

Hepatic venous pressure gradient measurement: Time to learn!

Ashish Kumar, Praveen Sharma, Shiv Kumar Sarin

Department of Gastroenterology, G B Pant Hospital, New Delhi, India

Portal hypertension is a clinical syndrome defined by a pathological increase in portal pressure. The development of cirrhosis of the liver is characterized by clinical manifestations related to portal hypertension like esophageal varices, ascites, bleeding, and encephalopathy. Direct measurement of portal pressure is invasive, inconvenient, and clinically impractical. Currently, the most commonly used parameter is the Hepatic Venous Pressure Gradient (HVPG), i.e., the difference between the wedged (WHVP) and the free hepatic venous pressures. HVPG represents the gradient between pressures in the portal vein and the intra-abdominal portion of inferior vena cava. When blood flow in a hepatic vein is stopped by a wedged catheter, the proximal static column of blood transmits the pressure from the preceding communicated vascular territory (hepatic sinusoids) to the catheter. Thus, WHVP reflects hepatic sinusoidal pressure and not the portal pressure itself. In the normal liver, due to pressure equilibration through interconnected sinusoids, wedged pressure is slightly lower than portal pressure, though this difference is clinically insignificant. In liver cirrhosis, the static column created by balloon inflation cannot be decompressed at the sinusoidal level due to disruption of the normal intersinusoidal communications; therefore, WHVP gives an accurate estimation of portal pressure in cirrhosis. The normal HVPG value is between 1 to 5 mmHg. Pressure higher than this defines the presence of portal hypertension, regardless of clinical evidence. HVPG ≥ 10 mmHg (termed clinically significant portal hypertension) is predictive of the development of complications of cirrhosis, including death. HVPG above 12 mmHg is the threshold pressure for variceal rupture. The main advantages of HVPG are its simplicity, reproducibility, and safety. This review summarizes the technique of the HVPG measurement.

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Portal hypertension is a clinical syndrome defined by a pathological increase in the portal venous pressure (PVP). The development of cirrhosis of the liver is characterized by clinical manifestations related to portal hypertension like esophageal varices, ascites, bleeding, and encephalopathy.¹ Because most complications of liver cirrhosis are related to portal hypertension, it would follow that a high portal pressure would be predictive of decompensation. Hallion and Francois-Frank² attempted the first PVP measurement in 1896 by inserting a cannula into the mesenteric vein of a dog and connecting it to a water manometer. This direct measurement of PVP was invasive, inconvenient, and clinically impractical. In 1951, Myers and Taylor³ first described the measurement of wedged hepatic venous pressure (WHVP), which reflected sinusoidal pressure, an indirect measure of PVP. Since then, the measurement of WHVP has been proven to be safe and closely correlated with the directly measured PVP. Currently, the most commonly used parameter is not the WHVP but the HVPG, the difference between the WHVP and the free hepatic venous pressure (FHVP). In older literature, HVPG was frequently referred as 'correct-

ed' WHVP or 'corrected' sinusoidal pressure.⁴

The HVPG represents the gradient between portal vein and intra-abdominal vena cava pressure (Fig 1). When blood flow in a hepatic vein is stopped by a wedged catheter, the proximal static column of blood transmits the pressure from the preceding communicated vascular territory (the hepatic sinusoids) to the catheter. Thus, the WHVP reflects the hepatic sinusoidal pressure and not the portal pressure itself. In the normal liver, due to the pressure equilibration through the interconnected sinusoids, WHVP is slightly lower than portal pressure, though this difference is usually insignificant.⁵ The normal HVPG value lies between 1 to 5 mm Hg. Pressure higher than this value defines the presence of portal hypertension, regardless of clinical features.⁶ In liver cirrhosis, the static column created by balloon inflation cannot be decompressed at the sinusoidal level due to disruption of the normal intersinusoidal communications. Therefore, in cirrhosis WHVP gives an accurate estimation of portal pressure. This is true for the most common etiologies of liver cirrhosis, alcoholic and viral-related.⁷ It is also well known that a threshold value of HVPG is required for the develop-

ment of varices and variceal bleeding.⁸ HVPG above 5 mmHg in the absence of clinical manifestations is considered as subclinical portal hypertension. An HVPG ≥ 10 mmHg (termed 'clinically significant portal hypertension' or CSPH) predicts the development of complications of cirrhosis, including death.⁹ HVPG above 12 mmHg is the threshold level for variceal rupture.^{8,10}

