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**Pancreas transplantation with grafts from donors deceased after circulatory death (DCD): 5 years single center experience**

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| | | |
|---|---|---|
| DBD | - | Donation after brain death |
| DCD | - | Donation after cardiac death |
| DGF | - | Delayed graft function |
| EGF | - | Early graft failure |
| ESOT | - | European Society of Organ Transplantation |
| ICU | - | Intensive care unit |
| MOD | - | Multi organ donation |
| PAK | - | Pancreas after kidney |
| PDRI | - | Pancreas donor risk index |
| SPK | - | Simultaneous pancreas kidney |
| WVS | - | Withdrawal of ventilator support |
| WIT | - | Warm ischemia time |

**Abstract**

**Introduction** Donation after circulatory death (DCD) pancreas transplantation has been shown to be an additional way to deal with donor organ shortages. The results of five year DCD pancreas transplantation are presented.

**Methods** A retrospective, single center analysis (2011 – 2015) was performed to compare the results of donation after brain death (DBD) to DCD pancreas transplantation.

**Results** During the study period, 104 pancreas transplantations (83 from DBD and 21 from DCD) were performed. Median pancreas donor risk index (PDRI) was 1.47, (DBD 1.61 vs. DCD 1.35 (p=0.144)). Without the factor DCD, PDRI from DCD donors was significantly lower (DBD 1.61 vs DCD 0.97 (p<0.001). Donor age was the only donor related risk factor associated with pancreas graft survival (HR 1.06, p=0.037). Postoperative bleeding and kidney DGF occurred more frequently in recipients from DCD (p=0.006). However, DCD pancreata had a lower incidence of thrombosis. Kidney and pancreas graft survival were equally good in both groups.

**Conclusion** Pancreas transplantation from DCD donors yields comparable results to DBD donors when PDRI of DCD are relatively low. Most DCD donors are younger donors with trauma as cause of death. These DCD pancreas grafts may be a better option to cope with increasing organ shortages than exploring the limits with older (and higher PDRI) DBD donors.

## Introduction

Pancreas transplantation from donation after brain death (DBD) has been steadily improving over the last decades with good long-term outcome in terms of patient and graft survival.(1-3) Simultaneously, the number of patients and time on the waiting list increased in the Eurotransplant area.(4, 5) Unfortunately, suitable DBD organs matching this need remained stagnant.(5) Pancreatic grafts from donation after circulatory death (DCD) have been shown to be suitable for transplantation and may provide an additional organ source.(6-11)

The first DCD pancreas transplantation in our center was performed in 2011.(8) In 2015, 52% of all donor procedures in The Netherlands were DCD, and 9/20 (45%) of pancreas transplantations at our institute were from DCD procedures.(12)

The warm ischemic period during graft procurement is generally believed to inflict a higher degree of ischemia reperfusion injury and subsequently post-reperfusion graft pancreatitis and thrombosis. This makes transplant professionals reluctant to accept DCD grafts for transplantation. In general, peri-pancreatic infections occur in approximately 35% of all pancreas transplantations, but the question is whether these are all clinically significant. (13, 14) However, with careful DCD donor selection, the detrimental effects of warm ischemia on the allograft may be limited.

This study investigates whether the use of DCD pancreas donors is feasible when careful donor selection, indicated by the Pancreas Donor Risk Index (PDRI), is performed. More specifically, short term outcome (90 days patient and graft survival and complications, specifically post reperfusion graft pancreatitis, peri-pancreatic infection, bleeding, graft thrombosis) were investigated.

## Methods

All consecutive primary pancreas transplantations performed at Leiden University Medical Center from January 2011 until December 2015 were included in this study. Follow up was collected until May 1$^{st}$ 2016. Standard SPK transplantations were performed using a midline incision. The kidney was first transplanted in the left iliac fossa, followed by the pancreas on the right anastomosed on the iliac artery and caval vein. Exocrine drainage was performed by duodeno-enterostomy. All patients

received alemtuzumab induction therapy (15 mg subcutaneous on both the day of the transplantation and first postoperative day). Standard maintenance immunosuppression consisted of tacrolimus (Prograft) (twice daily 5mg based on trough levels 8-12 ug/l until 6 weeks, from then trough levels 5-10 ug/l) or cyclosporine (trough levels 150-200 ug/l until 6 weeks, from then trough levels 100-150 ug/l) combined with mycophenolate mofetil (twice daily 500mg when tacrolimus was prescribed and twice daily 1000mg when cyclosporine was prescribed), with or without addition of steroids. Standard anticoagulant therapy after pancreas transplantation consisted of subcutaneous low molecular weight heparin (nadroparin) 2850IE twice daily. If indicated prior to transplantation, therapeutic doses were prescribed (e.g. in case of atrial fibrillation or previous deep venous thrombosis or pulmonary embolisms).

