

Sedation in the Electrophysiology Laboratory: A Multidisciplinary Review

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Approaches to sedation in the electrophysiology (EP) laboratory range from intravenous sedation techniques to general anesthesia (GA) (Table 1¹). Although the provision of GA requires a dedicated anesthesia provider, controversy exists as to whether intravenous sedation in this complex patient population can or should be undertaken by a proceduralist-directed nurse-administered (PDNA) model. In a recent survey of 38 national academic centers, electrophysiologists noted that in 26.3% of EP cases, an anesthesiologist is warranted most of the time or always due to patient complexity,² yet there is also ample literature espousing the safety and benefits of PDNA sedation for these cases.^{3–7} Moreover, intended sedation plans may require modification over the course of a procedure. A 2-year review of 208 adults undergoing catheter ablation (including for supraventricular tachycardia, atrial tachycardia, atrial flutter, premature ventricular contraction, and ventricular tachycardia) using a variety of sedation strategies demonstrated that of the 186 non-GA cases, 20 required conversion to GA and 54 needed additional airway interventions.⁸

This article provides a comprehensive interdisciplinary review of sedation practices for EP procedures. It examines different care models (dedicated anesthesiology team, ad hoc anesthesiology, PDNA approach), discusses evidence for and against various sedation practices based on procedure type, reviews existing sedation guidelines, and offers practical recommendations for the clinical electrophysiologist.

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EP Procedures—Evidence for Sedation Practices

Atrial Fibrillation Ablation

Catheter ablation of atrial fibrillation (AF) is increasingly prevalent and in select patients may be offered as first-line therapy.⁹ Because these procedures are lengthy and necessitate patient immobility at certain critical times, deep sedation or GA is typically required. Among Heart Rhythm Society/European Heart Rhythm Association expert consensus statement task force members, ≈50% reported using GA for all AF cases.¹⁰ Although an approach that both prevents inadequate sedation (a common shortcoming of benzodiazepine-opiate regimens) and the adverse hemodynamic and respiratory effects of deep sedation has been sought, no such ideal regimen exists. Several sedation strategies for AF ablation have been studied and are summarized below.

Propofol versus opiate-benzodiazepine in AF ablation

A 2007 trial by Tang et al¹¹ prospectively randomized 120 patients undergoing AF ablation to 2 sedation strategies, both using a PDNA model: propofol infusion targeting deep-sedation versus a combination of midazolam–fentanyl for conscious sedation. In the propofol group, 2 patients required positive pressure ventilation (PPV) for persistent desaturation compared with 1 patient in the midazolam–fentanyl group. Additionally, 1 of the 2 propofol group patients suffered a cardiac arrest and required intubation and defibrillation. The proceduralists were less satisfied with propofol, primarily because of catheter instability from airway obstruction and delays in managing sedation-related complications. Though small, this trial is noteworthy because it was one of the first to prospectively compare 2 fundamentally different sedation techniques for AF ablation and report key associated outcomes. In 2011, Kottkamp et al⁶ performed a prospective observational study of 650 patients undergoing AF ablation under deep sedation using midazolam and fentanyl followed by a propofol infusion; a PDNA model was used for sedation administration. Overall, 2.3% of the subjects developed significant hypotension, 15% required vasoactive drug administration, and 1.5% developed significant hypoxia. Moreover,

Table 1. Spectrum of Analgesia to Anesthesia

	Minimal Sedation (Anxiolysis)	Moderate Sedation (Conscious Sedation)	Deep Sedation	General Anesthesia
Responsiveness	Normal response to verbal stimulation	Purposeful response to verbal or tactile stimulation	Purposeful response after repeated or painful stimulation	Unarousable even with painful stimulus
Airway	Unaffected	No intervention required	Intervention may be required	Intervention often required
Spontaneous ventilation	Unaffected	Adequate	May be adequate	Frequently inadequate
Cardiovascular function	Unaffected	Usually maintained	Usually maintained	May be impaired

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1.2% required PPV but no patients required intubation or the assistance of an anesthesia provider. The authors concluded that PDNA deep sedation for AF ablation is safe and practical.

