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Overall approach to therapy — Optimal therapy, including the choice of agent and the blood pressure goal, varies according to the specific hypertensive emergency [7,8].

It is generally unwise to lower the blood pressure too quickly or too much, as ischemic damage can occur in vascular beds that have grown accustomed to the higher level of blood pressure (ie, autoregulation). For most hypertensive emergencies, mean arterial pressure should be reduced gradually by about 10 to 20 percent in the first hour and by a further 5 to 15 percent over the next 23 hours [9].

The major exceptions to gradual blood pressure lowering over the first day are:

- The acute phase of an ischemic stroke – The blood pressure is usually **not** lowered unless it is ≥185/110 mmHg in patients who are candidates for reperfusion therapy (table 1) or ≥220/120 mmHg in patients who are not candidates for reperfusion therapy. (See "Initial assessment and management of acute stroke", section on 'Blood pressure management'.)
- Acute aortic dissection – The systolic blood pressure is **rapidly** lowered to a target of 100 to 120 mmHg (to be attained in 20 minutes) [9]. (See "Management of acute aortic dissection".)
- Intracerebral hypertension – Goals of antihypertensive therapy in such patients are variable and are discussed elsewhere. (See "Spontaneous intracerebral hemorrhage: Treatment and prognosis", section on 'Blood pressure'.)

After a suitable period (often 8 to 24 hours) of blood pressure control at target in the intensive care unit, oral medications are usually given and the initial intravenous therapy is tapered and discontinued.

Neurologic emergencies — Severe hypertension with acute neurologic signs or symptoms is usually the most complicated and difficult clinical scenario, as the differential diagnosis includes varied conditions that have disparate treatments, only some of which routinely involve lowering the blood pressure:

- Ischemic stroke – Patients with acute ischemic stroke-in-evolution are most often **not** given antihypertensive drugs unless they are candidates for tissue plasminogen activator and their initial blood pressure is ≥185/110 mmHg or if their initial blood pressure is ≥220/120, even if they are not candidates for reperfusion therapy. The preferred antihypertensive medications in this setting are discussed in detail elsewhere. (See "Initial assessment and management of acute stroke", section on 'Blood pressure management'.)
- Hemorrhagic stroke – Management of blood pressure in patients with spontaneous intracerebral hemorrhage and subarachnoid hemorrhage is complicated by competing risks (eg, reducing cerebral perfusion) and benefits (eg, reducing further bleeding). An international trial involving 2839 subjects with onset of symptoms less than six hours prior to presentation and a baseline blood pressure of 150 to 200 mmHg found that lowering blood pressure (within one hour) to <140 mmHg was safe and produced nonsignificant benefits on death and major disability [10].United States guidelines indicate that blood pressure-lowering therapy should be given in this setting if there are no contraindications [10]. Intravenous labetalol and nicardipine are most often used as first-line agents, although

