



Simultaneous Pancreas and Kidney Transplantation

22

Wen Xie, Rami Kantar, Laura DiChiacchio,
and Joseph R. Scalea

Contents

History and Biology of Simultaneous Pancreas Kidney Transplantation	272
A New Day: Kelly and Lillehei	272
Stalwart Progress Towards Success	272
Indications for Simultaneous Pancreas Kidney Transplantation	272
Selection Criteria: Be Thoughtful and Smart	272
Pretransplant Cardiac Risk Assessment	273
Donor Selection Criteria	273
Contraindications to SPK	274
SPK Patient Advocacy: Insights of Organ Allocation to Support Your Patient	274
Outcomes of Simultaneous Pancreas Kidney Versus Kidney Only Transplantation	274
Diligence Yields Improvement	274
Surgical Techniques of Simultaneous Pancreas and Kidney Transplantation	275
Elegance and Challenge	275
Pancreas Procurement: An Unappreciated Nuance of the Successful SPK	275
Implantation of the Pancreas and Kidney: Standard Approaches Yield Standard Results	277
Pancreas Ahead of Kidney	277
Ipsilateral SPK Considerations	277
To Roux or Not to Roux	278
Hemostasis and a Word of Warning	278
The Kidney Transplant: Right or Left	278
Intra- vs. Extraperitoneal Graft Placement	278
Portal Versus Systemic Venous Drainage	279
SPK and Portal Drainage	279
Bladder Versus Enteric Drainage	279
Bladder Related SPK Insights	279
Jejunal Versus Duodenal Drainage	279
Pancreas Re-transplantation	280
SPK After Prior Transplantation	280
References	280

W. Xie · R. Kantar · L. DiChiacchio
Division of Transplantation, Department of Surgery, University of
Maryland School of Medicine, Baltimore, MD, USA
e-mail: wen.xie@surgery.ufl.edu

J. R. Scalea (✉)
Department of Surgery, Division of Transplantation,
Medical University of South Carolina, Charleston, SC, USA
e-mail: scalea@musc.edu

History and Biology of Simultaneous Pancreas Kidney Transplantation

A New Day: Kelly and Lillehei

In December 1966, William Kelly and Richard Lillehei transplanted the first human pancreas as part of a simultaneous pancreas kidney (SPK) transplant procedure performed in a 28-year-old patient at the University of Minnesota (see Chap. 5) [1]. While the patient unfortunately succumbed to a fatal pulmonary embolus, the procedure marked a revolution in the surgical management and treatment of patients with diabetes [1]. To this date, pancreas transplantation without concomitant kidney transplantation remains the only near-curative option for patients with complicated insulin-dependent diabetes [2]. The initial enthusiasm triggered by the technical success of the procedure was markedly challenged by a significant rate of medical and surgical complications resulting from graft rejection and loss [2]. Nevertheless, an improved understanding of transplantation biology, coupled with the innovation of novel and more potent immunosuppressant agents—most notable agents being cyclosporine in 1983 and antithymocyte globulin in 1999—facilitated the reemergence of pancreas with or without kidney transplantation as a surgical option for patients with complicated insulin-dependent diabetes mellitus [2].

Stalwart Progress Towards Success

Advances in transplantation immunology and biology were accompanied by notable technical modifications, refinements, and improvements to the procedure of pancreas transplantation [2]. The initial transplant reported by William Kelly and Richard Lillehei consisted of a duct-ligated segmental pancreatic graft, transplanted with a kidney from a deceased donor in a 28-year-old uremic patient with type 1 diabetes [1, 3]. The second transplant by the same group in 1966 included the donor's entire pancreas with duodenal segment transplanted to a 32-year-old recipient's left iliac fossa extraperitoneally (see Chap. 5) [1, 3, 4]. The first pancreatic transplant with urinary drainage was performed in November 1971 by Marvin Gliedman from New York, with drainage through the native ureter [3, 5]. Merkel subsequently performed a segmental pancreas transplant alone with end-to-side ductoenterostomy in 1973 [3, 6]. The technique of bladder drainage of pancreatic exocrine secretions was initially reported in 1983 by Hans Sollinger at the University of Wisconsin, and transiently became the drainage procedure of choice from the mid-1980s to the mid-1990s as it allowed for monitoring of urinary amylase levels as a marker for rejection of the pancreatic graft [2]. The sur-

gical approach of whole organ pancreaticoduodenal grafts with enteric drainage of pancreatic exocrine secretions, as initially described by Richard Lillehei, was reintroduced in 1984 by Thomas Starzl and remains the more popular drainage approach to this date [3, 7]. The substantial complications associated with bladder drainage of pancreatic exocrine secretions including dehydration, hematuria, acidosis, urinary tract infections, and bladder injury have in fact resulted in conversion from bladder to enteric drainage in a significant number of patients (see Chap. 29) [2]. All of these refinements coupled with the innovations in transplantation immunology and biology resulted in improved outcomes and a progressive reemergence of SPK transplantation as the surgical cure for diabetes mellitus [2].

Indications for Simultaneous Pancreas Kidney Transplantation

Selection Criteria: Be Thoughtful and Smart

Selection criteria for SPK, pancreas alone (PTA), and pancreas after kidney (PAK) transplantation vary between transplant centers. SPK transplantation is usually offered for patients with insulin-dependent diabetes and advanced chronic kidney disease (CKD) or end-stage renal disease (ESRD), with some patients receiving PAK transplantation (see Chap. 23) [8–10]. Patients who suffer from hypoglycemic unawareness and have brittle diabetes with wide fluctuations in blood glucose levels, or very poor quality of life resulting from their diabetes may be candidates for pancreas transplantation alone (PTA) (see Chap. 24) [8–10].

Recipient native renal function determines the type of transplant an individual receives. Patients who are approaching ESRD and have living kidney donors may consider undergoing a preemptive living donor kidney transplant followed by a PAK at a later time; there is well-established patient and graft survival benefits of using transplantation as the primary mode of renal replacement therapy [11–13]. The effects on survival also carry over to the kidney graft in preemptive SPK transplants [14, 15]. So, if a patient can be maintained off dialysis and has a GFR <20 mL/min, waiting for a preemptive SPK transplants may provide the same benefit without having to utilize their living donor. Some additional considerations favoring waiting for preemptive SPK transplant over living donor kidney transplant (LDKT) followed by PAK include avoidance of risks associated with a second surgery and decreasing immunological challenges. However, if patient is unable to stay free of dialysis, a PAK transplant can still provide some benefit on kidney graft survival [16].