The prognostic role of HVPG for portal hypertensive bleeding is further strengthened by results of several prospective cohort studies, randomized controlled trials, and meta-analyses showing that a reduction of HVPG to <12 mmHg or a reduction of HVPG by 20% of baseline considerably reduces the risk of bleeding.¹¹⁻¹⁴ Accordingly, sequential HVPG measurements, before and at a time point after drug administration, should serve to identify patients not achieving this target HVPG reduction and therefore to tailor drug therapy or switch to another form of therapy.^{14,15,16}

The main advantages of the hepatic vein catheterization technique are its simplicity, reproducibility, and safety.

HVPG measurement

The technique

HVPG measurement is done after an overnight fast, under conscious sedation and monitoring for vital signs (including heart rate, arterial blood pressures, digital oxygen saturation, and ECG). Under local anesthesia and aseptic conditions, a venous introducer is placed in the right jugular, in the femoral, or in the antecubital vein using the Seldinger technique. Doppler ultrasound can be used to facilitate venous localization and puncture. The advantages and disadvantages of each of these three approaches are given in Table 1.

With the introducer in place, a 7-French balloon-tipped catheter (Swan-Ganz or Goodale Lubin) is advanced through it under fluoroscopic guidance and hooked

into a hepatic vein. Once the tip of the catheter is inside the hepatic vein 3 cm to 4 cm from its opening into the IVC, the position of catheter and caliber of vein is confirmed by injection of contrast. The FHVP is measured by keeping the tip of the catheter 'free' in the hepatic vein, and the WHVP is measured after inflating the catheter balloon. The wedged pressure should be recorded after the pressure tracing is stable, which may require up to 2 minutes in some cases. After measuring the pressure, adequate occlusion of the hepatic vein should be checked by (i) inability to withdraw blood on suction through the catheter, (ii) slow injection of 2-5 mL of contrast dye with the balloon inflated should show the typical 'wedged' pattern (sinusoidogram), without any reflux of the contrast or washout through communications with other hepatic veins, and (iii) lack of venous wave form. If adequate occlusion is not achieved, the reading should be discarded and a fresh reading is taken and occlusion reconfirmed. WHVP reading should always be taken before injecting the contrast; otherwise the value would be falsely high (a) because of the injected contrast, and (b) because the contrast is not a good medium for transmitting the pressure.

Three readings are taken and their mean value is used. If these readings differ by more than 1 mmHg, all readings need to be obtained again. The catheter should be carefully rinsed with heparinized normal saline before taking each set of readings. Permanent tracings should be obtained using a multichannel recorder. The total time required for the procedure ranges from 10-20 minutes. The rate of successful hepatic catheterization is greater than 95%.

Although HVPG is an easy and simple technique, accurate measurements require specific training, as the procedure differs from those used in heart catheterization laboratories and intensive care units. To achieve reliable and reproducible results, meticulous attention to detail is required. Table 2 lists a series of practical tips to ensure accurate measurements.^{17,18,19}

Table 1: Differences in various approaches for hepatic hemodynamic assessment

Factor	Antecubital approach	Jugular approach	Femoral approach
Ease	Easy cannulation	Technically difficult	Easy cannulation
Tolerability of procedure	Good since the sterile field does not include the face	Patient may feel uncomfortable as face needs to be covered	Fair
Liver biopsy	Not possible	Possible (trans-jugular)	Not possible
Post-procedure precautions	Can stand or walk shortly after the procedure	Needs to restrict neck movement for 2-3 hours	Patient needs to be in bed for 4 to 12 hours depending on coagulation impairment
Complications	Air embolism, infection	Pneumothorax, hemothorax, arrhythmias, air embolism, infection, carotid arterial puncture	Deep vein thrombosis, infection, femoral arterial puncture

