Caring for Transplant Recipients in a Nontransplant Setting

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CE Continuing Education

* This article has been designated for CE credit. A closed-book, multiple-choice examination follows this article, which tests your knowledge of the following objectives:

1. Understand transplant-specific issues, including key interventions
2. Analyze the role of immunosuppressive therapy unique to transplant recipients
3. Explore the potential complications associated with organ transplantation

Scientific and technical advancements have made organ transplantation the treatment of choice for increasing numbers of patients with end-stage organ failure. As a result, the number of organ transplantations performed annually has increased. Because of this increase, more transplant recipients than before receive ongoing healthcare in local community settings and return to the transplant center for periodic checkups or treatment for marked complications. A transplant recipient may be admitted to a local hospital for a diagnosis unrelated to transplantation and routinely treated in that setting, but regardless of whether the admitting diagnosis is transplant related, the patient's status as a transplant recipient must be considered when the plan of care is designed.

In this article, we address transplant-specific issues, assessment of patients who are transplant recipients, and key interventions to help healthcare providers successfully care for these patients in collaboration with the transplant center. We focus on those aspects of care that are unique to transplant recipients, including organ rejection and immunosuppressive drug therapy, risk of infection, physical and psychosocial problems common in all transplant recipients, and organ-specific assessments and nursing interventions for recipients of heart, lung, liver, kidney, and pancreas transplants.

Organ Rejection and Immunosuppressive Therapy

The immune system evolved to protect the host from microbial invasion by recognizing as "nonself" any foreign material (antigen) that enters the body and then eliminating or neutralizing the foreign material. Thus, the immune system of a transplant recipient is programmed to recognize the transplanted organ as nonself and to take action to eliminate or neutralize the graft, a process

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known as rejection. Immunosuppressive therapy is used to inhibit the immune system and protect the graft from this natural response.

Acute rejection of a transplanted organ involves interactions among antigens from the donor organ, the recipient's T cells and B cells, and chemical messengers known as cytokines or interleukins.[1] Because of the complexity of the process, many potential targets exist for intervention with immunosuppressive medications.

The first step in acute rejection involves preparation, or "presentation," of the donor antigens by cells that act as antigen-presenting cells; these cells may come from either the recipient or the donor. Next, recipient T cells, including helper ($CD4^+$) and cytotoxic ($CD8^+$) T cells, recognize donor antigens on the surface of the antigen-presenting cells. The antigen-presenting cells produce a cytokine called interleukin-1 (IL-1), which acts as a signal to activate the helper T cells. Antigen recognition and activation by IL-1 trigger helper T cells to secrete various cytokines, including IL-2, and also stimulate other T cells to express receptors specific for IL-2. IL-2 binds to these receptors and causes proliferation of cytotoxic T cells, which can bind directly to cells in the grafted organ and cause cell lysis.

Helper T cells produce other cytokines in addition to IL-2, including IL-4 and IL-5, that can cause transformation of B cells into plasma cells. Plasma cells produce antibodies that bind specifically to the target antigens on cells in the graft, leading to complement fixation and injury to the graft. Figure 1 illustrates the process of rejection and the sites of action of immunosuppressive medications.[2]

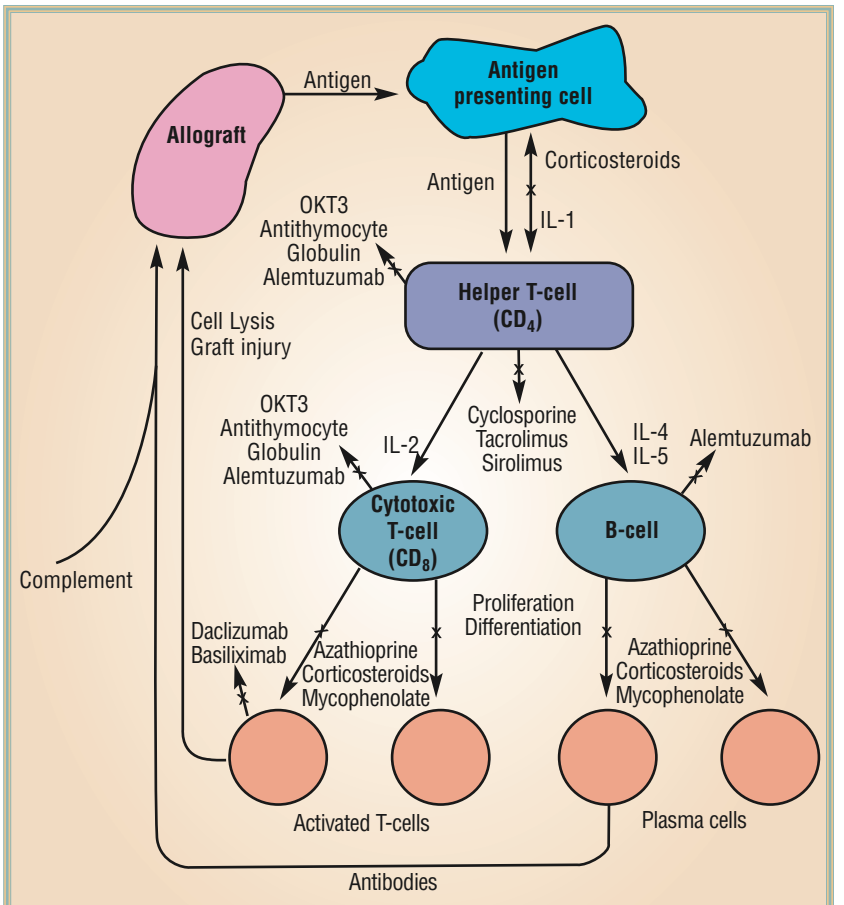

Figure 1 Sites of action for immunosuppressive agents.

Abbreviation: IL, interleukin.
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Immunosuppressive therapy can be divided into 3 general categories: induction, maintenance, and rejection. In all 3 categories, multiple drugs are generally used to target different steps in the rejection process. Induction therapy refers to the drugs and doses used in the immediate peritransplant period, when the risk of rejection is highest and potent immunosuppression is needed; such therapy may also be used when renal function is compromised. Although the risk of rejection is highest in the first 3 months after transplantation, the risk is present throughout the rest of the recipient's life, and therefore maintenance immunosuppression is continued indefinitely, usually with reduced doses of drugs. If episodes of organ rejection are diagnosed, rejection treatment is typically initiated at higher doses of drugs and/or with more potent intravenous immunosuppressants. After resolution of the acute rejection episode, doses typically are tapered down to maintenance therapy levels. Table 1 lists the actions, administration, and adverse effects of commonly used immunosuppressive drugs.[3-8]

Because the need for maintenance immunosuppression is lifelong, transplant recipients must continue to take their immunosuppressive medications during any illness or hospitalization. Maintaining a consistent blood level of immuno-

suppressive medication is critical; therefore, recipients must take or be given their medications on a strict schedule (ie, every 12 hours rather than simply twice a day). If a patient cannot take oral medication, parenteral alternatives may be available, and the transplant center should be contacted to discuss these alternatives. In addition, many drug interactions can affect blood levels of immunosuppressants[6,7] (Table 2); therefore, a pharmacist or a comprehensive reference of drug interactions should be consulted before any new medication is started in any patient who is a transplant recipient. Because target blood levels are often specific to a transplant center and the individual patient, and because multiple assays are available for immunosuppressive agents, monitoring the serum levels of immunosuppressants should always be undertaken in collaboration with the transplant center.

Risk of Infection

The need for immunosuppressive therapy leaves transplant recipients at risk for infection. Both common and rare types of infection can occur. Influencing factors include a recipient's past exposure to infections and overall preoperative health; exposure to new sources of infection related to the surgical procedure, the environment, and possible donor-transmitted infections; and the recipient's degree of immunocompromise, which is highest early after transplantation or after treatment for graft rejection.

Types and manifestations of infections follow a relatively predictable time course after transplantation.[9] In the early postoperative period, infections related to nosocomial organisms predominate; common infections include pneumonia, wound and urinary tract infections, and sepsis related to use of intravenous catheters and instrumentation.

Between 1 and 6 months after transplantation, infections caused by opportunistic organisms begin to appear. Causative organisms include cytomegalovirus (CMV), herpes simplex virus, varicella-zoster virus (shingles), *Pneumocystis carinii, Nocardia, Aspergillus, Candida,* and *Toxoplasma gondii.* Several strategies to prevent opportunistic infections are commonly used, including trimethoprim-sulfamethoxazole to prevent *P carinii* pneumonia,[10] nystatin oral solution or troche to prevent oral and esophageal candidal infections, and acyclovir for prophylaxis against herpes simplex virus.

