

Cardiac dysrhythmias during donor care

Organ procurement coordinators must treat various cardiac dysrhythmias (arrhythmias), including rhythm disturbances that may cause or follow a cardiac arrest, in about 15% to 50% of donors. Treatment decisions should be based on the particular dysrhythmia and its effect on donor blood pressure. Medications selected should be effective but short acting. In this article, data available in publications located through a PubMed search are reviewed and specific dysrhythmias that are likely to occur during donor care are described. Treatment recommendations are based on guidelines from the American Heart Association. (*Progress in Transplantation*. 2006;16:74-81)

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Notice to CE enrollees:

A closed-book, multiple-choice examination following this article tests your ability to accomplish the following objectives:

1. Discuss the dysrhythmias that may occur during donor care
2. Identify pharmacological considerations of managing cardiac dysrhythmias during donor care
3. Describe treatment strategies to manage specific dysrhythmias that are likely to occur during donor care

The incidence of cardiac dysrhythmias (arrhythmias) during donor care is difficult to determine. A search of the PubMed database and the authors' files produced few related citations¹⁻⁸ (Table 1). Similarly, publications about extended somatic support after brain death⁹⁻¹² did not identify dysrhythmias in that group. Data from Table 1 suggest that about 20% to 30% of donors experience a dysrhythmia. Sinus tachycardia (depending on the heart rate definition of tachycardia) appears most common, occurring in 20% to 50% of donors, followed by sinus bradycardia in 15%, atrial fibrillation in 10%, and other dysrhythmias less commonly. Treatment of dysrhythmias is not always necessary but may be required if the dysrhythmia causes a change in blood pressure.

Cardiac arrest does not preclude subsequent organ donation, even donation of the heart,¹³⁻¹⁶ but obviously requires treatment of the precipitating and possibly continuing dysrhythmia(s). Organ procurement and transplantation data from December 3, 2004, provided by the United Network for Organ Sharing (UNOS), indicate that 4.7% of donors in 2002 and 4.9% in 2003 had a cardiac arrest during their hospitalization before organ removal. In 3 other reports,^{1,16,17} 7%, 16%, and 25% of donors sustained a cardiac arrest. Solomon et al¹⁷ defined cardiac arrest as any episode of observed ventricular fibrillation or asystole; the type of arrest was not defined in other publications. Successful transplantation occurred after arrest times of more than 3 minutes,¹⁵ 4 to 90 minutes,¹⁴ and 18.8 (SD 14.6) minutes (in children).¹⁸ Some of these publications and the UNOS data indicate that some cardiac arrests occurred while the patient was being cared for before certification of brain death, but other arrests occurred after consent for donation had been given and while the donor was being prepared for organ removal. Therefore, organ procurement coordinators may be required to initiate treatment of significant dysrhythmias, including dysrhythmias that occur during a cardiac arrest.

Dysrhythmias are well documented during the evolution of brain death in experimental animals.¹⁹ The dysrhythmias appear to be influenced by the speed with which brain death is induced²⁰ and are more common immediately after brain herniation.^{19,21} Sinus brady-

Table 1 Publications citing dysrhythmias during donor care

| Reference | Study design | No. of patients | Comments |
|-----------------------------|----------------------------|-----------------|--|
| Nygaard et al ¹ | Retrospective | 114 | 27% had dysrhythmias; 25% had cardiac arrest; dysrhythmias not specified if before/after brain death; specific dysrhythmias not identified |
| Logigian et al ² | Prospective | 18 | Describes terminal rhythm changes after discontinuation of mechanical ventilation; before ventilation was stopped 2 patients (11%) had sinus bradycardia; 4 (22%) had sinus tachycardia, and 2 (11%) had atrial fibrillation |
| Drory et al ³ | Not specified | 28 | Sinus rate 90-122/min in 41%; sinus bradycardia in 14%; patients not supported as donors; "terminal" phase of brain death marked by atrial fibrillation in 58%, ventricular fibrillation in 17% |
| Muhlberg et al ⁴ | Retrospective | 144 | All supported with pulmonary artery and arterial catheters; cites high frequency of abnormal electrolyte levels; 30% had unspecified bradycardia |
| Dujardin et al ⁵ | Retrospective, consecutive | 66 | Ventricular dysrhythmias more common (32%) when decreased left ventricular function, occurred overall in 9 (14%) of 66 donors; donors with known heart disease or chest trauma were excluded |
| Griep et al ⁷ | Retrospective | 22 | Donors supported; sinus tachycardia (50%); atrial premature beats (number not specified); atrial tachycardia (9%) |
| Dosemeci et al ⁸ | Prospective | 94 | Nonspecified dysrhythmias in 21.3%; ventricular fibrillation cardiac arrest in 1%; bradycardia in 1%; sinus tachycardia in 15%; and atrial tachycardia in 4% (A. Ramazanoglu, L. Dosemeci, personal communication) |

cardia and tachycardia and ventricular premature beats occur most consistently in these experiments.^{21,22} Similarly, anecdotal comments from several publications cited in Table 1 suggest a greater predilection to dysrhythmias in humans soon after brain death and less so during subsequent donor care.