The most basic requirements for SPK are very simple: kidney failure and insulin-dependent diabetes. SPK and

PAK transplantation are performed most commonly in patients with type 1 diabetes, as the insulin resistance and high insulin need of type 2 diabetic patients was historically thought to be too high to be overcome by the pancreas graft. However, with increasing experience, we now know SPK and PAK transplantation can be quite successfully performed in patients with non-type 1 diabetes, who have an insulin requirement and meet certain selection criteria [8–10]. Indeed, the outcomes of SPK among patients with type 2 diabetes are now excellent compared to patients with type 1 diabetes (see Chaps. 66 and 71) [8, 9]. Evidence of patient and graft survival benefits with SPK transplants in patients with type 2 diabetes vs. kidney transplant alone has been mixed; outcomes are definitively better compared to deceased donor kidney transplant (DDKT) and may be as good as LDKT [17–19]. With the added quality of life benefits of freedom from insulin and dialysis, eligible patients with type 2 diabetes and ESRD should certainly be considered for SPK transplantation. Candidates with type 2 diabetes for SPK most commonly meet the following criteria: body mass index (BMI) less than 30 kg/m², age less than 65 years, on insulin with a total daily insulin requirement <1 unit/kg/day, kidney failure, and a fasting C-peptide level less than 10 ng/mL [8–10].

The majority of SPK transplants are usually performed after patients have initiated dialysis treatment. It is important to note that while the majority of patients who undergo SPK transplantation have diabetic nephropathy, patients may have nephropathy from other coexisting conditions. Therefore, the patient's CKD or ESRD does not need to be a result of diabetic nephropathy to qualify for SPK transplantation if the patient suffers from insulin-dependent diabetes [8–10].

While the above selection criteria are important to consider, it is critical that the accepting surgeon be thoughtful about candidacy. For example, BMI alone in an otherwise well-appearing and very functional young person should not preclude transplantation. Alternatively, chronologic age alone should not preclude SPK in older patients who are very functional (see Chap. 66). Further, C-peptide has highly variable methods of testing and diagnosis and should not itself guide judgment about acceptance or denial for SPK transplantation.

Pretransplant Cardiac Risk Assessment

Coronary artery disease is common in patients with diabetes and kidney failure (see Chap. 27). Further, because SPK is a major vascular operation, patients undergoing SPK carry the highest cardiac risk according to revised cardiac risk index (RCRI) [20–22]. Moreover, cardiac stress testing can be inaccurate in the diabetic kidney failure patient [23–25]. For

these reasons, the authors stress the importance of assessing not only cardiac function and stress response, but also coronary architecture. In the authors' center, coronary calcium scoring as well as left heart catheterization is considered for SPK candidates. This protocolized approach has yielded a near-zero rate of significant postoperative myocardial ischemia [26].

Donor Selection Criteria

In many ways, pancreas donor selection has been made artificially challenging. There is substantial literature addressing donor selection criteria (see Chap. 12). The informed transplant surgeon, however, should take note that many of these publications restrict use of potential SPK transplants by suggesting that organs need to be perfect. The challenge is that, particularly with the pancreas, there remains substantial subjectivity in visual pancreas inspection and thus acceptance.

In the overwhelming majority of SPKs, the pancreas and kidney are from the same deceased donor. In these cases, the decision to accept an SPK is largely driven by pancreas, rather than kidney, quality. This is because surgeons tend to be more selective for the pancreas rather than the kidney. At the authors' center, the inclusion criteria for pancreas acceptance include: KDPI <60% and BMI <40. This has the effect of including largely younger, thinner donors.

Visual inspection of the pancreas is important. Once the donor is entertained by the surgeon, they should request a visual description of the donor organ from the recovering surgeon, if they themselves are not procuring. Common visual descriptors of pancreatic transplants include edema, fat, vasculature, and texture. Indeed, it is quite common for more selective surgeons to rule out a pancreas for transplant based on "edema" or "fat content," yet these are understudied, and what studies exist include few objective metrics. What is important for the accepting surgeon to evaluate is a hard, shrunken pancreas consistent with prior pancreatitis or a pancreas encapsulated in abdominal fat. Further, arterial supply to the pancreas can be quite important. For example, if the liver is being recovered for transplant, but there is a replaced right hepatic artery that travels through the head of the pancreas, the pancreas is commonly not usable. It is advised that novice, accepting pancreas surgeons discuss complex cases with senior partners prior to declining their acceptance.

There are several important considerations for the acceptance of the SPK. Critically, the SPK includes a pancreas and kidney. Thus, both organs must be of acceptable quality. Further, because delayed graft function of the kidney after SPK can cause surgical challenges such as pancreas swelling and significant reactive ascites from poor fluid clearance, it is

optimal to choose kidneys that work immediately. For the pancreas, avoiding donors with a history of pancreatitis is advised.

Reports of pancreas donation from donors <10 years old did show some good results, but may be technically difficult because of the size of the vessels and thus increased thrombosis risk in theory (see Chap. 29) [27]. Similarly, pancreas grafts from obese donors with BMI >30 kg/m² have worse short- and long-term outcomes due to a variety of factors including graft quality, increased risk of technical failure, and surgical complications (see Chap. 66). Pancreas from DCD donors have been demonstrated to have similar outcomes as DBD donors if selected for carefully and procured by an experienced surgeon (see Chap. 68). HLA-matching also appears to have less effect on graft outcomes with HLA-mismatching having no impact on long-term graft survival despite more episodes of rejection (see Chap. 66) [28]. So, while the transplant community awaits more defined consensus on expanded donor criteria, careful considerations of all of the donor and recipient factors, even when utilizing non-standard donors, can lead to success.

Efforts to increase the donor pool, particularly for hypersensitized recipients, have prompted interest in living-donor SPK transplants (LDSPK) (see Chaps. 37–39). In this case, a single kidney and the tail of the living donor's pancreas are procured for transplant. Although technical failure rates were high with initial attempts, the success rate is now reasonably high and with meticulous selection. Indeed, there is a true risk of development of diabetes in the donor, although this is likely minimal [29, 30]. The process of obtaining both grafts from the same donor while ensuring good recipient outcome requires experience and resources that is not widely available.

An alternative to obtaining both graft from the same living donor is a simultaneous deceased donor pancreas and living donor kidney (SPLK) transplant [31]. This allows the recipient to be free from insulin and dialysis with a single procedure without the complications of a living donor pancreas transplant. The logistics of this procedure are challenging as they require the donor to be on call, and as many as three simultaneous ORs. Nonetheless, in several studies, the long-term survival of the kidney in SPLK exceeds that of SPK from a deceased donor.

Contraindications to SPK

With regard to recipients, severe vascular disease may prohibit successful SPK transplantation. For example, concentric bilateral iliac arteriosclerosis, or the absence of suitable outflow from prior caval ligation or thrombosed iliac veins may contraindicate transplantation. Further extensive prior abdominal surgery with limited access to an enteric conduit

would also preclude an SPK transplant. Relative contraindications include multiple prior transplants as well as inflammatory bowel disease, and these should be assessed on a case by case basis. There are few absolute contraindications to SPK donors. However, these include the transmission of HIV or cancer from donor to recipient, as is true for other solid organs as well. The transplantation of a pancreas from a donor with diabetes is also contraindicated.

