslant$2 h after the start of a specific drug treatment or therapeutic endoscopy.
- Development of hypovolaemic shock.
- 3 g drop in Hb (9% drop of Ht) within any 24 h period if no transfusion is administered. This time frame needs to be further validated.
- The potential value of an index of blood transfusion requires prospective validation (5;D).

Baveno V definitions and criteria for failure of secondary prophylaxis

- Failure to prevent re-bleeding is defined as a single episode of clinically significant re-bleeding from portal hypertensive sources after day 5 (5;D).
- Clinically significant re-bleeding: recurrent melena or hematemesis resulting in any of the following:

1. hospital admission,
2. blood transfusion,
3. 3 g drop in Hb,
4. death within 6 weeks.

Areas requiring further study (5;D)

- Prospective validation of Baveno IV and V criteria and comparison with Baveno II and III definitions.
- Interactions of time events with prognostic factors.
- Definition and usefulness of a transfusion index for failure criteria:
  Clinical applicability.
  Appropriate for randomised trials.
  Expected response to transfusions/within determined policy of transfusion.

Therapeutic options in patients with portal hypertension

Pre-primary prophylaxis (prevention of the formation of varices)

Background

- Prevention of the development of complications of portal hypertension is an important area of research (5;D).
- Hepatic venous pressure gradient (HVPG) $\geqslant$10 mm Hg is predictive of varices formation and decompensation (1b;A).

Recommendations for management

- All cirrhotic patients should be screened for varices at diagnosis (5;D).
- Pre-primary prophylaxis should only include patients without gastro-esophageal varices (5;D).
- Treatment of underlying liver disease may reduce portal hypertension and prevent its clinical complications (1b;A).
- There is no indication, at this time, to use beta-blockers to prevent the formation of varices (1b;A).
- HVPG measurement in pre-primary prophylaxis may be recommended only in the context of clinical trials (5;D).

Areas requiring further study (5;D)

- Basic mechanisms in the development and progression of portal hypertension.
- Non-invasive techniques to identify patients with clinically significant portal hypertension.
- The impact of treating the underlying chronic liver disease in the development of varices and other portal hypertensive related complications.
- Treatments to prevent the development of varices and other portal hypertensive related complications in different risk groups (e.g. patients with HVPG between 6 and 10 mm Hg and those with HVPG $\geqslant$10 mm Hg).

Prevention of the first bleeding episode

Patients with small varices

- Patients with small varices with red wale marks or Child C class have an increased risk of bleeding (1b;A) and should be treated with nonselective beta-blockers (NSBB) (5;D).

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- Patients with small varices without signs of increased risk may be treated with NSBB to prevent progression of varices and bleeding (1b;A). Further studies are required to confirm their benefit.

Patients with medium-large varices

- Either NSBB or endoscopic band ligation (EBL) is recommended for the prevention of the first variceal bleeding of medium or large varices (1a; A).
- The choice of treatment should be based on local resources and expertise, patient preference and characteristics, side effects, and contra-indications (5;D).
- Carvedilol is a promising alternative (1b;A) which needs to be further explored.
- Shunt therapy, endoscopic sclerotherapy, and isosorbide mononitrate alone should not be used in the prophylaxis of first variceal bleeding (1a;A).
- There is insufficient data to recommend the use of NSBB in combination with Isosorbide-5-Mononitrate (ISMN), spironolactone, or EBL for primary prophylaxis (1b;A).

Patients with gastric varices

- Despite the absence of specific data on prophylactic studies, patients with gastric varices may be treated with NSBB (5;D).

Role of HVPG measurement

- In centers where adequate resources and expertise are available, HVPG measurements should be routinely used for prognostic and therapeutic indications (5;D).
- Controlled trials using pharmacological therapy in primary prophylaxis should include HVPG measurements (5;D).
- A decrease in HVPG of at least 20% from baseline or to $\leqslant$12 mm Hg after chronic treatment with NSBB is clinically relevant in the setting of primary prophylaxis (1a;A).
- Acute HVPG response to intravenous propranolol may be used to identify responders to beta-blockers, specifically a decrease in HVPG of 10% or to $\leqslant$12 mm Hg may be relevant in this setting (1b;A).