An observational study by Salukhe et al⁴ comprised 1000 consecutive patients undergoing AF ablation via propofol infusion with the intent of achieving deep sedation; fentanyl and midazolam were administered as adjunctive agents. The authors did not list any exclusion criteria. There were no premature procedure terminations related to sedation, but adverse sedation-related events were common and managed by cessation of propofol in nearly all cases. Cessation of the propofol infusion was required in 15.6% of their patients (13.6% for hypotension, 1.9% for respiratory depression, and 0.1% for hypersalivation). In the 19 patients with respiratory depression, SaO₂ recovered after cessation of propofol except in 1 patient who required 4 minutes of PPV. Lastly, Wutzler et al¹² reported on 401 patients stratified by age (≤ 50 years, 51–74 years, and ≥ 75 years) to PDNA deep sedation with the use of propofol, midazolam, and piritramide. There was no difference in intraprocedural or sedation complications except for significantly greater rates of hypotension in the oldest group. There was also a higher incidence (0.0% versus 2.2 versus 9.4%, $P=0.004$) of postprocedure adverse events such as respiratory infection, renal failure, and delirium among the elderly. One patient required PPV for prolonged hypoxia following a contrast reaction. Though the authors concluded that even in elderly patients (≥ 75 years-old) PDNA deep sedation using propofol and other agents is safe for AF ablation, their findings suggest that the elderly may not uniformly tolerate a PDNA strategy and may be more prone to a variety of sedation-related complications.

See Table 2 for a summary of EP sedation strategies.

Dexmedetomidine in AF ablation

Dexmedetomidine, an α -2 adrenergic agonist with a short distribution half-life (6 minutes), provides sedation and analgesia with fewer respiratory depressant effects compared to other agents. Side effects of this medication include bradycardia, cardiac conduction abnormalities, and hypotension.

Two recently published prospective trials examined the use of dexmedetomidine for AF ablation. The first, by Cho et al,¹³ randomized 90 patients to receive either midazolam and remifentanyl versus dexmedetomidine and remifentanyl. Moderate-to-deep sedation was targeted and administered by an anesthesiologist. There were significantly higher rates of desaturation in the midazolam–remifentanyl group versus the dexmedetomidine–remifentanyl group (2.2% versus 33.3%, $P<0.001$). The proceduralists, who were blinded to the sedation regimen, were more satisfied ($P<0.001$) with dexmedetomidine–remifentanyl, which achieved a deeper level of sedation despite the use of one third less remifentanyl. However, there was significantly more hypotension at 60 and 120 minutes in the dexmedetomidine–remifentanyl group. The second trial by Sairaku et al⁵ randomized 87 patients to dexmedetomidine with rescue thiamylal (a barbiturate) versus thiamylal only targeting moderate sedation using a PDNA model; both groups also received pentazocine (an opioid analgesic). The incidence of hypotension and bradycardia were similar between groups. However, the dexmedetomidine group experienced fewer apneic events ($P<0.0001$), fewer lower body movements ($P=0.0098$), and desaturations ($P=0.049$).

GA compared to sedation in AF ablation

A prospective trial by Di Base et al¹⁴ compared sedation with fentanyl or midazolam ($n=128$) versus GA ($n=129$). The authors did not state who provided sedation services in the fentanyl–midazolam group. The procedure time was shorter in the GA group (2.4 ± 1.4 versus 3.6 ± 1.1 hours, $P<0.001$) and more patients were arrhythmia free at follow-up in the GA group (88% versus 69%, $P<0.001$). The benefit of GA was attributed to increased catheter stability with consistent, regular thoracic excursions, and less patient movement. However, in a second randomized trial, when subjects were examined with capsule endoscopy, the same authors found a higher incidence of esophageal injury with GA compared to conscious sedation (48% versus 4%, $P<0.001$). This associated finding with GA was ascribed to reduced esophageal motility

Table 2. Proceduralist-Directed Nurse-Administered Sedation for Atrial Fibrillation Ablation