Older publications^{21,23} describing the "natural history" of brain death emphasize the inevitability of cardiac collapse and terminal dysrhythmias within hours to, at most, a few days after brain death. That data must be reconsidered, however, as donor care provided in those earlier years did not aggressively support somatic organ function as is customary now.

As brain death evolves, it causes significant physiological changes in cytokine production and cardiac histopathology, enzyme release, and contractility.²⁴⁻²⁶ Paramount among these changes are high concentrations of catecholamines circulating in the blood and dysfunction of the sympathetic and parasympathetic nervous systems. Hypotension resulting from reduced myocardial contractility and the conflicting effects of hormonal and nervous system changes after brain death may cause coronary artery constriction and ischemia-induced dysrhythmias. Changes in plasma fluid and electrolyte concentrations may also predispose organ donors to instability in myocardial depolarization and repolarization processes.²⁷ Vasoactive drugs required to provide inotropic support or peripheral vasoconstriction may also stimulate increased heart rate (positive chronotropic effect) and changes in cardiac

depolarization/repolarization. Such influences may create a physiological environment that predisposes the heart to dysrhythmias.

Clinical Interventions

This discussion highlights the changes in cardiac rate and rhythm reported in Table 1 and such changes encountered in the authors' experience. Readers are assumed to have a fundamental knowledge of electrocardiography, the ability to perform or assist in electrical cardioversion, and the support structure of a hospital or organ procurement organization (OPO) that has physicians readily available for consultation. It is assumed that interventions have been completed or are underway to minimize the effects of vasoactive drugs and to optimize blood oxygen and electrolyte levels, pH, and blood pressure.²⁸ These suggestions are based on the assumption that treatment will be needed for a relatively short time (<24 hours) before organ removal.

Pharmacological Considerations

The pharmacological "half-life" of a medication means the time required, after the medication is discontinued, for drug metabolism and excretion to reduce the blood concentration of the medication to half its original therapeutic level. Accepted pharmacological principles indicate that about 4 half-lives are needed to ensure complete elimination of a drug. For example, if a medication's pharmacological half-life is 10 hours, 40 hours will be needed to eliminate the drug



Figure 1 Sinus bradycardia.



Figure 2 Sinus tachycardia.

from the circulation. Medication half-life is dependent on such factors as patient/donor age, general metabolic rate (as affected by body temperature), how the drug is metabolized (eg, by liver, kidney), and in which body compartments (eg, fat stores, entire body water) the drug is distributed. In general, it is reasonable to select medications with short half-lives during donor care so as to provide “drug-free” organs for transplantation.

The importance of clearing the donor’s circulation of medication is unclear, however, because the amount of any drug in a single implanted organ is unknown. For example, amiodarone is very lipid soluble and, accordingly, has a very long half-life because it is reabsorbed and recirculated from its lipid reservoirs throughout the body. This characteristic would seemingly be a disadvantage during donor care. However, most amiodarone would remain in the donor’s lipid stores after organ removal. Therefore, although the concentration of amiodarone, or any drug, in a single transplanted organ is unknown, it is most likely low enough not to endanger the recipient.²⁹ Similarly, many harmful effects from drugs depend on the length of time the medication has been given. During donor care, the duration of treatment is expected to be brief. Therefore, although this issue has not been studied, the risk to transplant recipients from drugs administered during donor care to treat dysrhythmias is likely to be very low.

Supraventricular Dysrhythmias

Bradycardia. Sinus bradycardia (Figure 1) is encountered during the evolution of brain death, commonly as part of the Cushing reflex (hypertension and

bradycardia). Nonsinus (eg, junctional) bradycardia may represent an “escape” mechanism should the sinus node fail. The initial treatment for both would ordinarily be administration of atropine (0.5-1.0 mg) via the intravenous route. However, if vagal nuclei in the brain have already been compressed, atropine may not have an effect,³⁰ and medications with a direct positive chronotropic effect on the heart must be used. The most effective is isoproterenol (Isuprel, 1.0 μ g intravenously, may be repeated each minute to increase heart rate). Isoproterenol may induce hypotension and ventricular dysrhythmias, and it must be used cautiously. Unfortunately, isoproterenol is commonly nonformulary in many hospitals because it is seldom prescribed. Other agents with positive chronotropic actions include any of the commonly used inotropic agents (ie, dopamine, epinephrine, or dobutamine) in standard doses used for hypotension.