Infection with CMV, a herpesvirus, is an important viral infection in transplant recipients, not only with regard to the development of infectious disease syndromes but also because of its association with acute and chronic rejection, allograft dysfunction, and opportunistic superinfections. Once infected, patients harbor the inactive virus for life, and the virus can be reactivated after transplantation. Patients can also be exposed to CMV through the transplanted organ or through blood transfusions from donors previously exposed to CMV. Signs and symptoms of CMV infection include fever with constitutional symptoms, leukopenia, thrombocytopenia, mild lymphocytosis, and mild hepatitis.[9] Tissue invasion can cause localized disease, often in the gastrointestinal tract (diarrhea, erosion, and ulceration) and lungs (pneumonitis). Patients with the greatest risk for CMV disease (50%-75%) are (1) recipients who are seronegative for the virus who receive a transplant from a donor who is seropositive for CMV and (2) seropositive recipients who require antilymphocyte antibody therapy because of graft rejection.[11] For patients at highest risk, protocols incorporating ganciclovir[12] and hyperimmune globulin have been used for prophylaxis. The recent availability of oral valganciclovir has greatly simplified protocols for CMV prophylaxis and treatment. Preemptive treatment strategies involve periodic surveillance with use of techniques such as plasma polymerase chain reaction[13] and antigenemia testing,[14,15] a rapid diagnostic assay used to detect CMV protein in peripheral blood leukocytes before clinical disease is apparent. Antigenemia testing is useful for early diagnosis of CMV infection and for monitoring the effectiveness of treatment.[16] The use of CMV-negative or leukocyte-reduced blood cell products is recommended to reduce the possibility of new exposures to CMV.

After the first 6 months after transplantation, most infections are community acquired. These infections, although similar to those that occur the general population, place transplant recipients at increased risk because of the recipients' immunocompromised state. Vaccinations should be given only with approval from the transplant center, and live-virus vaccines are contraindicated in immunosuppressed patients. Transplant recipients are advised to avoid contact with ill persons and environments high in dust or mold, use good handwashing practices, obtain prophylactic antibiotics as recommended before dental procedures, and report signs and symp-

Table 1 Classification, dosage, administration, and adverse effects for commonly used immunosuppressive drugs[3-8]

Drug, generic names (brand names)	Classification and action	Usual dosage range	Administration	Adverse effects
Cyclosporine (original formulation: Sandimmune; microemulsion formulation: Neoral, Gengraf) Original and microemulsion formulations are *not* interchangeable	Calcineurin inhibitor Inhibits T-cell proliferation and differentiation	3-10 mg/kg per day orally in divided doses, adjusted according to blood levels Intravenous dose is typically 1/3 of the oral dose	Oral form available as capsules or liquid, usually given every 12 h Oral liquid should be mixed with milk, chocolate milk, apple juice, or orange juice in a glass container Intravenous form should be diluted in isotonic sodium chloride solution or 5% dextrose in water, prepared in a glass container and non–polyvinyl chloride tubing, and administered over 2-6 hours or as a continuous infusion over 24 hours Contact transplant center for preferred intravenous administration method before starting intravenous therapy	Most common: tremor, hirsutism, gingival hyperplasia, hypertension, nephrotoxic effects, hyperlipidemia, hypomagnesemia, hyperuricemia Other effects: headache, numbness, tingling, nausea, vomiting, diarrhea, hepatotoxic effects, increased risk of infection, lymphoma
Tacrolimus, FK-506 (Prograf)	Calcineurin inhibitor Inhibits T-cell proliferation and differentiation	0.05-0.2 mg/kg per day orally in divided doses, adjusted according to blood levels Intravenous dose is 25% of the total daily oral dose	Oral form usually given every 12 hours Intravenous form diluted in isotonic sodium chloride solution or 5% dextrose in water, prepared in a glass or non–polyvinyl chloride container, and given through a dedicated intravenous line as a continuous infusion over 24 hours Contact transplant center for preferred intravenous administration method before starting intravenous therapy	Most common: constipation, nausea, diarrhea, abdominal pain, headache, insomnia, tremor, hyperglycemia, hypertension, nephrotoxic effects, hypomagnesemia, hyperuricemia, hyperlipidemia, thrombocytopenia Other effects: vomiting, arrhythmias, hepatotoxic effects, hyperkalemia, anemia, leukocytosis, flushing, itching, hair loss, neurotoxic effects (including seizures, aphasia, confusion, psychosis, paresthesias), photophobia, blurred vision, tinnitus, increased risk of infection, lymphoproliferative disorders
Azathioprine (Imuran)	Antiproliferative Inhibits purine metabolism, blocks most T-cell functions, inhibits primary antibody synthesis, decreases number of circulating monocytes and granulocytes	2-5 mg/kg per day, adjusted according to white blood cell count Intravenous and oral doses are the same	Usually given as a single daily dose; can be given in divided doses or with food to decrease gastrointestinal intolerance Intravenous dose should be diluted in 5% dextrose in water or isotonic sodium chloride solution and infused over 30-60 minutes	Most common: nausea, vomiting, neutropenia, thrombocytopenia Other effects: anemia, hepatotoxic effects, pancreatitis, alopecia, increased risk of infection and malignant neoplasms
Mycophenolate mofetil (CellCept), Mycophenolic acid (Myfortic)	Antiproliferative Selectively inhibits de novo pathway of purine synthesis; more specific and potent inhibition of T- and B-cell proliferation than azathioprine	Mycophenolate mofetil: 500-1500 mg twice a day Intravenous and oral doses are the same Mycophenolic acid: 360-720 mg orally twice a day	Twice-daily dose usually given every 12 hours Do not crush, break, chew, or open capsules Intravenous dose should be diluted in 5% dextrose in water to 6 mg/mL concentration and infused over at least 2 hours Avoid skin contact with intravenous solution	Most common: constipation, diarrhea, nausea, vomiting, neutropenia, anemia Other effects: abdominal pain, headache, confusion, tremor, hypertension, peripheral edema, gastrointestinal bleeding, increased cough, acne, rash, tremor, changes in phosphorus or potassium levels, insomnia, thrombocytopenia, hyperlipidemia, increased risk of infection or malignant neoplasms
Sirolimus, Rapamycin (Rapamune)	Proliferation signal inhibitor Inhibits T-cell progression from G_1 to S phase, blocking lymphocyte proliferation Inhibits vascular response to injury	1-8 mg orally once daily	Must be taken at least 4 hours after cyclosporine dose Should be taken consistently with or without food Oral solution should be mixed in water or orange juice	Most common: nausea, diarrhea, anemia, thrombocytopenia, neutropenia, hyperuricemia, hyperlipidemia, hypertension Other effects: arthralgia, asthenia, headache, interstitial lung disease, hepatotoxic effects, peripheral edema, rash, insomnia, acne, delayed wound healing, thromboembolism, lymphocele, nephrotoxic effects (only when given in combination with cyclosporine or tacrolimus), increased risk of infection and malignant neoplasms
Methylprednisolone/prednisone (Solu-Medrol, Deltasone)	Corticosteroid Depletes lymphocytes, inhibits macrophages, affects T-cell activation	Prednisone: 2.5-100 mg/d orally, in single or divided doses Methylprednisolone: 250-1000 mg/d intravenously	Oral dose usually given after meals Intravenous dose can be given via infusion over several minutes; doses ≥500 mg should be given over at least 30 minutes	Most common: gastrointestinal upset, anxiety, insomnia, osteoporosis, Cushing syndrome, hyperglycemia, weight gain Other effects: mood swings, growth depression, hypertension, sodium and fluid retention, impaired skin healing, skin atrophy, cataracts, glaucoma, adrenocortical insufficiency, gastric ulceration, acne, hirsutism, increased risk of infection
Antithymocyte globulin, equine (ATGAM)	Polyclonal antibody Depletes lymphocytes	10-15 mg/kg intravenously daily for 14 doses and then 10-15 mg/kg every other day for 14 days (up to 21 doses)	Diluted in isotonic sodium chloride solution, 5% dextrose in water and 0.45% sodium chloride solution, or 5% dextrose in water and 0.2% sodium chloride solution to a maximum concentration of 4 mg/mL; mixture should not be shaken Dose infused via high-flow central vein over at least 4 hours via a 0.22- to 1-µm filter Skin testing before therapy recommended: intradermal dose of 0.1 mL of 1:1000 dilution in isotonic sodium chloride solution with a contralateral injection of isotonic sodium chloride solution as control Can premedicate with acetaminophen, diphenhydramine, and/or corticosteroids to reduce incidence of infusion-related reactions	Most common: fever, chills, pruritus, rash, urticaria, leukopenia, thrombocytopenia, arthralgias, headache, nausea, vomiting, diarrhea Other effects: serum sickness, anaphylaxis, pulmonary edema, apnea, acute renal failure, back pain, chest pain, seizures, paresthesia, confusion, dizziness, syncope, increased risk of infection and malignant neoplasms
Antithymocyte globulin, rabbit; RATG (Thymoglobulin)	Polyclonal antibody Depletes lymphocytes	1.5 mg/kg per day intravenously for 7-14 days	Diluted in 50-500 mL isotonic sodium chloride solution or 5% dextrose in water to a final concentration of ~0.5 mg/mL Mixed gently by inverting the container only once or twice First dose infused over 6 hours; subsequent doses over 4 hours through a 0.22-µm filter via a central catheter or a percutaneously inserted central catheter	Most common: fever, chills, abdominal pain, nausea, diarrhea, dyspnea, dizziness, thrombocytopenia, leukopenia, myalgias/arthralgias, headache, hypertension, tachycardia, peripheral edema, hyperkalemia, infections Other effects: anaphylaxis, serum sickness, anemia, periorbital edema, seizures, congestive heart failure, bradycardia, myocarditis, hyperglycemia, increased risk of malignant neoplasms