Areas requiring further study

- Studies evaluating the use of carvedilol.
- Studies evaluating novel therapeutic options.

Treatment of acute bleeding from varices

Blood volume restitution

- The goal of resuscitation is to preserve tissue perfusion. Volume restitution should be initiated to restore and maintain hemodynamic stability.
- PRBC transfusion should be done conservatively at a target hemoglobin level between 7 and 8 g/dl., although transfusion policy in individual patients should also consider other factors such as co-morbidities, age, hemodynamic status and ongoing bleeding (1b;A).
- Recommendations regarding management of coagulopathy and thrombocytopenia cannot be made on the basis of currently available data (5;D).
- PT/INR is not a reliable indicator of the coagulation status in patients with cirrhosis (1b;A).

Antibiotic prophylaxis

- Antibiotic prophylaxis is an integral part of therapy for patients with cirrhosis presenting with upper gastrointestinal bleeding and should be instituted from admission (1a;A).
- Oral quinolones are recommended for most patients (1b;A).
- Intravenous ceftriaxone should be considered in patients with advanced cirrhosis (1b;A), in hospital settings with high prevalence of quinolone-resistant bacterial infections and in patients on previous quinolone prophylaxis (5;D).

Prevention of hepatic encephalopathy

- Recommendations regarding management and prevention of encephalopathy in patients with cirrhosis and upper GI bleeding cannot be made on the basis of currently available data (5;D).

Assessment of prognosis

- HVPG $\geqslant$20 mm Hg, Child-Pugh class C, and active bleeding at endoscopy are the variables most consistently found to predict 5-day treatment failure (2b;B).
- Child-Pugh class C, MELD score $\geqslant$ 18, and failure to control bleeding or early re-bleeding are the variables most consistently found to predict 6-week mortality (2b;B).

Timing of endoscopy

- Patients with GI bleeding and features suggesting cirrhosis should have upper endoscopy as soon as possible after admission (within 12 h) (5;D).

Pharmacological treatment

- In suspected variceal bleeding, vasoactive drugs should be started as soon as possible, before endoscopy (1b;A).
- Vasoactive drugs (terlipressin, somatostatin, octreotide, vapreotide) should be used in combination with endoscopic therapy and continued for up to 5 days (1a;A).

Endoscopic treatment

- Endoscopic therapy is recommended in any patient who presents with documented upper GI bleeding and in whom esophageal varices are the cause of bleeding (1a;A).
- Ligation (EVL) is the recommended form of endoscopic therapy for acute esophageal variceal bleeding, although sclerotherapy may be used in the acute setting if ligation is technically difficult (1b;A).
- Endoscopic therapy with tissue adhesive (e.g. *N*-butyl-cyanoacrylate) is recommended for acute bleeding from isolated gastric varices (IGV) (1b;A) and those gastro-esophageal varices type 2 (GOV2) that extend beyond the cardia (5;D).
- EVL or tissue adhesive can be used in bleeding from gastro-esophageal varices type 1 (GOV1) (5;D).

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Early TIPS placement

- An early TIPS within 72 h (ideally ⩽24 h) should be considered in patients at high-risk of treatment failure (e.g. Child-Pugh class C <14 points or Child class B with active bleeding) after initial pharmacological and endoscopic therapy (1b;A).

Use of balloon tamponade

- Balloon tamponade should only be used in massive bleeding as a temporary "bridge" until definitive treatment can be instituted (for a maximum of 24 h, preferably in an intensive care facility) (5;D).

Use of self-expandable metal stents

- Uncontrolled data suggest that self-expanding covered esophageal metal stents may be an option in refractory esophageal variceal bleeding, although further evaluation is needed (4;C).