SPK Patient Advocacy: Insights of Organ Allocation to Support Your Patient

Organ allocation can dramatically affect the ability for a patient to receive a kidney transplant, a pancreas transplant, or both. For example, there are approximately 90,000 patients in the United States listed for a kidney transplant at the time of this writing (December 2020). In comparison, only several thousand are listed for an SPK transplant. Importantly, as of 2020, the availability of the pancreas guides the allocation of the kidney such that the kidney follows the pancreas (see Chap. 7). To this end, the wait times for SPK are far shorter than are the wait times for kidney transplantation alone. To advocate for the best patient outcome, it is often beneficial to offer SPK to eligible patients who will not survive long enough to receive a kidney transplant alone. A common scenario encountered in our hospital is a 60-year-old diabetic patient not yet on dialysis. In this case, the candidate will need to survive about 6 years on dialysis and insulin, in the hopes of receiving a deceased donor kidney alone. Alternatively, if the patient were to be offered an SPK, he may be transplanted sooner with better quality organs (e.g., lower kidney donor profile index, (KDPI)), avoiding the attendant risks of chronic renal failure. To this end, the larger SPK operation represents an “investment” that the patient makes up-front to avoid the challenges of remaining on the waitlist.

Outcomes of Simultaneous Pancreas Kidney Versus Kidney Only Transplantation

Diligence Yields Improvement

Whereas diabetes and renal failure were near uniformly fatal in the early days of pancreas transplantation, mortality following pancreas transplantation is now estimated to be 4% at 1 year and 9% at 5 years, largely driven by cardiovascular deaths [32]. The biggest improvements in SPK outcomes were realized in the mid-1990s, when tacrolimus' availability was mirrored by improvements in surgical techniques (see Chap. 66).

SPK transplant recipients have improved long-term survival as compared to deceased-donor kidney only transplant recipients at 8-year follow-up (72% vs. 55%) [32]. Data also suggest that patients undergoing SPK transplantation have comparable long-term survival when compared to patients undergoing living-donor kidney transplantation at 8-year follow-up (72%) [18]. The 1-year and 10-year graft survival rates for SPK transplant recipients are estimated to be 86% and 54%, respectively [33]. Better graft outcomes are observed in cases of younger donors, younger recipients with lower BMI as well as lower cardiovascular disease burden, with the most common reported causes of graft loss following pancreas transplantation being thrombosis (31%), chronic rejection (21%), and acute rejection (15%) [34]. Successful pancreas transplantation leads to long-term independence from insulin requirement, improved glucose metabolism, improved lipid metabolism, as well as improved endothelial function [35–39]. Successful pancreas transplantation also results in multiple improvements in the microvascular complications resulting from chronic diabetes, including the prevention and improvement of diabetic nephropathy, as well as the stabilization and improvement of diabetic neuropathy [40–48]. To date, data are insufficient or equivocal regarding the impact of pancreas transplantation on diabetic retinopathy and the chronic macrovascular complications resulting from diabetes (see Chaps. 60, 62, and 63 [40–48]).

Surgical Techniques of Simultaneous Pancreas and Kidney Transplantation

Elegance and Challenge

Surgical transplantation of a pancreas and kidney combines challenging elements of general surgery, urology, and vascular surgery. It is an exciting and educational procedure that can be safely performed by competent residents and fellows under the watchful eye of a skilled staff member. Indeed, the SPK is among the most gratifying operations we perform, as it transforms the life of the patient with diabetes and renal failure.

Since the first pancreas transplant in 1966, surgical techniques for SPK have undergone significant transformation which contributed to improved outcomes (see Chap. 29). Several considerations need to be taken into account when performing SPK transplants: Pancreatic procurement, vascular inflow and outflow, pancreatic exocrine drainage, and placement of the kidney. However, optimization of a successful SPK transplant begins with meticulous graft preparation on the “backbench”. Indeed, this operation is as elegant as it is technically challenging.

Pancreas Procurement: An Unappreciated Nuance of the Successful SPK

Pancreas procurement is covered elsewhere in this textbook (see Chaps. 14–16). The generally accepted technique for pancreas procurement results in a graft which includes a segment of the donor duodenum cradling the head of the pancreas, the whole pancreas including the portal vein, splenic artery and the superior mesenteric artery (SMA) stumps, as well as the spleen (Fig. 22.1).

The preparation of the donor pancreas for an SPK is very similar to that of a solitary pancreas transplant. Occasionally the placement of the kidney will affect the backtable preparation of the pancreas, however. For example, if the surgeon is planning an ipsilateral SPK, they may want to consider portal venous drainage of the pancreas. If so, the pancreas backbench procedure will need to include the extension of the arterial Y graft in order to have sufficient length on the artery.

Preparation of the pancreas allograft begins by inspection to assess graft quality inclusive of anatomic variants and surgical damage (see Chap. 17). Next, the spleen is removed during which time the splenic vessels are ligated, or doubly ligated in the case of the splenic artery and vein. Care should be taken during graft preparation to avoid cutting into the pancreatic parenchyma in order to prevent pancreatic ductal leaks. The approach to peripancreatic fat varies, but the authors’ practice is to excise all excess fat, to avoid significant reperfusion injury (Fig. 22.2).

The duodenal stump is then addressed. The duodenum, functionally, does not need to be very long in the recipient. It is simply the conduit of pancreatic effluent to the recipient jejunum. Excess duodenum is trimmed using GIA staplers and a short segment of duodenum around the pancreatic head should remain for anastomosis. The staple lines may



Fig. 22.1 Pancreas fully prepared for transplantation. Duodenal staple lines oversewn, nice demonstration of donor Y-graft



Fig. 22.2 Peripancreatic fat. This organ is quite usable, but the fat should be cut away prior to transplantation



Fig. 22.3 Interposition graft for external iliac artery Y-graft for recovery related arterial dissection first identified on the backtable

be reinforced with interrupted or running suture, imbricating the two stapled ends of the duodenum. The primary reason to oversee the ends is to prevent bleeding rather than leak.

Donor SMA and splenic artery stumps should be of good quality and adequate length to allow for reconstruction via a Y graft. Because most pancreas donors are young and healthy, donor vessel quality is not typically a concern. However, it is not unreasonable to decline the transplant of a pancreas for low quality, atherosclerotic, or damaged vessels. Damage during recovery may not only affect the external aspect of the vessels, but the intima as well. For example, arterial dissections may occasionally occur from too much traction on the vessels during explant. Complex arterial reconstructions can be attempted, such as backtable interposition grafts using redundant additional iliac vessel under the instruction of experienced pancreas surgeons (Fig. 22.3).

The common iliac artery with its bifurcation to the internal and external iliac arteries is the most commonly used conduit for the Y-graft. However, if the donor iliac arteries are heavily calcified or are not available, the brachiocephalic trunk, carotid arteries, or arteries from banked donor vessels can also be considered. Depending on the size

match, the internal iliac artery is typically anastomosed to the splenic artery and the external iliac artery to the SMA using fine, prolene sutures. It is important to remember that the flow of a vessel is proportional to diameter, but inversely so to the length. So, keeping your inflow short is generally preferred. The vessels should be trimmed to minimize the amount of redundancy and risk of kinking, but there should be enough length to avoid excess tension on the anastomosis.