Continued

Table 1 *Continued*

Drug	Classification and action	Usual dose range	Administration	Adverse effects
			Can premedicate with acetaminophen, diphenhydramine, and/or corticosteroids to reduce incidence of infusion-related reactions	
Muromonab-CD3, OKT3 (Orthoclone OKT3)	Monoclonal antibody Blocks T-cell function	5 mg/d intravenously for 10-14 days	Patients should be premedicated with methylprednisolone (8 mg/kg), acetaminophen, and diphenhydramine 1-4 hours before administration Before OKT3 is given, patient's temperature must be reduced to 37.8ºC (100ºF) and volume overload/uncompensated heart failure must not be present Solution should not be shaken Administered via intravenous infusion over <1 min For the first few doses, patient should be closely monitored in a facility equipped for cardiopulmonary resuscitation	Most common: cytokine release syndrome (fever, headache, rigor, chills, tremor, nausea, vomiting, abdominal pain, myalgia, arthralgia, rash); occurs with the first 2-3 doses Other effects: severe cytokine release syndrome (pulmonary edema, hypotension, hypertension, tachycardia, tachypnea, respiratory failure, cardiac arrest, arrhythmias, decreased urine output), dizziness, seizures, cerebral edema, encephalopathy, anaphylaxis, aseptic meningitis, hearing loss, impaired vision, aplastic anemia, neutropenia, thrombocytopenia, increased risk of infection and lymphoma
Daclizumab (Zenapax)	Monoclonal antibody IL-2 receptor blocker; inhibits IL-2–dependent T-cell activation	1 mg/kg intravenously for 5 doses First dose given within 24 hours before surgery; subsequent doses, every 14 days	Each dose diluted in 50 mL isotonic sodium chloride solution (mixed gently, not shaken) and infused over 15 minutes through a peripheral or central venous catheter	Most common: gastrointestinal distress Other effects (frequency in clinical trials similar to that of placebo): hypertension, hypotension, chest pain, tachycardia, peripheral edema, dyspnea, pulmonary edema, tremor
Basiliximab (Simulect)	Monoclonal antibody; IL-2 receptor blocker; inhibits IL-2–dependent T-cell activation	20 mg intravenously for 2 doses First dose given approximately 2 hours before transplantation; second dose, 4 days later	Diluted to a volume of 50 mL in isotonic sodium chloride solution or 5% dextrose in water Mixed gently (should not be shaken) and infused over 20-30 min through peripheral or central venous catheter	Most common: gastrointestinal distress Other effects (frequency in clinical trials similar to that of placebo): anemia, hypertension, headache, pulmonary edema, insomnia, asthenia, dizziness, dyspnea, fever, tremor
Alemtuzumab (Campath)	Monoclonal antibody Induces cell lysis of lymphocytes	20-30 mg intravenously once daily (frequency and duration of therapy vary by transplant center)	Desired dose diluted in 100 mL of isotonic sodium chloride solution or 5% dextrose in water Mixed gently (should not be shaken) Should be protected from light Infused over 2 hours Patients premedicated with diphenhydramine and acetaminophen 30 minutes before first dose, at any dose escalation, and as clinically indicated	Most common: anemia, neutropenia, thrombocytopenia, infection, infusion-related reactions (hypotension, bronchospasm, shortness of breath, fever, chills, rigor, rash), nausea, vomiting, diarrhea, headache, fatigue Other effects: dysthesias, dizziness, tremor, peripheral edema, bronchitis, pneumonitis, hypertension, hypotension, tachycardia, myalgias, skeletal pain

toms of infection promptly. Care providers should maintain a high index of suspicion, because the usual signs and symptoms of infection, such as inflammation and fever, can be masked by immunosuppressive drugs. Aggressive investigation of seemingly minor signs and symptoms or findings may be warranted.

Other Problems Common in Transplant Recipients

Several other medical problems are common in transplant recipients. These include renal dysfunction, hypertension, diabetes, hyperlipidemia, osteoporosis, malignant neoplasms, and difficulties in psychosocial adjustment.

Renal Dysfunction

Renal dysfunction is one of the most common adverse effects of treatment with cyclosporine and tacrolimus. The mechanisms are largely related to vasoconstrictive effects on afferent arterioles and to tubulointerstitial fibrosis.[17] Monitoring and control of blood levels of cyclosporine and tacrolimus, use of calcineurin inhibitor–free immunosuppression protocols, and avoidance of additional nephrotoxic agents are approaches to preserve renal function.

Hypertension

Hypertension develops in many transplant recipients and is associated with immunosuppressive medications, especially cyclosporine and, to a lesser degree, tacrolimus, sirolimus, and corticosteroids. Other factors may include preexisting hypertension, worsening renal function, and volume expansion. Control of hypertension can be challenging and often requires the use of several agents.

Diabetes

Several immunosuppressive medications, especially corticosteroids and tacrolimus, are associated with hyperglycemia. Many transplant recipients require therapy, including dietary modification, use of oral hypoglycemics, and/or use of insulin.

Hyperlipidemia

Hyperlipidemia is common after transplantation. Development can be multifactorial; risk factors include a history of ischemic coronary disease, corticosteroid therapy, high blood glucose levels, and weight gain.[18] Lipid-lowering 3-hydroxy-methylglutaryl coenzyme A reductase inhibitors (statins) are effective in lowering lipid levels,[19] although combining cyclosporine or tacrolimus and a statin increases the risk of rhabdomyolysis and requires close follow-up for signs and symptoms of muscle weakness and elevated levels of creatine kinase and liver enzymes.

Osteoporosis

Before transplantation, transplant recipients often have decreased bone density, which is related to multiple factors, including inactivity, low calcium intake, cachexia, and prolonged loop diuretic therapy. Transplant recipients experience further loss of bone mass within months after transplantation[20]; reductions are marked in older patients despite calcium supplementation.[21] Cyclosporine, tacrolimus, and corticosteroids all have been associated with bone loss. Compression fractures are common. Preventive strategies include weight-bearing exercise, calcium supplementation, reduction in or withdrawal of corticosteroid therapy, and postmenopausal estrogen replacement. Bisphosphonates, calcitriol,[22,23] and calcitonin nasal spray have been useful for the prevention and treatment of transplant-related osteoporosis.

Malignant Neoplasms

Malignant neoplasms, a major cause of late death in transplant recipients, are a consequence of chronic immunosuppression. The incidence increases with intensity of immunosuppression and cumulative years of exposure. The most common tumors involve the skin and lips[24]; the next most common are lymphoproliferative disorders, mainly non-Hodgkin lymphoma. The frequency of lymphoproliferative malignant tumors is lowest in kidney recipients and highest in heart and lung recipients and children.[25] An increased incidence of lymphoma has been observed in patients who received muromonab-CD3 (OKT3) or antithymocyte glob-

Table 2 Drug interactions for commonly used immunosuppressive agents* [6,7]

