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Donations After Circulatory Death in Liver Transplant

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Abstract

The supply of liver grafts for treatment of end-stage liver disease continues to fall short of ongoing demands. Currently, most liver transplants originate from donations after brain death. Enhanced utilization of the present resources is prudent to address the needs of the population. Donation after circulatory or cardiac death is a mechanism whereby the availability of organs can be expanded. Donations after circulatory death pose unique challenges given their exposure to warm ischemia. Technical principles of donations after circulatory death procurement and pertinent studies investigating patient outcomes, graft outcomes, and complications are highlighted in this review. We also review associated risk factors to suggest potential avenues to achieve improved outcomes and reduced complications. Future considerations and alternative techniques of organ preservation are discussed, which may suggest novel strategies to enhance preservation and donor expansion through the use of marginal donors. Ultimately, without effective measures to bolster organ supply, donations after circulatory death should remain a consideration; however, an understanding of inherent risks and limitations is necessary.

Keywords

Donor selection; Tissue and organ procurement

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Introduction

Liver transplant remains the only cure for end-stage liver disease.[1] Historically, organs were obtained for transplant after cardiac arrest of donors until 1968, when the Committee of Harvard Medical School promoted the acceptance of brain death.[2] With this recognition, organ donation after brain death (DBD) became widespread among transplant centers, with DBD accounting for 92.1% of transplants.[3] Unfortunately, the present volume of transplants is insufficient to meet the demands. In the United States, 15027 individuals were registered on the liver transplant wait list in 2013, while only 5921 hepatic grafts were transplanted.[4] Given this discrepancy, the use of organs after circulatory arrest (cardiac death) of donors (DCD) that fail to meet the full criteria of brain death or deemed neurologically intact have grown in acceptance.[5] Donations after circulatory death account for 12.1% of total transplants, which is up from 1.9% in 2000.[3] However, there is potentially room to further expand its use, given that 10.5% of potentially suitable DCDs are discarded.[3]

Donations after circulatory death are classified into 5 categories depending on the circumstances of death (Table 1).[6,7] Donations after circulatory death differ from DBDs based on the length of warm ischemic time (WIT), which represents the time between cardiac death and organ cooling during procurement. Unlike DCDs, DBDs do not usually have WIT before procurement. Notably, controlled DCDs have WIT that can be estimated, resulting in reduced ischemic insult relative to uncontrolled donors, where the WIT is often inexact and extended, making assessment of injury difficult.

The outcome of DCD transplants is organ dependent. Renal transplants performed from uncontrolled DCDs have equivalent outcomes compared with controlled DCDs.[8] Furthermore, renal grafts with uncontrolled DCDs were found to have comparable results to DBDs, suggesting renal tolerance of ischemia.[9] Conversely, liver grafts are more susceptible to ischemia, which can lead to unfavorable outcomes.[10] As a result, category 3 donors are most often used for liver transplants to ensure a controlled environment where WIT can be closely monitored. In addition, the percentage of unused livers from DCDs has risen consistently for several years.[11] Given the underuse of DCDs for transplants and the continued demand for obtaining suitable organs, we highlight the current procedures and pertinent literature related to DCD outcomes, complications, risk factors, and future considerations, including novel methods of hepatic organ preservation.

Procedure of donations after circulatory death

Rapid recovery techniques are used for DCD procurement, and these may also involve premortem cannulation.[12,13] Commencement of the procedure involves withdrawal of ventilator and perfusion support, during which the patient is monitored until the end of cardiorespiratory function. Administration of heparin at the time of withdrawal has met resistance, given the potential for hastened death; however, its use is still considered standard practice.[14] On declaration of death, 5 minutes should elapse to ensure the absence of autoresuscitation.[15]

Donations after circulatory death for liver transplant can be separated into 3 main phases based on organ temperature. The first phase represents WIT after withdrawal of

cardiopulmonary support and lasts until cold flush of the organ.[15] This phase includes organ procurement. To minimize WIT, premortem cannulation can be performed through access of the femoral artery and vein before withdrawal of support.[12] If premortem cannulation is not performed, aortic and portal system cannulation is completed after declaration of death. After explantation, the liver is flushed with cold preservation solution, thus ending WIT and commencement of the second phase, cold ischemic time (CIT). Cold ischemic time lasts until the organ is successfully transplanted in the recipient, which is largely governed by transportation time of organs. Last, another WIT period ensues after reperfusion of the organ.

Outcomes

Several database analyses have shown inferior patient and graft survival for DCD liver transplants.[16–19] An analysis by Abt and associates revealed DCD liver transplants had 1- and 3-year graft survival rates of 70.2% and 63.3% compared with DBD grafts, which were 80.4% and 72.1% ($P = .003$ and $P = .012$).[16] Similarly Mateo and associates found reduced graft survival for DCD compared with DBD (1- and 3-year graft survival rates for DCD of 71% and 60% vs DBD having 80% and 72%; $P < .001$).[17] The disparity between DCD and DBD liver grafts is further supported by 2 Scientific Registry of Transplant Recipients analyses. The first study by Merion and associates showed an 85% higher risk of graft failure for DCD over DBD, with 3-month, 1-year, and 2-year survival rates of 83.0%, 70.1%, and 60.5% versus 89.2%, 83.0%, and 75.0% ($P < .001$).[18] Selck and associates found similar trends, with 6-month, 1-year, 2-year, and 3-year graft survivals of 79%, 72%, 63%, and 57% versus 88%, 84%, 78%, and 74% for DCD and DBD ($P < .001$).[19] The higher graft failure associated with DCD was suggested to occur within the first 180 days.[19]

Akin to graft survival, several single-center studies (Table 2) have suggested reduced patient survival associated with DCD liver transplants relative to DBD.[20–22] Foley and associates reported reduced patient survival at 1 and 3 years (80% and 68% with DCD vs 91% and 84% with DBD; $P = .002$) and diminished graft survival at 1 and 3 years (67% and 56% with DCD vs 86% and 80% with DBD; $P = .0001$) associated with DCD donors.[20] In contrast, Skaro and associates found no significant difference in patient survival, although lower 1- and 3-year graft survival rates in DCD relative to DBD were found (61.3% and 52.6% with DCD vs 85.2% and 74.2% with DBD; $P = .005$).[21] Furthermore, there was a 3.2-fold higher risk of retransplant among DCD livers compared with DBD.[21] Similarly, de Vera and associates did not find a significant difference in patient survival; however, 1-, 5-, and 10-year graft survival rates were significantly worse for DCD compared with DBD transplants (69%, 56%, and 57% vs 82%, 73%, and 63%; $P < .0001$).[22] Furthermore, other evidence suggests a potential link between DCD liver transplants and acute kidney injury in patients with no history of renal damage.[23]