FHVP

FHVP is usually close to the IVC pressure, the difference being less than 2 mmHg. Occasionally, the difference is greater than 2 mmHg. In these situations, partial or complete wedging of the catheter, inadequate placement of the catheter, or a hepatic vein obstruction must be ruled out. Because of this, it is very useful to measure the IVC pressure (at the level of the hepatic vein ostia) before measuring the hepatic venous pressures. Measurements in IVC below the hepatic vein ostia may overestimate the IVC pressure due to compression of the IVC by a hypertrophied liver.¹⁹

FHVP versus IVC pressure versus RA pressure

A few authors suggest that IVC pressure (above the liver) rather than FHVP should be recorded.²⁰ We believe that the true gradient is provided by pressure measured at RA rather than FHVP or IVC. Portal hypertension is defined by an increase in pressure in portal vein which is usually caused by downstream obstruction (Figure 1); this obstruction may be located anywhere from the portal vein to

sinusoids to hepatic veins to IVC to the right atrium. The gradient between the portal vein and the right atrium, across the obstructive lesion(s) determines the flow of blood through porto-systemic shunts including the esophageal varices. When there is no obstruction between the hepatic vein and the right atrium, the gradient between these two sites (HV to RA gradient) is 1-2 mmHg. However, this gradient is higher if there is some obstruction between these two sites (e.g. IVC web or, more commonly, hypertrophied caudate lobe compressing the IVC). This gradient is also falsely high when the HV pressure is recorded in a thin vein leading to partial wedging of the catheter. This may be obviated by measuring pressure in the IVC or RA than in the free hepatic vein. This issue is particularly important when measuring the effect of drug therapy on HVPG because the expected changes are small in magnitude (2-6 mmHg).²⁰

Thus, the use of RA pressure in place of FHVP has the following advantages: (1) The total gradient between portal vein and the heart, which determines the risk of flow

