Case 2018-5
A patient with complete heart block and pacemaker implant presented for routine CABG. Post-induction placement of a pulmonary artery catheter resulted in bradycardia, hypotension, asystole and ultimately Vfib. Emergency sternotomy was performed and the patient was placed on cardiopulmonary bypass. CABG was performed and the patient survived but with mild anoxic brain damage. Investigation revealed that the pacemaker electrode had been dislodged, probably by the pulmonary artery catheter. The electrode had not been securely attached to the myocardium. The electrodes had been inserted about a month prior to surgery. These electrodes were designed to lay flat against the myocardium relying on pannus growth to slowly fix them to the myocardium.

Discussion
This case presents an opportunity to discuss the intraoperative loss of pacemaker function. While this scenario occurred in the setting of planned cardiac surgery, there are issues pertinent to the general anesthesiologist, including care of the pacemaker-dependent patient and the risks of central line placement in the presence of a new device. In this case, the patient’s life was saved by the swift response of the team in instituting emergency cardiopulmonary bypass; however, the patient still sustained harm in the form of mild anoxic brain damage. In the general O.R. setting where cardiopulmonary bypass is not readily available as a rescue therapy, the need for adequate preparation in the care of these patients is even greater.

Complete heart block (CHB), also known as third-degree atrioventricular (AV) block, is a severe bradyarrhythmic disorder that results in the inability of electrical impulses to travel from the atria to the ventricles due to lack of conduction through the AV node. This condition may result from myriad causes, including ischemic heart disease and fibrosis of the conduction system, and can also occur after procedures such as cardiac valve replacement or implantation, ventricular septal defect closure and septal ablation for hypertrophic cardiomyopathy. CHB may be deliberately induced with AV nodal ablation in situations such as persistent atrial fibrillation with an uncontrollable rapid ventricular response.

The electrocardiogram demonstrates atrial activity (p waves) and ventricular activity (QRS complexes) that occur independently of one another with an atrial rate that is typically faster than the ventricular rate. CHB may be hemodynamically stable or unstable depending on the level of the block and the location of the escape rhythm, which can occur at the level of the AV node or bundle of His (narrow QRS and higher escape rates of 40-60 bpm) or below the bundle of His (wider QRS and rates of 40 bpm or less).
However, ventricular escape rhythms can become unreliable even in what initially appears to be hemodynamically stable CHB. Thus, CHB represents a Class I indication for permanent pacemaker placement and is indicated even when the patient is asymptomatic, particularly when the escape rhythm is below the AV node and less than 40 bpm or when there is documented asystole greater than or equal to 3.0 seconds. In this case, it appears that the patient was completely pacemaker-dependent due to the hemodynamic instability that occurred after loss of lead contact.

A comprehensive discussion of the perioperative management of cardiac rhythm devices is beyond the scope of this article. However, the Heart Rhythm Society and ASA have published guidelines on this topic from which a few general points regarding the care of the pacemaker-dependent patient will be emphasized. Monopolar electrocautery may be erroneously detected by the pacemaker as native patient electrical activity (oversensing) and can result in pacemaker inhibition. The risk of pacemaker inhibition is higher in procedures above the umbilicus and of greater consequence in the pacemaker-dependent patient, who may experience significant bradycardia or asystole with prolonged electromagnetic interference. Application of a magnet to a permanent pacemaker generally results in closure of a magnetic switch that turns off sensing and initiates asynchronous pacing. While procedures below the umbilicus may be managed expectantly with availability of a magnet if clinically significant oversensing occurs, surgery above the umbilicus may require pacer inhibition during electrocautery use with a regular magnet or sterile magnet if the pacer is within the operative field. Otherwise, lack of access to the pacemaker or lack of patient tolerability of the magnet-associated rate may mandate perioperative device reprogramming. Alternatively, use of short electrocautery bursts limited to 4-5 seconds each may be well tolerated even by pacemaker-dependent patients and presents another strategy for perioperative management. Of note, bipolar electrocautery does not cause electromagnetic interference. In addition, while it is generally assumed that a magnet will not initiate asynchronous pacing when a pacemaker is associated with an implantable cardioverter-defibrillator (ICD), the release of new devices and individual patient device reprogramming may result in a large variability of pacemaker-ICD response to a magnet.