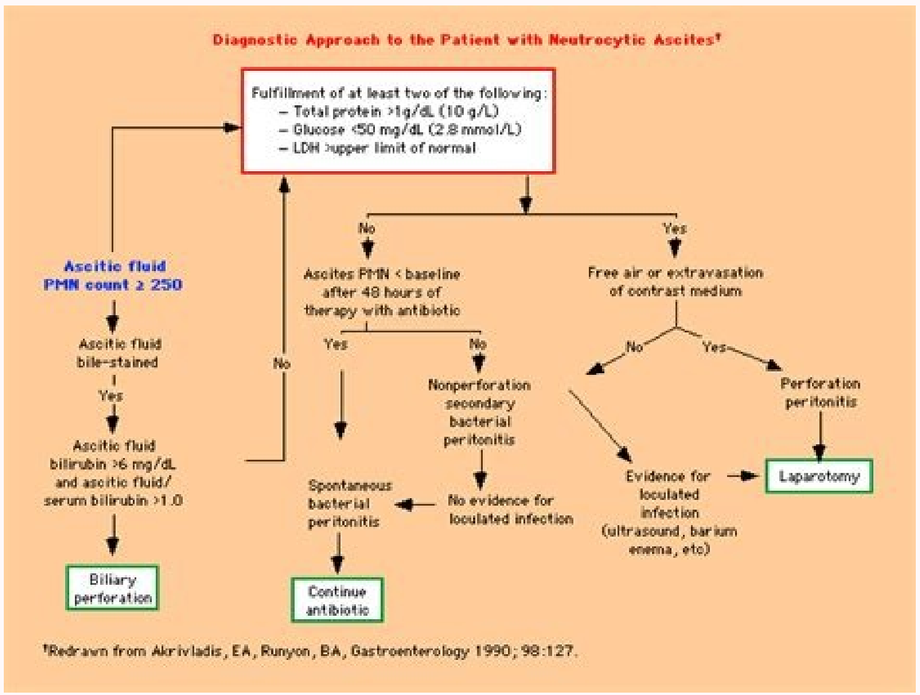
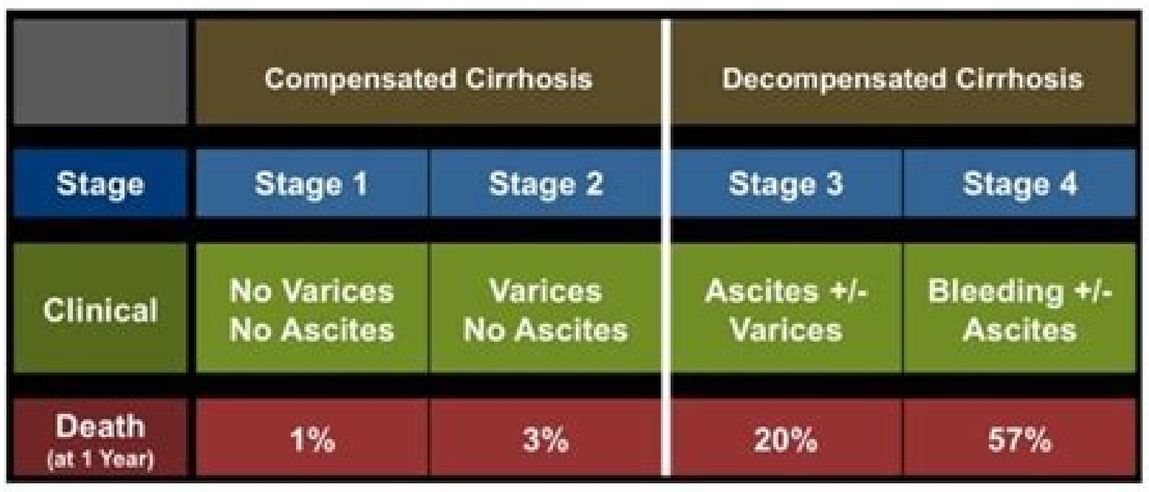


Figure 2 Four-Stage Cirrhosis Classification System

Patients with cirrhosis can be subcategorized as having four stages, with stages 1 and 2 classified under Compensated category and stages 3 and 4 in the Decompensated category. The risk of death increases significantly with each more advanced stage.

Source: D'Amico G, Garcia-Tsao G, Pagliaro L. Natural history and prognostic indicators of survival in cirrhosis: a systematic review of 118 studies. J Hepatol. 2006;44:217-31.