Drug	Interactions that increase drug level	Interactions that decrease drug level	Other drugs whose level/effect is increased by drug	Other drugs whose level/effect is decreased by drug	Interactions producing additive toxic effects
Cyclosporine	Acetazolamide, allopurinol, amiodarone, amlodipine, amprenavir/fosamprenavir, azithromycin, bromocriptine, carvedilol, ceftriaxone, chloramphenicol, chloroquine, cimetidine, cisapride, clarithromycin, clonidine, colchicine, dalfopristin/quinupristin, danazol, diltiazem, doxorubicin, doxycycline, erythromycin, felodipine, fluconazole, fluvoxamine, glipizide, glyburide, imatinib, isoniazid, itraconazole, ketoconazole, mestranol, methotrexate, methylprednisolone, methyltestosterone, metoclopramide, metronidazole, nefazodone, nelfinavir, nicardipine, nifedipine, norfloxacin, oral contraceptives, pentazocine, prednisone, propafenone, ritonavir, saquinavir, stanozolol, tacrolimus, tamoxifen, telithromycin, testosterone, ticarcillin, verapamil, voriconazole Also: grapefruit juice, fatty foods, milk	Bosentan, bupropion, carbamazepine, cholestyramine, clindamycin, dexamethasone, nafcillin, nevirapine, octreotide, omeprazole, orlistat, oxcarbazepine, phenobarbital, phenytoin/fosphenytoin, primidone, rifabutin, rifampin, sulfadiazine, sulfamethoxazole/trimethoprim, terbinafine, warfarin Also: St. John's wort, garlic, red wine, chronic alcohol use	Bosentan, caspofungin, colchicine, digoxin, etoposide, methotrexate, nifedipine, pravastatin, sirolimus, tolterodine, tretinoin	Mycophenolate mofetil, warfarin	Nephrotoxic effects: angiotensin-converting enzyme inhibitors, acyclovir, amikacin, amphotericin, ganciclovir, gentamicin, melphalan, nonsteroidal anti-inflammatory drugs, tacrolimus, tobramycin Myositis: statins Increased serum levels of potassium: angiotensin-converting enzyme inhibitors, amiloride, spironolactone Hepatotoxic effects: valproic acid Neurotoxic effects: acyclovir, ganciclovir, imipenem, quinolones
Tacrolimus	Amprenavir/fosamprenavir, basiliximab, bromocriptine, chloramphenicol, cimetidine, clarithromycin, clotrimazole, cyclosporine, dalfopristin/quinupristin, danazol, diltiazem, erythromycin, ethinyl estradiol, fluconazole, itraconazole, ketoconazole, methylprednisolone, metoclopramide, metronidazole, nefazodone, nelfinavir, nicardipine, nifedipine, omeprazole, ritonavir, saquinavir, telithromycin, theophylline, verapamil, voriconazole	Carbamazepine, caspofungin, nafcillin, nevirapine, oxcarbazepine, phenytoin/fosphenytoin, phenobarbital, primidone, rifabutin, rifampin Also: St. John's wort	Ziprasidone		Nephrotoxic effects: angiotensin-converting enzyme inhibitors, acyclovir, amikacin, amphotericin, cyclosporine, ganciclovir, gentamicin, nonsteroidal anti-inflammatory drugs, tobramycin Myositis: statins Increased serum levels of potassium: angiotensin-converting enzyme inhibitors, amiloride, spironolactone
Azathioprine	Allopurinol, mesalamine, sulfasalazine			Cyclosporine, warfarin	Bone marrow suppression: angiotensin-converting enzyme inhibitors, ganciclovir, sulfamethoxazole/trimethoprim Hepatotoxic effects: methotrexate
Mycophenolate mofetil/mycophenolic acid		Antacids, cholestyramine, cyclosporine, iron	Acyclovir, ganciclovir	Oral contraceptives	
Sirolimus	Amprenavir/fosamprenavir, cyclosporine, diltiazem, erythromycin, itraconazole, ketoconazole, saquinavir, telithromycin, voriconazole Also: grapefruit juice	Carbamazepine, nafcillin, nevirapine, oxcarbazepine, phenobarbital, phenytoin/fosphenytoin, primidone, rifabutin, rifampin Also: St. John's wort			
Prednisone/methylprednisolone	Clarithromycin, itraconazole, ketoconazole, ritonavir Methylprednisolone only: aprepitant, dalfopristin/quinupristin, diltiazem	Butalbital, carbamazepine, oxcarbazepine, phenobarbital, phenytoin/fosphenytoin, primidone, rifampin	Cyclosporine, tacrolimus	Bupropion, quetiapine	Hyperglycemia: gatifloxacin Prolonged weakness/myopathy: atracurium, cisatracurium, pancuronium, rocuronium, vecuronium Peripheral edema: montelukast Gastrointestinal distress: aspirin
Basiliximab			Tacrolimus		

*Blank cells indicate that no drug interactions of this type have been documented.

ulin for induction immunosuppression, but not in those who received antibodies to IL-2 receptors.[25] The risk is also increased in patients who receive repeated courses of muromonab-CD3.[26] Incidences of other common tumors (eg, breast, cervix, lung, colon, prostate) are similar to the incidences in the general population,[24] although the tumor course may be more aggressive because of immunosuppressive medications. All transplant recipients require comprehensive physical examination and screening at recommended intervals. Treatment for established tumors often involves reduction of immunosuppression therapy, with careful follow-up to screen for graft rejection.

Difficulties in Psychosocial Adjustment

Most transplant recipients return to a full life after transplantation; many return to work or school within 3 to 6 months, although some remain disabled for medical reasons. Barriers to full rehabilitation include the inability to find work, concerns about future medical needs, and fear of loss of disability income or insurance. Depression and anxiety are common because of disruptions in family dynamics, financial concerns, physical discomfort, and setbacks after transplantation.[27] The adverse effects of immunosuppressive agents, including cushingoid appearance, hirsutism, acne, mood disorders, growth retardation in children, cataracts, and osteoporosis, also present challenges that affect body image and the recipients' sense of well-being. In some recipients, perceived overwhelming physical or psychosocial challenges may lead to noncompliance with posttransplant medications.

Organ-Specific Considerations

In addition to problems common to many transplant recipients, organ-specific issues must be considered. In the remainder of this article, we provide information specific to heart,

Table 3 Nursing care for patients who are organ transplant recipients

Key patient-specific information	Key assessments	Key interventions	Call the transplant center for
All transplant patients			
Notify transplant center of admission and request key patient-specific information: Type of transplant Date of/time since transplantation Primary diagnosis before transplantation History of graft rejection and other complications Current medications and usual schedule, confirming: • Immunosuppression medications, schedule, and doses • Formulation or brand of immunosuppression medications usually taken • Recent blood levels of immunosuppressive drugs, if known	Vital signs Weight Signs and symptoms of infection	Maintain immunosuppressive drugs on patient's usual schedule Protect patient from obvious sources of infection Use universal precautions Aggressively investigate signs and symptoms of infection Evaluate any new drug for nephrotoxic effects If procedure that requires contrast medium is scheduled, consider hydration and acetylcysteine (Mucomyst) for renal protection Adjust drug dosages as recommended according to renal function Ensure that blood products administered have reduced numbers of leukocytes and are free of cytomegalovirus	Body temperature >37.8°C (>100°F); if ≥38.4°C (≥101.5°F), call immediately Nausea, vomiting, or diarrhea lasting >24 hours Patient cannot take immunosuppressive medication or missed doses Starting or stopping any medications to review for drug interactions Monitoring of immunosuppressive drug levels Adjustment of doses of immunosuppressive drugs Before administration of vaccines (live vaccines contraindicated)
Organ-specific assessments			
Organ- and patient-specific key information	**Key assessments**	**Signs and symptoms of rejection**	**Call the transplant center for**
Heart			
Usual heart rhythm and blood pressure range Any known abnormalities of cardiac function Whether coronary artery vasculopathy is present	Heart rate and rhythm Blood pressure Cardiac and respiratory system examinations	Fluid gain, edema Shortness of breath Fatigue Decreased exercise tolerance Abdominal bloating S_3 gallop New bradycardia Atrial fibrillation or flutter Decrease in blood pressure Decreased ejection fraction, hemodynamic compromise (late signs)	Signs or symptoms of heart failure or rejection Suspected ischemic events Hemodynamic compromise New abnormal cardiac findings or results on cardiac tests
Lungs			
Type of transplant (single, bilateral, living lobar, or heart-lung) Usual spirometry results and oxygenation	Cardiac and respiratory system examinations Spirometry Chest radiograph Sputum culture Oxygenation	Cough Dyspnea Fever Fatigue Adventitious lung sounds (crackles, wheezes) Infiltrates or effusions on chest radiograph Hypoxemia Decreases in pulmonary function tests	Signs or symptoms of lung rejection or infection Chest pain Changes from baseline in blood pressure, heart rate, or resting respiratory rate
Liver			
Results of baseline liver chemistry tests Catheters or drains Bile duct reconstruction	Results of liver chemistry tests Renal function Prothrombin time and international normalized ratio Bile drainage	Elevated levels of liver enzymes Fatigue Fever Tenderness or pain over the liver Dark or tea-colored urine White or clay-colored stools Yellow eyes Yellow skin/jaundice Ascites Pruritus	Significant change in levels of liver enzymes Sudden increase or decrease in catheter drainage Signs or symptoms of graft rejection Change in mental status

Continued

lung, liver, kidney, and pancreas transplant recipients. Table 3 outlines key information on recipients, transplant-specific assessments, and interventions to help nurses design a plan of care for transplant recipients being treated in a nontransplant setting.

Heart Transplantation

Heart transplantation is the preferred treatment for a select group of patients with end-stage heart disease. The most common diagnoses in such patients are cardiomyopathy (46%) and coronary artery disease (45%); smaller numbers of patients have valvular (3%) or congenital heart disease (2%) or require retransplantation (2%).[28] Current survival rates reported by the International Society for Heart and Lung Transplantation are 83% at 1 year after transplantation and 72% at 5 years; 50% of patients survive more than 9.4 years.[28]

In the standard orthotopic surgical technique originally described by Lower and Shumway, cuffs of the recipient's right and left atria are attached to the right and left atria of the donor heart at suture lines, with anastomosis of the aorta and pulmonary artery completing the procedure.[29] Currently, the bicaval technique in which the intact right atrium of the donor heart is preserved by anastomoses at the recipient's superior and inferior vena cavae[30] is used more often. Figure 2 illustrates both surgical techniques.[31]

The transplanted heart is denervated; that is, it is completely disconnected from the direct autonomic nerve innervation of the sympathetic and parasympathetic (vagal) chains that influence the heart's rate, electrical conduction speed, and contractile force. As a result, the resting heart rate is higher than normal, usually 90/min to 110/min. During stress or exercise, acceleration of the heart rate is blunted and delayed. After the completion of exercise, deceleration of the heart rate is gradual and prolonged. Blunting of the maximum heart rate is associated with limitations in maximum oxygen consumption, to approximately 64% of predicted values,[32] and thus lower exercise capacity. The lack of direct sensory input results in a lack of angina; however, evidence indicates that reinnervation can occur over time.[33] The denervated heart has an altered response to cardiac drugs whose actions are mediated through the autonomic nervous system. For example, atropine is ineffective when used to treat bradycardia in a transplant recipient because this drug works by blocking parasympathetic input. In heart transplant recipients with cardiac denervation, isoproterenol is the drug of choice for bradycardia because it has direct stimulatory effects on cardiac adrenergic receptors.