Should the surgeon prefer portal venous drainage, the arterial conduit will need to be extended using the segment of external iliac artery discarded after it was cut back from the anastomosis to the donor graft SMA (Fig. 22.4). In this case, the Y-graft common iliac artery is anastomosed end-to-end with the divided segment of the external iliac artery.

The portal vein should be short. The portal vein is mobilized from surrounding connective tissue such that there is no twisting or kinking when the vein is distended after reperfusion. Once this is complete, the graft is tested for leaks.

The kidney allograft should be cleaned of any excess perinephric fat. Identifying and clearing fat off of the renal hilum structures to ensure adequate length without twisting



Fig. 22.4 Extension of common iliac vessel for portal venous drainage technique. Artery should be quite long to reduce tension on the SMV anastomosis

or kinking of the vessels is critical for laminar flow. Because the surgeon is performing an SPK, not just a kidney transplant alone, it is important to stop and strategize about the amount of target vessel required for dissection in the recipient ahead. For example, if the kidney has multiple arteries, it may affect the surgeon's decision to place it on the left, right, or perhaps more proximally or distally on the recipient's iliac system. Each of these elements is critical to the success of the SPK.

Implantation of the Pancreas and Kidney: Standard Approaches Yield Standard Results

Several approaches to the SPK transplant have been debated over the years (see Chap. 29). In this regard, the best approach is the standard approach with which the surgeon is most familiar. At present, the commonest approach to SPK is via a midline laparotomy from the xyphoid process to the symphysis pubis. This is the current practice of the authors.

We commonly state that the pancreas allograft is placed in the right iliac fossa, but given systemic outflow to the cava and short portal vein, the organ sits just right of midline. A Cattell-Braasch maneuver for right-sided medialization of the viscera exposes the right common iliac vessels and the IVC. The IVC is cleared of retroperitoneal tissue just above its bifurcation and the right common iliac artery is also dissected free as it crosses over the IVC to allow for adequate space for clamping of the vessels and anastomosis. The pancreas allograft is positioned in the "head up" position, with the pancreatic head cephalad. Some surgeons prefer "head down" as this allows them access to the bladder, should bladder drainage of the pancreatic effluent be required. However, in the modern era there is almost no reason to do so. In support of the "head down" approach, it may allow for a more anatomic side-to-side duodenjejunostomy for enteric drainage, a bit more distal on the recipient's small bowel. The authors use a "head up" approach.

Pancreas Ahead of Kidney

For a standard systemically drained pancreas, and a left-sided kidney, the pancreas is transplanted first (see Chap. 29). The authors believe this is wise, as it provides more time after reperfusion to allow the small vessels of the pancreas to distend and open up. In this way, the surgeon can be more comfortable that hemostasis is achieved during a singular operation.

Vascular anastomoses of the pancreas begin with donor portal vein to the IVC in an end-to-side fashion. The portal vein should be trimmed so that after completion of the anastomosis, the pancreas is anchored in place without significant movement or kinking of the vein, minimizing the risk of venous thrombosis. The portal vein can also be anastomosed to the common or external iliac vein. Arterial inflow of the pancreatic allograft comes from the right common iliac artery through its anastomosis with the Y-graft. Upon vascular reperfusion, care should be taken to achieve adequate hemostasis as there can be significant bleeding from the residual peripancreatic tissue.

Ipsilateral SPK Considerations

If the surgeon is performing an ipsilateral SPK with systemic drainage (see Chap. 29), care must be taken to ensure the pancreas is high enough up on the cava, so not to crowd the subsequently transplanted kidney. If the pancreas is sewn to the common or external iliac vein on the right, it will likely require that the kidney is transplanted on the left.

To Roux or Not to Roux

While enteric drainage was debated widely, it is now standard to perform a duodenojejunostomy versus bladder drainage. Enteric drainage can be done with or without a Roux-en-Y loop. The benefit of a Roux-en-Y loop at the index operation is that it reduces bowel contents passing by the pancreatic-enteric anastomosis, perhaps reducing the risk of a bowel leak. However, the leak rate is quite low in the modern era, and as such does not warrant (in the authors' opinion) routine additional enteric anastomoses during the primary operation.

First, a segment of jejunum approximately 40 cm from the ligament of Treitz is identified. The segment of bowel should comfortably reach the donor duodenum under no tension. Clamping proximal and distal recipient bowel is done to avoid spillage. The donor and recipient bowel segments are positioned so that the anastomosis is located on the antimesenteric edge. A two layered, hand-sewn anastomosis is done, leaving an adequate lumen for pancreatic exocrine drainage.

Hemostasis and a Word of Warning

Once the pancreas transplant is performed, and prior to the kidney transplant, it is the firm recommendation of the authors that time is spent thoughtfully assessing the transplanted pancreas for hemostasis. The kidney will take approximately 45–60 min to transplant, and during this time, the surgeon will have limited ability to see the pancreas. In this regard, it is possible to lose substantial blood if your pancreas is not hemostatic. Beyond bleeding, it is critical that the surgeon consider the pancreas lie at this point in the operation. Indeed, to perform the kidney transplant, the surgeon will need to retract the pancreas laterally, to the right. As such, it is critical that one appreciate how much laxity and stretch can be applied to the newly transplanted pancreas. It is possible to crush, thrombose, or injure the pancreas while retracting it in order to perform the kidney transplant.

The Kidney Transplant: Right or Left

The renal allograft can be placed on the left or the right. It is very common to place the kidney on the left; however, this requires that the surgeon dissects the left-sided iliac vessels. Alternatively, it is possible to place the kidney on the right (ipsilaterally), regardless of whether the pancreas is on the vena cava or the SMV. In this case, minimal additional dis-

section is required. However, placement of the kidney and pancreas together can sometimes be a challenge. When both organs are placed on the same side, they need to “nest” well, and they are likely to touch. It is important for ipsilateral transplants to envisage the geometry of the organs' end position prior to sewing them in. If they are too crowded once they are reperfused, tension or pressure on either organ may lead to leaks, bleeds, or thromboses. Presuming the surgeon elects to place the kidney distal to the pancreas, there is no risk of reducing blood flow to the transplanted pancreas while clamping the external iliac arteries for the kidney. However, clamping the distal vessels after the pancreas transplant does create temporary artificial pancreatic graft hypertension and may lead briefly to swelling or bleeding.

Regardless of sidedness, mobilization of the external iliac vessels, the venous, followed by the arterial anastomosis is completed. The ureter is implanted via a cystotomy at the dome of the urinary bladder. The use of a ureteral stent is optional. The kidney may be “retroperitonealized” by either raising a flap of the peritoneum or by tacking the descending/sigmoid mesentery to the pelvic side wall.