	Compensated Cirrhosis		Decompensated Cirrhosis	
Stage	Stage 1	Stage 2	Stage 3	Stage 4
Clinical	No Varices No Ascites	Varices No Ascites	Ascites +/- Varices	Bleeding +/- Ascites
Death (at 1 Year)	1%	3%	20%	57%

ALF etiologies

- Viruses
  - Hepatitis A, B, D, or E viruses
  - Cytomegalovirus
  - Epstein-Barr virus
  - Herpes simplex virus
  - Varicella zoster virus
  - Parvovirus
- Drug-induced liver injury
  - Acetaminophen
  - Non-acetaminophen (eg, isoniazid, phenytoin, valproate, propylthiouracil, nitrofurantoin)
  - Recreational drugs (eg, cocaine, MDMA)
- Autoimmune hepatitis
- Ischemic/congestive hepatitis
- Budd-Chiari syndrome
- Wilson disease
- *Amanita phalloides*
- Pregnancy (eg, acute fatty liver of pregnancy, HELLP syndrome)
- Heat stroke
- Malignant infiltration
- Seronegative (indeterminate)

MDMA indicates 3,4-methylenedioxy-N-methylamphetamine.

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Diagnosis of spontaneous bacterial peritonitisBruce A Runyon, MDUpToDate performs a continuous review of over 330 journals and other resources. Updates are added as important new information is published. The literature review for version 13.1 is current through December 2004; this topic was last changed on September 14, 2004. The next version of UpToDate (13.2) will be released in June 2005. INTRODUCTION — Spontaneous bacterial peritonitis (SBP) is defined as an ascitic fluid infection without an evident intraabdominal surgically-treatable source [1]. The presence of SBP, which almost always occurs in patients with cirrhosis and ascites, is suspected because of suggestive signs and symptoms (show table 1). (See "Clinical manifestations of spontaneous bacterial peritonitis"). The diagnosis is established by a positive ascitic fluid bacterial culture and an elevated ascitic fluid absolute polymorphonuclear leukocyte (PMN) count (250 cells/mm3). This topic review will focus on the major issues involved in establishing the diagnosis of SBP, beginning with the safety of paracentesis. The distinction of SBP from secondary bacterial peritonitis and from alcoholic hepatitis in patients with cirrhosis and ascites will also be discussed. This topic is also discussed in an official guideline issued by the American Association for the Study of Liver Diseases (see "AASLD guideline: Management of adult patients with ascites due to cirrhosis"). SAFETY OF PARACENTESIS — The criteria for the diagnosis of SBP require that abdominal paracentesis be performed and ascitic fluid be analyzed before a diagnosis of SBP can be made. A "clinical diagnosis" of SBP without paracentesis is not adequate. Many physicians inappropriately avoid performing minor procedures on patients with ascites because of coagulopathy and fear of hemorrhagic complications. Approximately 70 percent of patients with ascites have an abnormal prothrombin time [2]. However, the fear of hemorrhagic complications is unfounded except in the setting of clinically apparent disseminated intravascular coagulation or clinically apparent fibrinolysis [3]. These conditions occur in fewer than 1 in every 1000 paracenteses. Furthermore, the number of patients who require transfusion of red blood cells for a paracentesis-related hemorrhage is less than 1 percent [2,4]. In a study involving 1100 large-volume paracenteses, patients tolerated the procedure without transfusions despite an INR as high as 8.7 and platelet count as low as 19,000 [3]. Thus, the benefits of diagnostic paracentesis outweigh the risks in almost all circumstances. The general indications for abdominal paracentesis are listed in Table 2 (show table 2). Some physicians transfuse plasma or platelets prior to paracentesis in patients with coagulopathy. This practice is not supported by data and would lead to transfusion of approximately 140 units of plasma to prevent transfusion of only two units of red cells [2,4,5]. We have never given prophylactic plasma or platelets prior to abdominal paracentesis in the thousands of procedures that we have performed. Traumatic paracentesis occurs when the bowel is entered by the paracentesis needle. This complication is recognized when air, enteric fluid, or frank stool is aspirated during attempted paracentesis. It usually occurs when the operator is inexperienced, the needle is placed too close to a surgical scar (with bowel adherent to the abdominal wall), or ileus is present. ASCITIC FLUID ANALYSIS — Immediately after the paracentesis needle and attached syringe are withdrawn from the abdomen, the "skin" needle should be removed and replaced with a sterile needle to minimize the risk of skin flora growing