In contrast, a review of the United Network for Organ Sharing database found the use of DCD livers in pediatric patients to be comparable with DBD, with 1- and 3-year graft survival rates of 89.2% and 79.3% for DCD versus 75.6% and 65.8% for DBD ($P = .3$).[24] Additionally, many single-center studies (Table 2) have suggested that both graft function and patient survival are comparable between DCD and DBD liver transplants.[25–29] Meurisse

and associates reported DCD liver transplants had 1, 3, and 5-year graft survival rates of 90%, 82%, and 82% and patient survival rates of 93%, 85%, and 85%, which were similar to DBD liver transplant survival rates of 85%, 74%, and 68% ($P = .524$) and patient survival rates of 88%, 78%, and 72% ($P = .348$).[25] Grewal and associates were able to achieve comparable long-term outcomes between DCD and DBD liver transplants with 1-, 3-, and 5-year patient and graft survival rates for DCD transplants (91.5%, 88.1%, and 88.1% and 79.3%, 74.5%, and 71.0%) vs DBD transplants (87.3%, 81.1%, and 77.2% and 81.6%, 74.7%, and 69.1%).[27] These findings are supported by Fujita and associates and Vanatta and associates.[28,29] Other studies suggest that the patient and graft survival rates after 1 year with DCD liver transplants are 100%; however, these studies contain small sample sizes and no comparisons to DBD.[30,31]

Risk factors and complications

Donor and recipient risk factors leading to complications after DCD liver transplants have been identified (Table 3). Donor age > 50 years, WIT > 30 minutes, and CIT > 8 hours are documented risk factors for graft failure.[16,19,20,32,33] Donor weight > 200 kg has been reported as well.[34,35] Notably, Lee and associates found a significantly increased risk of graft loss when WIT was > 15 minutes.[33] Importantly, true warm ischemia (time from significant hypotension to cold flush) has been used interchangeably with total WIT (period from extubation to cold flush), which may lead to inaccuracies in the interpretation of studies.[14] A recommendation for the division of WIT into 2 distinct phases has been proposed, with phase 1 including the period from withdrawal of support to cardiopulmonary cessation and phase 2 including the time from loss of circulation to cold flush.[14] Halazun and associates[36] also stressed the need for distinction between warm ischemia during organ procurement and warm ischemia after reperfusion. Furthermore, Taner and associates suggested a link between the duration of asystole-to-cross clamping and the development of ischemic cholangiopathy ($P < .05$).[37] A CIT of < 8 hours is preferred for DCD liver transplants, as Chan and associates[35] and Foley and associates[32] have revealed an increased risk of ischemic cholangiopathy with prolonged CIT ($P = .05$). To minimize CIT, some institutions start explantation of the recipient once the donor is deemed suitable.[32] Detry and associates noted that a short WIT of < 30 minutes and CIT of < 5 hours resulted in 100% patient and graft survival at 1 year.[31]

Several recipient characteristics can increase the risk of poor DCD outcomes, including high Model for End-stage Liver Disease scores, which have been associated with graft failure and biliary complications in some studies, whereas other studies refute this claim.[26,38,39] In addition, hepatitis C virus-positive recipients may be at increased risk for hepatitis C virus recurrence.[40,41] Recently, Croome and associates found that transplant recipients with hepatocellular carcinoma may have inferior survival rates after DCD transplant ($P = .049$).[42] Black recipient race and recipient body mass index > 30 kg/m^2 have also been indicated as adverse outcomes with DCDs.[22,34,37,43]

There are various complications associated with DCD liver transplants (Table 4). Primary nonfunction, delayed graft function, and hepatic artery thrombosis have been associated with DCD livers, and these complications may contribute to higher rates of retransplant. Biliary

complications have also been suggested to occur with increased frequency in DCDs.[20,25,26,44] A single-center study showed an overall biliary complication rate of 50% for DCDs versus 28.3% for DBDs ($P = .012$) with biliary stricture rates of 46.7% and 26.5% ($P = .018$).[25] Croome and associates reported that 25% of DCD liver transplants developed biliary complications compared with 13% of DBD liver transplants ($P = .062$).[26] These trends are supported by Foley and associates and Chan and associates.[32,35] Therefore, routine use of T-tube insertion in DCD transplants has been suggested for biliary drainage and early detection of leaks.[15] However, to date, there are inconclusive data to support routine use of T-tubes in DCD transplant procedures.[45]

Of the possible biliary complications, ischemic cholangiopathy, also referred to as ischemic-type biliary strictures, is more prevalent in DCD transplants than in DBDs.[32] Ischemic-type biliary stricture is regarded as the most severe biliary complication, as it can lead to intrahepatic bile duct strictures, hepatic abscesses, and hepatic necrosis, resulting in retransplant.[34,37] Extended CIT and donor age correlate with an increased incidence of ischemic-type biliary strictures, whereas ischemia reperfusion injury can exacerbate these problems.[32,35,46]

Transplant procedures using DCD organs result in greater overall financial costs. The 1-year cost after liver transplant has been calculated at 82 730 Euros for DBD versus 101 805 Euros for DCD ($P = .001$).[47] Not surprisingly, this analysis revealed increased complications associated with DCD grafts. It is important to note that patient and graft survival rates were not different between DCD and DBD grafts in the aforementioned study. Similarly, Jay and associates[48] found increased 1-year posttransplant costs for DCD recipients (125% of DBD cost). The cost remained higher in DCD (120% of DBD) transplants despite the exclusion of retransplants, which is a major risk for DCD livers but occurs infrequently.[48]

In light of increased complications associated with DCD liver transplants, the optimal recipient of these grafts remains unclear. High-risk recipients are subjected to unnecessary risk when exposed to a DCD liver transplant, and low-risk recipients may be able to physically withstand possible complications resulting from DCD liver transplants.[19] Vanatta and associates achieved comparable graft and patient survival rates between DCD and DBD liver transplants in recipients having low Model for End-stage Liver Disease scores ($P = .444$ and $P = .295$).[29] Donors were young with total WIT < 30 minutes and CIT of approximately 5 hours.[29] Conversely, the increased risk of DCD transplants may pose a favorable risk-to-benefit ratio only for high-risk recipients.[49] Although the exact indication for DCD transplant remains unclear, attempts to minimize complications through focus on risk factor mitigation is a wise strategy.