Intra- vs. Extraperitoneal Graft Placement

Some of the challenges of intraperitoneal placement of the allografts include difficulties in subsequent graft biopsies which can be challenging if not impossible and risks associated with entering the peritoneum such as bowel injury and secondary bowel obstruction. Extraperitoneal graft placement in SPK transplants has been described and can be done through two Gibson incisions [49] or via a lower midline incision (see Chap. 29) [50]. The peritoneum is swept medially to reveal the iliac vessels bilaterally to which the vascular anastomosis for the grafts is performed. This could require the pancreas graft to be placed head down towards the pelvis. As such, exocrine drainage would require a duodenocystostomy [51, 52] (bladder) or duodenoileostomy after the peritoneum is opened [49]. Extraperitoneally placed pancreas graft has the benefit of being easy to image using ultrasound, as there is minimal interference from overlying bowel gas, and allows for safe and easy biopsies [51]. Kidney grafts would be placed in the contralateral iliac fossa.

As described above, a generous midline incision has the benefit of excellent exposure of the vascular anastomosis sites. When placing the organs intraperitoneally, the bowels are more easily accessible for duodenal anastomosis and any peri-graft fluid can be absorbed by the peritoneum [49]. In addition, no muscles are divided which can reduce postoperative pain. Further, midline incisions have a decreased likelihood of wound infection compared to Gibson incisions

[53]. Technical errors leading to surgical complications can have significant consequences such as diffuse intra-abdominal infection, significant bleeding, and bowel obstruction requiring reexploration. On the other hand, the peritoneum is capable of managing infection, in general, better than the retroperitoneum. Regardless of location, the authors suggest avoiding infection after a pancreas transplant whenever possible.

Portal Versus Systemic Venous Drainage

In general for SPK and solitary pancreas transplantation, the consensus on the ideal venous drainage for a pancreas transplant has trended towards systemic drainage (see Chap. 29). However, there are certainly benefits of both techniques, particularly for the SPK when compared with solitary pancreas transplantation [54]. Systemic venous drainage is achieved as described earlier and can be to the vena cava or the iliac veins. On the other hand, portal venous drainage, established by Gaber et al., generally suggests outflow to the superior mesenteric vein [43].

The rationale for portal drainage is based on the venous outflow of the native pancreas. In the native pancreas, pancreatic outflow gets first pass metabolism through the liver. When the pancreatic allograft is sewn to the cava (systemic drainage), first pass metabolism does not occur. Indeed, in the native pancreas, the liver clears approximately 50% of secreted insulins. Portal drainage of the pancreas transplant is thought to avoid hyperinsulinemia and its complications such as insulin resistance and dyslipidemia due to a more physiologic insulin delivery [55, 56]. In addition, Philosophe et al. found that grafts with portal venous drainage experienced significantly lower rates of rejection and significantly higher graft survival at 36-months follow-up. However, more recent data show similar graft survival regardless of the type of venous drainage [57–59], and systemic venous drainage has again become more common (see Chap. 66) [33].

A common technique for portal drainage is anastomosis end-to-side to the recipient superior mesenteric vein below the transverse mesocolon. Then, through a small window in the mesentery of the ileum or jejunum, the end of the Y-graft is passed through and anastomosed to the right common iliac artery [60]. The pancreas is positioned with the duodenum cephalad and exocrine drainage is managed through an enteric anastomosis.

SPK and Portal Drainage

This technique has particular relevance in the SPK. For example, if the surgical team is planning to use right-sided (ipsilateral) arterial inflow for both the pancreas and the kid-

ney, using portal drainage may be favorable. Indeed, ipsilateral placement avoids crowding of the pancreas with kidney, even when both organs are on the same side. Because the portal drained pancreas resides among loops of bowel, it may be harder to see with an ultrasound or more challenging to biopsy. For patients with thick mesenteries (high BMI), prior gastric bypass, or congenital malrotation, portal drainage is generally not recommended as the geometry of the SMV's lie may be unfavorable.

Bladder Versus Enteric Drainage

After 1984, pancreas transplant enteric effluent was commonly managed using a bladder anastomosis [61–64]. While bladder drainage allowed monitoring of urinary markers of rejection and placed the allograft in an optimal position for immune monitoring via transvesical biopsy, cystitis and urethritis, bladder leaks, and metabolic acidosis from loss of bicarbonate were common (see Chap. 29) [10, 65]. Up to 40% of allografts utilizing bladder drainage thus met criteria for conversion to enteric drainage [2, 10]. In the mid-1990s when thymoglobulin induction therapy became standardized, the benefits of urinary immune monitoring no longer outweighed the risks associated with bladder drainage, and the general preference for exocrine drainage shifted to enteric drainage. Indeed, enteric drainage achieves a more physiologic exocrine profile. UNOS data from 2006 to 2016 demonstrate that over 90% of pancreas grafts during this time period utilized enteric over bladder drainage (see Chap. 66) [2, 66].

Bladder Related SPK Insights

For patients with SPK, as compared to solitary pancreas transplantation, there are specific concerns to remember with regard to bladder drainage. For example, if the kidney transplant fails but the pancreas still works, the surgeon should be prepared that the patient will still have 500–1000 mL per day of pancreatic effluent in the bladder, which is not diluted by urine. In rare circumstances, this condition may lead to bladder ulceration, pain, rupture, and emergency surgery (Fig. 22.5).

Jejunal Versus Duodenal Drainage

During an SPK, enteric drainage may be via the duodenum or jejunum. While a donor duodenum to recipient jejunal anastomosis is more common, there are some centers who prefer recipient duodenal outflow (see Chaps. 31 and 32).

The disadvantage of jejunal drainage is the lack of access for endoscopic immune monitoring of the allograft,

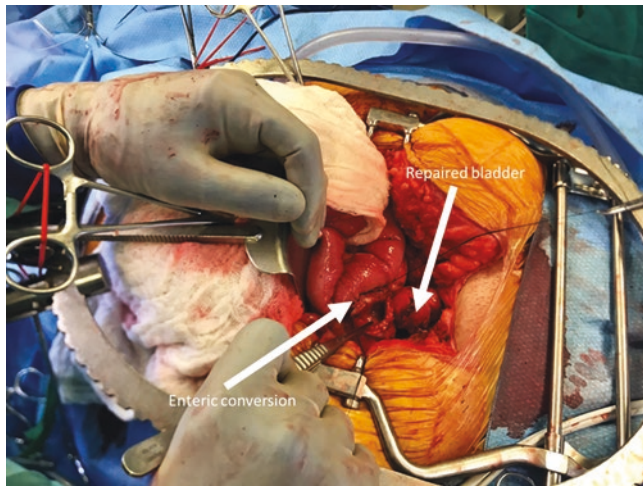


Fig. 22.5 Enteric conversion of bladder drained pancreas who presented, after kidney failure, with perforated bladder requiring emergency surgery. Pancreas was still functioning

as well as jejunal tethering. Indeed, duodenal drainage utilizing anastomosis of the donor duodenum to the second or third portions (D2/D3) of recipient duodenum is an alternative enteric drainage modality to address this disadvantage. This was first described by De Roover et al. from the group in Liege, Belgium in 2007 [67]. The technical feasibility of this approach and the theoretical advantages of duodenal drainage are described [67, 68]. While there are clear advantages regarding the technical ease of immune monitoring using endoscopic biopsy, this approach has not demonstrated improvement in clinical outcomes and there is a theoretical increased risk in complications from enteric leak [68, 69]. A recent single institution retrospective review of 241 pancreas transplants, including 125 utilizing duodenoduodenostomy and 116 with duodenojejunostomy, demonstrated comparable patient and graft survival with a median follow-up of 59 months [70]. These outcomes continue to require further study in a prospective fashion. The vast majority of initial pancreas transplants at this time continue to utilize jejunal drainage. The feasibility of duodenal drainage does, however, expand options in pancreas re-transplantation [68, 70, 71]. In the context of SPK, either approach is appropriate, and the surgeon should choose the procedure with which they are most comfortable.