External, transcutaneous pacing may be considered for treating refractory sinus bradycardia, nonsinus bradycardia, or conduction disorders within the heart if chronotropic medications are ineffective.³¹ Rarely, temporary transvenous pacing may be required if medications fail and/or transcutaneous pacing is ineffective. Assistance from a physician is needed for insertion of the pacing wire and initiation of therapy. OPO protocols should guide the sequential treatment of significant bradycardia.

Sinus Tachycardia. Sinus tachycardia (Figure 2) is common because of persistently elevated concentrations of catecholamines in the blood. Tolerance of rapid heart rates depends on donor age, historical or



Figure 3 Atrial fibrillation.

current intrinsic cardiac function, abnormal anatomy or function of the heart valves, and so on. Some donors may tolerate heart rates greater than 130/min without adverse effect, whereas congestive heart failure and hypotension may be precipitated in older donors or in any donor if cardiac function is already compromised. OPO guidelines should specify when treatment of sinus tachycardia should be initiated.

Common causes of sinus tachycardia in patients (ie, pain, anxiety, fear) do not apply to donors. Hyperthyroidism would be rare and would most likely be known beforehand, and fever after brain death is unusual. Hypovolemia, anemia, and high catecholamine levels in circulating blood, however, are common causes of tachycardia among donors and should be excluded by appropriate evaluations. Vasoactive drugs often cause sinus tachycardia and should be reduced to the lowest effective dose.

Esmolol (Brevibloc), a β -receptor blocker, is considered the drug of choice for “harmful” levels of sinus tachycardia. The effects of esmolol begin about 10 minutes after the start of its infusion and are gone 15 to 30 minutes after its infusion is stopped.³² An initial intravenous “loading” dose of 250 to 500 $\mu\text{g}/\text{kg}$ is given for 1 minute, or as quickly as possible while the mean arterial blood pressure is maintained above 65 mm Hg. Thereafter, an intravenous infusion is begun at 25 $\mu\text{g}/\text{kg}$ per minute and titrated to a desirable heart rate as determined by OPO guidelines. The infusion rate should always be less than 300 $\mu\text{g}/\text{kg}$ per minute.³² All β -receptor antagonists, such as esmolol, labetalol, or metoprolol risk hypotension as an important side effect, especially if hypovolemia is present. Bronchospasm may be precipitated in donors with preexisting asthma or bronchospastic chronic bronchitis. β -blockers may also be relatively contraindicated when cardiac contractility is low as indicated by the ejection fraction on an echocardiogram, low blood pressure, or intravascular hemodynamic measurements.

The duration of the effect of β -blockers other than esmolol may be too long. Labetalol is considered relatively short-acting, as repeated small boluses or an infusion,³³ but its effect continues for at least 2 to 4 hours after discontinuation. Most cardiac transplantation

programs prefer that a donor heart not be suppressed by medications during removal and implantation.

Diltiazem, a calcium channel antagonist, can also be used to urgently treat sinus tachycardia.³⁴ A 10-mg bolus is administered slowly and an intravenous infusion of 5 to 15 mg/h is used to titrate the heart rate downward. A higher infusion rate, up to 30 mg/h, was used in a recent study.³⁴ The half-life of a bolus of diltiazem is about 3.4 hours but increases to 4.1 to 4.9 hours when an infusion continues more than 24 hours.

Atrial Fibrillation. The irregularly irregular R-R intervals of this supraventricular dysrhythmia are characteristic (Figure 3). Atrial fibrillation is common because of preexisting or concurrent heart disease or cardiac “stress” factors present after brain death. Other causes, such as pulmonary embolism and hyperthyroidism, are less likely. Atrial fibrillation may cause decreased cardiac output because the atrial contraction at the end of diastole is lost, thus decreasing end-diastolic volume (preload). Formation of a thrombus within the noncontracting atrium could cause embolization to the lung or donated organs but this scenario would be unlikely during the short duration of donor care. Anticoagulation is, therefore, not recommended.