Future considerations

Several methods to minimize the detrimental outcomes of DCD transplants have been proposed. Hypothermic machine perfusion has resulted in superior graft survival and reduced delayed graft function relative to static cold storage (SCS) in DCD renal grafts.[50] For hepatic grafts, SCS involves vascular flush with cold preservation solution, followed by placement into cold slush to minimize metabolism.[51,52] Hypothermic machine perfusion consists of constant perfusion of preservation solution administered via the hepatic artery

and portal vein. Guarrera and associates have successfully used hypothermic machine perfusion in clinical trials with extended criteria donors, including DCDs, and have shown reduced biliary complications compared with SCS.[53] Dutkowski and associates recently demonstrated comparable clinical results between unperfused DBD livers and DCD livers after hypothermic machine perfusion.[54] Similarly, Dries and associates used a DCD porcine liver model to show reduced arteriolonecrosis of the peribiliary vascular plexus with hypothermic machine perfusion compared with SCS ($P = .024$).[55] Experimental rat models have revealed that hypothermic oxygenated perfusion can protect from biliary injury after a period of warm ischemia.[56] In addition, Lee and associates[57] have demonstrated that rat livers undergoing hypot hermic machine perfusion for 5 hours can tolerate warm ischemia for 30 minutes, unlike models preserved with cold preservation solution alone.

Normothermic machine perfusion involves administration of warmed (37°C) preservation solution and provides an optimal metabolic environment, allowing for preservation, monitoring, and resuscitation of marginal organs.[58,59] In a porcine model, after 1 hour of warm ischemia and 24 hours of cold storage, viability of livers could be achieved with normothermic machine perfusion, whereas livers preserved with SCS alone were not viable ($P < .05$).[58] These results are supported by Xu and associates[59] and Schon and associates.[60] Normothermic machine perfusion was also shown to reduce liver and bile duct injury in a porcine DCD liver model.[61] Recovery of DCD livers and successful transplant after an extended WIT has also been recorded in rat models.[62] Rat livers subjected to 45 minutes of warm ischemia and 2 hours of cold storage were successfully recovered using normothermic machine perfusion and transplanted.[63] Rat survival was 100% after 4 weeks, whereas rats receiving SCS livers died within 12 hours.[63]

Subnormothermic machine perfusion is an intermediate strategy, which entails graft cooling to 21°C, thereby permitting ongoing assessment of graft function and viability while minimizing metabolic demands.[64] Dries and associates[65] have demonstrated the feasibility of using subnormothermic machine perfusion to assess and preserve livers. A porcine DCD liver model showed reduced bile duct injury with use of subnormothermic machine perfusion.[66] Likewise, several other groups have suggested that subnormothermic machine perfusion can recover and allow transplant of ischemic livers with promising results.[64,67] Notably, successful preservation of rat livers for up to 4 days can be achieved using supercooling and subsequent subnormothermic machine perfusion.[68] This method has the potential to prolong transport of DCD livers by 3- to 4-fold.[68]

Conclusions

Increasing the supply of hepatic grafts to meet the demands of those with end-stage liver disease remains a formidable challenge. Continued expansion of DCD graft utilization represents a potential avenue to help satisfy organ demands. Outcomes of DCD liver transplants have improved over time, with evidence to support noninferiority of DCD to DBD with respect to patient and graft outcomes. These outcomes are attributed, in part, to meticulous adherence to strict protocols and sound surgical techniques. Donations after circulatory death are subject to several complications, including biliary complications, primary nonfunction, and delayed graft function. As such, mitigating donor and recipient

risk factors such as advanced donor age, WIT, and CIT are of prime importance. Ongoing investigations into improved preservation techniques and organ repair should continue and may ultimately negate the risk of using DCD livers. In the interim, the use of DCD livers remains a viable mechanism to expand the current donor pool; however, its use will need to be tempered against inherent shortcomings.

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References

1. Berlakovich GA. Clinical outcome of orthotopic liver transplantation. Int J Artif Organs. 2002; 25(10):935–938. [PubMed: 12456034]
2. A definition of irreversible coma. Report of the Ad Hoc Committee of the Harvard Medical School to Examine the Definition of Brain Death. JAMA. 1968; 205(6):337–340. DOI: 10.1001/jama.1968.03140320031009 [PubMed: 5694976]
3. Saidi RF, Markmann JF, Jabbour N, et al. The faltering solid organ donor pool in the United States (2001–2010). World J Surg. 2012; 36(12):2909–2913. DOI: 10.1007/s00268-012-1748-0 [PubMed: 22933050]
4. Kim WR, Lake JR, Smith JM, et al. OPTN/SRTR 2013 Annual Data Report: liver. Am J Transplant. 2015; 15(Suppl 2):1–28. DOI: 10.1111/ajt.13197
5. Rady MY, Verheijde JL, McGregor J. Organ donation after circulatory death: the forgotten donor? Crit Care. 2006; 10(5):166.doi: 10.1186/cc5038 [PubMed: 17020597]
6. Kootstra G, Daemen JH, Oomen AP. Categories of non-heart-beating donors. Transplant Proc. 1995; 27(5):2893–2894. [PubMed: 7482956]
7. Sanchez-Fructuoso AI, Prats D, Torrente J, et al. Renal transplantation from non-heart beating donors: a promising alternative to enlarge the donor pool. J Am Soc Nephrol. 2000; 11(2):350–358. [PubMed: 10665943]
8. Hoogland ER, Snoeijs MG, Winkens B, Christaans MH, Van Heurn LW. Kidney transplantation from donors after cardiac death: Uncontrolled versus controlled donation. Am J Transplant. 2011; 11(7):1427–1434. DOI: 10.1111/j.1600-6143.2011.03562.x [PubMed: 21668628]
9. Reznik ON, Skvortsov AE, Reznik AO, et al. Uncontrolled donors with controlled reperfusion after sixty minutes of asystole: a novel reliable resource for kidney transplantation. PLoS One. 2013; 8(5):e64209.doi: 10.1371/journal.pone.0064209 [PubMed: 23737973]
10. Cursio R, Gugenheim J. Ischemia-reperfusion injury and ischemic-type biliary lesions following liver transplantation. J Transplant. 2012; 2012:164329.doi: 10.1155/2012/164329 [PubMed: 22530107]
11. Orman ES, Barritt AS, Wheeler SB, Hayashi PH. Declining liver utilization for transplantation in the United States and the impact of donation after cardiac death. Liver Transplant. 2013; 19(1):59–68. DOI: 10.1002/lt.23547
12. Reich, D. Non-heart-beating donor organ procurement. In: Humar, A.Matas, A., Payne, W., editors. Atlas of Organ Transplantation. London, UK: Springer; 2006.
13. D'Alessandro AM, Hoffmann RM, Knechtle SJ, et al. Successful extrarenal transplantation from non-heart-beating donors. Transplantation. 1995; 59(7):977–982. DOI: 10.1097/00007890-199504150-00009 [PubMed: 7709458]
14. Bernat JL, D'Alessandro AM, Port FK, et al. Report of a National Conference on Donation after cardiac death. Am J Transplant. 2006; 6(2):281–291. DOI: 10.1111/j.1600-6143.2005.01194.x [PubMed: 16426312]
15. Reich DJ, Mulligan DC, Abt PL, et al. ASTS recommended practice guidelines for controlled donation after cardiac death organ procurement and transplantation. Am J Transplant. 2009; 9(9): 2004–2011. DOI: 10.1111/j.1600-6143.2009.02739.x [PubMed: 19624569]