**Data collection**

Donor, recipient and transplant related risk factors are shown in Tables 1-3. Follow up data included: peak serum amylase and drain fluid amylase levels during the first 3 postoperative days, surgical and percutaneous re-interventions, patient and pancreas and kidney graft survival (including causes of graft failure). Pancreas graft failure was death censored and defined as return to exogenous insulin therapy. Minimal follow up was 90 days, to allow for analysis of early pancreas graft failure (EGF).(15) Kidney graft failure (death censored) was defined as need for renal replacement therapy or relisting on the kidney transplant waiting list.

**Analysis**

Donor warm ischemia time was calculated from the time of withdrawal of ventilatory support (WVS) until the start of organ cold perfusion. Functional warm ischemia time was considered to start when systolic blood pressure < 50 mmHg, in line with Eurotransplant and British Transplantation Society guidelines.(16, 17) Post reperfusion graft pancreatitis was defined as an increased serum amylase levels (> 250U/L) in combination with drain fluid amylase levels (>3000U/L), not requiring additional interventions.(18) Peri-pancreatic infection was defined as any peri-pancreatic infection, including abscess, infected fluid collection or hematoma, requiring surgical intervention or radiological, percutaneous drainage (Clavien-Dindo grade IIIa/b).(14, 18) All other surgical complications, such as bleeding, anastomotic

leakage, graft thrombosis, graft loss, and Clavien-Dindo grade III or higher were analysed. Other complications, such as pneumonia, post-operative wound infection and urinary tract infection were not included in the database. Delayed kidney graft function (DGF) was defined as the need for renal replacement therapy within the first week after transplantation. Patient and graft survival were estimated using the Kaplan-Meier method.

**Organ procurement**

Standard DCD organ procurement in The Netherlands starts with withdrawal of ventilatory support at the ICU. No ante-mortem interventions (heparin administration or femoral artery cannulation) are legally allowed in The Netherlands. Following cardiac arrest, a 5-minute 'no touch'-period is mandatory and when auto resuscitation does not occur within this period, the declaration of death is issued. Upon arrival in the operating room, a rapid laparotomy is carried out. The aorta is cannulated, the inferior caval vein vented and pressurized infusion of ice-cold preservation solution is started. This marks the end of the first warm ischemic period WIT. The remaining procedure, as well as DBD organ procurement, is performed as described in the ESOT MOD learning course.(19) Of note, in both DCD and DBD procedures mobilization of the pancreas was performed only after cold perfusion. Procurements were carried out by independent procurement teams, sometimes consisting of a local team, as was described elsewhere.(20) All organs were cold stored on ice in University of Wisconsin (UW) solution or histidine-tryptophan-ketoglutarate (HTK) solution.

**Results**

In the five-year study period (2011 –2015), 83 DBD (76 SPK, 7 PAK) and 21 DCD (20 SPK, 1PAK) primary pancreas transplantations were performed. All DCD donors were Maastricht category III. From the 83 DBD grafts, 3 were from another country and all other grafts, including all 21 DCD grafts, were from The Netherlands. Our local team procured 31/104 (30%). Of 21 DCD grafts, 8 (38%) were procured locally, compared to 23/83 (28%) DBD grafts (p=0.353). Four pancreatic grafts were initially bladder drained with conversion to enteric drainage in a second operation in two cases, as described before.(21) All other grafts were anastomosed to the terminal ileum. Donor, recipient and transplant demographics are shown in Table 1-3. There

was no significant difference in steroid-free immunosuppression between both groups (90% in DBD vs. 86% in DCD, p=0.073). Mean duration of follow up was 2.6 years for DBD organ recipients and 2.2 years for DCD organ recipients (p=0.2).