Pancreas Re-transplantation

Pancreas re-transplantation has been considered of higher risk for technical failure and rejection in comparison to kidney re-transplantation; however, as the frequency of re-transplantation increases, more studies have reported favorable outcomes in carefully selected patients (see Chaps. 66 and 70) [72–76]. In

a large series described by Rudolph et al., risk of technical failure and patient death in pancreas re-transplantation is similar to primary pancreas transplantation [77]. Timing of re-transplantation does remain controversial, with conflicting evidence in small series describing early versus delayed re-transplantation [78–80]. Early allograft loss is most frequent due to allograft thrombosis, which occurs in 5–10% of pancreas transplants [2]. Technical considerations including geometric positioning of the allograft to avoid twisting or torsion of the vascular supply are paramount to the prevention of pancreas allograft thrombosis, as is the prevention of intraoperative and postoperative hypoperfusion. The technical approach to re-transplantation must consider these same factors and also address any technical contributions to failure of the primary allograft [65, 76, 77]. Use of portal venous drainage and considering alternate approaches to enteric drainage are options to improve technical outcomes in re-transplantation. While a left-sided pancreas allograft can be placed, this must be done with caution given the geometric position and angling of the vascular anastomoses required, increasing the risk of thrombosis and allograft loss. Individualization of the surgical approach and the timing of re-transplantation should be performed given the patient's comorbidities and etiology of initial allograft failure. Pancreas re-transplants following surgical complications in the first allograft have significantly improved allograft survival, while those placed following a nonsurgical failure trend toward reduced survival [81, 82]. Importantly, re-transplantation of the pancreas allograft in patients with a kidney allograft has been shown to prolong survival of the kidney allograft [73].

SPK After Prior Transplantation

Re-transplantation with SPK after SPK, or SPK after kidney transplant, carries specific risks largely related to patient selection and anatomy. For example, if your patient had a prior successful SPK that failed after many years, they will have also to be subject to immunosuppression for a very long time. In this way, understanding frailty and tissue quality is important. With regard to anatomy, it is critical ahead of SPK after kidney or SPK transplant to obtain cross-sectional imaging to ensure the surgeon has a thoughtful plan for operative execution. To this end, the authors highly recommend a senior partner to assist with preoperative and more complex pancreatic transplant cases.

References

1. Kelly WD, Lillehei RC, Merkel FK, Idezuki Y, Goetz FC. Allotransplantation of the pancreas and duodenum along with the kidney in diabetic nephropathy. *Surgery*. 1967;61(6):827–37.

