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I Swear It Will Only Take 15 Minutes: Anesthesia/Sedation for TEE/Cardioversion

Julia Metzner, M.D.
University of Washington, Seattle, WA

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Stem Case and Key Questions Content

You are the weekend on-call anesthesiologist getting ready for an unusually busy schedule, when the cardiology fellow calls to ask for your assistance with a Transesophageal Echocardiogram (TEE)/ Cardioversion, he is hoping to get it started before the scheduled surgeries and says it will only take 15 minutes.

1. What are your options for where and how you can provide care for this patient? Will it actually take only 15 minutes?

This 45 year old, 72 Kg male patient was admitted from an outside hospital with atrial fibrillation and an acute exacerbation of chronic congestive heart failure. He has a prior diagnosis of restrictive cardiomyopathy of unknown etiology resulting in Heart Failure with Normal Ejection Fraction (HFNEF), however the most recent Trans-Thoracic Echocardiography (TTE) at the outside hospital indicated that the patient's ejection fraction is 15%. When you go to the ICU to see the patient you note that he is jaundiced, dyspneic after speaking one or two sentences, has an SpO2 of 96% with nasal cannula, and has a PA catheter in place.
His past medical history is remarkable for HFNEF and Depression.
Medications: Sertraline, Coumadin (last taken 24 hours ago), Enalapril, Carvedilol, Furosemide.
Receiving infusions of: IV Dobutamine 3 mcg/kg/min and IV Nesiritide 0.01mcg/kg/min.
His vital signs are: pulse irregular, 120 bpm; BP 104/55; respirations 16/min; SpO2 96% on 30% oxygen
Labs: Hct 31%; Bilirubin 8.2 mg/dl (normal 0.3-1 mg/dl); PT 21.6; INR 2.0; PTT 52; Plts 98.
Chest X-ray: Moderate cardiac enlargement, an Implantable Cardioverter Defibrillator (AICD) in situ. Bilateral mild diffuse lung disease with left lower lung consolidation indicating atelectasis or aspiration.
Right heart catheterization results: RA Mean: 17 mmHg, RV: 59/17 mmHg, PA: 59/41/47 mmHg, Wedge (PCWP): 23 mmHg Fick Cardiac Output: 3.6 liters/minute Cardiac Index: 1.7 liters/minute/M2 Pulmonary artery saturation: 47%

2. Is this patient healthy enough for sedation or anesthesia for a TEE? Does he have any contraindications to TEE? What will the TEE provide that a TTE will not? Would more specific and comprehensive information on his anticoagulation therapy change your thinking on these issues?

3. What would you expect his pulmonary artery saturation to be if he was not acutely ill? How would this differ from venous oxygen saturation if it were measured in the SVC or right atrium?

4. What is nesiritide?

5. Does the presence of the AICD need to be factored in the plans for cardioversion? Could you use the AICD for cardioversion?
After debating the need for the TEE with the cardiology fellow, you are approaching the patient to discuss your anesthetic plan. During the discussion, the wife says that she has heard that the TEE will help understand what the cause of the heart failure is and asks you what does "diastolic heart failure" mean.

6. How would you explain diastolic dysfunction/diastolic heart failure/HFNEF to her without using the term lusitropy?

7. What is your anesthetic plan and does the possible presence of diastolic heart failure specifically influence this plan? Would the presence of GERD affect your plan, what about obesity and potential difficult ventilation and laryngoscopy?
After weighing the potential risks and benefits of various sedation regiments you decide on dexmedetomidine sedation.

8. What are the likely hemodynamic effects of dexmedetomidine? What infusion dose would you use?
The dexmedetomidine is started and the patient's oropharynx is sprayed with local anesthetic; during the scope insertion the patient gags, chokes, and does not tolerate the scope insertion. The scope is removed and the oropharynx is sprayed again. After a bolus of etomidate 10 mg the scope is inserted successfully, but with nasal prongs in place the patient's saturation drops to 80%. The scope is removed and with bag/mask ventilation the saturation returns to the mid 90s.

9. What are your options now for restarting this TEE?
During the TEE some blood is noted around the bite block and is suctioned. After the scope is removed the patient requires further suctioning and coughs up some blood. The patient is successfully cardioverted to sinus rhythm. Eventually the patient returns to the ICU with SPO2 in the low 90s on a regular face mask. 30 minutes later, you receive a stat page to the ICU. The patient appears cyanotic and SPO2 on a non-rebreather mask is 85%. An ABG shows pH, 7.42; PO2, 128; PCO2, 34.