Primary Author	Year	Study Design	Patients	Medications	BMI	Capnography/ Monitoring and Airway	Intended Level of Sedation	Sedation-Related Outcomes
Tang ¹¹	2007	RCT Deep sedation vs conscious sedation	120	Propofol infusion vs midazolam+fentanyl	n/a	None	Deep vs conscious sedation	Propofol; Hypotension – 28% (vs 16%), Hypoxia – 21.7% (vs 6.7%) 2 pts bag mask (vs 1 pt), 1 cardiac arrest
Kottkamp ⁶	2011	Observational No exclusions reported	650	Propofol, midazolam, fentanyl	28	None Oropharyngeal airway	Deep sedation	Hypotension (≤70 mm Hg) 2.3%, ephedrine bolus 15%, Hypoxia (<85%) 1.5%, mask ventilation 1.2%
Salukhe ⁴	2012	Observational Exclusion; No exclusions reported	1000	Propofol infusion+fentanyl, midazolam if propofol stopped	n/a	None	Deep sedation	Propofol cessation – 13.6% hypotension 1.9% respiratory depression (1 pt bag-mask vent)
Wutzler ¹²	2013	Observational 3 groups by age Exclusion not defined	401	Propofol, midazolam, pirritramide	27.65	None Oropharyngeal airway in all	Deep sedation	1 hypoxic event related to contrast reaction, no hypotension (≤80 mm Hg or ≥40% change)
Sairaku ⁵	2014	RCT Exclusion; OSA	87	Dex vs thiamylal Both groups pentazocine, rescue thiamylal	23.75	None SD-101 portable respiratory monitor	Moderate sedation	Dex; Less apnea, higher RASS, lower movement index, shorter procedure, trend for hypotension

BMI indicates body mass index; n/a, not applicable; OSA, obstructive sleep apnea; RASS, Richmond Agitation Sedation Scale; RCT, randomized controlled trial.

and swallowing.¹⁵ There are other noteworthy drawbacks to the use of GA for AF ablation procedures. An absence of patient feedback might mask intraprocedural complications. Because of the possibility of phrenic nerve injury, nondepolarizing muscle relaxants should be avoided to preserve diaphragmatic function, especially for cryoballoon AF ablation where the incidence of phrenic nerve injury is higher. Even mechanical ventilation may negatively impact catheter stability, an issue that has been successfully addressed by utilizing high-frequency jet ventilation.¹⁶

Summary: AF ablation

In the aggregate, the literature on PDNA with propofol demonstrates a relatively low cumulative incidence of PPV and intubation, but a significant overall associated incidence of hypoxia and hypotension with this technique. A major concern is propofol's narrow therapeutic window; thus it seems prudent to question the generalizability of these findings and the underpowered nature of these analyses for rare but catastrophic events. Although the literature on dexmedetomidine for AF ablation is limited, outcomes from existing trials suggest it may be utilized as part of an effective and safe sedation regimen for these procedures. Moreover, because dexmedetomidine is associated with less respiratory depression than other agents, it may be a useful alternative to propofol, particularly when moderate sedation is targeted and/or the sedation will be administered without an anesthesia provider.

The use of a PDNA model for AF ablation is supported by the literature, particularly when moderate sedation is targeted. Nonetheless, GA or deep sedation by an anesthesia provider may be preferred, especially for cases that are prolonged, complex, being performed in the elderly patient, or in those with significant comorbidities.

Supraventricular Tachycardia Ablation

Evidence supports the use of moderate sedation with benzodiazepines and opiates for supraventricular tachycardia (SVT) ablation using a PDNA model,^{17,18} and this approach is frequent during SVT ablation.² With appropriately trained personnel and proper monitoring, this strategy is associated with few sedation-related adverse events.⁷ Inadequate analgesia is the most common disadvantage of this strategy, which might be exacerbated by either medication tolerance or a prolonged procedure time. However, these considerations must be balanced against the need for arrhythmia inducibility, which can be challenging with deeper sedation levels. Successful SVT induction use of GA with volatile anesthetics has been described,¹⁹ but any degree of sedation is thought to have the potential to reduce arrhythmia inducibility, with

benzodiazepine-opiates possibly having less of an impact.²⁰ When an anesthesiology provider is needed for SVT ablation, a primary goal is balancing patient comfort with a level of sedation that allows arrhythmia induction.