Long-term patient studies, not including donors, have shown little benefit to converting the heart to a normal sinus rhythm if the ventricular rate is controlled.³⁵ The decision to attempt cardioversion or to simply provide medication to control heart rate should be determined by each OPO. If cardioversion to normal sinus rhythm is desired, electrical or pharmacological methods are available. Urgent or emergent electrical cardioversion is indicated when the ventricular response rate during atrial fibrillation is very rapid ($>130/\text{min}$) and causes significant hypotension. The method for cardioversion is described in the guidelines from the American Heart Association.³⁶ Pharmacological conversion and rate control are attempted with several drugs (Table 2). OPO protocols for specific medications should define when and which medications should be used and/or discontinued before organ removal. Use of a heart for transplantation that remains in atrial fibrillation was not reported in any publications we found, but if normal sinus rhythm resumes, perhaps the heart could be reconsidered.

Table 2 Possible medications for pharmacological cardioversion or ventricular rate control in atrial fibrillation^{35,37-41}

| Medication | Dose and method of administration | Comments |
|------------|---|--|
| Esmolol | Same as recommended for sinus tachycardia (see text) | Digoxin recommended if heart failure present. (digoxin 0.50 mg intravenous, then 0.25 mg intravenous in 2 hours) |
| Amiodarone | 150 mg for 30 min; then infuse 1 mg/min for 6 hours, then 0.5 mg/min | Preferred when left ventricular failure present; ~48% success; may cause hypotension, bradycardia, or atrioventricular block; extended half-life |
| Diltiazem | Initial bolus 0.25-0.35 mg/kg; follow with intravenous infusion of 5-15 mg/h titrated to heart rate or conversion | ~94% response; hypotension (13% of patients); intravenous administration of calcium may decrease any hypotension that occurs |
| Ibutilide | 1-mg bolus for 10 min (may repeat once after 10 min) | Use only for cardioversion; does not control heart rate; concerns: prolongs QT interval; torsade de pointes (~4% of cases); ventricular tachycardia in 1.5%-2.6% of cases; less hypotension after administration of ibutilide than after administration of amiodarone; elimination half-life 6 hours |

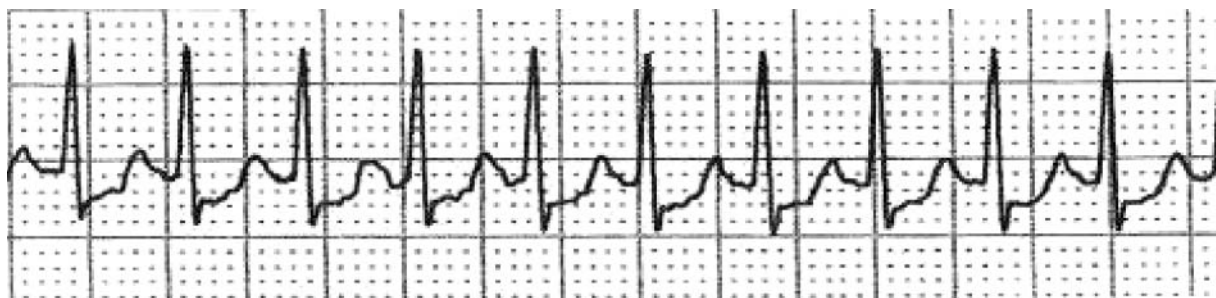


Figure 4 Supraventricular tachycardia.

Atrial Tachycardia. Dysrhythmias that show a narrow QRS complex, a regular R-R interval, and rapid ventricular response (Figure 4) may indicate an atrial or perhaps nodal tachycardia, both often referred to as supraventricular tachycardia.⁴² The rate and hemodynamic response (hypotension) of supraventricular tachycardia dictates the urgency and choice of therapeutic options. Severe hypotension demands emergent synchronized electrical cardioversion.³⁶ Significant but less severe hypotension usually allows pharmaceutical treatment. Administration of 6 mg of adenosine as a rapid intravenous bolus is recommended, followed by a second dose of 12 mg in 2 minutes if the heart has not converted to a sinus rhythm or the ventricular rate has not slowed.⁴³ Administration of diltiazem should follow conversion to sinus rhythm so as to decrease the incidence of recurrent supraventricular tachycardia. The assistance of a physician should be sought when adenosine is administered because of its potential to induce other dysrhythmias. If administration of adenosine is not successful, diltiazem or esmolol should be tried, and if these fail, amiodarone is recommended.⁴³

Other Supraventricular Dysrhythmias. Other rhythm changes during donor care may indicate other atrial or nodal ectopic foci or conduction abnormali-

ties. These causes of rhythm changes appear, from reports in Table 1, to be uncommon. A physician should be consulted to determine the diagnosis and available treatment options.