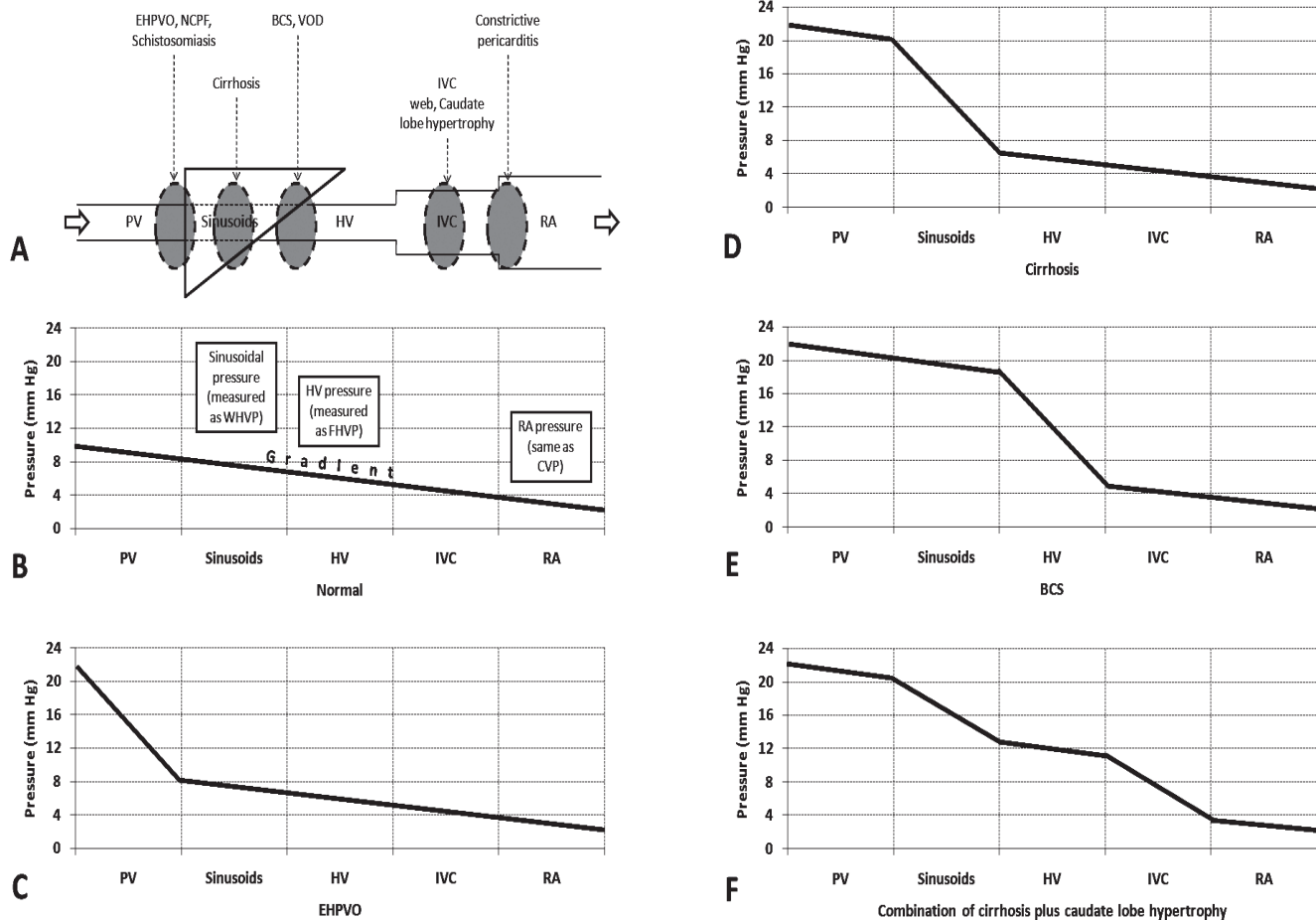


Figure 1: Schematic diagram representing various levels of obstruction in portal and hepatic circulation, and corresponding changes in pressure

through porto-systemic shunts, is determined, rather than just the gradient across the liver; (2) Falsely high measurements of FHVP due to the use of a thin hepatic vein are obviated; (3) Greater accuracy in assessing the response to a drug therapy; (4) Greater reproducibility of measurements; and (5) In patients having combination of obstructive lesions (e.g. cirrhosis with caudate lobe hypertrophy), using RA pressure would give actual gradient while traditional HVPG would miss the gradient across the IVC.

When reporting results of HVPG, it is important to mention whether FHVP, IVC or RA has been used as the reference point. Acronyms such as HVPG_h, HVPG_i, and HVPG_r can be used to indicate HVPG values derived using HV, IVC, and RA, respectively, as reference points.

WHVP

When the hepatic vein is occluded, a continuous column of fluid between the catheter and the sinusoids is formed. This results in a WHVP reading that is equal to the

Table 2: Tips for accurate HVPG measurement

Before the procedure

1. Check the indication for HVPG measurement.
2. Ensure absence of contraindications (severe cardiac or pulmonary disease, known hypersensitivity to contrast agents, pregnancy, encephalopathy).
3. Explain the procedure to the patient and take consent.
4. Check coagulation profile; if grossly deranged, arrange fresh frozen plasma.
5. Assess need for transjugular liver biopsy and plan access accordingly.
6. Fasting for 6 hours, with adequate hydration
7. Part preparation.
8. Preferred sedation: midazolam 0.1-0.2 mg/kg intravenously

Required setup

1. Expertise and a cardiology / intervention radiology back-up service
2. Facilities for good fluoroscopy, with/without Doppler ultrasound
3. Continuous monitoring of heart rate, heart tracing, respiration and digital oxygen saturation
4. Adequate resuscitative measures to be readily available
5. A recorder capable of accurately measuring pressures in the venous range and of producing a permanent tracing of pressure values.
6. A quartz pressure transducer that can detect changes in venous pressure (arterial transducers are not suitable).
7. An occlusion catheter (e.g. Swan-Ganz catheter or 7F Goodale Lubin catheter).

Calibration of pressure monitor

1. Use scale with upper limit of 30 or 60 mmHg.
2. Use slow recording speed (1-5 mm/s).
3. Calibrate transducer with known external pressure (e.g. 13.6 cm column of water should read 10 mmHg).
4. The transducer should be at the level of right atrium (mid-axillary line).
5. Set the zero reference point also at mid-axillary line.
6. The tube connecting the catheter and the transducer should be as short in length as possible, and at the same level as the transducer.
7. The tube should be flushed with heparinized saline and should be free of air bubbles.
8. All connections should be tight to prevent any leakage.