10. What is your differential diagnosis?

Model Discussion Content
Preprocedural Considerations
Transesophageal Echo(TEE)/cardioversion is a routine procedure and might be performed in the ICU or the PACU and one has to weigh the risk and trouble of transporting the patient against the potential suboptimal nature of the environment and staffing for recovery. The most likely barrier to providing care in the ICU is the staffing ratio for recovery. The options range from minimal sedation with local anesthesia to the oropharynx, deep propofol sedation all the

way through to endo-tracheal intubation, with patient considerations being the obvious factor in helping to decide which technique is appropriate. A study of sedation for TEE revealed a range of 6-45 minutes of TEE time with a few extra minutes for cardioversion and recovery [1]. Most likely the major time drain will be coordinating the presence of all the personnel involved. The situation described raises challenging issues because nobody is supposed to know more about cardiac health than the cardiologists and if they say this patient needs a TEE then the temptation is to think that they have also thought about the safety of anesthesia for TEE in this situation relative to other management options. After all, they are often the source of notes saying “This patient is cleared for surgery and anesthesia; avoid hypoxemia, hypotension and hypercarbia.” If you don’t have a good understanding of TEE in this situation then you won’t be in a position to start the conversation with the cardiologist about options. In addition to potential difficulty in tolerating the sedation or anesthesia that is needed for a comfortable TEE, specific contraindications to consider are: conditions such as esophageal diverticulum, large hiatal hernia, recent esophageal or gastric surgery, esophageal varices, a history of odynophagia or dysphagia, cervical arthritis, a history of mediastinal irradiation, oropharyngeal deformities, and severe coagulopathy [2]. The incidence of complications associated with TEE, vary according to the definition of complication and the study cited, with one large study indicating an incidence of less than 0.2% for major complications while another generated a higher incidence of major upper GI complications of 1.2% [3,4]. The complications range from brief episodes of desaturation and sore throat all the way to life threatening retropharyngeal hematoma and Mallory-Weiss tear with major hemorrhage [5]. If you are doing a TEE for intraoperative management and decision-making for cardiac anesthesia then it is clear that it is your responsibility to assess the patient for contraindications, but in this situation is it your responsibility to double check the cardiologist’s work? The patient is jaundiced and the likely cause with severe heart failure is congestive hepatopathy but with hepatic pathology the possibility of esophageal varices is raised. The other question is whether this coagulation profile constitutes a severe coagulopathy with its abnormalities in platelets, intrinsic and extrinsic pathways. Both the possibility of esophageal pathology and whether the degree of coagulopathy poses a hazard to this patient for TEE should be addressed. A discussion of the value of TEE when compared to TTE must be framed in terms of the patient specific clinical circumstances and goals of the assessment. For example, for patients with an unknown source of stroke, TEE reveals the potential source in approximately 40% of patients where TTE failed [6]. In the specific situation of reducing the risk of stroke with cardioversion, TEE is more effective in visualizing thrombi in the left atrial appendage. The incremental increase in diagnostic accuracy of TEE versus TTE for types of mitral valve lesions is less conclusive as studies fall on both sides [7,8]. For diagnosis of infective endocarditis TEE is superior to TTE because of better image quality, particularly when vegetations are small [9]. For our patient, if the anticoagulation has been adequate for three weeks (INR >2) and optimal TTE imaging can be obtained for assessment of overall ventricular function or to assign a new diagnosis other than worsening HF due to atrial fibrillation, then there may not be much of an incremental advantage to adding TEE in this situation. This should at least be discussed with cardiology team, particularly if you have significant concerns regarding the safety of the sedation/anesthesia for TEE.

Mixed Venous Oxygen Saturation (SvO2) measurement in patients with CHF
SvO2 measurement is an accepted method for monitoring patients with CHF. The normal SvO2 is = 75%, a 5-10% increase or decrease being considered significant. The central venous oxygen saturation-ScvO2 measures the hemoglobin saturation of blood in the SVC and is lesser than SvO2 by about 2-5%. This small difference is normal, and explained by the following: the

CVC tip resides in the SVC, which collects blood from the brain and upper part of the body. The PAC sampling site is in the pulmonary artery (PA), which collects mixed venous blood that drains from the whole body, including SVC and IVC. The blood that drains from the lower part of the body into the IVC has a higher venous saturation level than the blood in the SVC, mostly due to highly saturated (92%) renal venous blood. Since the RA receives blood from both, the difference between the measured SvO2-ScvO2 at this level should be minimal [10].