Ventricular Dysrhythmias

When the QRS complex is wide (>0.12 seconds), either a supraventricular rhythm with aberrant conduction or a ventricle-based dysrhythmia is present. Differentiation between these 2 mechanisms is beyond the scope of this discussion.⁴⁴ Importantly, however, supraventricular foci with aberrant conduction through the heart may not require therapy unless the rhythm is sustained or causes hypotension.⁴⁴

Ventricular dysrhythmias may occur as single beats, groups of 2 or more beats, or as a sustained rhythm at various rates of ventricular contraction, including ventricular tachycardia⁴⁵ (Figure 5). Loss of an organized focus for depolarization may allow "degeneration" of ventricular tachycardia to ventricular fibrillation, wherein the ventricles cease contraction.

Ventricular fibrillation demands immediate electrical defibrillation and initiation of cardiopulmonary resuscitation.³⁶ Amiodarone is recommended at the higher bolus dose of 300 mg intravenously if the ven-

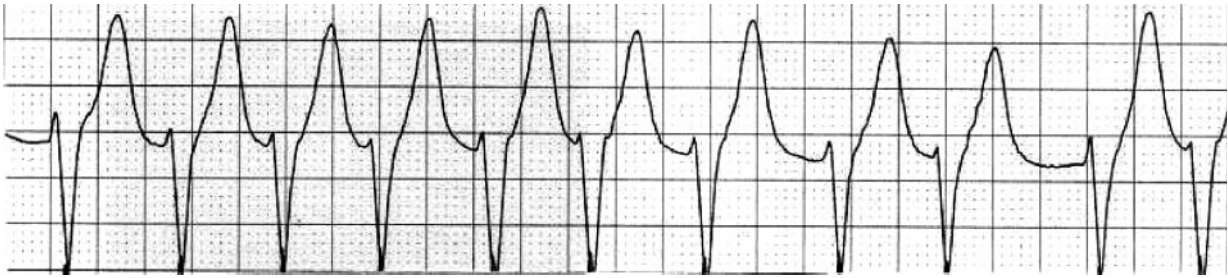


Figure 5 Ventricular tachycardia.

tricular fibrillation is resistant to defibrillation.^{45,46} Treatment of ventricular tachycardia and other ventricular dysrhythmias depends on their rate and hemodynamic effect (hypotension).

Treatment of occasional premature ventricular contractions, groups of such contractions, and nonsustained ventricular tachycardia is considered optional,⁴⁷ although attempts to suppress the latter rhythm with amiodarone may be desirable.⁴⁶ Sustained ventricular tachycardia causing loss of arterial blood pressure should be treated as ventricular fibrillation with full cardiac resuscitation including synchronized cardioversion. When blood pressure is sustained in the presence of recurrent or continuing ventricular tachycardia, use of several drugs, including amiodarone, is recommended in the guidelines of the American Heart Association.⁴³ No studies of sustained or recurrent ventricular tachycardia during donor care were located, but use of amiodarone would seem appropriate until a physician can be consulted.

Summary

Dysrhythmias occurring during donor care appear more likely to occur soon after brain death. With the exception of sinus tachycardia, the incidence of dysrhythmias is relatively low during donor care (15%-20%). The occurrence of cardiac arrest and associated dysrhythmias is also uncommon at 5% to 15%. Avoiding electrolyte changes, hypoxemia, or hypotension is fundamental to preventing dysrhythmias. Although not all cardiac rhythm abnormalities require treatment, associated hypotension caused by the dysrhythmia often forces intervention. In general, short-acting intravenous medications are preferred but may not be available for all rhythm changes. Only the intravenous route is appropriate during donor care. The organ procurement coordinator should be familiar with the guidelines of the American Heart Association, particularly the medications recommended and the adverse effects of medications described therein. OPO protocols should direct appropriate treatments for commonly encountered dysrhythmias. In-house physician support that is immediately available should also be part of OPO support and hospital development. Although uncommon, dysrhythmias may rapidly cause hypotension that risks recovery of all organs.