Approximately 30% to 40% of heart transplant recipients experience a rejection episode during the first year after transplantation,[28] most often during the first 6 months.[34] Rejection is diagnosed via endomyocardial biopsy, in which 4 to 6 samples of myocardial tissue are obtained for histological examination. Biopsy samples are interpreted according to a standardized scale; the degree of interstitial cellular infiltrate and the presence of myocyte damage or necrosis are determined. A total of 4 numeric grades ranging from no rejection (grade 0) to the most severe (grade 4) have been described,[35] but this scale is being revised to simplify

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Table 3 *Continued*

Organ- and patient-specific key information	Key assessments	Signs and symptoms of rejection	Call the transplant center for
Kidney			
Baseline serum level of creatinine Baseline weight Calculated estimated creatinine clearance by Cockcroft-Gault formula: Males: (140 – age) × (lean body weight in kilograms) ÷ (stable creatinine level × 72) Females: Calculate as for males × 0.85	Accurate inputs and outputs, daily weights Blood pressure Serum levels of electrolytes, urea nitrogen, and creatinine Volume status Evaluation for signs and symptoms of urinary tract infection Bedside bladder ultrasounds if urinary residuals suspected Renal transplant ultrasound	Increased creatinine level Decreased urine output Fever, chills Graft tenderness, swelling Edema, pulmonary edema Hypertension Proteinuria Hematuria Signs and symptoms of uremia	Creatinine level greater than baseline level Signs or symptoms of graft rejection Need for a biopsy
Pancreas			
Pancreas transplant category Simultaneous pancreas and kidney Pancreas after kidney Pancreas alone Surgical technique of exocrine drainage: bladder or enteric Baseline renal function	Renal function Serum levels of amylase and lipase Urine level of amylase Blood glucose levels Character of stool and urine Abdominal or urinary tract complaints	Elevated serum levels of amylase Elevated serum levels of lipase Decrease in urinary levels of amylase (if graft bladder drained) Hyperglycemia	Acute abdominal symptoms Signs or symptoms of rejection Laboratory values outside of patient's normal values Bleeding

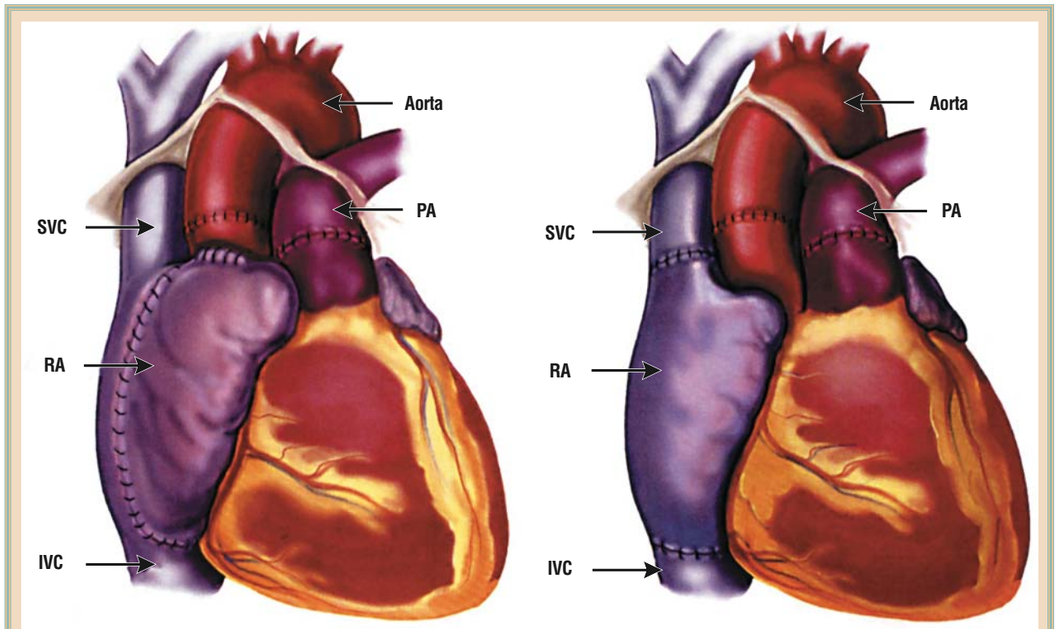

Figure 2 Surgical techniques in heart transplantation. Left, Lower-Shumway orthotopic technique. Right, Bicaval modification.

Abbreviations: IVC, inferior vena cava; PA, pulmonary artery; RA, right atrium; SVC, superior vena cava.
Adapted from Chen et al,[31] copyright 1999 Landes Bioscience, with permission from Sage Publications.

the rejection grades to 3 categories: mild, moderate, and severe.[36]

Cardiac allograft vasculopathy (CAV) is an accelerated form of diffuse and obliterative arteriosclerosis that remains a principal limiting factor in long-term survival in heart transplant recipients.[28] CAV occurs in more than 40% of recipients within 5 years after transplantation[37] and can result in clinical events ranging from myocardial ischemia and infarction to congestive heart failure, ventricular arrhythmias, and sudden death. The etiology is thought to involve immunological and nonimmunological mechanisms, resulting in vascular endothelial injury and a localized sustained inflammatory response[38] that leads in turn to proliferation and thickening of the vascular intima and, ultimately, to narrowing of the vessel lumen along its entire length. Prevention and treatment involve strategies to manage nonimmunological risk factors, including smoking cessation; exercise; weight loss; control of hypertension; use of cholesterol-lowering drugs, usually statins; close diabetes management; and daily administration of low-dose aspirin. Modification of the immunosuppressive drug regimen to include the proliferation signal inhibitors sirolimus or everolimus may reduce the incidence and severity of CAV,[39] slow CAV disease progression,[40] and reduce lesions in patients with established disease.[41] Revascularization procedures such as angioplasty and coronary artery bypass graft surgery benefit only a minority of heart transplant recipients with focal disease and are thought to be only palliative for recipients with diffuse CAV. The use of intracoronary stents shows promise for improved long-term vessel patency and improved clinical outcome.[42] Retransplantation is the ultimate option in certain patients.

Lung Transplantation

Lung transplantation is primarily indicated in patients with chronic lung disease whose condition deteriorates despite maximal therapy and who, without transplantation, would die of end-stage lung disease; rarely is it indicated for pulmonary patients who are acutely or critically ill.[43] The most common diagnoses in patients who have lung transplantation are chronic obstructive pulmonary disease (39% of recipients), idiopathic pulmonary fibrosis (17%), cystic fibrosis (16%), α_1-antitrypsin deficiency emphysema (9%), and primary pulmonary hypertension (4.2%).[44] Since 1995, slightly more than 50% of all lung transplants have been performed in patients 50 to 64 years old who had either chronic obstructive pulmonary disease or idiopathic pulmonary fibrosis.[44]

Survival after lung transplantation has improved significantly; current rates are 74% at 1 year, 58% at 3 years, 47% at 5 years, and 24% at 10 years. Survival is related, at least in part, to the pulmonary disease that led to transplantation: survival rates at 10 years remain highest in recipients who had cystic fibrosis, whereas rates for recipients who had chronic obstructive pulmonary disease rank near the bottom.[44]

The first successful single-lung transplants, in 2 patients with idiopathic pulmonary fibrosis, were performed in 1983 by the Toronto Lung Transplant Group.[45] Double-lung transplantation, in which 2 lungs are transplanted as a group (en bloc) with a tracheal anastomosis, was first accomplished in 1985.[46] The en bloc procedure was indicated for patients with bronchiectasis (eg, patients with cystic fibrosis) but was complicated by problems with the tracheal anastomosis that resulted in dehiscence and death. The procedure was then modified in 1989 to a bilateral sequential lung transplant, in which each lung is removed and attached separately.[47] Single-lung transplantation incorporating bronchial anastomoses via a telescoping technique with the recipient and donor airway was later used to avoid airway complications.[48] Figure 3 illustrates the surgical technique of single-lung transplantation.[49]

Living-donor lobar transplantation was first performed in a 12-year-old girl with bronchopulmonary dysplasia.[50] Adolescents who receive lung transplants from living donors have posttransplant survival rates similar to those of adolescents who receive lung transplants from

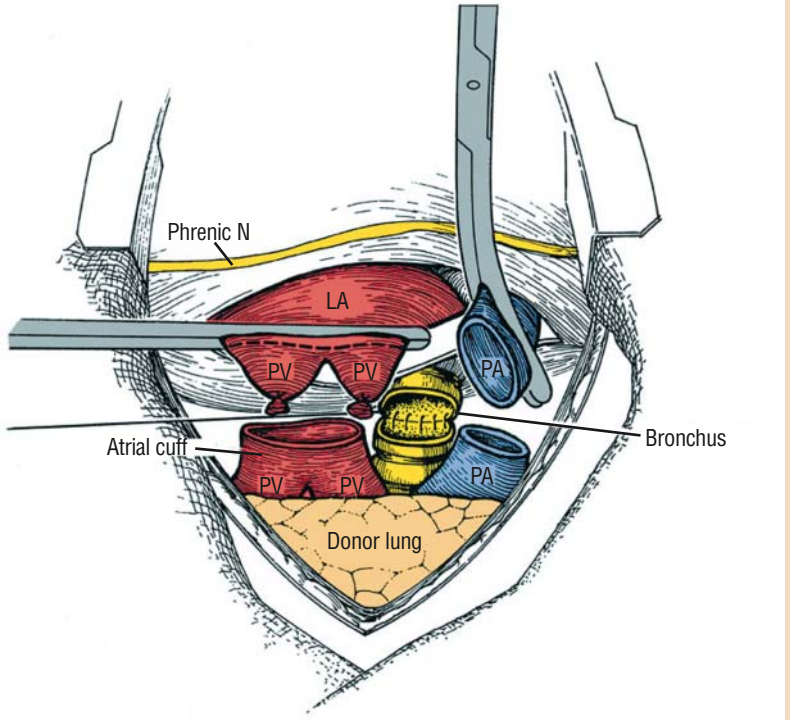

Figure 3 Surgical technique in single-lung transplantation.