Median PDRI of all pancreata was 1.47 (0.68 – 2.48). No statistical significant difference in PDRI of DBD grafts compared to DCD grafts (1.61 vs. 1.35, p=0.143) was observed. However, if donor type was excluded from the PDRI calculation, the difference between DBD and DCD was significant (1.61 vs 0.97 respectively, p<0.001). DCD donors were significantly younger than DBD donors (27 (11 – 47) years vs 43 (10 – 60) years (median (range), p=0.001)). Stroke was the leading cause of death in DBD (65%), whereas DCD donors died from trauma or anoxia in 66% of the cases (p=0.001). Median donor WIT of DCD grafts was 31 (15 – 45) minutes, median functional WIT was 27 (12 – 42) minutes. (Table 3)

**Graft pancreatitis and peri-pancreatic infection**

Post-reperfusion graft pancreatitis occurred in 47 patients (45%), of which 27 resolved spontaneously without interventions. The remaining 20 recipients developed (infected) fluid collections that required intervention (either percutaneous or surgical drainage). Peri-pancreatic infection that was not preceded by post-reperfusion graft pancreatitis occurred in 10 patients (Table 4). There was no statistical difference in the incidence of graft pancreatitis between DBD and DCD graft recipients. Logistical regression analysis did not show an association between donor WIT with post reperfusion pancreatitis and peri-pancreatic infection. From 30 patients that suffered from peri-pancreatic infection, 2 lost their graft within 90 days due to thrombosis.

**Other early postoperative outcome**

Relaparotomy was required in 32/104 patients (31%). In 17 patients, a re-operation was required due to postoperative bleeding. This occurred significantly more frequent in recipient of DCD organs (11% vs. 38%, p=0.005). DBD organ recipients lost 9 grafts (7 due to thrombosis, 1 due to bleeding and 1 due to anastomotic leakage), versus none of the DCD organ recipients (p=0.198). Of all 96 SPK recipients, 17 (16%) suffered from kidney delayed graft function (DGF). Kidney DGF occurred significantly more frequently with kidneys from DCD donors (13% vs. 35%, p=0.043). There was a statistically significant association with kidney DGF and re-interventions

for bleeding (6/17), compared to recipients with immediate kidney function who required fewer reinterventions (10/80, p=0.032). Prescription of steroids as part of initial immunosuppression was not associated with thrombosis (p=0.314) One recipient with a DBD SPK died during the initial hospital stay due to systemic inflammatory response syndrome following two exploratory laparotomies for anastomotic leakages.

**Long term outcome**

Mean duration of follow up was 2.5 years (SD 1.3 years). Kaplan Meier estimated patient survival after 90 days, 1 year and 2 years was 98.8%, 97.5% and 94.5% for DBD recipients versus 100% for DCD recipients after 2 years (p=0.268) (Fig 1). Kaplan Meier estimated pancreas graft survival after 90 days, 1 year and 2 years was 89.2%, 85.5% and 85.5% for DBD organs and 100%, 100% and 93.3%, respectively, for DCD organs (p=0.428) (Fig. 2). For recipients with functioning grafts (insulin independence) at three months (n=95), data on HbA1c levels were available in 81/95 (85%). Mean HbA1c was 33 mmol/mol (SD 4mmol/mol) in the DBD group and 32 mmol/mol (SD 5 mmol/mol) in the DCD group (p=0.45). Kaplan Meier estimated kidney graft survival after 90 days, 1 year and 2 years was 98.7%, 96.0% and 94.1% for DBD kidneys and 100%, 93.8% and 93.8% for DCD kidneys (p=0.342) (Fig 3).

In univariate survival analysis, analyzing the complete cohort, donor age was a significant risk factor for pancreas graft failure (HR 1.06, 95% CI 1.00 – 1.11, p=0.037). Also, PAK was a significant risk factor for pancreas graft failure compared to SPK ($Chi^2$ 11.80, p=0.001). DCD, as stated above, and donor cause of death ($Chi^2$ 3.51, p=0.320) were not associated with pancreas graft survival. Using a previously described PDRI cut-off of 1.24 (22), high PDRI was identified as a risk factor for pancreas graft failure ($Chi^2$ 4.61, p=0.032). Numbers were too small to analyse PDRI as a continuous variable and to perform multivariate Cox-regression analysis.

**Discussion**

This study compares the outcome of DCD pancreas transplantation to DBD pancreas transplantation in a recent cohort. This study shows that pancreas transplantation

from young (mainly low PDRI) donors, either DCD or DBD, yields good results. Consequently, DCD grafts with low PDRI should certainly be considered for transplantation.