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References

1. Nygaard CE, Townsend RN, Diamond DL. Organ donor management and organ outcome: a 6-year review from a level I trauma center. *J Trauma*. 1990; 6:728-732.
2. Logigian EL, Ropper AH. Terminal electrocardiographic changes in brain-dead patients. *Neurology*. 1985;35:915-918.
3. Drory Y, Ouaknine G, Kosary IZ, Kellermann JJ. Electrocardiographic findings in brain death: description and presumed mechanism. *Chest*. 1975;67:425-432.
4. Muhlberg J, Wagner W, Rohling R, Link J, Neumayer HH. Hemodynamic and metabolic problems in the preparation for organ donation. *Transplant Proc*. 1986;18:391-393.
5. Ouaknine GE. Cardiac and metabolic alterations in brain death: discussion paper. *Ann NY Acad Sci*. 1978;315:252-256.
6. Dujardin KS, McCully RB, Wijdicks EFM, et al. Myocardial dysfunction associated with brain death: clinical, echocardiographic, and pathologic features. *J Heart Lung Transplant*. 2001;20:350-357.
7. Griep RB, Stinson EB, Clark DA, Dong E, Shumway NE. The cardiac donor. *Surg Gynecol Obstet*. 1971;133:792-798.
8. Dosemeci L, Yilmaz M, Cengiz M, Dora B, Ramazanoglu A. Brain death and donor management in the intensive care unit: experiences over the last 3 years. *Transplant Proc*. 2004; 36:20-21.
9. Shewmon DA. Chronic "brain death": meta-analysis and conceptual consequences. *Neurology*. 1998;51:1538-1545.
10. Powner DJ, Bernstein IM. Extended somatic support for pregnant women after brain death. *Crit Care Med*. 2003;31:1241-1249.
11. Kinoshita Y, Okamoto K, Yahata K, et al. Clinical and pathological changes of the heart in brain death maintained with vasopressin and epinephrine. *Path Res Pract*. 1990;186:173-179.
12. Iwai A, Sakano T, Uenishi M, Sugimoto H, Yoshioka T, Sugimoto T. Effects of vasopressin and catecholamines on the maintenance of circulatory stability in brain-dead patients. *Transplantation*. 1989;48:613-617.
13. Sadowski J, Wierzbicki K, Przybylowski P, et al. Does the episode of cardiopulmonary resuscitation in cardiac donors increase the risk of heart transplantation? *Przegl Lek*. 2003; 60:76-79.
14. Totsuka E, Fung JJ, Urakami A, et al. Influence of donor cardiopulmonary arrest in human liver transplantation: possible role of ischemic preconditioning. *Hepatology*. 2000;31:577-580.
15. Sweeney MS, Lammermeier DE, Frazier OH, Burnett CM, Haupt HM, Duncan JM. Extension of donor criteria in cardiac transplantation: surgical risk versus supply-side economics. *Ann Thorac Surg*. 1990;50:7-11.