2. Samoylova ML, Borle D, Ravindra KV. Pancreas transplantation: indications, techniques, and outcomes. *Surg Clin North Am*. 2019;99(1):87–101.
3. Han DJ, Sutherland DE. Pancreas transplantation. *Gut Liver*. 2010;4(4):450–65.
4. Lillehei RC, Idezuki Y, Feemster JA, et al. Transplantation of stomach, intestine, and pancreas: experimental and clinical observations. *Surgery*. 1967;62(4):721–41.
5. Gliedman ML, Gold M, Whittaker J, et al. Clinical segmental pancreatic transplantation with ureter-pancreatic duct anastomosis for exocrine drainage. *Surgery*. 1973;74(2):171–80.
6. Merkel FK, Ryan WG, Armbruster K, Seim S, Ing TS. Pancreatic transplantation for diabetes mellitus. *IMJ Ill Med J*. 1973;144(5):477–9, passim.
7. Starzl TE, Iwatsuki S, Shaw BW Jr, et al. Pancreaticoduodenal transplantation in humans. *Surg Gynecol Obstet*. 1984;159(3):265–72.
8. Gruessner AC, Laftavi MR, Pankewycz O, Gruessner RWG. Simultaneous pancreas and kidney transplantation—is it a treatment option for patients with type 2 diabetes mellitus? An analysis of the International Pancreas Transplant Registry. *Curr Diab Rep*. 2017;17(6):44.
9. Stratta RJ, Rogers J, Farney AC, et al. Pancreas transplantation in C-peptide positive patients: does “type” of diabetes really matter? *J Am Coll Surg*. 2015;220(4):716–27.
10. Al-Qaoud TM, Odorico JS, Redfield RR III. Pancreas transplantation in type 2 diabetes: expanding the criteria. *Curr Opin Organ Transplant*. 2018;23(4):454–60.
11. Kasiske BL, Snyder JJ, Matas AJ, Ellison MD, Gill JS, Kausz AT. Preemptive kidney transplantation: the advantage and the advantaged. *J Am Soc Nephrol*. 2002;13(5):1358–64.
12. Mange KC, Joffe MM, Feldman HI. Effect of the use or nonuse of long-term dialysis on the subsequent survival of renal transplants from living donors. *N Engl J Med*. 2001;344(10):726–31.
13. Papalois VE, Moss A, Gillingham K, Sutherland DE, Matas AJ, Humar A. Pre-emptive transplants for patients with renal failure an argument against waiting until dialysis. *N Engl J Med*. 2001;344(10):726–31.
14. Parajuli S, Swanson KJ, Patel R, et al. Outcomes of simultaneous pancreas and kidney transplants based on preemptive transplant compared to those who were on dialysis before transplant—a retrospective study. *Transpl Int*. 2020;33(9):1106–15.
15. Huang E, Wiseman A, Okumura S, Kuo HT, Bunnapradist S. Outcomes of preemptive kidney with or without subsequent pancreas transplant compared with preemptive simultaneous pancreas/kidney transplantation. *Transplantation*. 2011;92(10):1115–22.
16. Fridell JA, Niederhaus S, Curry M, Urban R, Fox A, Odorico J. The survival advantage of pancreas after kidney transplant. *Am J Transplant*. 2019;19(3):823–30.
17. Wiseman AC, Gralla J. Simultaneous pancreas kidney transplant versus other kidney transplant options in patients with type 2 diabetes. *Clin J Am Soc Nephrol*. 2012;7(4):656–64.
18. Alhamad T, Kunjal R, Wellen J, et al. Three-month pancreas graft function significantly influences survival following simultaneous pancreas-kidney transplantation in type 2 diabetes patients. *Am J Transplant*. 2020;20(3):788–96.
19. Rayhill SC, D’Alessandro AM, Odorico J, Knechtle S, Pirsch J, Heisey D, Kirk A, Van der Werf W, Sollinger H. Simultaneous pancreas-kidney transplantation and living related donor renal transplantation in patients with diabetes: is there a difference in survival? *Ann Surg*. 2000;231(3):417–23.
20. Ahn JH, Park JR, Min JH, et al. Risk stratification using computed tomography coronary angiography in patients undergoing intermediate-risk noncardiac surgery. *J Am Coll Cardiol*. 2013;61(6):661–8.
21. Bauer SM, Cayne NS, Veith FJ. New developments in the preoperative evaluation and perioperative management of coronary artery disease in patients undergoing vascular surgery. *J Vasc Surg*. 2010;51(1):242–51.
22. Boersma E, Kertai MD, Schouten O, et al. Perioperative cardiovascular mortality in noncardiac surgery: validation of the Lee cardiac risk index. *Am J Med*. 2005;118(10):1134–41.
23. Nerlekar N, Mulley W, Rehmani H, et al. Feasibility of exercise stress echocardiography for cardiac risk assessment in chronic kidney disease patients prior to renal transplantation. *Clin Transpl*. 2016;30(10):1209–15.
24. Ramakrishna G, Miller TD, Breen JF, Araoz PA, Hodge DO, Gibbons RJ. Relationship and prognostic value of coronary artery calcification by electron beam computed tomography to stress-induced ischemia by single photon emission computed tomography. *Am Heart J*. 2007;153(5):807–14.
25. Schermund A, Stang A, Mohlenkamp S, et al. Prognostic value of electron-beam computed tomography-derived coronary calcium scores compared with clinical parameters in patients evaluated for coronary artery disease. Prognostic value of EBCT in symptomatic patients. *Zeitschrift fur Kardiologie*. 2004;93(9):696–705.
26. St Michel D, Donnelly T, Jackson T, et al. Assessing pancreas transplant candidate cardiac disease: preoperative protocol development at a rapidly growing transplant program. *Methods Protoc*. 2019;2(4):82.
27. Fernandez LA, Turgeon NA, Odorico J, et al. Superior long-term results of simultaneous pancreas–kidney transplantation from pediatric donors. *Am J Transplant*. 2004;4:2093–101.
28. Rudolph EN, Dunn TB, Mauer D, et al. HLA-A, -B, -C, -DR, and -DQ matching in pancreas transplantation: effect on graft rejection and survival. *Am J Transplant*. 2016;16(8):2401–12.
29. Zielinski A, Nazarewski S, Bogetti D, et al. Simultaneous pancreas-kidney transplant from living related donor: a single-center experience. *Transplantation*. 2003;76(3):547–52.
30. Sutherland DE, Radosevich D, Gruessner R, Gruessner A, Kandaswamy R. Pushing the envelope: living donor pancreas transplantation. *Curr Opin Organ Transplant*. 2012;17(1):106–15.
31. Farney AC, Cho E, Schweitzer E, et al. Simultaneous cadaver pancreas living-donor kidney transplantation: a new approach for the type 1 diabetic uremic patient. *Ann Surg*. 2000;232(5):696–703.
32. Reddy KS, Stablein D, Taranto S, et al. Long-term survival following simultaneous kidney-pancreas transplantation versus kidney transplantation alone in patients with type 1 diabetes mellitus and renal failure. *Am J Kidney Dis*. 2003;41(2):464–70.
33. Gruessner AC. 2011 update on pancreas transplantation: comprehensive trend analysis of 25,000 cases followed up over the course of twenty-four years at the International Pancreas Transplant Registry (IPTR). *Rev Diabet Stud*. 2011;8(1):6–16.
34. Axelrod DA, Sung RS, Meyer KH, Wolfe RA, Kaufman DB. Systematic evaluation of pancreas allograft quality, outcomes and geographic variation in utilization. *Am J Transplant*. 2010;10(4):837–45.
35. Robertson RP, Sutherland DE, Lanz KJ. Normoglycemia and preserved insulin secretory reserve in diabetic patients 10–18 years after pancreas transplantation. *Diabetes*. 1999;48(9):1737–40.
36. Robertson RP, Sutherland DE, Kendall DM, Teuscher AU, Gruessner RW, Gruessner A. Metabolic characterization of long-term successful pancreas transplants in type I diabetes. *J Investig Med*. 1996;44(9):549–55.
37. Larsen JL, Stratta RJ, Ozaki CF, Taylor RJ, Miller SA, Duckworth WC. Lipid status after pancreas-kidney transplantation. *Diabetes Care*. 1992;15(1):35–42.
38. La Rocca E, Secchi A, Parlavecchia M, et al. Lipid metabolism after successful kidney and pancreatic transplantation. *Transplant Proc*. 1991;23(1 Pt 2):1672–3.
39. Fiorina P, La Rocca E, Venturini M, et al. Effects of kidney-pancreas transplantation on atherosclerotic risk factors and endothelial