Multiple reports, as well as multiple recent meta-analyses, have shown that it is feasible to utilize DCD pancreata for vascularized pancreas transplantation.(6, 9-11, 23) Our results corroborate with these results. Even more, this study demonstrates that with careful donor selection, especially in terms of donor age, but also transplant type (SPK vs. PAK), results from DCD pancreas transplantation are comparable to those of DBD pancreas transplantation. DBD donors had other risk factors and were on average from older donors and had more frequently stroke as a cause of death. All DCD grafts were from The Netherlands, mostly from the western region (17/21), in an attempt to keep CIT as short as possible. Therefore, PDRI was not significantly different between DBD and DCD donors. But when the factor 'donor type' (DBD or DCD) was eliminated from the equation, the differences in PDRI were remarkable and showed that DCD donors with otherwise near-to-perfect characteristics were selected. These data indicate that DCD donors can be used for pancreas transplantation, especially with relatively low PDRI (in our study mean PDRI 1.35). The number of re-interventions (30.8%) is comparable to the number reported in most studies, which may be as high as 35% in pancreas transplantation.(24) In our opinion, and in accordance with the risk analysis in this study, DCD donors can be used in addition to DBD donors with more unfavorable donor characteristics.

Elaborating on individual risk factors such as age, this may be explained by the fact that young donors tend to have leaner pancreas grafts, with smooth intravascular lining. The absence of excessive peri-pancreatic fat may facilitate easier back table procedure (with construction of the Y-graft and trimming of excess fat). We hypothesize that these factors may prevent early fatty necrosis with subsequent peri-pancreatic infection and thrombosis. In terms of PDRI, a 28-year-old DCD donor bears a similar risk as a 41-year-old DBD donor. (7, 25)

The donor WIT we report is similar to that described in the large study from the UK (5), but longer than the 15 – 20 minutes that have previously been mentioned in studies from the United States. (6, 23, 26) Again, the current study shows that, even

with prolonged donor WITs, even up to 45 minutes (withdrawal of ventilatory support to cold perfusion) and, which may even be more important, prolonged periods of relative hypoperfusion (functional warm ischemia time up to 42 minutes) good results can be achieved. This has also been shown by another single center report in 2012, which reported donor WITs up to 110 minutes, albeit with very long agonal phase in at least one case. (9) Nevertheless, WIT should still be considered an important risk factor associated with postoperative complications such as kidney DGF.

An interesting observation was the higher risk of bleeding in DCD. It could be that the higher bleeding percentage in DCD recipients may be related to the higher percentage of kidney DGF in this group and subsequently anti-factor Xa accumulation or uremia associated thrombopathy. In this study, no anti-factor Xa was determined as a measure of nadroparin accumulation, nor were blood urea levels post transplantation registered. Therefore, it was not possible to proof these interactions. The clinical data show a higher percentage of bleeding in the kidney DGF group. The same mechanism may explain the difference in graft thrombosis, although this difference was not statistically significant. In those particular cases, following DCD pancreas transplantation, delayed or slower kidney graft function may have caused anti-factor Xa accumulation and subsequently, may have played a role in the prevention of pancreas graft thrombosis. We realize that the 10% risk of complete pancreas graft thrombosis in the DBD group seems rather high. However, one of cases with thrombosis did not lead to graft loss and was preserved with function with anti-coagulant treatment. Another explanation might be the relative high risk pancreas grafts that are being used in The Netherlands (medium PDRI 1.61 in this study). (27) We do not believe that procurement, back table preparation or transplantation caused the difference, since all are done the same for DBD and DCD.

The percentage of post-reperfusion graft pancreatitis in this study is 45%. In a review by Nadalin et al., post-reperfusion graft pancreatitis is thought to occur in up to 100% of pancreas transplantation and is usually self-limiting.(13) However, this difference could be explained by the definition. We arbitrarily defined post-reperfusion graft pancreatitis as elevated drain amylase levels in combination with elevated serum amylase.  Neither DCD nor the duration of donor WIT were found to be a risk factor for post-reperfusion pancreatitis or peri-pancreatic infection. In our series, of 48

patients that suffered from post reperfusion graft pancreatitis, only 20 (42%) also suffered from peri-pancreatic infection. This is 19% of our total population, which is similar to data reported in 2013.(14) Furthermore, 10/30 peri-pancreatic infections weren't preceded by any biochemical abnormalities. The clinical relevance of post-reperfusion graft pancreatitis is not entirely clear (13, 18) Interestingly, there were slightly more peri-pancreatic infections in DBD. Possibly, this is caused by the higher donor age in DBD.