New Therapeutic Options in CHF
Nesiritide (Natrecor) is a recombinant form of human brain natriuretic peptide (BNP), which is secreted by the ventricles in response to increased cardiac volume and pressure overload. Although does not affect cardiac contractility, improves the symptoms of venous congestion by acting as a powerful arterial-venous vasodilator. Nesiritide has been approved by the FDA for use in the short-term treatment of patients with acutely decompensated heart failure characterized by dyspnea at rest or by clinical evidence of fluid overload (NYHA IV). Nesiritide is available only in parenteral form. The recommended dosage of nesiritide is a 2 mcg/kg IV bolus, followed by an infusion of 0.01-0.03 mcg/kg/minute. The effect starts @ 15 min, the t ½ being 20 min, and the drug is cleared from plasma within 2 hours of stopping the IV infusion. Hemodynamic effects: decreases RAP, PCWP, PAP, MAP, and SVR; produces diuresis and effectively improves the symptoms of congestion. However, may cause symptomatic hypotension, which may necessitate prompt cessation of infusion or initiation of inotropic support, e.g. dobutamine. It helps reset the renin-angiotensin-aldosterone system (RAAS) in the setting of acute CHF. In its action as a vasodilator, nesiritide may potentiate an anesthetic agent's tendency to decrease SVR. Patients on nesiritide should have invasive hemodynamic monitoring to assure maintenance of adequate blood pressure during infusions [11].

Considerations for Cardioversion in Patients with Automatic Implantable Cardioverter Defibrillator(AICD)
There are many case reports from the 1970s and 80s describing damage to pacemaker systems after external cardioversion and a study from the 1990s systematically assessing pacemaker function after external cardioversion indicates an incidence of transient (up to 30 min) loss of capture in 50% of patients, a six-fold overall increase of ventricular pacing thresholds, sensing failure in 41% of patients, and three cases of pacemaker malfunction of which two required generator replacement [12,13]. The most recent study on this topic did not reveal any damage to the more modern devices using biphasic shocks and an antero-posterior defibrillator lead placement, and >8 cm distance for lead placement from the device [14]. AICDs have been used to cardiovert atrial fibrillation, however, even though lower electrical energy can be used, there is still significant discomfort associated with AICD discharge. The use of the AICD with sedation may be most worth considering in obese patients where transthoracic resistance will be higher, perhaps making external cardioversion less successful [15].

Diastolic Heart Failure
Although diastolic heart failure (DHF) as a clinical subset of the syndrome of heart failure has been recognized for 70 years, it seems to be more difficult to conceptualize than systolic heart failure (SHF) and DHF has received increasing attention in recent years [16]. Perhaps the easiest way to understand diastolic dysfunction is to conceive of the heart as a double action pump, both contraction and relaxation are energy dependent and active. Imagine the action of your cheeks working both when blowing out and sucking in; diastolic activity creates active suction to improve early diastolic filling and is not a simple passive return to a resting state. The

active suction pumping fails with diastolic heart failure (DHF) to a greater degree than the myocardial shortening during contraction. Higher pressure is required for filling, more of the filling occurs later in diastole; more time is required for filling and filling is much more dependent on atrial contraction. The stimuli and the signals that ultimately produce the DHF phenotype of chronic heart failure remain largely unknown, as the neurohormonal abnormalities in DHF and SHF appear to be similar. The management of acute decompensated DHF is aimed toward a reduction in pulmonary congestion and correction of any precipitating factors, such as hypertensive crises, myocardial ischemia, acute rhythm disturbances, and sepsis. Specific treatment of precipitating factors should be considered: vasodilators for hypertensive crisis, coronary revascularization, restoration of sinus rhythm, and hemodynamic optimization in septic shock [17, 18]. To a large extent none of these interventions are specifically different from considerations for the treatment of systolic heart failure; however, there is a suggestion that individuals with DHF are likely to be more susceptible to decompensation with over-diuresis. Another consideration is the possibility that inotropic agents may not provide as beneficial a response in DHF, particularly if they lead to an increase in heart rate, with levosimendan being an exception acting as it does a calcium sensitizer and improving contractility without impairing diastolic relaxation [19]. The use of CPAP by mask has been specifically investigated and found to be helpful in the treatment of DHF [20].