Abbreviations: LA, left atrium; PA, pulmonary artery; PHRENIC N, phrenic nerve; PV, pulmonary vein. Adapted from McCarthy et al,[49] copyright 1996 Futura Publishing Co Inc, with permission from Blackwell Publishing.

deceased donors.[51] Currently, living-donor lobar lung transplantation has become an option for potential lung transplantation patients who may not survive until a deceased-donor transplant is available.[52,53]

Postoperative pulmonary complications in lung transplant recipients include abnormalities in gas exchange, reperfusion edema, pulmonary emboli, and airway problems (eg, stenosis, bronchomalacia, exophytic granulation tissue, necrosis, and dehiscence).[54] Ineffective airway clearance related to denervation of the transplanted lung may contribute to pooling of secretions, leading to infection. Acute rejection is common, occurs at least once in most lung transplant recipients, and is usually reversible. Management of acute rejection consists of use of high-dose corticosteroids, optimization of maintenance immunosuppression, and additional immunotherapy, when necessary.[55]

During the first 30 days after lung transplantation, the most common cause of death is related to graft failure and infections other than CMV infection. CMV infection and acute rejection usually occur during the first year but generally are not fatal. After the first year, causes of death include infection and malignant tumors, but approximately 30% of deaths have been attributed to bronchiolitis obliterans syndrome.[44] Bronchiolitis obliterans syndrome, also called chronic rejection, is a term used to explain graft deterioration that affects the small airways and results in persistent airflow obstruction as indicated by spirometry (not histological examination) in the absence of infection or acute rejection.[56] Although the diagnostic criteria for the syndrome are based on a decrease in lung function as indicated by pulmonary function testing, histological changes such as fibrous scarring can be seen in the walls of the airway, or granulation tissue may obliterate the lumen of the airway. The term *bronchiolitis obliterans* is used when histological examinations show scarring that affects the small airways. Recurrent acute rejection or histologically severe rejection is a major risk factor for the late development of chronic rejection.[56]

Retransplantation has been performed in lung transplant recipients with severe early graft dysfunction or airway healing problems, but according to the Retransplant Registry, 63% of retransplantations occur in patients with bronchiolitis obliterans syndrome.[57] Early survival rates after retransplantation are lower than after the first transplantation, but the results for retransplantations performed because of chronic rejection do not differ from those for retransplantations performed because of other indications.[57]

Liver Transplantation

Liver transplantation is the recognized standard of care for patients with acute and chronic end-stage liver disease.[58] Advances in surgical and procurement procedures and in immunosuppressive medications have contributed to the current success of liver transplantation. Survival rates 1 and 5 years after transplantation are 86.5% and 72.6%, respectively; 55.3% of liver transplant recipients survive more than 10 years.[59]

The most common indications for liver transplantation in adults are chronic hepatitis, alcoholic liver disease, primary biliary cirrhosis, primary sclerosing cholangitis, and autoimmune hepatitis. The most common indications in children include cholestatic diseases such as biliary atresia and metabolic disorders such as α_1-antitrypsin deficiency.[60]

The persistent shortage of deceased donor livers has led to several surgical alternatives, including reduced-liver, split-liver, and living-donor liver transplantation. Reduced-liver transplantation involves the left lobe or the left lateral segment of the donor liver. Split-liver transplantation allows the left lobe to be used for a child and the right lobe for an adult. In living-donor transplantation, the left lobe is donated to a child or the right lobe is used for an adult recipient, because the left lobe typically would not supply sufficient liver mass for an average-sized adult patient.[61] Because of the unique ability of the liver to regenerate, in living-donor transplantation, both the donor's liver and the recipient's liver return to near-normal volume within 2 to 3 months after transplantation; most of the regeneration occur within the first week after resection or transplantation.[62]

The transplant operation begins with an incision in the abdomen, often referred to as a "Mercedes" or "inverted Y" incision. The removal of the diseased liver, the hepatectomy, is accomplished by locating the main liver arteries and veins and bile ducts and then clamping them. Simultaneously, the donor liver is prepared for transplantation. The donor gall bladder is removed. The donor liver is placed in the abdominal cavity and the vessels are anastomosed; then the bile duct is connected. The donor and recipient bile ducts may be anastomosed end to end, a procedure known as a choledochocholedochostomy, typically over a biliary stent or a T tube. If the recipient bile duct is not appropriate for end-to-end reconstruction, the donor bile duct is connected to the recipient's duodenum, a procedure called a choledochojejunostomy or Roux-en-Y procedure, and the recipient will not have a tube.[60] Figure 4 illustrates the surgical technique in liver transplantation.[63]

Liver function is monitored by using assays of prothrombin time or the international normalized ratio and liver chemistry tests (ie, assays of serum levels of alanine aminotransferase, aspartate aminotransferase, γ-glutamyl transpeptidase, alkaline phosphatase, total bilirubin, albumin, lactic dehydrogenase, and ammonia) and by checking for normal serum levels of lactate. Bile production through the T tube should begin immediately; thick, dark, gold, brown, or green bile is an indicator of good liver function. The T tube is left open to a drainage bag for about the first 5 to 7 days and then is capped off and left in place for 3 to 4 months; a cholangiogram is performed before the tube is removed.

The most common complications after liver transplantation are rejection of the graft, infection, mechanical problems, recurrent hepatitis, and psychological changes. Rejection occurs in up to 60% of all liver transplant recipients, often with at least a single episode within the first 3 months after transplantation.[58] A biopsy is the optimal test to differentiate among rejection, infection, and recurrent disease, all of which have similar signs and symptoms. Mechanical problems may include bleeding at the anastomosis site; hepatic artery thrombosis or stenosis; and bile duct leaks, strictures, or obstructions. Recurrent liver diseases, such as hepatitis, primary biliary cirrhosis, primary sclerosing cholangitis, autoimmune hepatitis, and nonalcoholic steatohepatitis are the most common causes of late liver failure. In recipients who had transplantation because of hepatitis C, the infection recurs within 2 years 100% of the time and may occur as early as a few weeks after transplantation.[64]

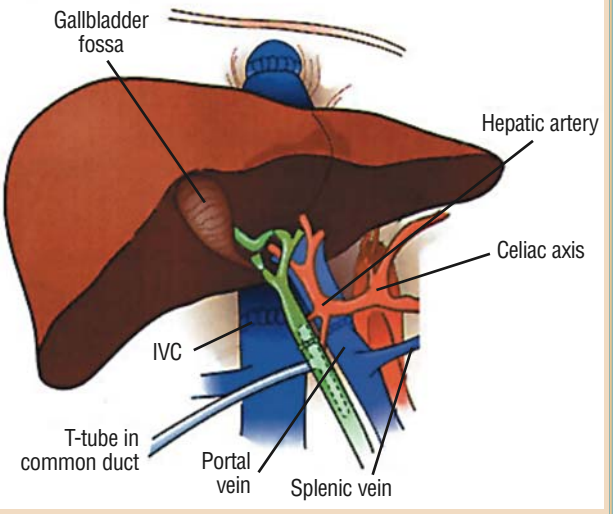

Figure 4 Surgical technique in liver transplantation.

