individuals who will definitely experience a perioperative cardiac event, and revascularisation inevitably means treatment of many patients in an attempt to prevent a few adverse outcomes. In any event, the modest survival benefit is unlikely to exceed the additional risk of a bypass operation that would not ordinarily have been done. These risk/benefit considerations apply particularly to patients with peripheral vascular disease, in whom coronary bypass surgery carries an especially high risk.

The cause of perioperative myocardial infarction probably differs from that of spontaneous myocardial infarction, with important management implications. Although infarction might be expected to occur during the stress of the operation itself, it is commonest on the second and third postoperative days, and is strongly predicted by episodes of ischaemia on postoperative Holter monitoring.[6] The peak burden of electrocardiographic ischaemia on postoperative days 2 and 3 is itself preceded by a peak in the amount of tachycardia on days 1 and 2.[7] A hypothesis proposed is that the stress response to surgery (eg, high circulating concentrations of catecholamines, prothrombotic tendency) results in increased myocardial oxygen demand and increased shear stresses on atherosclerotic plaques, leading to secondary plaque rupture.

The emphasis in the perioperative management of patients at high cardiac risk should therefore be directed away from mechanical revascularisation towards improved control of myocardial oxygen demand and protection of vulnerable atherosclerotic plaques. Aggressive modification of coronary risk factors with anti-smoking advice, cholesterol lowering, and control of hypertension are essential.[8] Perioperative haemodynamic monitoring, meticulous pain control, and the careful use of β-blockers are also crucial. Mangano and colleagues[9] showed that atenolol given intravenously at induction of anaesthesia and continued during the hospital stay reduced overall mortality by 55% over the subsequent 2 years. This result was due mainly to improved survival over the first 8 months, possibly as a result of a reduction in shear stresses on vulnerable atherosclerotic plaques postoperatively.[9]

Despite advances in perioperative management, there continues to be pressure for patients at high cardiac risk to be identified preoperatively. The data obtained by Lee and colleagues emphasise that for most patients cardiac risk-stratification before elective non-cardiac surgery requires only a knowledge of the risk associated with the procedure and a simple clinical assessment. Resources should be directed away from the unnecessary investigation of low-risk individuals, towards improved perioperative management for those at high risk.

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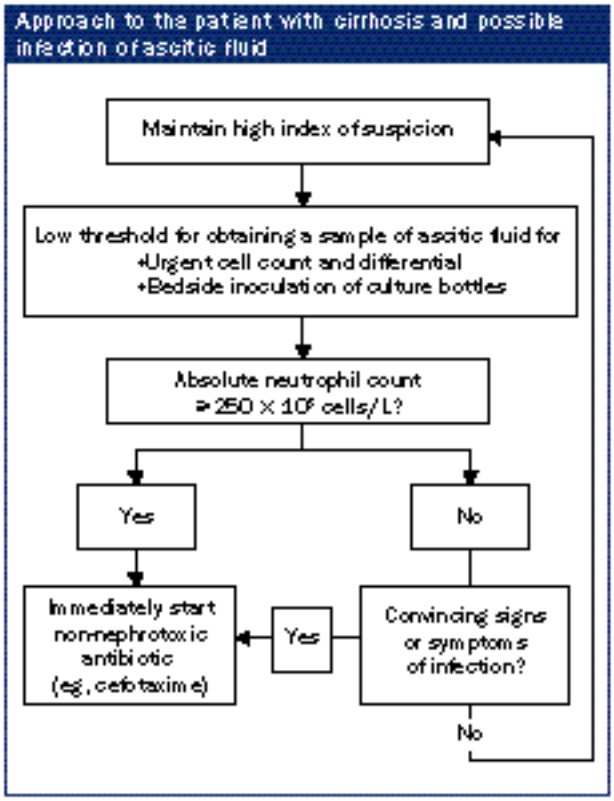
Albumin infusion for spontaneous bacterial peritonitis

Spontaneous bacterial peritonitis is a common complication of ascites and cause of death of patients with cirrhosis.[1] The survival of a patient with spontaneous bacterial peritonitis depends on an aggressive approach to diagnosis and treatment[2] (see figure). This strategy has been shown to reduce infection-related mortality to 5% or less.[3] However, about 40% of patients do not survive the admission to hospital during which the infection is detected,[3] and renal failure and gastrointestinal bleeding are the commonest causes of death.[4] This infection may lead to a deterioration in renal function by increasing the peripheral vasodilatation and renal vasoconstriction that are present in patients with advanced cirrhosis.[4]

A recent randomised controlled trial[5] lends support to an association between circulatory dysfunction and bacterial peritonitis in patients with cirrhosis. The study showed that renal impairment can be prevented by intravenous albumin infusion—1·5 g per kg bodyweight within 6 h of detecting the infection and 1 g per kg on day three.[5] The infusion of albumin was associated with a stable plasma renin concentration. Patients who did not receive albumin experienced a significant increase in plasma renin concentrations. In-hospital mortality was 29% in the antibiotic-only group compared with 10% in the antibiotic-plus-albumin group. The latter rate is the lowest reported for patients with spontaneous bacterial peritonitis and the difference in survival rates was still statistically significant at 3 months. The study was not blinded or placebo controlled, but otherwise seems to have been very well done and provides some of the much needed evidence for making "evidence-based" decisions for these patients.