16. Abt PL, Desai NM, Crawford MD, et al. Survival following liver transplantation from non-heart-beating donors. Ann Surg. 2004; 239(1):87–92. DOI: 10.1097/01.sla.0000103063.82181.2c [PubMed: 14685105]

17. Mateo R, Cho Y, Singh G, et al. Risk factors for graft survival after liver transplantation from donation after cardiac death donors: An analysis of OPTN/UNOS data. Am J Transplant. 2006; 6(4):791–796. DOI: 10.1111/j.1600-6143.2006.01243.x [PubMed: 16539637]

18. Merion RM, Pelletier SJ, Goodrich N, Englesbe MJ, Delmonico FL. Donation after cardiac death as a strategy to increase deceased donor liver availability. Ann Surg. 2006; 244(4):555–562. DOI: 10.1097/01.sla.0000239006.33633.39 [PubMed: 16998364]

19. Selck FW, Grossman EB, Ratner LE, Renz JF. Utilization, outcomes, and retransplantation of liver allografts from donation after cardiac death: implications for further expansion of the deceased-donor pool. Ann Surg. 2008; 248(4):599–607. DOI: 10.1097/SLA.0b013e31818a080e [PubMed: 18936573]

20. Foley DP, Fernandez LA, Leverson G, et al. Donation after cardiac death: the University of Wisconsin experience with liver transplantation. Ann Surg. 2005; 242(5):724–731. DOI: 10.1097/01.sla.0000186178.07110.92 [PubMed: 16244547]

21. Skaro AI, Jay CL, Baker TB, et al. The impact of ischemic cholangiopathy in liver transplantation using donors after cardiac death: The untold story. Surgery. 2009; 146(4):543–553. DOI: 10.1016/j.surg.2009.06.052 [PubMed: 19789011]

22. De Vera ME, Lopez-Solis R, Dvorchik I, et al. Liver transplantation using donation after cardiac death donors: long-term follow-up from a single center. Am J Transplant. 2009; 9(4):773–781. DOI: 10.1111/j.1600-6143.2009.02560.x [PubMed: 19344466]

23. Leithead, Ja, Tariciotti, L., Gunson, B., et al. Donation after cardiac death liver transplant recipients have an increased frequency of acute kidney injury. Am J Transplant. 2012; 12(4):965–975. DOI: 10.1111/j.1600-6143.2011.03894.x [PubMed: 22226302]

24. Abt P, Kashyap R, Orloff M, et al. Pediatric liver and kidney transplantation with allografts from DCD donors: A review of UNOS data. Transplantation. 2006; 82(12):1708–1711. DOI: 10.1097/01.tp.0000254762.95625.d0 [PubMed: 17198264]

25. Meurisse N, Vanden Bussche S, Jochmans I, et al. Outcomes of liver transplantations using donations after circulatory death: A single-center experience. Transplant Proc. 2012; 44(9):2868–2873. DOI: 10.1016/j.transproceed.2012.09.077 [PubMed: 23146544]

26. Croome KP, McAlister V, Adams P, Marotta P, Wall W, Hernandez-Alejandro R. Endoscopic management of biliary complications following liver transplantation after donation from cardiac death donors. Can J Gastroenterol. 2012; 26(9):607–610. [PubMed: 22993731]

27. Grewal HP, Willingham DL, Nguyen J, et al. Liver transplantation using controlled donation after cardiac death donors: an analysis of a large single-center experience. Liver Transpl. 2009; 15(9):1028–1035. DOI: 10.1002/lt.21811 [PubMed: 19718636]

28. Fujita S, Mizuno S, Fujikawa T, et al. Liver transplantation from donation after cardiac death: a single center experience. Transplantation. 2007; 84(1):46–49. DOI: 10.1097/01.tp.0000267424.88023.7b [PubMed: 17627236]

29. Vanatta JM, Dean AG, Hathaway DK, et al. Liver transplant using donors after cardiac death: A single-center approach providing outcomes comparable to donation after brain death. Exp Clin Transplant. 2013; 11(2):154–163. DOI: 10.6002/ect.2012.0173 [PubMed: 23480344]

30. Bartlett A, Vara R, Muiesan P, et al. A single center experience of donation after cardiac death liver transplantation in pediatric recipients. Pediatr Transplant. 2010; 14(3):388–392. DOI: 10.1111/j.1399-3046.2009.01206.x [PubMed: 20519017]

31. Detry O, Seydel B, Delbouille M-H, et al. Liver transplant donation after cardiac death: experience at the University of Liege. Transplant Proc. 2009; 41(2):582–584. DOI: 10.1016/j.transproceed.2009.01.001 [PubMed: 19328931]

32. Foley DP, Fernandez LA, Leverson G, et al. Biliary complications after liver transplantation from donation after cardiac death donors: an analysis of risk factors and long-term outcomes from a single center. Ann Surg. 2011; 253(4):817–825. DOI: 10.1097/SLA.0b013e3182104784 [PubMed: 21475025]