Mid to long-term kidney, pancreas and patient survival were generally good. Although DCD organ recipients suffered from more postoperative bleeding and endured more kidney delayed graft function, this did not reflect in inferior long term outcome. All patients with functioning pancreas grafts at 90 days had good glycemic control and kidney function. Pancreas graft survival (insulin independence) was excellent, especially for the DCD recipients, even up to two years after transplantation. Kidney graft survival was also good in both groups.

Several limitations apply to this study. This is a retrospective database analysis with possible drawbacks that are characteristic of such studies. In addition, the data concern a single center and there was a relatively small number of patients in the study. This limited our ability to perform a multivariate risk factor analysis. Nevertheless, this is still one of largest single center reports on DCD pancreas transplantation that included all consecutive DCD pancreas transplantations in our center.(23) There is an ongoing discussion in the pancreas transplant community concerning the definition of pancreas graft failure. In this study, failure was defined as insulin independence (death censored). We appreciate that this is a subjective definition, which makes comparison difficult. However, this definition reflects the clinical situation of this patient, which is evaluated by a clinician. HbA1c levels, both at any time during follow and at start of exogenous insulin levels, facilitate comparison between different reports. We did not report HbA1c at the start of exogenous insulin therapy, since almost all had failed within 90 days (and HbA1c would thus reflect glycemic control from prior to the transplantation). Unpublished data from our center indicates that graft survival depends partially on the definition of failure. The protocol of immunosuppression changed over the course of the study. We now aim to transplant our patients in a steroid free regime, with only tacrolimus

and mycophenolate mofetil. There is no evidence that this change in protocol influenced our results with regards to graft survival.

We did not experience a high rate of complications leading to graft loss in the DCD donors. These data indicate that that DCD donors can be considered for pancreas donation with all parameters and possible risk factors taken into account. A pancreas graft from a young, lean, DCD donor after trauma, with short cold ischemia time may in fact yield better results than pancreas grafts from older DBD donors. All those parameters combined, that are reflected in a low PDRI, may be a better predictor than just DBD or DCD. In our opinion, such low PDRI DCD donors should not be precluded from vascularized pancreas donation beforehand.

**Conclusion**

Results from carefully chosen DCD donors for pancreas transplantation yield good results. Other factors than merely DCD are important in predicting outcome. We advocate that DCD pancreata, especially those with lower PDRI (younger donors and trauma as cause of death) should be considered for transplantation. This study shows that, although DCD recipients have more postoperative bleeding and kidney DGF, pancreas and kidney graft survival are at least equal to that of DBD recipients. Hopefully, these results will convince other transplant centers to utilize pancreata from DCD donors.