Management of treatment failures

- Persistent bleeding despite combined pharmacological and endoscopic therapy is best managed by TIPS with PTFE-covered stents (2b;B).
- Re-bleeding during the first 5 days may be managed by a second attempt at endoscopic therapy. If re-bleeding is severe, PTFE-covered TIPS is likely the best option (2b;B).

Areas requiring further study

- The need for correction of coagulation disorders. Influence of coagulopathy and thrombocytopenia on outcome.
- Improve prognostic models: Better stratification of risk to determine timing of the initial endoscopy, duration of drug therapy and type of treatment.
- Treatment and prevention of HE.
- Best antibiotic.
- Role of self-expandable esophageal stents.
- Treatment of gastric varices.
- Treatment of paediatric patients: no studies define the best approach.
- Treatment of bleeding ectopic varices like duodenal varices.
- Role of erythromycin before endoscopy.

Prevention of re-bleeding

Time to start secondary prophylaxis

- Secondary prophylaxis should start as soon as possible from day 6 of the index variceal episode (5;D).
- The start time of secondary prophylaxis should be documented.

Patients with cirrhosis

- Combination of beta-blockers and band ligation is the preferred therapy as it results in lower re-bleeding compared to either therapy alone (1a;A).
- Hemodynamic response to drug therapy provides information about re-bleeding risk and survival (1a;A).
- The addition of ISMN to beta-blockers may improve the efficacy of treatment in hemodynamic non-responders (5;D).

Patients with cirrhosis who are unable or unwilling to be treated with EVL

- Beta-blockers with Isosorbide Mononitrate is the preferred option (1a;A).

Patients with cirrhosis who have contra-indications or intolerance to beta-blockers

- Band ligation is the preferred treatment (5;D).

Patients who fail endoscopic and pharmacological treatment for the prevention of re-bleeding

- Transjugular Intra-hepatic Porto-systemic Shunt (TIPS) with Polytetrafluoroethylene (PTFE)-covered stents is effective and is the preferred option. Surgical shunt in Child-Pugh A and B patients is an alternative if TIPS is unavailable (2b;B).
- Transplantation provides good long-term outcomes in appropriate candidates and should be considered (2b;B). TIPS may be used as a bridge to transplantation (4;C).

Patients who have bled from isolated gastric varices type I (IGV1) or gastro-oesophageal varices type 2 (GOV2)

- *N*-butyl-cyanoacrylate (1b;A) or TIPS (2b;B) are recommended.

Patients who have bled from gastro-oesophageal varices type 1 with (GOV1)

- May be treated with *N*-butyl-cyanoacrylate, band ligation of oesophageal varices or beta-blockers (2b;B).

Patients who have bled from portal hypertensive gastropathy

- Beta-blockers (1b;A) should be used for prevention of recurrent bleeding.

Patients in whom beta-blockers are contraindicated or fail and who cannot be managed by non-shunt therapy

- TIPS (4;C) or surgical shunts (4;C) should be considered.

Non-cirrhotic portal hypertension

Similar to Baveno IV, a session in Baveno V was devoted to non-cirrhotic portal hypertension, focusing on the Budd-Chiari syndrome and extra-hepatic portal vein obstruction.

Budd-Chiari syndrome [BCS – hepatic venous outflow tract obstruction (HVOTO)]

Definition

- Budd-Chiari syndrome can be located from the level of the small hepatic veins to the level of the termination of inferior vena cava into the right atrium.
- BCS is a heterogeneous condition with regard to causes and pathogenesis.
- BCS is considered secondary when the mechanism for HVOTO is compression/invasion by a benign or malignant tumour, abscess or cyst.
- BCS is considered primary otherwise.