Anesthetic Management

The effect of anesthetic agents on LV diastolic dysfunction is not well known. Anesthesiologists involved in the perioperative care of patients with cardiovascular disorders routinely manage these patients appropriately, perhaps without realizing the stages of diastolic dysfunction. Considering serious lack in the literature, it would be hard to argue against choices as continuous infusion of midazolam, propofol or dexmedetomidine (dex). Regardless of the chosen regimen, careful titration to the desired effect and continuous hemodynamic ad respiratory monitoring is strongly advised. It is important to realize that the hemodynamic effects of dex depend significantly on the dosing regimen and whether a bolus dose is employed before the maintenance infusion. Based on a study performed by Ebert et al [21] in healthy volunteers using a target-controlled infusion system to provide increasing concentrations of dex (0.7 to 15 ng/mL; for reference: a bolus of 1 µg/kg will result in a plasma concentration of 0.9 ng/ml), at low concentrations there is a decrease in MAP (13%) followed by progressive increase (12%) but no changes in CVP, PAP, SVR and PVR have been observed. Increasing concentrations produced progressive decreases in HR (maximum 29%) and CO (35%) and did increase all other hemodynamic variables, including MAP, PAP, SVR/PVR. These findings suggest, that low doses of dex, in the range of 0.2-0.7 µg/kg/ hour should be chosen in order to preserve hemodynamic stability in this severely compromised patient. The hemodynamic effects of a bolus of dex have shown a biphasic response. An acute and rapid (over 10 min) IV injection of 1 µg/kg will initially and transiently increase BP by approx 15-20% and decrease heart rate by approx 30%. This initial increase in BP is probably due to the vasoconstrictive effects of dex when stimulating peripheral alpha-2 receptors. Heart rate returns to baseline by 15 minutes, but BP gradually declines to approx 15% below baseline by 1 hour. To attenuate these effects, bolusing should be omitted or its dose reduced to 0.5 µg/kg and administered over 20-30 minutes. The use of dex infusions to assist on TEE examination has been recently described (1 µg/kg bolus over 15 min, followed by 0.2 µg/kg/hour infusion) and showed better hemodynamic variables and improved patient satisfaction than with benzodiazepine and narcotics alone, with no superimposed respiratory depression [22].

TEE-related Complications
Depending on the type of sedation used, desaturation to <80% has been documented to occur in between 13-20% of TEE examinations [23]. At the point of scope insertion if a large bolus of anesthetic is administered the patient may become apneic and with or without bolus anesthetic administration may develop obstruction or laryngospasm. In a patient with marginal FRC there may not be adequate O2 reserves to avoid hypoxemia before obstruction or apnea resolves. If the problem is simply hypopnea and not complete apnea then increasing FIO2 could alleviate the problem. The presence of the scope of course makes it more difficult to deliver a higher FIO2. There are purpose made commercial masks with a diaphragm/orifice for insertion of a TEE probe or endoscope. Tse et al describe a technique that involves using a plastic bag taped over the patient's head and draped over the face to increase FIO2 under the drape [24]. (www.TSEmask.com) A non-rebreather mask can also be modified by cutting a large opening in it and creating a tegaderm diaphragm to allow passage of the scope. Attempting to reinsert the scope with maximum pre-oxygenation, avoidance of prolonged apnea or hypopnea, and maintaining higher FIO2 post insertion may overcome the problem. Although it may seem too crowded in the oropharynx for this solution to work some clinicians have documented the successful use of deep propofol sedation and LMA placement with TEE, both as a primary plan and rescue [23].Without the ABG results or if we assume the ABG results are in error then the likely cause of the cyanosis and low O2 saturation would be hypoxemic respiratory failure due to aspiration, pulmonary edema, or worsening atelectasis. If the ABG results are accurate then with a PaO2 of 128 the O2 saturation should, of course, be 100. Standard pulse oximetry measures the relative absorbance of 2 wavelengths (660 nm and 940 nm) of light to differentiate oxyhemoglobin from deoxyhemoglobin; however, methemoglobin absorbs both of these wavelengths equally, such that at high levels of methemoglobin, the pulse oximeter reads a saturation of 85%. If co-oximetry is available this could confirm the diagnosis.

Methemoglobinemia becomes a consideration if benzocaine was the spray used for the topical anesthesia of the oropharynx. Another possible cause of methemoglobinemia that would need to be considered in this setting is the administration of nitrates (nitroglycerine or nitroprusside [25] Methylene blue is the treatment for symptomatic treatment of methemoglobinemia as it is metabolized to form a reducing agent that facilitates the reduction of methemoglobin. Patients with methemoglobin levels of 20% to 30% or higher should receive IV methylene blue at a dose of 1 to 2 mg/kg over 5 minutes. Patients with a deficiency of G6PD should not receive methylene blue because this can result in hemolysis. In our patient, a history of SRI anti-depressant therapy complicates matters as there is a risk for serotonin toxicity (clonus, hyperreflexia, pyrexia and altered mental state) when methylene blue is administered to patients taking SRI therapy [26,27]. This interaction appears to be related MAO inhibition activity that methylene blue has only been relatively recently recognized to possess. Other situations an anesthesia provider might encounter the use of methylene blue are localization of parathyroid glands and treatment resistant vasoplegia, particularly post cardiopulmonary bypass [28].

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