The use of intravenous infusions of albumin in clinical practice is controversial. The Cochrane meta-analysis,[6] which reviewed the use of albumin in many disorders, concluded that albumin may increase mortality. This conclusion led to a very heated debate in the medical and lay press. However, rather than drawing one conclusion about the value and safety of albumin from 30 distinctly different randomised studies, a more useful approach may be to focus on each specific setting in which albumin is used. The randomised trial of albumin infusion carried out by Pau Sort and colleagues[5] to prevent renal failure and death in patients with spontaneous bacterial peritonitis is very encouraging. The recommended initial dose of albumin is more than 100 g for an average man, and 50–100 g for the day 3 infusion. Although this regimen is very expensive, it would be justified if further trials confirm the impressive survival advantage. As the



investigators suggest, studies of lower doses of albumin or synthetic plasma expanders are warranted.[5]

These same investigators have consistently championed the use of albumin after large-volume paracentesis in patients with cirrhosis and tense ascites.[7] This strategy has been shown to help prevent the increase in plasma renin concentrations that can occur if a large volume of fluid is removed without albumin infusion.[8] The increase in plasma renin concentration has been associated with an increase in mortality.[9] However, no randomised trial has shown a direct association between paracentesis without albumin replacement and an increase in mortality: the association is an indirect one.The studies that do propose an association have been criticised for including patients who had diuretic-sensitive ascites.[10] Patients with diuretic-sensitive ascites are most appropriately treated with a sodium-restricted diet and oral diuretics.[2] Large-volume paracentesis should be used only for initial treatment of tense ascites (with removal of 2–4 L) or for treatment of diuretic-resistant ascites (removal of nearly all ascitic fluid every 2 weeks).[2] Too frequently, large-volume paracentesis is repeated in diuretic-sensitive patients who seem refractory to diuretics, but in fact are simply not eating a sodium-restricted diet.The sodium concentration in a 24 h urine sample can indicate non-compliance with the diet.[2] Many patients make little effort to comply with the diet or do not realise that foods such as dill pickles, soy sauce, or buttermilk are extremely high in sodium. Diet education by a dietician can lead to successful diuretic treatment and prevent the need for therapeutic paracentesis and the debate associated with it.

Immediate infusion of albumin, in addition to broad-spectrum antibiotic treatment at the time of detection of spontaneous bacterial peritonitis,can maximise survival of patients with this common complication of cirrhosis. The benefits of albumin infusion for other disorders must be weighed against the cost and potential hazards before its use can be recommended or opposed.

Whether albumin infusion after each sequential therapeutic paracentesis improves the survival of patients with truly diuretic-resistant ascites remains to be proved.

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New treatments for pulmonary fibrosis?

Idiopathic pulmonary fibrosis (IPF) is a life-threatening syndrome. Various factors, such as aspiration and exposure to wood dust or metal dust, may lead to a common clinical and histological expression of lung disease. After initial epithelial-cell injury and inflammation, the lung is reorganised by a destructive fibroproliferative response. In 65% of IPF patients, histological evaluation reveals usual interstitial pneumonia (UIP). At low magnification, UIP shows between-field variability in features such as honeycomb lung, interstitial fibrosis, and normal lung.[1] UIP reduces the surface area of the lung and obliterates pulmonary vessels, which results in ventilation-perfusion mismatch, hypoxaemia, and breathlessness. Patients with an UIP pattern of IPF have a mean age of 65 years, do not respond to steroids, and have a median survival of only 2·8 years.[2] A minority of patients present with the clinical syndrome of IPF, but histological examination reveals either the more recently described non-specific interstitial pneumonia (NSIP) or desquamative interstitial pneumonia (DIP). NSIP is a diagnosis of exclusion, characterised by the absence of the typical pattern of UIP and the presence of uniform fibrosis with cellular infiltration of the alveolar walls.[1] DIP is characterised by excess numbers of macrophages in the alveolar spaces. Patients with NSIP and DIP are significantly younger than those with UIP. Their mean age is 57 years, and they tend to respond to oral corticosteroids and have a median survival of more than 10 years.[2] An appreciation of potential histological subgroups (not all patients are fit for lung biopsy), demographics,and survival patterns are important in the evaluation of outcomes of treatment.