in the cultures. Appropriate handling of the ascitic fluid that is aspirated is essential to optimize the results [5]. Otherwise, a negative culture may be obtained, possibly leading to the diagnosis of culture negative neutrocytic ascites. Most such patients actually have SBP. (See "Spontaneous bacterial peritonitis variants"). Handling of the ascitic fluid — Handling of the ascitic fluid sample should include the following steps: Appropriately prepared blood culture bottles should be inoculated preferably with at least10 mL of fluid. However, some bottles will not permit more than the recommended amount of fluid to be instilled without infringing upon the "head space" in the bottle in which case the upper end of the recommended amount of fluid should be instilled. These bottles should be sent for bacterial culture.Both the use of blood culture bottles and the volume of the sample are important. Sending a syringe or tube of fluid to the laboratory for culture dramatically decreases the sensitivity of the results since SBP is a low-colony-count monomicrobial infection similar to bacteremia [5]. Thus, culturing ascitic fluid as if it were blood (with bedside inoculation of ascitic fluid into blood culture bottles) has been shown to increase the culture-positivity of the ascitic fluid of patients with an ascitic fluid PMN count 250 cells/mm3 (in the absence of prior antibiotic treatment, pancreatitis, tuberculous peritonitis, or malignancy-related ascites) from about 50 percent (with the conventional method) to approximately 80 percent [1]. The volume of fluid cultured also has a dramatic impact on the sensitivity of the culture in detecting bacterial growth. In one report, for example, inoculation of 10 or 20 mL of fluid into 100 mL blood culture bottles led to a much higher culture-positivity rate than a 1 mL inoculum (93 versus 53 percent) [5]. Approximately 1 mL of fluid should be injected into a "purple-top" EDTA tube for cell count. An accurate cell count cannot be obtained if the fluid is allowed to clot or submitted in a nonanticoagulated tube. Several mL of fluid should be injected into a "red-top" tube for chemistries. Chemistries should include albumin, so that the serum-ascites albumin gradient can be determined, total protein concentration, glucose, lactate dehydrogenase, and amylase. If the fluid is dark orange or brown, a bilirubin concentration should be obtained. A separate syringe or tube of fluid should be sent for Gram stain. The latter test will not be performed if a separate sample of fluid is not submitted with the request for Gram stain. Laboratories will not perform a Gram stain on the culture bottles until they have incubated for 12 to 24 hours. Centrifugation of the fluid prior to performing a Gram stain is not necessary since it does not increase the yield and may decrease the likelihood of obtaining a positive culture [5].Cell count — Analysis of the ascitic fluid consists of routine, optional, and unusual tests (show table 3). The cell count and differential should be ordered "stat," otherwise many laboratories will prioritize the cell count below routine peripheral blood counts. The absolute PMN count in the ascitic fluid is calculated by multiplying the total white blood cell count (or total "nucleated cell" count) by the percentage of PMNs in the differential. The cell count and differential are performed manually (meaning that a technician must be removed from other duties to perform the count) without formal quality control. The accuracy of these tests is totally dependent upon the skill and interest of the medical technologist. Automated cell counts appear to be accurate and, once the technique is validated, may become the standard approach [6]. The automated approach has the potential to improve reliability and accuracy, and the speed with which results are obtained [7]. One potential source of error in the PMN count is that hemorrhage into the ascitic fluid, as in a traumatic paracentesis, leads to red cell and white cell entry into the fluid. A corrected PMN count should be calculated if there is bloody fluid: one PMN is subtracted from the absolute PMN count for every 250 red cells/mm3. PMNs lyse rapidly, much more so than red cells. Thus, if the bleeding episode occurred prior to rather than during paracentesis, the PMNs that entered the fluid may have lysed and the corrected PMN count may be a negative number. The results should be available in one to four hours. Delayed results lead to delayed detection of infection, delayed initiation of empiric treatment, and decreased chance for survival of the patient. The results of