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Aetiology

- Myeloproliferative diseases should be investigated in all patients with primary BCS, first by testing for V617F JAK2 mutation in peripheral blood. When V617F JAK2 is undetectable, further tests for myeloproliferative diseases should be performed (e.g. molecular testing and bone marrow biopsy) (2b;B).
- When liver synthetic function is impaired, low plasma levels of antithrombin, protein C, and protein S should not be considered as specific for an inherited defect unless it is already known in family members. Similarly, anticardiolipin antibodies at low titres and increased serum homocysteine levels may not reflect underlying prothrombotic conditions (3b;B).

Diagnosis

- BCS is diagnosed by the demonstration of an obstruction of the venous lumen, or by the presence of hepatic vein collaterals (4;C).
- Liver biopsy is not necessary to make a diagnosis of BCS when vascular imaging has demonstrated obstruction of the hepatic venous outflow tract (4;C).
- Liver biopsy is the only means to make a diagnosis of BCS of the small intra-hepatic veins (4;C).
- Hepatic nodules are frequent and most often are benign. HCC may occur and therefore the patient should be referred to centers experienced in managing BCS (5;D).

Management

- Controlled clinical trials for BCS have not been performed, hence the current recommendations for therapy are based on cohort studies and expert opinion (5;D).
- Management of BCS should be undertaken at centers with experience in this condition.
- Anticoagulation should be recommended to all patients, in the absence of major contra-indications (5;D).
- Previous bleeding related to portal hypertension is not considered a major contra-indication for anticoagulation, provided appropriate prophylaxis for recurrent bleeding is initiated (5;D).
- Complications of portal hypertension may be treated as recommended for the other types of liver diseases (5;D).
- Stenoses that are amenable to percutaneous angioplasty/stenting should be actively looked for, and treated accordingly (5;D).
- TIPS insertion should be attempted by experts when angioplasty/stenting is not feasible, and when the patient does not improve on medical therapy (5;D).
- Liver transplantation should be considered in patients with manifestations refractory to the above procedures (5;D).
- More data are needed to provide a definition of treatment failure (5;D).
- The response to treatment should be closely monitored by assessing sodium and water balance, serum ALT levels, serum bilirubin level and the occurrence of complications of treatment (5;D).
- A satisfactory long term control of the disease is indicated by the absence of clinically detectable ascites, jaundice, encephalopathy, gastrointestinal bleeding, and bacterial infection together with a good performance status, regardless of liver tests (4;C).

Extra-hepatic portal vein obstruction (EHPVO)

Definition

- EHPVO is defined by obstruction of the extra-hepatic portal vein with or without involvement of the intra-hepatic portal veins and does not include isolated thrombosis of splenic vein or superior mesenteric vein (SMV).
- EHPVO is characterized by features of recent thrombosis or of portal hypertension with portal cavernoma as a sequel of portal vein obstruction.
- Presence of cirrhosis and/or malignancy should be stated.

Aetiology

- EHPVO is a heterogeneous entity with regards to causes and pathogenesis.
- EHPVO is frequently associated with one or several risk factors for thrombosis which may be occult at presentation.
- Presence of cirrhosis, malignancy and other intra-abdominal causes such as inflammation, trauma, etc. do not exclude the presence of systemic risk factors.

Clinical presentation

- Recent EHPVO: can be assumed when patients present with symptoms such as abdominal pain, ascites, or fever in the absence of portal cavernoma and porto-systemic collaterals. Patients also can be asymptomatic (5;D).
- Chronic EHPVO: is associated with portal cavernoma.

Diagnosis

- EHPVO is diagnosed by Doppler US, CT, or MRI, which demonstrate portal vein obstruction, presence of intraluminal material or portal vein cavernoma.

Natural history

- The natural course of EHPVO is mainly determined by the presence or absence of associated diseases such as cirrhosis or malignancy.
- Most patients with EHPVO in the absence of cirrhosis and malignancy have a relatively benign course.
- Morbidity is mainly related to variceal bleeding, recurrent thrombosis, symptomatic portal biliopathy, and hypersplenism.