## References


1. Sutherland DE, Gruessner RW, Dunn DL, Matas AJ, Humar A, Kandaswamy R, et al. Lessons learned from more than 1,000 pancreas transplants at a single institution. Annals of surgery. 2001;233(4):463-501.
2. Sollinger HW, Odorico JS, Becker YT, D'Alessandro AM, Pirsch JD. One thousand simultaneous pancreas-kidney transplants at a single center with 22-year follow-up. Annals of surgery. 2009;250(4):618-30.
3. Kopp WH, Verhagen MJ, Blok JJ, Huurman VA, Fijter JWd, Koning EJd, et al. Thirty Years of Pancreas Transplantation at Leiden University Medical Center: Long-Term Follow-Up in a Large Eurotransplant Center. Transplantation. 2015;99(9):e145-51.
4. Annas GJ. The paradoxes of organ transplantation. American Journal of Public Health. 1988;78(6):621-2.
5. Samuel U. Eurotransplant Annual Report 2015. 2016.
6. Muthusamy AS, Mumford L, Hudson A, Fuggle SV, Friend PJ. Pancreas transplantation from donors after circulatory death from the United Kingdom. American journal of transplantation : official journal of the American Society of Transplantation and the American Society of Transplant Surgeons. 2012;12(8):2150-6.
7. Axelrod DA, Sung RS, Meyer KH, Wolfe RA, Kaufman DB. Systematic evaluation of pancreas allograft quality, outcomes and geographic variation in utilization. American journal of transplantation : official journal of the American Society of Transplantation and the American Society of Transplant Surgeons. 2010;10(4):837-45.
8. Blok JJ, Ringers J, Schaapherder AF, Dubbeld J, Baranski AG, de Fijter JW, et al. Report of the first five DCDD pancreas transplants within the Eurotransplant region; excellent results with prolonged first warm ischemia times. Transplant international : official journal of the European Society for Organ Transplantation. 2013;26(4):e31-3.
9. Qureshi MS, Callaghan CJ, Bradley JA, Watson CJ, Pettigrew GJ. Outcomes of simultaneous pancreas-kidney transplantation from brain-dead and controlled circulatory death donors. The British journal of surgery. 2012;99(6):831-8.
10. Siskind E, Akerman M, Maloney C, Huntoon K, Alex A, Siskind T, et al. Pancreas transplantation from donors after cardiac death. An update of the UNOS database. Pancreas. 2014;43(4).
11. van Loo ES, Krikke C, Hofker HS, Berger SP, Leuvenink HG, Pol RA. Outcome of pancreas transplantation from donation after circulatory death compared to donation after brain death. Pancreatology. 2017;17(1):13-8.
12. Eurotransplant. 1092P Data report - The Netherlands. 2016.
13. Nadalin S, Girotti P, Konigsrainer A. Risk factors for and management of graft pancreatitis. Current opinion in organ transplantation. 2013;18(1):89-96.
14. Singh RP, Vrakas G, Hayek S, Hayek S, Anam S, Aqueel M, et al. Clinically significant peripancreatic fluid collections after simultaneous pancreas-kidney transplantation. Transplantation. 2013;95(10):1263-9.
15. Finger EB, M. RD, Dunn TB, Chinnakotla S, Sutherland DE, Matas AJ, et al. A Composite Risk Model for Predicting Technical Failure in Pancreas Transplantation. American Journal of Transplantation. 2013;13(7):1840-9.
16. Transplantation From Donors After Circulatory Death British Transplantation Society Guidelines. 2015.
17. Eurotransplant Manual. Chapter 9: Donor. 2016.

18. Dindo D, Demartines N, Clavien P-A. Classification of Surgical Complications. Annals of surgery. 2004;240(2):205-13.
19. Foundation DT. E Learning MOD - Procurement Surgery [Available from: https://www.mod-surgery.org/website/.
20. de Boer JD, Kopp WH, Ooms-de Vries K, Haase-Kromwijk BJ, Krikke C, de Jonge J, et al. Abdominal organ procurement in The Netherlands - An analysis of quality and clinical impact. LID - 10.1111/tri.12906 [doi]. (1432-2277 (Electronic)).
21. van de Linde P, van der Boog PJ, Baranski AG, de Fijter JW, Ringers J, Schaapherder AF. Pancreas transplantation: advantages of both enteric and bladder drainage combined in a two-step approach. Clinical transplantation. 2006;20(2):253-7.
22. Blok JJ, Kopp WH, Verhagen MJ, Schaapherder AF, Fijter JWd, Putter H, et al. The Value of PDRI and P-PASS as Predictors of Outcome After Pancreas Transplantation in a Large European Pancreas Transplantation Center. Pancreas. 2016;45(3).
23. Shahrestani S, Webster AC, Lam VW, Yuen L, Ryan B, Pleass HC, et al. Outcomes From Pancreatic Transplantation in Donation After Cardiac Death: A Systematic Review and Meta-Analysis. Transplantation. 2016.
24. Troppmann C. Complications after pancreas transplantation. Current opinion in organ transplantation. 2010;15(1):112-8.
25. Mittal S, Sharples E, Lee F, Reddy S, Sinha S, Friend P, et al. App to reality: snapshot validation of the US Pancreas Donor Risk Index in a UK center. The Journal of surgical research. 2013;183(2):841-5.
26. Bellingham JM, Santhanakrishnan C, Neidlinger N, Wai P, Kim J, Niederhaus S, et al. Donation after cardiac death: a 29-year experience. Surgery. 2011;150(4):692-702.
27. Kopp WH, de Vries E, de Boer J, Putter H, Schareck W, Samuel U, et al. Donor risk indices in pancreas allocation in the Eurotransplant region. Transplant international : official journal of the European Society for Organ Transplantation. 2016;29(8):921-9.