33. Lee K-W, Simpkins CE, Montgomery RA, Locke JE, Segev DL, Maley WR. Factors affecting graft survival after liver transplantation from donation after cardiac death donors. Transplantation. 2006; 82(12):1683–1688. DOI: 10.1097/01.tp.0000250936.73034.98 [PubMed: 17198260]
34. Mathur AK, Heimbach J, Steffick DE, Sonnenday CJ, Goodrich NP, Merion RM. Donation after cardiac death liver transplantation: predictors of outcome. Am J Transplant. 2010; 10(11):2512–2519. DOI: 10.1111/j.1600-6143.2010.03293.x [PubMed: 20977642]
35. Chan EY, Olson LC, Kisthard JA, et al. Ischemic cholangiopathy following liver transplantation from donation after cardiac death donors. Liver Transplant. 2008; 14(5):604–610. DOI: 10.1002/lt.21361
36. Halazun KJ, Al-Mukhtar A, Aldouri A, Willis S, Ahmad N. Warm ischemia in transplantation: search for a consensus definition. Transplant Proc. 2007; 39(5):1329–1331. DOI: 10.1016/j.transproceed.2007.02.061 [PubMed: 17580133]
37. Taner CB, Bulatao IG, Willingham DL, et al. Events in procurement as risk factors for ischemic cholangiopathy in liver transplantation using donation after cardiac death donors. Liver Transplant. 2012; 18(1):101–112. DOI: 10.1002/lt.22404
38. Habib S, Berk B, Chang CC, et al. MELD and prediction of post-liver transplantation survival. Liver Transplant. 2006; 12(3):440–447. DOI: 10.1002/lt.20721
39. Desai NM, Mange KC, Crawford MD, et al. Predicting outcome after liver transplantation: utility of the model for end-stage liver disease and a newly derived discrimination function. Transplantation. 2004; 77(1):99–106. DOI: 10.1097/01.TP.0000101009.91516.FC [PubMed: 14724442]
40. Yagci G, Fernandez LA, Knechtle SJ, et al. The impact of donor variables on the outcome of orthotopic liver transplantation for hepatitis C. Transplant Proc. 2008; 40(1):219–223. DOI: 10.1016/j.transproceed.2007.11.058 [PubMed: 18261591]
41. Mawardi M, Aba Alkhail FA, Katada K, et al. The clinical consequences of utilizing donation after cardiac death liver grafts into hepatitis C recipients. Hepatol Int. 2011; 5(3):830–833. DOI: 10.1007/s12072-010-9242-y [PubMed: 21484141]
42. Croome K, Wall W, Chandok N, Beck G, Marotta P, Hernandez-Alejandro R. Inferior survival in liver transplant recipients with hepatocellular carcinoma receiving donation after cardiac death liver allografts. Liver Transpl. 2013; 19(11):1214–1223. DOI: 10.1002/lt.23715 [PubMed: 23907778]
43. Hong JC, Yersiz H, Kositamongkol P, et al. Liver transplantation using organ donation after cardiac death: a clinical predictive index for graft failure-free survival. Arch Surg. 2011; 146(9):1017–1023. DOI: 10.1001/archsurg.2011.240 [PubMed: 21930997]
44. Maheshwari A, Maley W, Li Z, Thuluvath PJ. Biliary complications and outcomes of liver transplantation from donors after cardiac death. Liver Transplant. 2007; 13(12):1645–1653. DOI: 10.1002/lt.21212
45. Seehofer D, Eurich D, Veltzke-Schlieker W, Neuhaus P. Biliary complications after liver transplantation: old problems and new challenges. Am J Transplant. 2013; 13(2):253–265. DOI: 10.1111/ajt.12034 [PubMed: 23331505]
46. Sanchez-Urdazpal L, Gores GJ, Ward EM, et al. Ischemic-type biliary complications after orthotopic liver transplantation. Hepatology. 1992; 16(1):49–53. DOI: 10.1002/hep.1840160110 [PubMed: 1618482]
47. Van Der Hilst CS, Ijtsma AJC, Bottema JT, et al. The price of donation after cardiac death in liver transplantation: A prospective cost-effectiveness study. Transpl Int. 2013; 26(4):411–418. DOI: 10.1111/tri.12059 [PubMed: 23398215]
48. Jay CL, Lyuksemburg V, Kang R, et al. The increased costs of donation after cardiac death liver transplantation: caveat emptor. Ann Surg. 2010; 251(4):743–748. DOI: 10.1097/SLA.0b013e3181d3d3da [PubMed: 20224367]
49. Dubbeld J, van Hoek B, Ringers J. Use of a liver from donor after cardiac death: is it appropriate for the sick or the stable? Curr Opin Organ Transplant. 2011; 16(2):239–242. DOI: 10.1097/MOT.0b013e3283447acd [PubMed: 21415827]