All patients must have heart rate monitoring that allows for the identification of a pulse (the true perfused heart rate) either by pulse oximetry or intraarterial pressure monitoring. In addition, chemical or transcutaneous methods of rescue should be available, particularly if the patient is high risk. In this case, despite the fact that the patient was known to have complete heart block with a planned intravascular access procedure that could interact with the pacemaker wires, it appears that pacer pads were not placed on the patient and that transcutaneous pacing was not attempted. Transcutaneous pacing could have provided immediate heart rate support and temporized the situation until a more durable solution (i.e., transvenous pacer) could be obtained. In the absence of pacer pads, atropine (0.5 mg q3-5 min to a total of 3 mg) should be administered to decrease vagal tone, although the adequacy of response may depend on the level of conduction block. In addition, chronotropic β1 agonists such as dopamine (3-20 mcg/kg/min), dobutamine (5-20 mcg/kg/min) or epinephrine can be administered as well.

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No mention was made of the presence or absence of an interrogation report, but lack of a device report could represent a systems issue that may have contributed to this case. Patients undergoing elective surgery should have a device check within the past 12 months (within six months with ICD) identifying important features such as type of device, manufacturer, model, indication for device, battery age, programming mode, pacemaker dependence, underlying rhythm, magnet response, and lead alerts and age (particularly if less than 3-12 months old). The age of the leads is important information as physical lead dislodgement is possible during placement of a central venous catheter (CVC) and is thought to be more likely in the setting of new leads. The presence of such a report would have alerted the team to the date of pacemaker placement or whether there were new leads in the presence of a previously placed generator, a much less obvious situation. This knowledge may have allowed for a better risk/benefit assessment of placing a pulmonary artery catheter (PAC) and may have triggered a more robust back-up plan if a PAC was deemed crucial to the care of the patient. Other red flags aside from lead age include documentation of difficulty in lead placement or need for multiple revisions due to lead migration. Use of alternative forms of cardiac output assessment such as pulse contour analysis (FloTrac) or impedance (Cheetah) devices could have obviated the need for a PAC.

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There is very little data on the impact of CVC or PAC placement on rates of pacer lead dislodgement. Baseline rates of lead dislodgement are suggested by one study of cardiac-resynchronization therapy (which involves the placement of leads in the right atrium, right ventricle and coronary sinus for left ventricular pacing), which reported that 7.6 percent of patients had lead dislodgement at six months, with 2.9 percent involving the left ventricular lead.\(^4\) Left ventricular lead dislodgement, in particular, has been reported to be as high as 6-8 percent at six months.\(^5\) When a CVC without a PAC is indicated in the patient with new pacer leads, placement in the internal jugular or subclavian location contralateral to the origin of the leads with avoidance of unnecessary advancement of the guidewire to avoid contact with the pacemaker wires may be helpful (e.g., right internal jugular or subclavian CVC with left subclavian origin leads, left internal jugular or subclavian CVC with right subclavian origin leads). Alternatively, placement of the line in the femoral location may completely avoid the possibility of lead dislodgement.\(^6\) The safety of high-risk PAC placements can be augmented with fluoroscopy as well as the availability of personnel and materials allowing for emergent transvenous pacer placement. Finally, despite successful placement of a PAC, removal of the PAC may still result in disturbance of the leads. If there is any question of the possibility of lead dislodgement, a chest X-ray or ultimately device interrogation may provide reassurance of pacer lead integrity.

In summary, this case illustrates the high degree of caution that is necessary in the care of the pacemaker-dependent patient. An adequate back-up plan in the event of loss of pacing by chemical or electrical means should be in place, particularly if there is a risk of lead dislodgement due to the need for a CVC or PAC.

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References
2. Crossley GH, Poole JE, Rozner MA, et al. The Heart Rhythm Society (HRS)/American Society of Anesthesiologists (ASA) Expert Consensus Statement on the perioperative management of patients with implantable defibrillators, pacemakers and arrhythmia monitors: facilities and patient management: executive summary this document was developed as a joint project with the American Society of Anesthesiologists (ASA), and in collaboration with the American Heart Association (AHA), and the Society of Thoracic Surgeons (STS). *Heart Rhythm*. 2011;8(7):e1-e18.