**Table 1. Demographics of donors after brain death and donors after circulatory death**

| | DBD | | DCD | | |
|---|---|---|---|---|---|
| | n | % | n | % | p-value |
| Gender | | | | | **0.037** |
| Male | 27 | 32% | 12 | 57% | |
| Female | 56 | 68% | 9 | 43% | |
| Cause of death | | | | | **<0.001** |
| Stroke | 54 | 65% | 5 | 24% | |
| Trauma | 22 | 26% | 7 | 33% | |
| Anoxia | 3 | 4% | 7 | 33% | |
| Other | 4 | 5% | 2 | 10% | |

| | Median | Min - max | Median | Min - max | |
|---|---|---|---|---|---|
| Age | 43 | 10 - 60 | 27 | 11 - 47 | **0.003** |
| BMI | 23 | 17 - 29 | 22 | 18 - 29 | 0.329 |
| ICU days | 2 | 0 - 13 | 3 | 0 - 7 | **0.009** |
| Creatinine (mg/dL) | 0.64 | 0.35 - 4.65 | 0.67 | 0.43 - 1.13 | 0.523 |
| PDRI | 1.61 | 0.68 - 2.48 | 1.35 | 1.03 - 2.44 | 0.143 |
| PDRI (donortype excluded) | 1.61 | 0.68 - 2.48 | 0.97 | 0.74 - 1.75 | **<0.001** |

* Difference measured using Chi-square for categorical and Mann-Whitney for continuous variables

**Table 2. Demographics of recipients of DBD or DCD organs**

| | DBD | | DCD | | |
|---|---|---|---|---|---|
| | n | % | n | % | p-value |
| Gender | | | | | 0.526 |
| Male | 45 | 46% | 13 | 62% | |
| Female | 38 | 54% | 8 | 38% | |
| Coronary artery disease | 11 | 13% | 3 | 14% | >0.999 |
| Cerebrovascular disease | 10 | 13% | 1 | 5% | 0.455 |
| Peripheral vascular disease | 29 | 35% | 8 | 38% | 0.816 |
| Sensitized (PRA>5%) | 17 | 21% | 5 | 24% | 0.771 |
| End stage renal disease (SPK recipients) | | | | | 0.609 |
| Pre-emptive | 36 | 47% | 7 | 35% | |
| Hemodialysis | 24 | 32% | 8 | 40% | |
| Peritoneal dialysis | 16 | 21% | 5 | 25% | |
| | Median | Min - max | Median | Min - max | |
| Age | 43 | 25 - 64 | 43 | 28 - 55 | >0.999 |
| BMI | 25 | 17 - 35 | 26 | 17 - 34 | 0.625 |

* Difference measured using Chi-square for categorical and Mann-Whitney for continuous variables

**Table 3. Demographics of transplantations of DBD or DCD organs**

| | DBD | | DCD | | |
|---|---|---|---|---|---|
| | n | % | n | % | p-value |
| Transplant type | | | | | >0.999 |
| SPK | 76 | 92% | 20 | 95% | |
| PAK | 7 | 8% | 1 | 5% | |
| PTA | 0 | 0% | 0 | 0% | |
| Perfusion solution | | | | | 0.075 |
| UW | 74 | 89% | 15 | 71% | |
| HTK/Other | 9 | 11% | 6 | 29% | |
| Anticoagulant therapy | | | | | 0.180 |
| Nadroparin 2850IE | 8 | 9% | 0 | 0% | |
| Nadroparin 5700IE | 71 | 86% | 21 | 100% | |
| Nadroparin 11400IE** | 4 | 5% | 0 | 0% | |
| Immunosuppression | | | | | 0.073 |
| Cyclosporin + Mycophenolate | 1 | 1% | 0 | 0% | |
| Cyclosporin + Mycophenolate + Prednisone | 2 | 2% | 3 | 14% | |
| Tacrolimus + Mycophenolate | 74 | 89% | 18 | 86% | |
| Tacrolimus + Mycophenolate + Prednisone | 6 | 7% | 0 | 0% | |

| | Median | Min - max | Median | Min - max | |
|---|---|---|---|---|---|
| Pancreas CIT (hr) | 10 | 4 - 14 | 11 | 7 - 15 | 0.143 |
| Pancreas donor functional WIT (min) *** | | | 27 | 12 – 42 | n/a |
| Pancreas donor WIT (min)**** | | | 31 | 15 - 45 | n/a |
| Pancreas recipient WIT (min) | 26 | 14 - 64 | 25 | 10 - 41 | 0.613 |

* Difference measured using Chi-square for categorical and Mann-Whitney for continuous variables