Abbreviation: IVC, inferior vena cava.
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Combination therapy with long-acting interferon and ribavirin is the current treatment of choice for recurrent hepatitis C.[65]

Kidney Transplantation

Kidney transplantation offers improved quality of life[66] and long-term survival for patients with end-stage renal disease.[67] In a population of long-term dialysis patients, Wolfe et al[67] found that dialysis patients on the transplant waiting list had a lower mortality rate than did nonlisted dialysis patients, most likely reflecting that healthier patients are placed on the waiting list. However, a significant long-term survival benefit was detected in patients who received their first deceased-donor kidney transplant. Although the mortality rate in these recipients was higher initially after transplant surgery than in patients who remained on the waiting list, long-term mortality was 48% to 82% lower.[67]

The primary diagnoses in patients on the waiting list for kidney transplants are diabetes (26%), glomerular disease (22%), hypertension (19%), and polycystic kidney disease (6%).[59] Patients can receive kidneys from living or deceased donors. The percentage of transplants from living donors has steadily increased, to more than 40% of all kidney transplants performed annually.[59] The survival rates at 1 and 5 years after receipt of a deceased-donor kidney are 94.5% and 81%, respectively, compared with 97.6% and 89.8% after receipt of a living-donor kidney. Rates of graft survival at 1 and 5 years are 89.0% and 66.2%, respectively, for deceased-donor kidney recipients compared with 94.6% and 79.2% for living-donor kidney recipients.[59]

The surgical procedure is performed through an incision in the right or left iliac fossa. The extraperitoneal space is preserved, the renal artery is anastomosed to the external iliac artery, and then the renal vein is anastomosed to the external iliac vein. Next a neocystostomy is performed to prevent reflux.[68] An indwelling ureteral stent is commonly placed and subsequently is removed several days[69] or weeks after transplantation. Surgical complications after transplantation are relatively rare but may include urological (urine leaks, reflux, obstruction), vascular (renal vein thrombosis, renal artery thrombosis, renal artery stenosis), or wound complications (infection, lymphoceles).[68] Figure 5 illustrates the surgical technique in kidney transplantation.[70]

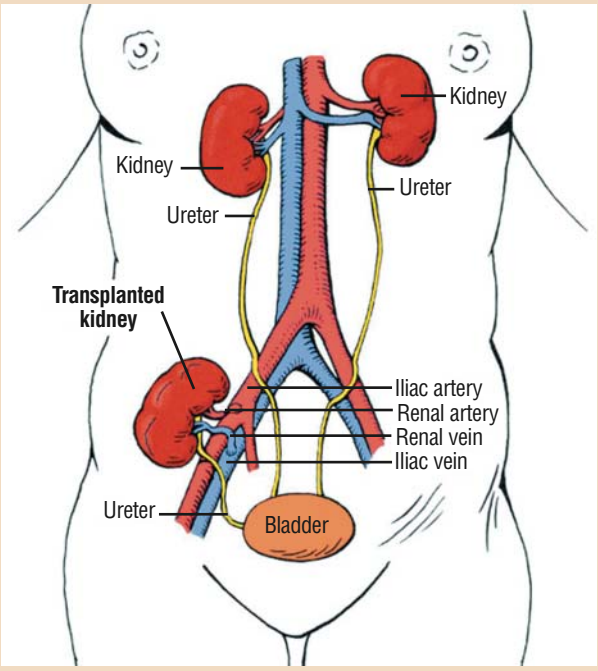

Figure 5 Surgical technique in kidney transplantation.

Adapted from Humar and Dunn,[70] copyright 2005, with permission from the McGraw Hill Companies.

Rejection rates in the first year after transplantation have decreased dramatically, to 15% in 2002.[59] Diagnosis of rejection is made on the basis of percutaneous biopsy of the allograft with local anesthesia and ultrasound guidance. Multiple core biopsy samples are obtained and are histologically graded by using the standardized Banff classification scheme. The 6 Banff diagnostic categories are normal; antibody-mediated rejection; borderline changes suggestive of acute cellular rejection; acute, active cellular rejection; chronic/sclerosing allograft nephropathy; and other.[71] Some infections, such as those caused by CMV and polyomavirus, may mimic rejection but can be recognized on the basis of biopsy results and treated. Treatment for polyomavirus infection has been a challenge, but reduction of immunosuppression therapy is currently indicated to avoid loss of the transplanted kidney.[72] Examination of biopsy samples may also reveal recurrence of primary disease or de novo renal diseases.

Chronic allograft nephropathy is a common cause of late graft failure. It begins with a deterioration of renal function and is commonly associated with proteinuria and hypertension.[73] Histological characteristics are nonspecific, and the course is unpredictable, with renal function fluctuating.[74] Risk factors for

chronic allograft nephropathy include delayed graft function immediately after transplantation, acute rejection, serum levels of creatinine greater than 177 µmol/L (2.0 mg/dL) by 6 months, African-American ethnicity, donor age greater than 50 years,[75] and long-term use of calcineurin inhibitor drugs used for immunosuppression.[76]

Preservation of long-term graft function involves, in addition to immunosuppressive medications to prevent rejection, interventions to control hypertension, lipid levels, blood glucose levels, and proteinuria. Smoking cessation, weight control, a low-cholesterol and low-sodium diet, and exercise are also important. If renal dysfunction occurs, care providers should consider all possible prerenal (eg, hypotension; dehydration; renovascular disease; infection; medications; and nephrotoxic drugs, including calcineurin inhibitors), renal (eg, acute tubular necrosis, rejection, pyelonephritis, contrast nephropathy, atheroemboli, polyomavirus and CMV infections), and postrenal (eg, obstruction, dysfunctional bladder, and ureteral or urethral strictures) causes and should correct the found cause to return the recipient to baseline renal function. If graft loss occurs, retransplantation may be considered.

Pancreas Transplantation

Many patients with type 1 diabetes and some with type 2 diabetes experience wide fluctuations in blood glucose levels despite standard insulin regimens. These patients are at risk for serious consequences such as seizure or coma and for secondary complications such as blindness, amputation, nerve damage, and renal failure. Pancreas transplantation may be a practical alternative, resulting in stable blood glucose levels and improved quality of life. As reported to the International Pancreas Transplant Registry, currently the rate of survival 1 year after pancreas transplantation is more than 95%.[77] Pancreas transplantation is offered in 3 categories: simultaneous pancreas and kidney transplantation, pancreas transplantation after kidney transplantation, and pancreas transplantation alone. Graft survival at 1 year is higher in recipients who have simultaneous pancreas and kidney transplantation (85%) than in recipients who have pancreas transplantation after kidney transplantation (78%) or pancreas transplantation alone (77%) groups.[77]

The denervated donor pancreas is placed heterotopically, most often on the right side of the pelvis and usually through a midline incision.[78] The arterial and venous blood vessels of the donor pancreas are anastomosed to the blood vessels of the recipient's pancreas, and insulin is secreted into the recipient's circulation as needed to maintain euglycemia. The pancreas contains 2 types of tissue: endocrine tissue, which contains the insulin-producing beta cells of the islets of Langerhans, and exocrine tissue, which contains the acinar cells that produce the digestive enzymes secreted through the pancreatic duct. The enzymes that are produced by the exocrine tissue of the transplanted pancreas must be managed and safely excreted by enteric or bladder drainage (Figures 6A and 6B).[79] In enteric drainage, the most common surgical technique used, the deceased-donor pancreas, along with a segment of donor duodenum, is anastomosed to the small intestine. Pancreatic fluid and enzymes are excreted into the gastrointestinal tract. In bladder drainage, the deceased-donor pancreas, along with a segment of donor duodenum, is anastomosed to the dome of the bladder. Pancreatic fluid and enzymes are excreted into the urine. Urinary amylase levels may be monitored to detect graft rejection.

Rejection of a transplanted pancreas can be difficult to diagnose and is usually evident first in the exocrine pancreas. Signs and symptoms may include elevated levels of serum amylase and lipase and, in a bladder-drained pancreas, a decrease in the level of urinary amylase. An increase in the level of blood glucose indicates altered function of the endocrine pancreas and is usually a late sign of rejection. Once hyperglycemia becomes evident, the rejection process is probably irreversible.[80] A pancreas biopsy can provide a definitive diagnosis of rejection but should be done by experienced personnel in a transplant setting. Any rejection treatment should be decided on and managed by the transplant team. Rejection rates at 1 year after transplantation range from 2% to 8% and vary slightly according to pancreas transplantation surgical category.[77]

The rate of pancreas graft loss in the first year after transplantation ranges from 6% to 8%; the most common cause is technical failure.[77] Any pancreas transplant recipient who has acute abdominal signs and symptoms, such as severe pain or hematuria, or sudden onset of hyperglycemia may have pancreas graft thrombosis.[80] Generally, this situation requires immediate exploratory laparotomy to remove the pancreas before necrosis occurs. Whenever