Pressure recording

1. Record the IVC pressure on the tracing at the level of the liver before catheterizing the hepatic vein.

2. Catheterize preferably the main right hepatic vein.
3. Do not advance the catheter too far into the hepatic vein when measuring the free pressure (FHVP), which should not be more than 1 mmHg greater than the IVC pressure. Greater differences require withdrawal of the catheter closer to the IVC for an accurate measurement of FHVP.
4. Some workers prefer measuring RA pressure (or suprahepatic IVC pressure) in place of FHVP. The location of the reference point (HV, IVC or RA) used for measuring HVPG should be clearly indicated (HVPG_h, HVPG_i, or HVPG_r, respectively) and the same location should be used for studying the effects of drugs.
5. Venous pressure should be allowed to stabilize for at least 1 minute for WHVP and 15 seconds for FHVP (some patients may require longer periods).
6. After recording the WHVP, confirm adequate occlusion of the hepatic vein by: (a) failure to withdraw blood on suction, (b) slow injection of at least 2-5 mL of contrast dye which should show the typical 'wedged' pattern (sinusoidogram), without any reflux of the contrast or washout through communications with other hepatic veins, and (c) absence of wave form.
7. If occlusion was inadequate, discard the reading and take a fresh reading by re-occlusion at a different site, or with greater balloon volume.
8. Take three sets of readings after flushing the catheter with saline especially after using the contrast each time.
9. Take a mean of the three readings. If any two readings differ by more than 1 mmHg, discard all readings and start afresh. Such variations indicate mistakes in the recording procedure.
10. Register all ongoing events (e.g. coughing, talking or slight movement) on the tracing.
11. All measurements should be recorded permanently. Do not rely on digital readings on the screen. These are instantaneous readings and may not be representative (because of cough, movement) of the correct measurement.

After the procedure

1. Ensure adequate hemostasis after removing the introducer.
2. Ensure immobilization of the part for at least 6 hours after the procedure.
3. Palpate the abdomen, dorsalis pedis pulse and assess limb sensations over the next 24 hours.

sinusoidal pressure. In normal liver, the low-resistant sinusoidal network dissipates most of the pressure back up from the wedged catheter. In this situation, since there is no direct connection between the catheter and the portal tributaries, the pressure reading reflects the sinusoidal pressure, which is slightly lower than the portal venous pressure.¹⁷

There are two techniques for measuring WHVP: catheter advancement technique and balloon occlusion technique. In the former, the catheter is pushed down in the hepatic vein until it cannot be advanced further; this results in a complete obstruction of the venous flow and the pressure recorded in this occluded position is the WHVP.

The balloon occlusion technique was proposed and validated by Groszmann *et al.*²¹ It requires the use of a balloon tipped catheter; inflation and deflation of the balloon within the hepatic vein allows measurement of wedged and free pressures, usually in a large right lobar hepatic vein, without the need to advance and withdraw the catheter for each WHVP and FHVP determination. This technique is preferred because it allows serial measurements of FHVP and WHVP using the same catheter, inflated and deflated repeatedly. The catheter can also be left in place safely for several hours to permit monitoring of the effects of pharmacologic agents on portal hemodynamics. Balloon technique also avoids the decompressive effect of venous-to-venous shunts that are proximal to the balloon. Furthermore, using the conventional catheters, the WHVP is measured in a small hepatic venule. A recent study indicates differences in the values obtained when the catheter is wedged in different hepatic veins.²² These differences are probably due to the heterogeneity of sinusoidal involvement in diseases like liver cirrhosis.^{23,24} In contrast, the use of balloon catheter allows measurement in the hepatic veins at the lobar and sublobar levels. This measurement is an average of pressures in several segments of the liver and thus is likely to more closely represent the true portal venous pressure.¹⁷

WHVP versus HVPG

WHVP represents the absolute value of sinusoidal pressure, which is slightly lower than the actual portal vein pressure. In patients with liver cirrhosis, it is closely similar to intravariceal pressure.²⁵