There is a paucity of reports directly comparing specific agents, the team delivering sedation, or the level of sedation that inhibits arrhythmia induction. Concern for sedation medication interference in SVT ablation is derived from a few select studies demonstrating conduction slowing with remifentanyl²¹ and propofol,²² case reports of tachycardia termination on administration,²³ and the knowledge that some arrhythmias are catecholamine dependent. Lai et al examined 150 patients (143 adults, 7 children) presenting for SVT ablation under deep sedation using propofol. The first 20 had anesthesiology-administered sedation followed by PDNA sedation for the subsequent 130. The authors reported no sedation-related complications and out of 152 diverse arrhythmias, 148 were successfully induced using propofol, except ectopic atrial tachycardia in 4 of 7 pediatric patients.²⁴ Ketamine, an analgesic with sympathomimetic qualities, has been used without an anesthesiologist and has been demonstrated to enhance conduction and promote arrhythmia inducibility.²⁵ In contrast, dexmedetomidine has been eschewed for use during EP studies due to concern that rhythms will be noninducible.²⁶ However, when administered in typical sedation doses, dexmedetomidine's advantages include minimal respiratory depression. Review of the available literature suggests there may be an overemphasis on particular sedation agents in regard to arrhythmia inducibility.

Ventricular Tachycardia Ablation

Unstable hemodynamics, long procedure times, and patient comorbidities often result in the use of GA for ventricular tachycardia (VT) ablation. Nof et al²⁷ performed a retrospective and prospective study in patients with sustained VT associated with structural heart disease. In the retrospective comparison of 226 patients, there was no difference in VT inducibility, complications, or abolition of clinical VT, but there was a higher incidence of hemodynamic support in the GA group. In the smaller prospective group, noninvasive programmed stimulation was performed via the patient's implantable cardioverter defibrillator (ICD) during light sedation and GA in the same patient; 8% were no longer inducible with GA and 50% had different induced VT morphology, although this cannot be causally attributed to GA. It appears GA does not have a large effect on VT inducibility or procedural success and the benefits may outweigh the higher use of vasoactive agents used during GA. These data do not apply to idiopathic VT where a study of sedation strategies is ripe for investigation. Though VT storm has been managed

with deep sedation and GA,²⁸ VT ablation success does not seem to be impacted by the use of GA.

Electrical Cardioversion

Electrical cardioversion (EC) is frequently performed to restore sinus rhythm in patients with tachyarrhythmias. Although EC is a relatively simple procedure to perform, it is complicated by several factors: (1) A brief period of deep sedation or GA is necessary,²⁹ (2) hemodynamic and respiratory stability must be maintained despite the adverse side effects of the required sedation agents, and (3) It is frequently performed in areas remote from the operating room or EP laboratory where unique logistical challenges may exist. The ideal anesthetic agent for EC should have a short onset time and duration of action producing a rapid loss of consciousness and recovery with minimal adverse hemodynamic and respiratory effects. No such agent currently exists, and the optimal sedation regimen for these procedures remains controversial as does the question of whether an anesthesia provider should provide sedation or whether a PDNA model is appropriate. This section summarizes the literature on this topic and provides recommendations for the practitioner.

PDNA sedation for EC

PDNA sedation for EC procedures utilizing benzodiazepines as the primary sedative agent has been described in multiple reports. In 2001, Pugh et al³⁰ assessed using diazepam as the sole agent in 141 patients who underwent EC for atrial arrhythmias over a 15-month interval. Five- to 10-mg diazepam was initially administered followed by additional doses as needed. Respiratory depression and oxygen desaturation occurred in only 2 patients (1.4%) who both required flumazenil treatment, but no patient required PPV. Another study in 2001 reported PDNA with midazolam for EC in 149 consecutive patients to achieve deep sedation (initial 2.5-mg dose of midazolam with additional boluses up to a maximum of 12 mg as well as supplemental meperidine). Flumazenil was used in each case at the end of the procedure. There were no patients who required intubation, all patients found the procedure tolerable, and there was no reported recall.³¹