- function in patients with uremia and type 1 diabetes. *Diabetes*. 2001;50(3):496–501.
40. Fioretto P, Steffes MW, Sutherland DE, Goetz FC, Mauer M. Reversal of lesions of diabetic nephropathy after pancreas transplantation. *N Engl J Med*. 1998;339(2):69–75.
 41. Allen RD, Al-Harbi IS, Morris JG, et al. Diabetic neuropathy after pancreas transplantation: determinants of recovery. *Transplantation*. 1997;63(6):830–8.
 42. Aridge D, Reese J, Niehoff M, et al. Effect of successful renal and segmental pancreatic transplantation on peripheral and autonomic neuropathy. *Transplant Proc*. 1991;23(1 Pt 2):1670–1.
 43. Gaber AO, Cardoso S, Pearson S, et al. Improvement in autonomic function following combined pancreas-kidney transplantation. *Transplant Proc*. 1991;23(1 Pt 2):1660–2.
 44. Navarro X, Kennedy WR, Loewenson RB, Sutherland DE. Influence of pancreas transplantation on cardiorespiratory reflexes, nerve conduction, and mortality in diabetes mellitus. *Diabetes*. 1990;39(7):802–6.
 45. Navarro X, Sutherland DE, Kennedy WR. Long-term effects of pancreatic transplantation on diabetic neuropathy. *Ann Neurol*. 1997;42(5):727–36.
 46. Secchi A, Martinenghi S, Galardi G, Comi G, Canal N, Pozza G. Effects of pancreatic transplantation on diabetic polyneuropathy. *Transplant Proc*. 1991;23(1 Pt 2):1658–9.
 47. Pearce IA, Ilango B, Sells RA, Wong D. Stabilisation of diabetic retinopathy following simultaneous pancreas and kidney transplant. *Br J Ophthalmol*. 2000;84(7):736–40.
 48. Königsrainer A, Miller K, Steurer W, et al. Does pancreas transplantation influence the course of diabetic retinopathy? *Diabetologia*. 1991;34(Suppl 1):S86–8.
 49. Hakim NS, Zarka ZA, El-Tayar A, Mustafa N, Papalois VE. A new technique for kidney-pancreas transplantation. *Transplant Proc*. 2003;35(7):2803–4.
 50. Piovesan AC, Nahas WC, Antonopoulos IM, Mazzuchi E, Cocuzza MS. Complete extraperitoneal approach for kidney implant on simultaneous pancreas and kidney transplantation by midline incision. *Transplantation*. 2006;82(11):1552–4.
 51. Adamec M, Saudek F. Our experience with pancreatic graft extraperitoneal placement. *Transplant Proc*. 1997;29(7):3078.
 52. Delin G, Lulin M, Wu HX, Juzhong G. Experience with combined pancreatic-renal transplantation using extraperitoneal placement. *Transplant Proc*. 2000;32(7):2469.
 53. Dubernard JM, Traeger J, Martin X, Faure JL, Devonec M. Pancreatic transplantation in man: surgical technique and complications. *Transplant Proc*. 1980;12(4 Suppl 2):40–3.
 54. Boggi U, Amorese G, Marchetti P. Surgical techniques for pancreas transplantation. *Curr Opin Organ Transplant*. 2010;15(1):102–11.
 55. Carpentier A, Patterson BW, Uffelman KD, et al. The effect of systemic versus portal insulin delivery in pancreas transplantation on insulin action and VLDL metabolism. *Diabetes*. 2001;50(6):1402–13.
 56. Diem P, Abid M, Redmon JB, Sutherland DE, Robertson RP. Systemic venous drainage of pancreas allografts as independent cause of hyperinsulinemia in type I diabetic recipients. *Diabetes*. 1990;39(5):534–40.
 57. Petruzzo P, Da Silva M, Feitosa LC, et al. Simultaneous pancreas-kidney transplantation: portal versus systemic venous drainage of the pancreas allografts. *Clin Transpl*. 2000;14(4 Pt 1):287–91.
 58. Petruzzo P, Lefrancois N, Berthillot C, et al. Impact of pancreatic venous drainage site on long-term patient and graft outcome in simultaneous pancreas-kidney transplantation. *Clin Transpl*. 2008;22(1):107–12.
 59. Oliver JB, Beidas AK, Bongu A, Brown L, Shapiro ME. A comparison of long-term outcomes of portal versus systemic venous drainage in pancreatic transplantation: a systematic review and meta-analysis. *Clin Transpl*. 2015;29(10):882–92.
 60. Rogers J, Farney AC, Orlando G, Farooq U, Al-Shraideh Y, Stratta RJ. Pancreas transplantation with portal venous drainage with an emphasis on technical aspects. *Clin Transpl*. 2014;28(1):16–26.
 61. Sollinger HW, Lieberman LM, Kamps D, Warner T, Cook K. Diagnosis of early pancreas allograft rejection with indium-111-oxine-labeled platelets. *Transplant Proc*. 1984;16(3):785–8.
 62. Sollinger HW, Cook K, Kamps D, Glass NR, Belzer FO. Clinical and experimental experience with pancreaticocystostomy for exocrine pancreatic drainage in pancreas transplantation. *Transplant Proc*. 1984;16(3):749–51.
 63. Nghiem DD, Schulak JA, Corry RJ. Duodenopancreatectomy for transplantation. *Arch Surg*. 1987;122(10):1201–6.
 64. Nghiem DD, Corry RJ. Technique of simultaneous renal pancreatoduodenal transplantation with urinary drainage of pancreatic secretion. *Am J Surg*. 1987;153(4):405–6.
 65. El-Hennawy H, Stratta RJ, Smith F. Exocrine drainage in vascularized pancreas transplantation in the new millennium. *World J Transplant*. 2016;6(2):255–71.
 66. Sutherland DE, Gruessner RW, Dunn DL, et al. Lessons learned from more than 1,000 pancreas transplants at a single institution. *Ann Surg*. 2001;233(4):463–501.
 67. De Roover A, Coimbra C, Detry O, et al. Pancreas graft drainage in recipient duodenum: preliminary experience. *Transplantation*. 2007;84(6):795–7.
 68. Lindahl JP, Horneland R, Nordheim E, et al. Outcomes in pancreas transplantation with exocrine drainage through a duodeno-duodenostomy versus duodenojejunostomy. *Am J Transplant*. 2018;18(1):154–62.
 69. Horneland R, Paulsen V, Lindahl JP, et al. Pancreas transplantation with enteroanastomosis to native duodenum poses technical challenges—but offers improved endoscopic access for scheduled biopsies and therapeutic interventions. *Am J Transplant*. 2015;15(1):242–50.
 70. Walter M, Jazra M, Kykalos S, et al. 125 cases of duodeno-duodenostomy in pancreas transplantation: a single-centre experience of an alternative enteric drainage. *Transpl Int*. 2014;27(8):805–15.
 71. Lindahl JP, Reinholt FP, Eide IA, et al. In patients with type 1 diabetes simultaneous pancreas and kidney transplantation preserves long-term kidney graft ultrastructure and function better than transplantation of kidney alone. *Diabetologia*. 2014;57(11):2357–65.
 72. Parajuli S, Arpali E, Astor BC, et al. Concurrent biopsies of both grafts in recipients of simultaneous pancreas and kidney demonstrate high rates of discordance for rejection as well as discordance in type of rejection—a retrospective study. *Transpl Int*. 2018;31(1):32–7.
 73. Parajuli S, Arunachalam A, Swanson KJ, et al. Pancreas retransplant after pancreas graft failure in simultaneous pancreas-kidney transplants is associated with better kidney graft survival. *Transplant Direct*. 2019;5(8):e473.
 74. Gasteiger S, Cardini B, Gobel G, et al. Outcomes of pancreas retransplantation in patients with pancreas graft failure. *Br J Surg*. 2018;105(13):1816–24.
 75. Troppmann C. Complications after pancreas transplantation. *Curr Opin Organ Transplant*. 2010;15(1):112–8.
 76. Hollinger EF, Powelson JA, Mangus RS, et al. Immediate retransplantation for pancreas allograft thrombosis. *Am J Transplant*. 2009;9(4):740–5.

77. Rudolph EN, Finger EB, Chandolias N, Kandaswamy R, Sutherland DE, Dunn TB. Outcomes of pancreas retransplantation. *Transplantation*. 2015;99(2):367–74.
78. Fridell JA, Mangus RS, Hollinger EF, et al. The case for pancreas after kidney transplantation. *Clin Transpl*. 2009;23(4):447–53.
79. Sansalone CV, Maione G, Rossetti O, et al. Pancreas retransplantation: ideal timing and early and late results. *Transplant Proc*. 2006;38(4):1153–5.
80. Boudreaux JP, Corry RJ, Dickerman R, Sutherland DE. Combined experience with immediate pancreas retransplantation. *Transplant Proc*. 1991;23(1 Pt 2):1628–9.
81. Dunn TB. Life after pancreas transplantation: reversal of diabetic lesions. *Curr Opin Organ Transplant*. 2014;19(1):73–9.
82. Buron F, Thaunat O, Demuylder-Mischler S, et al. Pancreas retransplantation: a second chance for diabetic patients? *Transplantation*. 2013;95(2):347–52.