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Classification
In classifying EHPVO, the following characteristics should be specified (5;D):

- Site of PVT
- Presentation
- Type of underlying liver disease
- Degree of portal vein occlusion (incomplete or total)
- Extent of involvement of extra-hepatic portal venous system

Treatment: recent EHPVO: anticoagulation
- Recent EHPVO rarely resolves spontaneously.
- In non-cirrhotic patients with symptomatic recent EHPVO, low molecular weight heparin should be started immediately followed by oral anticoagulant therapy (2b;B). In asymptomatic patients, anticoagulation should be considered.
- Anticoagulation should be given for at least three months, unless an underlying persistent prothrombotic state has been documented, in which case life-long anticoagulation is recommended (5;D).
- Antibiotic therapy should be given if there is any evidence of SIRS/infection (5;D).

Treatment: chronic EHPVO: anticoagulation
- In patients with chronic EHPVO, there is no consensus on the indication for anticoagulant therapy.
- However, in those patients with a persistent documented prothrombotic state, anticoagulant therapy can be considered (5;D).
- There is insufficient evidence in favour of interventional therapy such as TIPS and local thrombolysis.

Treatment: bleeding
- For primary prophylaxis of variceal bleeding there is insufficient data on whether beta-blockers or endoscopic therapy should be preferred.
- For control of acute variceal bleeding, endoscopic therapy is effective (2b;B).
- For secondary prophylaxis endoscopic therapy is effective (2a;B). There is preliminary evidence to suggest that beta-blockers are as effective as endoscopic ligation therapy.
- Decompressive surgery or interventional radiological procedures should be considered for patients with failure of endoscopic therapy (5;D).
- Mesenteric-left portal vein bypass (Rex bypass) is preferred in managing bleeding from paediatric patients with chronic EHPVO, if feasible (2b;B).

Portal biliopathy-diagnosis
- Portal biliopathy is present in nearly all patients with EHPVO. In the majority, it is asymptomatic.
- MRCP is the first line of investigation.
- ERCP is only recommended if a therapeutic intervention is contemplated.

Portal biliopathy-treatment
Asymptomatic: No treatment (5;D).
Symptomatic:
- Bile duct stones: Endoscopic therapy.
- Common bile duct stricture: Endoscopic stenting; (3b;B) and porto-systemic shunt surgery should be considered, whenever possible, (3b;B). If not relieved by the above, hepatico-jejunostomy may be considered (3b;B).

Chronic EHPVO in children: treatment
- Mesenteric-left portal vein bypass (Rex bypass) should be considered in all children with complications of chronic EHPVO, who should be referred to centers with experience in treating this condition (5;D).

Unresolved issues and future studies
- Prospective data on the frequency and clinical profile of recent and chronic EHPVO.
- Natural history of EHPVO in children vs. adults; hepatic dysfunction.
- Primary prophylaxis of variceal bleeding.
- Case–control studies on frequency of prothrombotic states in EHPVO (particularly in the East), identification of high-risk population.
- Usefulness of long-term anticoagulants, emergency TIPS, Rex shunt surgery.
- Assessment of factors associated with treatment failure, disease progression and thrombosis recurrence.
- Experimental models of recent and chronic EHPVO.
- Management of ectopic varices.

Other issues

In Baveno IV, a session was devoted to predictive models in portal hypertension, during which classification stages of cirrhosis were proposed. Prospective validation of this classification is under way.

Conclusions

The purpose of the consensus definitions about the variceal bleeding episode is to use them in trials and other studies on portal hypertension, as well as in clinical practice. This does not mean that authors cannot use their own definitions, but they are encouraged to use and evaluate in parallel these Baveno V consensus definitions. This should result in some measure of standardisation and increased ease of interpretation among different studies. Equally important, if there are uniformly defined end-points, meta-analyses will be based on more homogeneous studies, which is an essential pre-requisite of this methodology. It is desirable that future studies be reported using these definitions as part of the evaluation. Change or refinement can then take place, as they have at Baveno V with respect to the previous consensus meetings, to ensure that the consensus definitions do have clinical relevance and are easily applied in practice.