50. Moers C, Smits JM, Maathuis M-HJ, et al. Machine perfusion or cold storage in deceased-donor kidney transplantation. N Engl J Med. 2009; 360(1):7–19. DOI: 10.1056/NEJMoa0802289 [PubMed: 19118301]
51. St Peter SD, Imber CJ, Friend PJ. Liver and kidney preservation by perfusion. Lancet. 2002; 359(9306):604–613. DOI: 10.1016/S0140-6736(02)07749-8 [PubMed: 11867131]
52. Latchana N, Peck JR, Whitson B, Black SM. Preservation solutions for cardiac and pulmonary donor grafts: a review of the current literature. J Thorac Dis. 2014; 6(8):1143–1149. DOI: 10.3978/j.issn.2072-1439.2014.05.14 [PubMed: 25132982]
53. Guarrera JV, Henry SD, Samstein B, et al. Hypothermic machine preservation facilitates successful transplantation of “orphan” extended criteria donor livers. Am J Transplant. 2015; 15(1):161–169. DOI: 10.1111/ajt.12958 [PubMed: 25521639]
54. Dutkowski P, Schlegel A, De Oliveira M, Mullhaupt B, Neff F, Clavien PA. HOPE for human liver grafts obtained from donors after cardiac death. J Hepatol. 2014; 60(4):765–772. DOI: 10.1016/j.jhep.2013.11.023 [PubMed: 24295869]
55. Op Den Dries S, Sutton ME, Karimian N, et al. Hypothermic oxygenated machine perfusion prevents arteriolonecrosis of the peribiliary plexus in pig livers donated after circulatory death. PLoS One. 2014; 9(2):e88521.doi: 10.1371/journal.pone.0088521 [PubMed: 24551114]
56. Schlegel A, Graf R, Clavien PA, Dutkowski P. Hypothermic oxygenated perfusion (HOPE) protects from biliary injury in a rodent model of DCD liver transplantation. J Hepatol. 2013; 59(5):984–991. DOI: 10.1016/j.jhep.2013.06.022 [PubMed: 23820408]
57. Lee CY, Jain S, Duncan HM, et al. Survival transplantation of preserved non-heart-beating donor rat livers: preservation by hypothermic machine perfusion. Transplantation. 2003; 76(10):1432–1436. DOI: 10.1097/01.TP.0000088674.23805.0F [PubMed: 14657681]
58. St Peter SD, Imber CJ, Lopez I, Hughes D, Friend PJ. Extended preservation of non-heart-beating donor livers with normothermic machine perfusion. Br J Surg. 2002; 89(5):609–616. DOI: 10.1046/j.1365-2168.2002.02052.x [PubMed: 11972552]
59. Xu H, Berendsen T, Kim K, et al. Excorporeal normothermic machine perfusion resuscitates pig DCD livers with extended warm ischemia. J Surg Res. 2012; 173(2):e83–e88. DOI: 10.1016/j.jss.2011.09.057 [PubMed: 22099594]
60. Schon MR, Kollmar O, Wolf S, et al. Liver transplantation after organ preservation with normothermic extracorporeal perfusion. Ann Surg. 2001; 233(1):114–123. DOI: 10.1097/00000658-200101000-00017 [PubMed: 11141233]
61. Boehnert MU, Yeung JC, Bazerbachi F, et al. Normothermic acellular EX vivo liver perfusion reduces liver and bile duct injury of pig livers retrieved after cardiac death. Am J Transplant. 2013; 13(6):1441–1449. DOI: 10.1111/ajt.12224 [PubMed: 23668775]
62. Izamis ML, Tolboom H, Uygun B, Berthiaume F, Yarmush ML, Uygun K. Resuscitation of ischemic donor livers with normothermic machine perfusion: a metabolic flux analysis of treatment in rats. PLoS One. 2013; 8(7):e69758.doi: 10.1371/journal.pone.0069758 [PubMed: 23922793]
63. Tolboom H, Milwid JM, Izamis ML, Uygun K, Berthiaume F, Yarmush ML. Sequential cold storage and normothermic perfusion of the ischemic rat liver. Transplant Proc. 2008; 40(5):1306–1309. DOI: 10.1016/j.transproceed.2008.03.100 [PubMed: 18589093]
64. Berendsen TA, Bruinsma BG, Lee J, et al. A simplified subnormothermic machine perfusion model restores ischemically damaged liver grafts in a rat model of orthotopic liver transplantation. Transplant Res. 2012; 1(1):6.doi: 10.1186/2047-1440-1-6 [PubMed: 23369351]
65. Op Den Dries S, Karimian N, Sutton ME, et al. Ex vivo normothermic machine perfusion and viability testing of discarded human donor livers. Am J Transplant. 2013; 13(5):1327–1335. DOI: 10.1111/ajt.12187 [PubMed: 23463950]
66. Knaak JM, Spetzler VN, Goldaracena N, et al. Subnormothermic ex vivo liver perfusion reduces endothelial cell and bile duct injury after donation after cardiac death pig liver transplantation. Liver Transpl. 2014; 20(11):1296–1305. DOI: 10.1002/lt.23986 [PubMed: 25179693]
67. Tolboom H, Izamis ML, Sharma N, et al. Subnormothermic machine perfusion at both 20°C and 30°C recovers ischemic rat livers for successful transplantation. J Surg Res. 2012; 175(1):149–156. DOI: 10.1016/j.jss.2011.03.003 [PubMed: 21550058]

68. Berendsen TA, Bruinsma BG, Puts CF, et al. Supercooling enables long-term transplantation survival following 4 days of liver preservation. Nat Med. 2014; 20(7):790–793. DOI: 10.1038/nm.3588 [PubMed: 24973919]

69. D'Alessandro AM, Hoffmann RM, Knechtle SJ, et al. Liver transplantation from controlled non-heart-beating donors. Surgery. 2000; 128(4):579–588. DOI: 10.1067/msy.2000.108421 [PubMed: 11015091]

70. Abt P, Crawford M, Desai N, Markmann J, Olthoff K, Shaked A. Liver transplantation from controlled non-heart-beating donors: an increased incidence of biliary complications. Transplantation. 2003; 75(10):1659–1663. DOI: 10.1097/01.TP.0000071487.73903.90 [PubMed: 12777852]

71. Manzarbeitia CY, Ortiz JA, Jeon H, et al. Long-term outcome of controlled, non-heart-beating donor liver transplantation. Transplantation. 2004; 78(2):211–215. DOI: 10.1097/01.TP.0000128327.95311.E3 [PubMed: 15280680]

72. Muiesan P, Girlanda R, Jassem W, et al. Single-center experience with liver transplantation from controlled non-heartbeating donors: a viable source of grafts. Ann Surg. 2005; 242(5):732–738. DOI: 10.1097/01.sla.0000186177.26112.d2 [PubMed: 16244548]

73. Pine JK, Aldouri A, Young AL, et al. Liver transplantation following donation after cardiac death: an analysis using matched pairs. Liver Transpl. 2009; 15(9):1072–1082. DOI: 10.1002/lt.21853 [PubMed: 19718634]

74. Doyle MBM, Collins K, Vachharajani N, et al. Outcomes using grafts from donors after cardiac death. J Am Coll Surg. 2015; 221(1):142–152. DOI: 10.1016/j.jamcollsurg.2015.03.053 [PubMed: 26095563]

75. Jay C, Ladner D, Wang E, et al. A comprehensive risk assessment of mortality following donation after cardiac death liver transplant - An analysis of the national registry. J Hepatol. 2011; 55(4):808–813. DOI: 10.1016/j.jhep.2011.01.040 [PubMed: 21338639]

Table 1

Categories of Donors After Cardiac Death[6,7]

Category	Description of Donor	Uncontrolled or Controlled Warm Ischemia
Category 1	Brought in dead	Uncontrolled
Category 2	Unsuccessful resuscitation	Uncontrolled
Category 3	Awaiting cardiac arrest	Controlled
Category 4	Cardiac arrest after brain death	Controlled
Category 5	Cardiac death in hospital as inpatient	Uncontrolled

Table 2

Single-Center Outcomes of Donations After Circulatory Death Versus Donations After Brain Death

