VOLUME - 11, ISSUE - 04, APRIL - 2022 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

Anaesthesiology

ANAESTHETIC CONSIDERATIONS FOR ORGAN HARVESTING IN A BEATING HEART BRAIN DEAD PATIENT.

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ABSTRACT Management of a beating heart brain dead donor requires an anesthesiologist to incorporate deep knowledge of the effects of brain death on various organ systems of the body. A specific peri-operative management is required during organ harvesting procedures. An anesthesiologist should be well versed in Intensive Care Unit (ICU) care procedures pertaining to various organ systems of the body before procurement of organs. Specific goals and strategies need to be implemented along with the surgical team to optimize donor outcome.

KEYWORDS : Beating Heart Brain Dead, Organ Harvesting, Donor, Anesthesiologist.

INTRODUCTION

A brain dead patient is the primary source of grafts for solid organ transplantation [1,2]. Appropriate management of organ donors from the diagnosis of brain death to the end of the organ procurement procedure is of importance to the anesthesiologist. This requires the anesthesiologist to optimize the functions of potential grafts. Intensive Care Unit (ICU) management is an additional and very important part in the management of such patients [3,4,5].

Brain Death and Anesthetic Implications

Nearly every organ system is affected by brain death. Normalization of the physiology of the donor is required to maximize the long-term viability of organs for donation [6].This strategy includes ICU care for optimization of the normal physiology before organ harvesting along with intra-operative management of such patients. The effects of brain death on various organ systems and their anesthetic implications are summarized below.

The Cardiovascular system:

1. Shortly after brain death, a prolonged period of increased sympathetic tone and cardiac output occurs as a result of a "catecholamine storm" [7]. This results in tachycardia and vasoconstriction resulting in profound hypertension.
2. Myocardial Injury also arises due to an increase in the systemic vascular resistance which may result in left ventricular failure, decreased cardiac output, and even mitral valve regurgitation [8].
3. To preserve the cardiac function an anesthesiologist needs to maintain the coronary perfusion pressure [9]. In the operating room it is managed with nitroprusside and/or esmolol.
4. Cardiac arrhythmias occur along with electrocardiogr aphic changes indicative of ischemia [8].
5. Following this after several hours of brain death a dramatic loss of sympathetic tone can occur and result in hemodynamic instability in such patients [10].
6. This loss of sympathetic tone must be managed in order to maintain the viability of the organ grafts [11].
7. Although organ procurement surgery is generally not a lengthy procedure, significant fluid shifts with considerable interstitial accumulation of fluid can occur from the abdominal and/or thoracic incision made for organ procurement.
8. Polyuria secondary to diabetes insipidus resulting from posterior pituitary infarction is common in brain death and can further contribute to hypovolemia [12].
9. Cardiovascular Goals should include a systolic blood pressure of $\geq$ 100 mmHg, a mean arterial pressure of $\geq$ 70 mmHg, with a heart rate of 60-120 beats per minute.
10. Intravascular Volume is maintained using infusion of crystalloids. Large volumes of 0.9% Normal Saline are avoided due to a risk of Hyperchloremic Acidosis. Where colloid resuscitation (Lung Procurement) is used HydroxyEthyl Strach (HES) is avoided.
11. Volume repletion should be guided by arterial waveform pulse pressure variation, central venous pressure, and urine output if diabetes insipidus is not an issue. Mixed Venous Oxygen Saturation of >60% can also be used as a parameter for viability.
12. Dopamine, phenylephrine, epinephrine, norepinephrine, and vasopressin are common agents and provide adequate hemodynamic support.
13. Vasopressin is the most ideal agent to maintain the vascular tone as it addresses the issues arising due to both blood pressure variation as well as the diabetes insipidus.
14. A balanced plan to maintain adequate blood pressure thus incorporates the replacement of intravenous fluid deficits while judiciously using vasopressors to counteract the decrease in systemic vascular resistance that occurs in patients after brain death.