Several definitions agreed upon in the previous Baveno workshops were taken for granted and not discussed in Baveno V. Interested readers can refer to the Baveno I–IV reports [2–4,7–10].

The suggestions about the topics of future studies reflect the opinions of the experts about the areas where new information is most needed.

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As long as new diagnostic tools and new treatments appear, they will have to be assessed in comparison with present-day standards.

Baveno V Faculty

The following were members of the Baveno V scientific committee

Jaime Bosch, Barcelona, Spain; Andrew K Burroughs, London, UK; Gennaro D'Amico, Palermo, Italy; Roberto de Franchis, Milan, Italy; Guadalupe Garcia-Tsao, West Haven CT, USA; Norman D Grace, Boston, MA, USA; Roberto J Groszmann, West Haven, CT, USA; Didier Lebrec, Clichy, France; Carlo Merkel, Padua, Italy; Massimo Primignani, Milan, Italy; Francesco Salerno, Milan, Italy; Shiv K Sarin, New Delhi, India; Thorkild IA Sørensen, Copenhagen, Denmark.

The following chaired sessions or symposia

Jaime Bosch, Barcelona, Spain; Andrew K Burroughs, London, UK; Juan Carlos Garcia-Pagàn, Barcelona, Spain; Guadalupe Garcia-Tsao, West Haven CT, USA; Roberto J Groszmann, West Haven, CT, USA; Loren Laine, Los Angeles, CA, USA; Didier Lebrec, Clichy, France; Carlo Merkel, Padua, Italy; Shiv K Sarin, New Delhi, India; Dominique Thabut, Paris, France; Dominique Valla, Clichy, France; Candid Villanueva, Barcelona, Spain.

The following participated in the presentations and the discussion as Panelists in the consensus sessions

Argentina: Julio Vorobioff, Rosario; *Belgium:* Frederik Nevens, Leuven; *Denmark:* Flemming Bendtsen, Copenhagen; *France:* Christophe Bureau, Toulouse, Paul Calés, Angers, Jean Pierre Vinel, Toulouse; *Germany:* Tilman Sauerbruch, Bonn; *India:* Ashish Kumar, New Delhi, Yogesh Chawla, Chandigarh; *Italy:* Giovanni Barosi, Pavia, Gennaro D'Amico, Palermo, Alessandra Dell'Era, Milan, Manuela Merli, Rome, Massimo Primignani, Milan; *Pakistan:* Shaha Abid, Karachi; *Spain:* Agustin Albillos, Alcalà de Henares, Angels Escorsell, Barcelona, Cristina Ripoll, Madrid; *Taiwan:* Gin Ho Lo, Taipei; *The Netherlands:* Harry Janssen, Rotterdam; *United Kingdom:* Peter Hayes, Edinburgh; *United States:* Norman D Grace, Boston, MA, Patrick Kamath, Rochester, MN.

The following gave review lectures

Juan G Abraldes, Barcelona, Spain; Rafael Bañares, Madrid, Spain; Annalisa Berzigotti, Barcelona, Spain; Laurent Castéra, Bordeaux, France; Roberto de Franchis, Milan, Italy; Gennaro D'Amico, Palermo, Italy; Juan Carlos Garcia-Pagàn, Barcelona, Spain; Simon Ling, Toronto, Canada; Brian S Mittmann, Los Angeles, CA, USA; Richard Moreau, Clichy, France; Massimo Pinzani, Florence, Italy; Marco Senzolo, Padua, Italy; Benjamin Shneider, Pittsburgh, PA, USA; Thorkild IA Sørensen, Copenhagen, Denmark; Armando Tripodi, Milan, Italy.

Conflict of interest

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