WHVP is known to determine the risk of variceal rupture, which is directly proportional to the intravariceal pressure as given by the Laplace equation:

$$T = (p_1 - p_2) \times r/w$$

where T is wall tension, p_1 is intravariceal pressure, p_2

is intra-luminal pressure in the esophagus, r is radius of the varix and w is the thickness of the varix wall (Figure 2). In normal circumstances, the intra-luminal pressure of esophagus (p_2) is the atmospheric pressure and is used as zero or the reference point during the measurement of HVPG. Hence, the variceal wall tension (T) is directly proportional to the intra-variceal pressure (p_1). In patients with liver cirrhosis, the intravariceal pressure (p_1) is almost similar to WHVP and hence the wall tension is directly proportional to WHVP (and not HVPG). Higher the WHVP, higher the wall tension and higher is the chance of variceal rupture.

In contrast, HVPG determines the risk of variceal formation or blood flow through the varices (Figure 3). HVPG represents the gradient between the portal and systemic circulations. As this gradient increases, new collaterals or shunts (including esophageal varices) open up between the portal and systemic circulation. Hence it is the HVPG (and not WHVP) which determines the variceal blood flow.

When reporting results of portal hemodynamics, both WHVP and HVPG should be reported, since one represents the risk of variceal rupture and the other represents the amount of variceal blood flow. However, HVPG has certain advantages over WHVP: (1) WHVP and FHVP are both affected by intra-abdominal pressure, presence of ascites, or the hydration status of the patient, but their gradient, the HVPG, is not; (2) the HVPG is not influenced by variations in the external zero reference point, another important source of error in WHVP and FHVP.¹⁷

Tolerability and safety

The procedure of HVPG measurement carries very little discomfort. Performed under slight conscious sedation

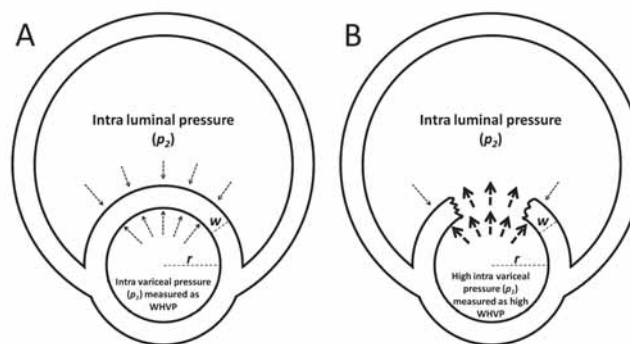


Figure 2: Schematic diagram representing effect of high intra-variceal pressure which causes rupture of varices. According to the Laplace law, the difference between intra-variceal pressure and esophageal intraluminal pressure is an important determinant for variceal rupture. WHVP correlates better with the intra-variceal pressure than does the HVPG