Study	Year	DCD or DBD (No. of Patients)	% Patient Survival at 1, 3, and 5 years	% Graft Survival at 1, 3, and 5 years	Donor Age, y	Recipient Age, y	Mean WIT, min	Mean CIT, min	Mean MELD
D'Alessandro et al.[69]	2000	DCD (19)	-, 72.6, -	-, 53.8, -	32.0 ± 15.1	47.4 ± 18.5	16.4 ± 10.9	474 ± 138	
		DBD (364)	-, 84.8, -	-, 80.9, -	32.8 ± 15.9	47.7 ± 14.3		504 ± 150	
Abt et al.[70]	2003	DCD (n=15)	79.0, 79.0, -	71.8, 71.8, -	30.1 ± 10.4		20.4 ± 6	366.4 ± 131	
		DBD (n=221)	90.9, 77.7, -	85.4, 73.9, -	38.4 ± 17.4			464.0 ± 149	
Manzarbeitia et al.[71]	2004	DCD (n=19)	89.5, 83.5 (2 year), -		33.95 ± 17.93		19.67 ± 7.68	574 ± 84	
		DBD (n=311)	84.2, 82.0 (2 year), -						
Muiesan et al.[72]	2005	DCD (n=32)	89.6, -, -	86.5, -, -	36 ± 14.9	38.4 (0.7–72)	14 ± 5.3	516 (312–858)	
		DBD (n=0)							
Fujita et al.[28]	2007	DCD (n=24)	86.8, 81.7, -	69.1, 58.6, -	31.6	51.1	12.8 ± 7.4	482	22.1
		DBD (n=1209)	84.0, 76.0, -	78.7, 70.2, -	35.1	42		497	23.6
Detry et al.[31]	2009	DCD (n=13)	100, 78.5 (2yr), -	100, 78.5 (2 y), -	51 (25–71)	55 (50–70)	<30	295	14.2 (8–25)
		DBD (n=0)							
Grewal et al.[27]	2009	DCD (n=108)	91.5, 88.1, 88.1	79.3, 74.5, 71.0	41 ± 17	55 ± 11	22.3	378 ± 102	17 ± 7.9
		DBD (n=1328)	87.3, 81.1, 77.2	81.6. 74.7. 69.1	48 ± 20	55 ± 10		429 ± 126	18 ± 8.6
Pine et al.[73]	2009	DCD (n=39)	82.1, 100, -	79.5, 63.6, -	41.85 (12–68)	50.59 (18–67)		352.21 (161–606)	14.23 (6–26)
		DBD (n=39)	68.2, 100, -	97.4, 97.4, -	42.87 (19–69)	49.38 (22–66)		593.87 (130–956)	15.23 (6–32)
Skaro et al.[21]	2009	DCD (n=32)	74.0, 74.0,-	61.3, 52.6, -	43.1 ± 17.6	53.1 ± 12.8	15.1 ± 4.6	330 ± 90	22.9 ± 10.2
		DBD (n=237)	90.4, 80.7, -	85.2, 74.2, -	44.7 ± 17.6	54.9 ± 10.0		312 ± 90	21.9 ± 10.1
de Vera et al.[22]	2009	DCD (n=141)	79, -, 70, 57 (10yr)	69, - 56, 44 (10y)	37.1 ± 15.9	53.1 ± 10.7	19.8 ± 8.8	657 ± 170	18.3 ± 9
		DBD (n=282)	85, -, 76, 64 (10yr)	82, -, 73, 63 (10 y)	39.1 ± 16.1	53.7 ± 9.3		636 ± 177	18.5 ± 9
Bartlett et al.[30] *pediatric	2010	DCD (n=14)	100, -, -	100, -, -	23 ± 10.64	7 (8 mo-16 y)	16 (11–29)	420 (330–504)	-
		DBD (n=0)							
Foley et al.[32]	2011	DCD (n=87)	84, -, 68, 54 (10yr)	69, -, 56, 43 (10 y)	35.8 ± 13.3	50.5 ± 13.1	20.8 ± 9.4	432 ± 138	19.7 ± 8.9
		DBD (n=1157)	91, -, 81, 67 (10yr)	86, -, 76, 60 (10 y)	36.5 ± 13.3	47.5 ± 16.7		498 ± 138	20.1 ± 8.7
Hong et al.[43]	2011	DCD (n=81)		78, 62, 53	36 ± 10	54 ± 11	26 ± 9	375 ± 138	24 ± 10
		DBD (n=0)							
Croome et al.[26]	2012	DCD (n=36)	91, 80 (2yr)	91, 73 (2 y)	41.8 ±13.19	53.6 ± 8	35 ± 18	336 ± 66	17.5 ± 8.3
		DBD (n=327)			45.93 ± 10.04	54.95 ± 10.07		432 ± 150	18.98 ± 9.79

Study	Year	DCD or DBD (No. of Patients)	% Patient Survival at 1, 3, and 5 years	% Graft Survival at 1, 3, and 5 years	Donor Age, y	Recipient Age, y	Mean WIT, min	Mean CIT, min	Mean MELD
Leithead et al.[23]	2012	DCD (n=88)	84.9, 78.0, -	83.7, 77.0, -	46.4 ± 16.1	55.7 ± 8.5	20.1 ± 9.0	448 ± 114	14 ± 5
		DBD (n=88)	90.5, 83.4, -	88.2, 81.3, -	50.4 ± 14.6	56.0 ± 9.2		498 ± 144	13 ± 4
Meurisse et al.[25]	2012	DCD (n=30)	93, 85, 85	90, 82, 82	51 (37–59)	60 (52–65)	24 (18–30)	414 (325–471)	15 (11–17)
		DBD (n=385)	88, 78, 72	85, 74, 68	53 (42–64)	58 (49–64)		516 (433–606)	16 (11–23)
Taner et al.[37]	2012	DCD (n=200)	92.6, 85.0, 80.9	80.9, 72.7, 68.9	40.3 ± 16.4	55.1 ± 9.4	25.3 ± 10.8	360 ± 90	17.9 ± 7.9
		DBD (n=1828)	89.8, 83.0, 76.6	83.3, 75.1, 68.6	46.6 ± 19.7	54.1 ± 10.4		420 ± 114	18.5 ± 8.6
Vanatta et al.[29]	2013	DCD (n=38)	92, 80, -	92, 74, -	33 ± 15	56 ± 8	21 ± 8	289 ± 97	16 ± 3
		DBD (n=76)	92, 86, -	91, 85, -	34 ± 15	55 ± 7		280 ± 11	18 ± 6
Doyle et al.[74]	2015	DCD (n=49)	95.9, 90.6, 87.1	93.6, 86.0, 79.5	29.1 ± 12.8	56.4 ± 10.4	33.6 ± 9.3	342 ± 114	18.7 ± 7.1
		DBD (n=98)	94.7, 83.5, 80.3	92.6, 81.4, 78.1	29.9 ± 12.1	54.9 ± 65.3		342 ± 132	18.3 ± 7.3