the cell count should be reviewed and a decision made about treatment within a few hours of the paracentesis. It is prudent to write the initial antibiotic order to be given "stat" to prevent an inadvertent delay in administration of the first dose. (See "Treatment and prophylaxis of spontaneous bacterial peritonitis"). The cell count, Gram stain, and culture provide the necessary information to make the diagnosis of SBP in most patients. In selected cases, however, one or more of the following tests may be useful. Serum-ascites albumin gradient — The serum-ascites albumin gradient indirectly measures portal pressure [8,9]. The albumin concentration of ascitic fluid and serum must be obtained on the same day. The ascitic fluid value is subtracted from the serum value to obtain the gradient (show table 4): If the difference (not a ratio) is >1.1 g/dL, the patient has portal hypertension, with 97 percent accuracy [9]. If the difference is 1 g/dL (10 g/L) Glucose concentration 5 ng/ml or ascitic fluid alkaline phosphatase >240 units/L were 92 percent sensitive and 88 percent specific for detecting gut perforation into ascitic fluid [27]. Imaging studies — Patients who meet the criteria for secondary bacterial peritonitis should undergo emergency plain and upright abdominal films and water-soluble gut contrast studies. Emergency laparotomy should be performed if free air or extravasation of contrast are documented [14]. Response to treatment — If neither free air nor extravasation of contrast is documented, emergency laparotomy cannot be justified even if suspicion of secondary bacterial peritonitis persists. Repeat paracentesis after 48 hours of treatment (with coverage of aerobic and anaerobic flora) can be helpful in this setting. If the follow-up PMN count is lower than the pretreatment value, and the initial culture grows only one organism, the patient probably has SBP; the second culture will probably be sterile. If the PMN count rises and the initial culture grows multiple organisms (especially if enterococcus or fungi are among the flora), the patient probably has nonperforation secondary peritonitis and warrants imaging studies in search for abscesses. Many of these patients will require laparotomy [14]. Survivors of laparotomy for surgical peritonitis in the setting of cirrhosis and ascites have been reported [23].This differential response to therapy assumes that an appropriate antibiotic has been administered. If an empiric antibiotic is chosen that does not penetrate ascitic fluid well or does not cover the flora fully, the response of a patient with SBP can mimic that of secondary bacterial peritonitis. Peritonitis developing during antibiotic treatment — SBP is so exquisitely sensitive to appropriate treatment that it does not develop in our experience in patients who are receiving broad-spectrum parenteral antibiotics [24]. (See "Treatment and prophylaxis of spontaneous bacterial peritonitis"). Thus, a search for a surgical source should be undertaken if such a patient develops bacterial peritonitis. Other conditions masquerading as secondary bacterial peritonitis — Occasionally peritoneal carcinomatosis or tuberculous peritonitis can be associated with neutrocytic ascites and an initial ascitic fluid analysis that meets criteria for secondary bacterial peritonitis. There are two clues that the diagnosis may not be surgical peritonitis: A predominance of non-PMNs on the differential white cell count The absence of feverIn addition, SBP that is detected late may also meet criteria for secondary bacterial peritonitis and may have a slow PMN response to therapy. The monomicrobial nature of the infection and the sterility of the second culture help provide evidence that SBP is the correct diagnosis. DISTINCTION FROM ALCOHOLIC HEPATITIS — The patient with alcoholic hepatitis warrants specific comment. These patients regularly develop fever, leukocytosis, and abdominal pain, which can masquerade as SBP. (See "Clinical manifestations and diagnosis of alcoholic liver disease"). Furthermore, they also can develop SBP. An important distinguishing point is that peripheral leukocytosis does not lead to an elevated ascitic fluid PMN count [28]. Thus, an elevated ascitic fluid PMN count must be presumed to represent SBP. Empiric antibiotic treatment (for presumed ascitic fluid infection) of patients with alcoholic hepatitis, who have fever and/or peripheral leukocytosis can be discontinued after 48 hours if ascitic fluid, blood, and urine cultures demonstrate no bacterial growth and the ascitic fluid PMN count is

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