A 2003 prospective single-blinded trial randomized 141 patients to diazepam versus midazolam sedation for EC of atrial arrhythmias.³² Sedation was administered using a PDNA titration protocol and an anesthesiologist was available in case of emergency. No major adverse events occurred, but 16 minor adverse events were reported with midazolam (20% hypotension, 3% desaturation) versus 9 with diazepam (7% hypotension, 0% desaturation) ($P=0.14$). Time to adequate sedation was shorter with midazolam, but the diazepam group had more rapid recovery (39 ± 24 minutes versus 77 ± 46

minutes, $P<0.0001$). There was no difference in awareness or other postprocedure issues at 24 and 48 hours. The investigators concluded that as a sole sedative for EC, both drugs were safe and well tolerated. A similar 2004 trial prospectively studied 368 consecutive patients over 1 year undergoing elective EC using only midazolam.³³ For the first 6 months an anesthesiologist was present; thereafter an anesthesiologist was only available on call. The mean dose of midazolam administered was 7 mg and all patients received flumazenil following the procedure. Two patients, 1 administered a total midazolam dose of 12 mg and the other 16 mg, were felt to have inadvertently transitioned to GA. Despite these occurrences, the investigators reported no episodes of apnea or other respiratory complications.

Notarstefano et al³⁴ examined EC in 202 patients over a 2-year period using midazolam via a PDNA model. Patients requiring EC for an atrial tachyarrhythmia or as part of an EP procedure were included. Fourteen patients were excluded for reasons that included the following: resting $SpO_2 < 90\%$, "serious" respiratory disease, weight > 130 kg, and ejection fraction $< 20\%$, and a known sedation-related complication. Adequate sedation was achieved in 99% of cases with no episodes of recall. Ten percent of subjects had transient SpO_2 decreases below 90%, which were all successfully managed with head and neck repositioning and flumazenil administration. No significant hemodynamic sequelae were observed and anesthesia assistance was never required.

Finally, a 2010 retrospective analysis of 624 elective ECs over a 6-year period examined PDNA for EC.²⁹ An anesthesiologist was not present but available if needed. Exclusion criteria included the following: severe obesity, significant respiratory disease, obstructive sleep apnea (OSA), dementia, seizure history, and severe or acute renal or liver impairment. The sedation protocol consisted of a midazolam bolus with supplemental bolus propofol. Flumazenil was administered at procedure conclusion in the majority of cases and no procedure was aborted due to inadequate sedation. There were no reported cases of over- or undersedation; however, transient apnea occurred in $< 3\%$ of subjects with all cases managed with PPV without incident without the need for an anesthesiologist. The authors concluded that PDNA is an appropriate model for sedation needs during outpatient EC in a selected population. However, they conceded their study may have been underpowered to assuredly claim the protocol was absolutely safe.²⁹ Similar to prior reports, the authors do not report the absolute number of patients excluded but based on the many comorbidities listed as exclusions, it is likely more than a trivial number. These excluded patients would certainly be more prone to sedation-related complications as compared to only those included in the current report.

Anesthesia provider administered sedation for EC

An early prospective trial in EC for AF and flutter randomized 44 patients to 1 of 4 sedative agents: sodium thiopental, etomidate, propofol, or midazolam.³⁵ All 4 groups received supplemental fentanyl. Exclusion criteria were hemodynamic instability, ventricular dysrhythmias, or signs of myocardial ischemia. All 4 agents provided an adequate level of sedation and there was no difference in the success rate of EC or energy level required. However, the use of midazolam resulted in significantly longer induction times, awakening times, and orientation times as compared to the other 3 agents. There was a significant decrease in blood pressure from baseline with midazolam (19%), thiopental (19%), and most marked for propofol (29%). There was no change in blood pressure with etomidate. The incidence of apnea was highest with propofol (58%) as compared to the other 3 agents (thiopental: 17%, etomidate: 10%, midazolam: 10%). The authors concluded that EC can be performed satisfactorily with all 4 agents but highlighted the inferior performance of midazolam as compared to the other agents in their trial.

