under saline solution to observe for bubbles. The donor left atrial cuff incorporating the pulmonary vein is connected to the native left atrium and the donor right or left pulmonary artery is connected to the native pulmonary artery. After completion of the vascular anastomoses, the lungs are gently reperfused. During this early reperfusion period, lung-protective ventilation strategies are employed and oxygen tension is reduced. The patient is transitioned to normal ventilation, drains are placed in the thoracic cavity, and the wounds are closed.

Induction of Immunosuppression Initiation of immunosuppression starts with induction of the patient under general anesthesia. Many programs utilize an induction agent (most commonly an IL-2 receptor/CD25 antagonist, but antithymocyte globulin, anti-CD52 monoclonal antibodies, or other induction agents may also be used), and systemic corticosteroids and purine modulators are administered after induction is complete. If an IL2 receptor antagonist is utilized for induction, a second dose is administered 4 days after the original dose. An additional dose of methylprednisolone is administered after allograft reperfusion in the operating room. Three-drug immune suppression is initiated with a calcineurin inhibitor, purine modulator, and continued systemic corticosteroids. In patients with severe acute renal dysfunction, calcineurin inhibitor initiation may be delayed.

Perioperative Considerations and Complications Early morbidity and mortality after lung transplant most commonly are sequelae of primary graft dysfunction or infection. Very rarely, hyperacute rejection has been observed; however, with the implementation of robust systems to ensure ABO and HLA compatibility at the time of transplant, the occurrence of hyperacute rejection is extremely uncommon. Primary graft dysfunction (PGD) encompasses a constellation of findings that result in poor early graft function after transplant. This phenomenon is often the consequence of ischemia-reperfusion injury in the allograft and is not related to infection or rejection. It is characterized by a diffuse pattern of infiltrates on the chest x-ray and poor pulmonary gas exchange with PaO_2:FiO_2 ratios <300, with severe PGD characterized by diffuse severe infiltrates