** These patients were on anticoagulation prior to transplantation

*** Withdrawal of ventilatory support - systolic blood pressure < 50 mmHg

**** Withdrawal of ventilatory support - organ cold perfusion

**Table 4. Early (<90 days) postoperative complications after DBD and DCD transplantation**

| | DBD | | DCD | | |
|---|---|---|---|---|---|
| | n | % | n | % | p-value |
| Thrombosis | | | | | 0.282 |
| Complete | 8 | 10% | 0 | 0% | |
| Partial | 24 | 29% | 7 | 33% | |
| Bleeding | 9 | 11% | 8 | 38% | **0.006** |
| Post reperfusion graft pancreatitis | 40 | 48% | 7 | 33% | 0.222 |
| Peri-pancreatic infection | 25 | 30% | 5 | 24% | 0.568 |
| Pancreas graft loss | 9 | 11% | 0 | 0% | 0.198 |
| Kidney delayed graft function | 10 | 13% | 7 | 35% | **0.041** |
| Patient death | 1 | 1% | 0 | 0% | >0.999 |

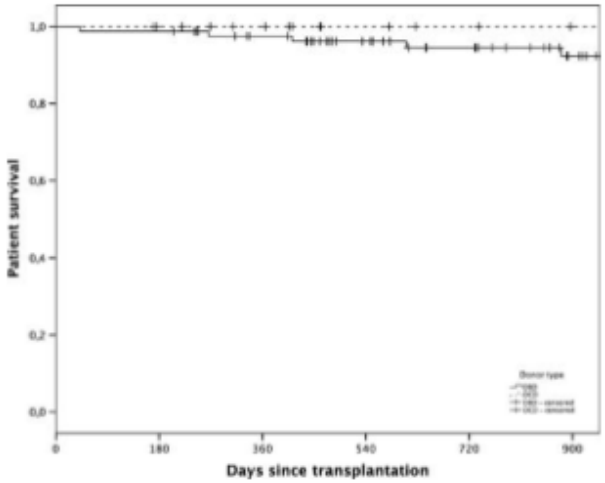


**FIGURE 1.** Kaplan-Meier estimated patient survival at 90 days, 1 year, and 2 years for DBD pancreas recipients versus DCD pancreas recipients.

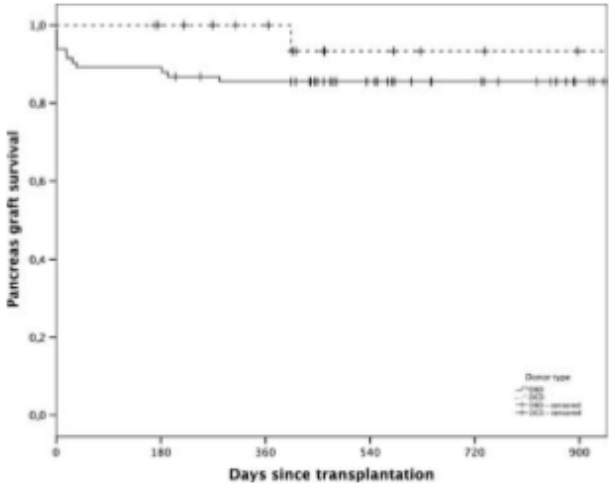


**FIGURE 2.** Kaplan-Meier estimated pancreas graft survival at 90 days, 1 year, and 2 years for DBD pancreas grafts versus DCD pancreas grafts.

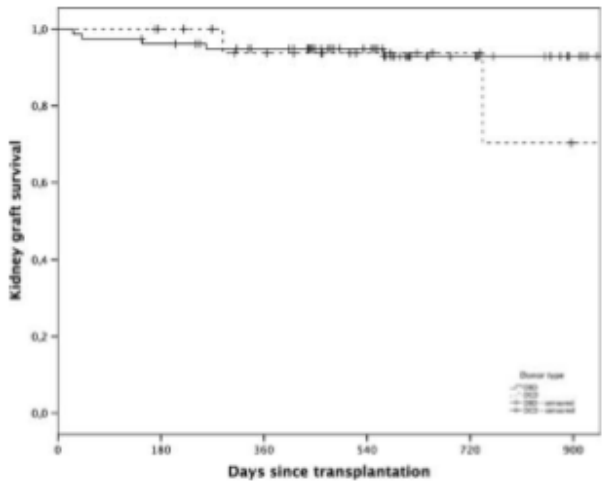


**FIGURE 3.** Kaplan-Meier estimated kidney graft survival at 90 days, 1 year and 2 years for DBD kidney grafts versus DCD kidney grafts.