The transplant professional counts heavily on the contributions of the pathologist for diagnosis before and after transplantation. While it is not the purpose of this manual to offer a description of all the conditions that lead to end-stage organ disease, it is important to offer an overview of posttransplant pathology as it pertains to diagnosis and management of acute rejection and other conditions that cause allograft dysfunction in the postoperative period. Finally, due to the sparse and often controversial information regarding pancreatic allograft biopsies, only liver and kidney transplantation are addressed here.

21.1. LIVER TRANSPLANT

The spectrum of liver transplant pathology begins with the examination of the recipient’s explanted liver. This is a valuable opportunity to study the primary disease in depth and to search carefully for other pathologic features, such as incidental hepatocellular carcinoma.

The donor liver can be evaluated rapidly before transplantation. Frozen-section technique is usually used for such an examination. The presence and the degree of steatosis (most importantly, macrovesicular steatosis), inflammation, necrosis, and fibrosis can be evaluated immediately by this technique. The liver with severe macrovesicular steatosis, usually involving more than 30% of parenchyma is at high risk for early graft failure (Fig. 21.1).

At the time of transplantation, a postperfusion liver biopsy (0 time) can be utilized to estimate the degree of preservation injury. The peak of morphologic changes, however, occurs after 48 hours, when centrizonal hepatocellular swelling
and cholestasis can be appreciated. The liver with a significant degree of macrovesicular steatosis is more susceptible to perfusion injury and a higher incidence of early graft failure.

After transplantation, monitoring of the hepatic functions and histopathologic examination of the allograft remain the most reliable methods for posttransplant management. The major causes of early allograft dysfunction are acute rejection, mechanical or technical complications, and infections. Acute graft rejection generally develops between 1 and 3 weeks posttransplant. Late acute graft rejection is possibly due to changes in immunosuppression regimen. Needle biopsy is used for accurate diagnosis of acute rejection. Fine-needle aspirate, although not widely accepted, has also been used in some transplant centers. Bile duct epithelium and blood vessels are the major targets of rejection. The three major diagnostic features are (1) mixed but predominantly lymphocytic portal infiltrate, (2) bile duct injury, and (3) endotheliatitis (Fig. 21.2). The portal infiltrate is composed of activated lymphocytes, polymorphonuclear leukocytes, and eosinophils. The bile duct epithelium shows nuclear crowding, cytoplasmic vacuolization, enlarged nuclei, and prominent nucleoli. Endotheliatitis consists of inflammatory infiltrate of the vessel wall, usually the portal vein, but sometimes the terminal venule, associated with endothelial cell swelling and separation.