The Respiratory System:

1. Brain death can lead to neurogenic pulmonary edema, as the initial increase in systemic vascular resistance may lead to increased blood volume within the venous system and subsequent pulmonary overload [13,14].
2. Pulmonary edema can also result from, or be exacerbated by, large-volume crystalloid resuscitation.
3. In addition, the release of catecholamines during the initial hypertensive and hyperdynamic period after brain death causes elevated cytokine levels and, thus, pulmonary endothelial damage and capillary disruption.
4. A lung-protective ventilatory strategy should be employed, with tidal volumes of 6-8 mLkg-1 and a positive end-expiratory pressure (PEEP) of 8-10 cm H2O, especially if lung procurement is planned [15].
5. If lung procurement for transplantation is planned, a minimally positive fluid balance should be maintained.
6. Keeping the central venous pressure (CVP) $\geq$ 10 mmHg increases the viability of Lung and Heart available for transplantation [16].

The Musculoskeletal System

1. Even in the setting of brain death, somatic movements can occur.
2. Skeletal muscle paralysis should be provided during organ procurement to optimize surgical conditions.
3. Additionally, muscle relaxation will ameliorate the somatic response to surgical stimulus mediated by spinal cord reflexes, which may persist even in the presence of brain death [17].

The Hematological System

1. The reported incidence of coagulopathy after brain injury

lies between10% to 80% [18].

2. Management in the ICU includes targeting a hemoglobin concentration of 9-10 gm per dl and no less than 7 gm per dl.
3. Platelets and plasma should be given when clinically significant bleeding is evident, but not to target a specific coagulation test level or platelet count.

The Endocrine System:

1. Impairment of the hypothalamic-pituitary axis occurs in the majority of patients with brain death and results in decreases in serum concentrations of anterior and/or posterior pituitary hormones.
2. There is reduction in tri-iodothyronine (T3), cortisol, and insulin after brain death.
3. Therapy using thyroid hormone, cortisol, and insulin improves the cardiovascular status and organ transplantation success rates while providing improved donor organ viability [Table 1].

Table 1: Hormone therapy for organ donors

Hormone	Dose
Thyroid hormone (tetraiodothyronine)	20 mcg iv bolus, 10 mcg/hr iv infusion
Vasopressin	1 U iv bolus, 2.4 U/hr iv infusion
Methylprednisolone	15 mg/kg iv q24hr

4. Hyperglycemia is associated with brain death. This causes a lower rate of organ suitability for transplantation and decreased renal graft survival. Serum glucose needs to be optimized to < 200 mg per dl.
5. Posterior pituitary infarction in the setting of brain death can lead to diabetes insipidus, producing large amounts of dilute urine and resultant serum hyperosmolarity. Arginine vasopressin (AVP) should be used to reduce urine output and prevent a further increase in serum osmolarity.
6. A hypotonic fluid or free water infusion may need to be used to correct hypernatremia such as 0.45% NaCl, as a maintenance fluid. Generally, correction should occur at a rate of 0.5-1.0 mEq per L per hr to avoid cerebral edema and its sequelae.

Other Anesthetic Considerations

1. Prior to the start of organ donation surgery, intravenous catheters and Central Venous Catheters should be in place for rapid large-volume intravascular fluid replacement.
2. An arterial catheter should be placed so that blood pressure can be continuously followed and managed intraoperatively.
3. For the procurement of heart, cold cardioplegia is utilized. Often Celsior solution is used for this procedure. The abdominal harvesting University of Wisconsin (UW) solution is used. For liver transplantation Celsior and Custodiol HTK solutions are used.

Summary and Conclusions

Anesthesiologists play a vital role in the management of organ donors, in the ICU as well s the operating room improving the outcome of the procedure. To manage the heart-beating brain dead donor, the anesthesiologist must incorporate knowledge of the effects of brain death on each organ system as well as the effects of the preoperative measures that the donor required in the ICU. It is also important to know which organs are going to be procured so that specific goals can be established and strategies can be implemented (e.g., lung-protective ventilation or intraoperative glycemic control) to optimize donor outcome [Table 2].

Table 2: Anesthetic Implications of Brain Death

System	Effects	Management
Cardiovascular System	Myocardial injury, Loss of vascular tone, Hypovolemia.	Restore intravascular volume . Use vasopressors as necessary to maintain adequate organ perfusion. Maintain SBP $\geq$ 100 mmHg, MAP $\geq$ 70, HR 60-120 beats per min.
Respiratory System	Increased pulmonary capillary permeability, Pulmonary edema.	'Lung-protective" ventilatory strategy: TV 6-8 mLkg-1 of predicted body weight, PEEP 8-10 cm H2O. Judicious intravenous fluid; CVP 4-8 (\10) mmHg.
Musculoskeletal System	Reflex somatic movements mediated by spinal reflexes.	Skeletal muscle paralysis.
Hematological System	Coagulopathy, disseminated intravascular coagulation.	Blood Transfusion. Correct coagulopathy with clotting factors or platelets if evidence of ongoing bleeding.
Endocrine System	Diabetes insipidus and obliteration of thyroid axis, Hyperglycemia, Hypernatremia.	Vasopressin. Insulin infusion to maintain serum glucose $\leq$ 180 mg per dl. Thyroxine or T3 infusion, corticosteroids.