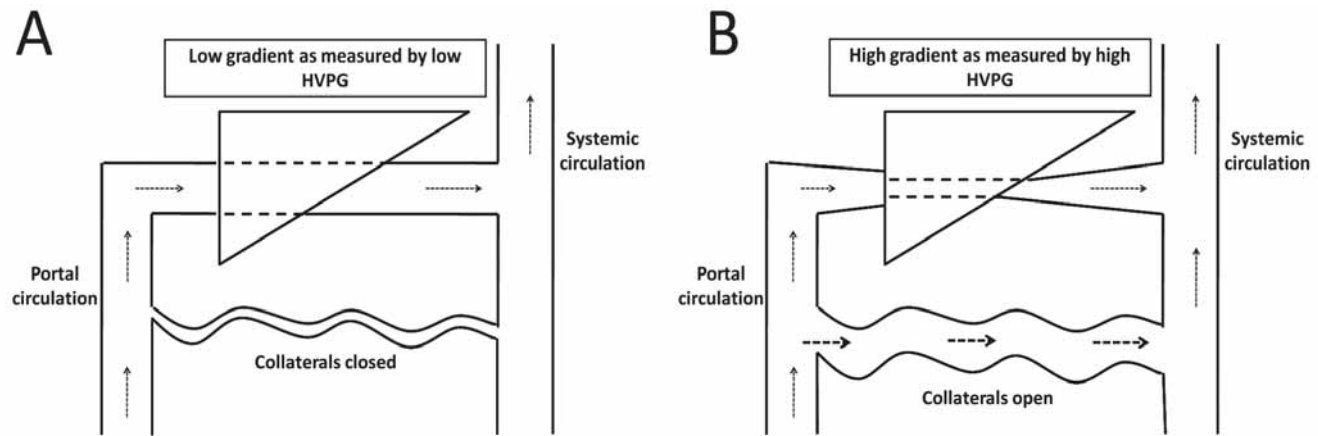


Figure 3: Schematic diagram representing the effect of gradient between the portal and the systemic circulations. High gradient causes more collaterals to form between portal and systemic circulation. Hence, high HVPG will result in higher chances of development and enlargement of varices

(midazolam 0.2 mg/Kg intravenously), its acceptability is comparable to that of upper gastrointestinal endoscopy. This kind of sedation does not influence HVPG measurement.^{19,26}

The procedure of measuring the HVPG has proved to be extremely safe.²⁰ No reports of mortality and serious complications have been published. In a sample of 1,000 patients with liver disease undergoing transjugular liver biopsy, it was reported that complications were related to the liver biopsy and not to the catheterization of hepatic veins.²⁷

Main complications of the HVPG measurement have been limited to local injury of the access vessel (femoral, jugular, or antecubital veins) and include leakage, hematoma, and rarely, vagal reactions, rupture of venous introducers, or arteriovenous fistulae (femoral vein to femoral artery or jugular vein to carotid artery). The risk of these complications is greatly reduced by performing deep venous puncture under Doppler ultrasonographic guidance. This is especially recommended in obese patients, when arterial palpation is difficult, or whenever the procedure is done by a trainee.¹⁹ Passage of the catheter through the right atrium may cause supraventricular arrhythmias (ectopic beats), which are self-limited in over 90% of occasions.

Our unit's experience has been positive after more than 1,000 such procedures with no major complications or procedure-related mortality.

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Correspondence to: Professor Sarin, Director and Head, Department of Gastroenterology, G B Pant Hospital, New Delhi 110 002, India. Fax: 91 (11) 2321 9710
E-mail: sksarin@nda.vsnl.net.in.

News and Notices

Enterocon 2008 - An international conference of Inflammatory bowel disease, and small large bowel endoscopy, and a workshop on Capsule Endoscopy is being organized by Apollo Gleneagles Hospitals, Kolkata, and will be held from August 2-4, 2008.

For details, contact: Dr M K Goenka (Chairperson, Organizing committee). E-mail: dr_mkgoenka@yahoo.co.in

The 5th SR Naik Memorial Workshop on 'Computers and Informatics in Medicine' will be held at the Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, August 30-31, 2008.

For details, contact: Dr. Rakesh Aggarwal, Department of Gastroenterology, SGPGI, Lucknow 226014. E-mail: rakesh@sppgi.ac.in, aggarwal.ra@gmail.com

The 49th Annual Conference of Indian Society of Gastroenterology and the Asia Pacific Digestive Week 2008 will be held at Ashok Hotel, New Delhi, India from September 12-16, 2008.

For details, contact: Dr. Rakesh Tandon, Chairman, Organizing Committee. Websites: www.apdw2008.net; www.isg.org.in. E-mail: apdw@apdw2008.net.

Medical Education Fellowships-2009: CMCL-FAIMER Regional Institute, Christian Medical College, Ludhiana

The CMCL-FAIMER regional Institute's Fellowship is a two-year fellowship program designed for Indian medical school faculties who have the potential to play a key role in improving medical education at their institutes. The program is uniquely designed to teach *education methods* and *leadership skills*, as well as to develop strong professional bonds with other medical educators. The fellowship is now running in its fourth year.

Sixteen fellowships are on offer for the year 2009. Requirements for selection are submission of a curriculum innovation project proposal and letter of support from applicant's institute. Limited funding is available to support fellows' travel, local expenses and course fee. Applications open from: July 1 to October 15, 2008

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