Abbreviations: CIT, cold ischemic time; DBD, donations after brain death; DCD, donation after circulatory (cardiac) death; MELD, model for end-stage liver disease; WIT, warm ischemic time

Table 3

Possible Risk Factors with Liver Transplant With Donations After Circulatory Death

Donor age[15,16,21,22,32,34,35,43,49,75]
Donor weight > 100 kg[34,35]
Recipient age > 60 years[34,75]
> 30 kg/m^2 Recipient body-mass index [22,43]
Recipient Black race[34,37]
Warm ischemic time > 30 minutes[20,22,32,34,43,70]
Asystole to cross-clamp duration[37]
Cold ischemic time > 8 hours[16,21,22,25,32,34,43]
Hepatocellular carcinoma[42,75]
MELD Score[17,21,22,34,49]
Hepatitis C[34,40,41,75]

Abbreviations: DBD, donations after brain death; DCD, donation after circulatory (cardiac) death; MELD, model for end-stage liver disease

Table 4

Single-Center Complications for Donations after Circulatory Death Versus Donations after Brain Death

Study	Complication	DCD	DBD	*P* Value
D'Alessandro et al.[69]				
	General biliary			
	Ischemic cholangiopathy	5.3	4.6	
	Hepatic artery thrombosis	0	3.8	
	Hepatic artery stenosis	10.5	5.2	
	Portal vein thrombosis	0	3.8	
	Primary nonfunction	10.5	1.3	.04
	Retransplantation	21.1		
Abt et al.[70]				
	General biliary	33.3	9.5	< .01
	Ischemic cholangiopathy	20	2.3	< .01
	Anastomotic biliary strictures	6.7	5.4	
	Vascular			
	Hepatic artery thrombosis	0	3.2	> .05
	Portal vein thrombosis	0	0.4	
	Primary nonfunction	6.7	3.6	> .05
Manzarbeitia et al.[71]				
	General biliary	10.5	13.8	.68
	Retransplantation	10.5	8.7	.68
Muiesan et al.[72]				
	General biliary	9.4		
	Anastomotic biliary strictures	6.3		
	Hepatic artery thrombosis	6.3		
	Retransplantation	3.1		
Fujita et al.[28]				
	General biliary	25	21	
	Ischemic cholangiopathy	12.5	9.1	.568
	Hepatic artery thrombosis	8.3	4.1	.312
	Portal vein thrombosis	4.2	2.6	.648
	Primary nonfunction	0	2.8	.405
	Retransplantation	20.8	9.4	.059
Chan et al.[35]				
	General biliary			
	Ischemic cholangiopathy	13.7	1	.001
	Anastomotic biliary strictures	9.8	78	NS
	Hepatic artery thrombosis	0	4.8	NS
	Primary nonfunction	0	3.2	NS
Detry et al.[31]				
	General biliary	15		

Study	Complication	DCD	DBD	*P* Value
	Anastomotic biliary strictures	7.7		
	Primary nonfunction	0		
	Retransplantation	0		
Grewal et al.[27]				
	General biliary			
	Ischemic cholangiopathy	8.3	1.9	.008
	Hepatic artery thrombosis	0.9	1.7	.469
	Primary nonfunction	3.7	1.4	.09
	Retransplantation	14.8	9.3	.107
	Recurrent hepatitis C	3.7	3.1	.449
Pine et al.[73]				
	General biliary	33.3	10.2	
	Ischemic cholangiopathy	20.5	0	.005
	Anastomotic biliary strictures	10.2	10.2	1
	Hepatic artery thrombosis	2.6	5.1	1
	Hepatic artery stenosis	12.8	0	.27
	Primary nonfunction	5.1	0	.494
Skaro et al.[21]				
	General biliary	53.1	21.5	< .001
	Ischemic cholangiopathy	37.5	1.7	< .001
	Hepatic artery thrombosis	9.4	3	.1
	Primary nonfunction	3.1	0.4	.22
	Retransplantation	21.9	6.8	.01
de Vera et al.[22]				
	General biliary	25	13	< .001
	Ischemic cholangiopathy	16.3		
	Hepatic artery thrombosis	6	6	1
	Primary nonfunction	12	3	< .001
	Retransplantation	18	7	< .001
Foley et al.[32]				
	General biliary	47	26	< .01
	Ischemic cholangiopathy	34	1	< .01
	Anastomotic biliary strictures	14	11	.37
	Hepatic artery thrombosis	8.5	2.9	.38
	Hepatic artery stenosis	5.7	10.5	.18
	Portal vein thrombosis	4	5	.54
	Primary nonfunction	1.2	2.3	.31
	Retransplantation	19	4.8	.0001
Hong et al.[43]				
	General biliary	29		
	Ischemic cholangiopathy	9.9		
Croome et al.[26]				

Study	Complication	DCD	DBD	*P* Value
	General biliary	25	13	.062
	Ischemic cholangiopathy	5.6	0	< .001
	Anastomotic biliary strictures	2.7	4	.7232
Leithead et al.[23]				
	Acute kidney injury	53.4	31.8	.035
Meurisse et al.[25]				
	General biliary	50	28.3	.012
	Ischemic cholangiopathy	33.3	12.5	.001
	Anastomotic biliary strictures	26.7	18.7	.287
	Primary nonfunction	0	0	
	Retransplantation	0	2.8	.175
Taner et al.[37]				
	General biliary	36.7		
	Ischemic cholangiopathy	12.6		
	Hepatic artery thrombosis	3.3		
	Hepatic artery stenosis	5.1		
	Primary nonfunction	2.3		
	Retransplantation	13.9		
Vanatta et al.[29]				
	General biliary	18.8	9.2	.225
	Ischemic cholangiopathy	7.9	1.3	.107
	Anastomotic biliary strictures	18.4	9.2	.225
	Hepatic artery thrombosis	0	3.9	.55
	Hepatic artery stenosis	10.5	6.6	.478
	Portal vein thrombosis	0	5.3	.299
	Primary nonfunction	2.6	1.3	1
Doyle et al.[74]				
	General biliary	34.6	22.4	
	Ischemic cholangiopathy	8.5	0	
	Anastomotic biliary strictures	16.3	12.2	.49
	Hepatic artery thrombosis	0	0	
	Portal vein thrombosis	0	0	
	Primary nonfunction	0	0	
	Acute kidney injury	16.3	4.1	.01

Abbreviations: DBD, donations after brain death; DCD, donation after circulatory (cardiac) death; NS, not significant