Additionally, anesthesiologists help to ensure that the highest quality and the most appropriate care is rendered to donors by working to establish protocols that maximize the number of available organs with the best chance of long-term graft viability.

REFERENCES

1. Anderson TA, Bekker P, Vagefi PA. Anesthetic considerations in organ procurment surgery: a narrative review. Can J Anesth. 2015;62:529–39.
2. Souter MJ, Eidbo E, Findlay JY, Lebovitz DJ, Moguilevitch M, Neidlinger NA, et al. Organ donor management: part 1. Toward a consensus to guide anesthesia services during donation after brain death. Semin Cardiothorac Vasc Anesth. 2017;22:211–22.
3. Niemann CU, Feiner J, Swain S, Bunting S, Friedman M, Crutchfield M, et al. Therapeutic hypothermia in deceased organ donors and kidney-graft function. N Engl J Med. 2015;373:405–14.
4. McKeown DW, Bonser RS, Kellum JA. Management of the heartbeating brain-dead organ donor. Br J Anaesth. 2012;108(Suppl 1):i96–107.
5. Boulard G, Guiot P, Pottecher T, Tencaillon A. Management of subjects in a state of brain death and the preservation of organs. Ann Fr Anesth Reanim. 2005;24:836–43.
6. Dictus C, Vienenkoetter B, Esmaeilzadeh M, Unterberg A, Ahmadi R. Critical care management of potential organ donors: our current standard. Clin Transplant 2009; 23(Suppl 21): 2-9.
7. Avlonitis VS, Wigfield CH, Kirby JA, Dark JH. The hemodynamic mechanisms of lung injury and systemic inflammatory response following brain death in the transplant donor. Am J Transplant 2005; 5(4 Pt 1): 684-93.
8. Novitzky D, Rhodin J, Cooper DK, Ye Y, Min KW, DeBault L. Ultrastructure changes associated with brain death in the human donor heart. Transplant Int 1997; 10: 24-32.
9. Szabo G. Physiologic changes after brain death. J Heart Lung Transplant 2004; 23(9 Suppl): S223-6.
10. Belzberg H, Shoemaker WC, Wo CC, et al. Hemodynamic and oxygen transport patterns after head trauma and brain death: implications for management of the organ donor. J Trauma 2007; 63: 1032-42.
11. Smith M. Physiologic changes during brain stem death—lessons for management of the organ donor. J Heart Lung Transplant 2004; 23(9 Suppl): S217-22.
12. Chen EP, Bittner HB, Kendall SW, Van Trigt P. Hormonal and hemodynamic changes in a validated animal model of brain death. Crit Care Med 1996; 24: 1352-9.
13. Novitzky D, Wicomb WN, Rose AG, Cooper DK, Reichart B. Pathophysiology of pulmonary edema following experimental brain death in the chacma baboon. Ann Thorac Surg 1987; 43: 288-94.
14. Bratton SL, Davis RL. Acute lung injury in isolated traumatic brain injury. Neurosurgery 1997; 40: 707-12.
15. Mascia L, Pasero D, Slutsky AS, et al. Effect of a lung protective strategy for organ donors on eligibility and availability of lungs for transplantation: a randomized controlled trial. JAMA 2010; 304: 2620-7.
16. Abdelnour T, Rieke S. Relationship of hormonal resuscitation therapy and central venous pressure on increasing organs for transplant. J Heart Lung Transplant 2009; 28: 480-5.
17. Gelb AW, Robertson KM. Anaesthetic management of the brain dead for organ donation. Can J Anaesth 1990; 37: 806-12.
18. Talving P, Benfield R, Hadjizacharia P, Inaba K, Chan LS, Demetriades D. Coagulopathy in severe traumatic brain injury: a prospective study. J Trauma 2009; 66: 55-61; discussion 61-2.