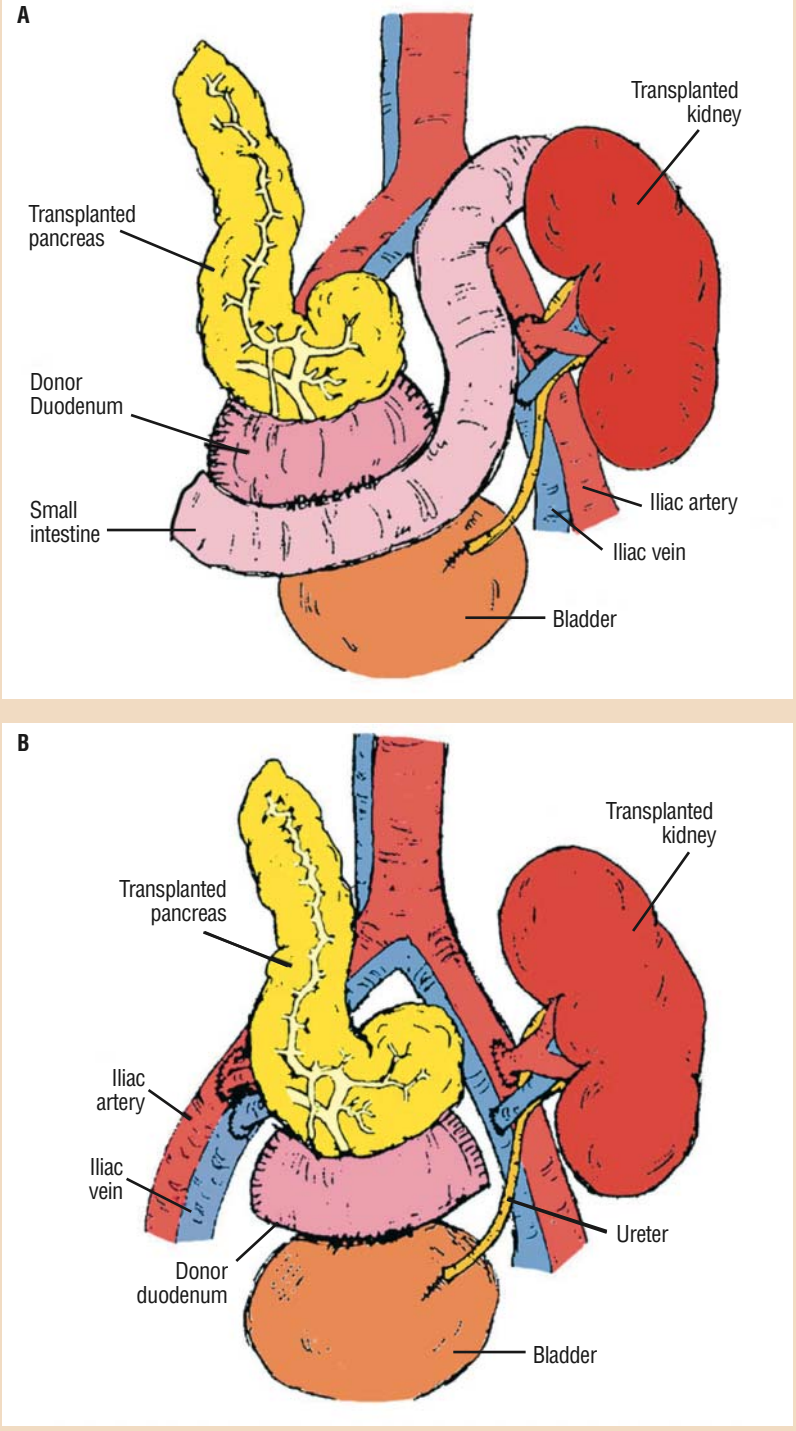

Figure 6 Surgical techniques in simultaneous pancreas-kidney transplantation. A, Enteric drainage of pancreatic exocrine secretions. B, Bladder drainage of pancreatic exocrine secretions.

Adapted from Steen,[79] copyright 1999, with permission from Lippincott Williams & Wilkins/American Association of Critical-Care Nurses.

possible, a transplant pancreatectomy should be performed at a transplant center by surgeons experienced in pancreas transplantation.

A pancreas recipient who has a bladder-drained graft must maintain a patent urinary tract at all times. A distended bladder can place stress on the anastomosis and create a leak. If the recipient cannot completely empty the bladder, a urinary catheter should be placed immediately. Of note, the urine in pancreas recipients is altered by the presence of pancreatic enzymes.

Other potential complications of pancreas transplantation include intra-abdominal infection, peritonitis, sepsis, graft pancreatitis, pancreatic fistula, pseudocyst, metabolic acidosis and dehydration, bleeding, recurrent urinary tract infection, hematuria, and dysuria. Diagnostic imaging studies are often key to identifying problems associated with the transplanted pancreas and may include ultrasound, computed tomography, magnetic resonance imaging, angiography, nuclear scintigraphy, and fluoroscopy.[81]

Conclusion

Transplant recipients often return to their community for ongoing healthcare. Regardless of whether the primary reason for admission to a community hospital or clinic is transplant related, the recipient's status as an organ transplant recipient must be taken into account when a plan of care is designed. The effectiveness of this care can be optimized by close collaboration between the community healthcare providers and the transplant center. Establishing communication early will help nurses obtain pertinent patient-specific information and history, gain knowledge of key transplant-specific assessments and monitoring, and obtain input from the transplant

center for ongoing management of the recipient's care. Community healthcare providers are key members of any recipient's transplant team and have a marked influence on recipients' overall health and outcomes after transplantation.

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CE Test Test ID C0622: Caring for Transplant Recipients in a Nontransplant Setting

Learning objectives: 1. Understand transplant-specific issues, including key interventions 2. Analyze the role of immunosuppressive therapy unique to transplant recipients 3. Explore the potential complications associated with organ transplantation

1. The process of rejection is identified by which of the following definitions?
a. The transplanted organ is recognized as nonself and the immune system protects it from other invading organisms.
b. The transplanted organ is not recognized until the recipient's immune system is triggered to recognize it and then protect it from the rest of the body's defenses.
c. The transplanted organ is recognized as nonself and the immune system builds a capsule around it to protect the body.
d. The transplanted organ is recognized as nonself and the immune system takes action to eliminate or neutralize the graft.

2. Which of the following helps activate helper T-cells?
a. Interleukin 3a (IL-3a)
b. Interleukin 1 (IL-1)
c. Interleukin 2 (IL-2)
d. Interleukin 5 (IL-5)

3. If organ rejection is diagnosed, which of the following best defines initial rejection treatment?
a. Induction combination therapy early, then single drug therapy for maintenance
b. Induction combination therapy with lower dose therapy, then maintenance therapy for 3 months
c. Higher dose of drugs and/or more potent intravenous immunosuppressants
d. Readministration of antiviral agents, and maintenance therapy at slightly higher doses

4. Which of the following best describes the opportunistic organism of infection between 1 and 6 months after transplantation?
a. Cytomegalovirus, *Pneumocysits carinii,* and *Candida*
b. *Streptococcus pneumoniae,* Trichomosis, and *Toxoplasma gondii*
c. Herpes simplex, *Staphylococcus epidermis,* and *Leptotrichia buccalis*
d. Rhinovirus, coronavirus, and *Eubacterium lentum*

5. Which of the following best describes the signs and symptoms of cytomegalovirus infection?
a. Afebrile, leukocytosis, and thrombocytosis
b. Leukopenia, mild lymphocytosis, and mild hepatitis
c. Thrombocytopenia, moderate lymphocytosis, and marked hepatitis
d. Leukocytosis, moderate lymphocytosis, and thrombopenia

6. Which of the following are transplant recipients advised to do after transplantation?
a. Avoid shellfish, wear a mask for the first 6 months, and use contact precautions
b. Avoid environments high in dust or mold, use good hand-washing practices, and report signs and symptoms of infections promptly
c. Avoid contact with pets, use well-ventilated areas, and obtain prophylactic antibiotics if exposed to persons with communicable infections
d. Avoid sun exposure because of immunosuppressive therapy and report any signs and symptoms of infection if not improved in 72 hours

7. What is the most common adverse effect of the use of cyclosporine or tacrolimus?
a. Cardiomyopathy
b. Splenomegaly
c. Hepatic failure
d. Renal dysfunction

8. Which of the following complications are noted to be a major cause of late death in transplant recipients as a result of chronic immunosuppression?
a. Cardiomegaly and renal failure
b. Diabetes with associated peripheral vascular disease
c. Malignant neoplasms
d. Hypertension and infection

9. Which of the following are identified as barriers to full rehabilitation?
a. Inability to find work, concerns about future medical needs, and depression and anxiety
b. Fear of not being accepted, fear of failure, and fear of organ rejection
c. Pain, questioning self-worth, and family social issues
d. Lack of support, pain, and organ rejection

10. Which of the following accurately describes characteristics of the transplanted (denervated) heart?
a. Resting heart rate lower than normal
b. Exaggerated heart rate response during stress or exercise
c. Blunting of maximum exercise heart rate resulting in limitations of maximal oxygen consumption
d. Lack of intrinsic heart rhythm necessitating pacemaker placement in most patients

11. Which of the following are considered common indications for liver transplantation?
a. Common biliary necrosis, hepatic encephalopathy, and acute hepatitis
b. Primary hepatitis, acute alcoholic liver disease, and chronic cholangitis
c. Penetrating trauma injury, coagulopathic disease, and autoimmune hepatitis
d. Chronic hepatitis, primary sclerosing cholangitis, and alcoholic liver disease

12. In living donor liver transplantation, the donor's and the recipient's livers usually return to near normal volumes within how long?
a. 2 to 3 months
b. 1 to 2 weeks
c. 6 to 8 weeks
d. 10 to 12 months

13. What is the most common cause of pancreatic graft loss in the first year?
a. Chronic rejection
b. Technical failure
c. Patient noncompliance
d. Insulin resistance

14. Which of the following is considered a late sign of pancreas rejection?
a. Elevated serum amylase and lipase
b. Decreased urine amylase in patients with bladder-drained pancreas
c. Increased urine amylase in patients with bladder-drained pancreas
d. Hyperglycemia

Test answers: Mark only one box for your answer to each question. You may photocopy this form.

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