16. Delaunay L, Denis V, Darmon PL, Catoire P, Bonnet F. Initial cardiac arrest is a risk factor for failure of organ procurement in brain-dead patients. *Transplant Proc.* 1996;28:2894.
17. Solomon NA, McGiven JR, Alison PM, et al. Changing donor and recipient demographics in a heart transplantation program: influence on early outcome. *Ann Thorac Surg.* 2004;77:2096-2102.
18. de Begona JA, Gundry SR, Razzouk AJ, Boucek MM, Kawauchi M, Bailey LL. Transplantation of hearts after arrest and resuscitation: early and long-term results. *J Thorac Cardiovasc Surg.* 1993;106:1196-1201.
19. Novitzky D, Wicomb WN, Cooper DKC, Rose AG, Fraser RC, Barnard CN. Electrocardiographic, hemodynamic and endocrine changes occurring during experimental brain death in the chacma baboon. *Heart Transplant.* 1984;4:63-69.
20. Shivalkar B, Van Loon J, Wieland W, et al. Variable effects of explosive or gradual increase of intracranial pressure on myocardial structure and function. *Circulation.* 1993;87:230-239.
21. Power BM, Van Heerden PV. The physiological changes associated with brain death: current concepts and implications for treatment of the brain dead organ donor. *Anaesth Intensive Care.* 1995;23:26-36.
22. Novitzky D, Horak A, Cooper DK, Rose AG. Electrocardiographic and histopathologic changes developing during experimental brain death in the baboon. *Transplant Proc.* 1989;21:2567-2569.
23. Black PM. Brain death. *N Engl J Med.* 1978;299:338-344.
24. Smith M. Physiologic changes during brain stem death: lessons for management of the organ donor. *J Heart Lung Transplant.* 2004;23:S217-S222.
25. Powner DJ. Treatment goals during care of adult donors that can influence outcomes of heart transplantation. *Prog Transplant.* 2005;15:226-232.
26. Szabo G. Physiologic changes after brain death. *J Heart Lung Transplant.* 2004;23:S223-S226.
27. Powner DJ, Kellum JA, Darby JM. Abnormalities in fluids, electrolytes, and metabolism of organ donors. *Prog Transplant.* 2000;10:88-96.
28. Powner DJ, Darby JM, Kellum JA. Proposed treatment guidelines for donor care. *Prog Transplant.* 2004;14:16-28.
29. Holt DW, Tucker GT, Jackson PR, et al. Amiodarone pharmacokinetics. *Am Heart J.* 1983;106:840-847.
30. Vaghadia H. Atropine resistance in brain-dead organ donors. *Anesthesiology.* 1986;65:711-712.
31. Trigano JA, Birkui PJ, Mugica J. Noninvasive transcutaneous cardiac pacing: modern instrumentation and new perspectives. *Pacing Clin Electrophysiol.* 1992;15:1937-1943.
32. Wiest D. Esmolol: a review of its therapeutic efficacy and pharmacokinetic characteristics. *Clin Pharmacokinet.* 1995;28:190-202.
33. Harrington C. Managing hypertension in patients with stroke: are you prepared for labetalol infusion? *Crit Care Nurse.* 2003;23:30-38.
34. Gabrielli A, Gallagher TJ, Caruso LJ, Bennett NT, Layon AJ. Diltiazem to treat sinus tachycardia in critically ill patients: a four-year experience. *Crit Care Med.* 2001;29:1874-1879.
35. Page RL. Newly diagnosed atrial fibrillation. *N Engl J Med.* 2004;351:2408-2416.
36. Guidelines 2000 for cardiopulmonary resuscitation and emergency cardiovascular care. Part 6, advanced cardiovascular life support: section 2, defibrillation. *Circulation.* 2000;102(suppl 1):I90-I94.
37. Marik PE, Zaloga GP. The management of atrial fibrillation in the ICU. *J Intensive Care Med.* 2000;15:181-190.
38. Rogers KC, Wolfe DA. Ibutilide: a class III rapidly acting antidysrhythmic for atrial fibrillation or atrial flutter. *J Emerg Med.* 2001;20:67-71.
39. Karth GD, Geppert A, Neunteufl T, et al. Amiodarone versus diltiazem for rate control in critically ill patients with atrial tachyarrhythmias. *Crit Care Med.* 2001;29:1149-1153.
40. Chow M. Intravenous amiodarone: pharmacology, pharmacokinetics and clinical use. *Ann Pharmacother.* 1996;30:637-643.
41. Hughes M, Binning A. Intravenous amiodarone in intensive care: time for a reappraisal? *Intensive Care Med.* 2000; 26:1730-1739.
42. Blomstrom-Lundqvist C, Scheinman MM, Aliot EM, et al. ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias—executive summary. *J Am Coll Cardiol.* 2003;42:1493-1531.
43. Guidelines 2000 for cardiopulmonary resuscitation and emergency cardiovascular care. Part 6, advanced cardiovascular life support: section 5, pharmacology I: agents for arrhythmias. *Circulation.* 2000;102(suppl 1):I112-I128.
44. Hudson KB, Brady WJ, Chan TC, Pollack M, Harrigan RA. Electrocardiographic manifestations: ventricular tachycardia. *J Emerg Med.* 2003;25:303-314.
45. Xavier LC, Kern KB. Cardiopulmonary resuscitation guidelines 2000 update: what's happened since? *Curr Opin Crit Care.* 2003;9:218-221.
46. Sarkozy A, Dorian P. Advances in the acute pharmacologic management of cardiac arrhythmias. *Curr Cardiol Rep.* 2003;5:387-394.
47. Katritsis DG, Camm AJ. Nonsustained ventricular tachycardia: where do we stand? *Eur Heart J.* 2004;25:1093-1099.

CE Test Test ID 4000-J43: Cardiac dysrhythmias during donor care

Learning objectives: 1. Discuss the dysrhythmias that may occur during donor care. 2. Identify pharmacological considerations of managing cardiac dysrhythmias during donor care. 3. Describe treatment strategies to manage specific dysrhythmias that are likely to occur during donor care.

1. What is the incidence of dysrhythmias during donor care?

- a. 10% to 20%
- b. 20% to 30%
- c. 30% to 40%
- d. 40% to 50%

2. Which of the following is the most frequent dysrhythmia experienced by donors?

- a. Sinus bradycardia
- b. Atrial fibrillation
- c. Sinus tachycardia
- d. Premature atrial contractions

3. What percentage of donors experience a cardiac arrest during hospitalization before organ removal?

- a. Less than 5%
- b. 5% to 10%
- c. 10% to 15%
- d. Up to 25%

4. Successful transplantation has occurred during what time frame after arrest?

- a. Less than 3 minutes
- b. 3 to 5 minutes
- c. 5 to 10 minutes
- d. Up to 90 minutes

5. How many half-lives are needed to ensure complete elimination of a drug?

- a. One
- b. Two
- c. Three
- d. Four

6. Which of the following is the most effective medication for the management of nonsinus (eg, junctional) bradycardia?

- a. Atropine
- b. Isuprel
- c. Amniodarone
- d. Epinephrine

7. Which of the following is not a common cause of sinus tachycardia in donors?

- a. Hypovolemia
- b. Anemia
- c. Vasoactive drugs
- d. Fever

8. What agent is considered the drug of choice for harmful levels of sinus tachycardia?

- a. Diltiazem
- b. Labetalol
- c. Esmolol
- d. Inderal

9. Urgent or emergent electrical cardioversion is indicated when atrial fibrillation occurs at what rate in conjunction with hypotension?

- a. Less than 100 beats/min
- b. 100 to 110 beats/min
- c. 110 to 120 beats/min
- d. Greater than 130 beats/min

10. What agent is indicated for the treatment of atrial tachycardia with severe hypotension?

- a. Diltiazem
- b. Labetalol
- c. Esmolol
- d. Adenosine

11. What is the incidence of cardiac arrest and associated dysrhythmias during donor care?

- a. Less than 5%
- b. 5%
- c. 5% to 10%
- d. 5% to 15%

12. A wide QRS complex of what measurement identifies a ventricular-based dysrhythmia?

- a. 0.08 seconds
- b. 0.10 seconds
- c. 0.12 seconds
- d. Greater than 0.12 seconds

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

- | | | | | | | | | | | | |
|-------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|--------------------------------|--------------------------------|--------------------------------|
| 1. <input type="checkbox"/> a | 2. <input type="checkbox"/> a | 3. <input type="checkbox"/> a | 4. <input type="checkbox"/> a | 5. <input type="checkbox"/> a | 6. <input type="checkbox"/> a | 7. <input type="checkbox"/> a | 8. <input type="checkbox"/> a | 9. <input type="checkbox"/> a | 10. <input type="checkbox"/> a | 11. <input type="checkbox"/> a | 12. <input type="checkbox"/> a |
| <input type="checkbox"/> b | <input type="checkbox"/> b | <input type="checkbox"/> b | <input type="checkbox"/> b | <input type="checkbox"/> b | <input type="checkbox"/> b | <input type="checkbox"/> b | <input type="checkbox"/> b | <input type="checkbox"/> b | <input type="checkbox"/> b | <input type="checkbox"/> b | <input type="checkbox"/> b |
| <input type="checkbox"/> c | <input type="checkbox"/> c | <input type="checkbox"/> c | <input type="checkbox"/> c | <input type="checkbox"/> c | <input type="checkbox"/> c | <input type="checkbox"/> c | <input type="checkbox"/> c | <input type="checkbox"/> c | <input type="checkbox"/> c | <input type="checkbox"/> c | <input type="checkbox"/> c |
| <input type="checkbox"/> d | <input type="checkbox"/> d | <input type="checkbox"/> d | <input type="checkbox"/> d | <input type="checkbox"/> d | <input type="checkbox"/> d | <input type="checkbox"/> d | <input type="checkbox"/> d | <input type="checkbox"/> d | <input type="checkbox"/> d | <input type="checkbox"/> d | <input type="checkbox"/> d |

Test ID: 4000-J43 Form expires: March 1, 2008 Contact hours: 1.5 Fee: \$11 Passing score: 9 correct (75%) AACN category: A ABTC category: I
 Test writer: Ruth Kleinpell-Nowell, RN, PhD, CS, CCNS

AMERICAN
ASSOCIATION
of CRITICAL-CARE
NURSES

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Program evaluation

| | Yes | No |
|--|--------------------------|--------------------------|
| Objective 1 was met | <input type="checkbox"/> | <input type="checkbox"/> |
| Objective 2 was met | <input type="checkbox"/> | <input type="checkbox"/> |
| Objective 3 was met | <input type="checkbox"/> | <input type="checkbox"/> |
| Content was relevant to my nursing practice | <input type="checkbox"/> | <input type="checkbox"/> |
| My expectations were met | <input type="checkbox"/> | <input type="checkbox"/> |
| This method of CE is effective for this content | <input type="checkbox"/> | <input type="checkbox"/> |
| The level of difficulty of this test was: | | |
| <input type="checkbox"/> easy <input type="checkbox"/> medium <input type="checkbox"/> difficult | | |
| To complete this program, it took me _____ hours/minutes. | | |

Name _____
 Address _____
 City _____ State _____ ZIP _____
 Social Security No. _____ Phone () _____
 If applicable: State(s) of licensure _____
 License number(s) _____
 ABTC certification number _____
 CPTC, expiration _____
 CCTC, expiration _____
 I would like to receive my certificate via e-mail.
 E-mail address: _____