A 2006 trial by Parlak et al³⁶ randomized 70 patients who required EC for AF to 1 of 4 groups based on age (<65 years, ≥65 years) and sedative (midazolam, propofol). Exclusions included the following: inability to cooperate, liver or renal insufficiency, electrolyte imbalances, acute respiratory symptoms, chronic obstructive pulmonary disease, hypotension, and unclear cardiac rhythms. All medications were administered by bolus injections. Mean induction time was similar in all 4 groups; however, mean recovery time was shorter in both propofol groups (18.8 minutes versus 40.3 minutes [<65 years] and 18.2 minutes versus 54.2 minutes [≥ 65 years], $P<0.001$). There were no hemodynamic differences between groups and no difference in the incidence of apnea. Interestingly, oxygen desaturations were higher in both midazolam groups as compared with their age-matched counterparts in the propofol groups ($P<0.05$). The authors concluded that propofol was a better option for the older patient because it was associated with shorter recovery times, fewer side effects, and increased comfort.³⁶

In 2011, Kalogridaki et al³⁷ performed a prospective trial comparing sedation with fentanyl/propofol versus fentanyl/etomidate in 46 patients who were undergoing EC for persistent AF. There was no difference in number of shocks needed to restore sinus rhythm or the number of unsuccessful cardioversion attempts. Patients in the etomidate group had a shorter time to loss of consciousness and to administration of the first shock. Patients in the propofol group had a more pronounced decline in systolic blood pressure. PPV was needed in 28% of patients in the propofol group versus 43% in the etomidate group ($P=0.360$). The investigators noted that both regimens provided excellent conditions for EC, but

etomidate facilitated a quicker induction time and provided more hemodynamic stability. They also concluded that when sedating patients for EC using these regimens, the presence of an anesthesiologist is necessary to recognize and manage potential airway obstruction or apnea.

Summary: electrical cardioversion

Sedating patients for EC is challenging and sedation goals for EC are different than for other EP procedures. EC is a brief but highly stimulating procedure necessitating both a short duration and deep level of sedation to mitigate discomfort, prevent recall, and facilitate rapid emergence and recovery.

Currently, sedation for EP procedures including EC is most often performed with benzodiazepines,³⁸ opioids,³⁸ and propofol.³⁹ At present there is insufficient evidence to support 1 specific drug regimen over another. Although benzodiazepines with or without opioids may seem appealing because of practitioner familiarity, ease of titration, and minimal adverse hemodynamic effects, these agents may not be ideal for EC.⁴⁰ Propofol, with its rapid onset and offset and ability to quickly achieve a deep plane of sedation, blunt or abolish laryngeal reflexes, and more reliably prevent recall, may in many instances be the superior choice.⁴⁰

As outlined above, multiple studies demonstrate that for EC, PDNA midazolam,^{31–34} diazepam,^{30,32} or even a combination of midazolam and propofol²⁹ is well tolerated. However, it is important to note that in a majority of these studies, patients at highest risk for sedation-related complications were excluded, yet adverse respiratory events still occurred.^{29,34} Arguments for PDNA are the difficulty involved in coordinating EP and anesthesiology teams, cost containment, decreased wait times, and possibly patient satisfaction.^{29,40} However, safety concerns about anesthetic drug administration by nonanesthesia providers,^{29,41} particularly when deep sedation is required, have been highlighted⁸ and have likely curtailed a more widespread adoption of this approach. There is no single ideal sedative agent for EC but as discussed above, propofol is likely superior as compared to benzodiazepines.

Pacemaker and ICD Implant

Modern pacemaker implantation is generally performed under PDNA moderate sedation, unless patient characteristics favor anesthesiology-administered sedation. The following discussion focuses on ICD and cardiac resynchronization therapy (CRT) device implantation. During the 1990s, as pulse generators became smaller and transvenous ICD leads increasingly prevalent, the venue for these cases shifted to the EP laboratory and away from the operating room, and with this shift came the concurrent use of “conscious sedation” for

these procedures. An early observational study by Fox et al⁴² of 500 ICD and CRT procedures with PDNA midazolam and opiates reported no deaths, no need for intubation, a single case requiring transient PPV, and flumazenil reversal in 7.5% of patients. Additionally, noncritical analgesic reversal with naloxone was performed in 2 cases.⁴² To date, there have been no prospective randomized trials specifically comparing PDNA to anesthesia-directed sedation strategies for ICD/CRT procedures. Nonetheless, observational studies contrasting various approaches to PDNA sedation in a broad range of EP procedures support this practice with low rates of episodic hypoxia (0–4.6%) with no reported need for PPV.^{7,43}

Use of GA may have several drawbacks for insertion of cardiovascular implantable electronic devices. A 1997 retrospective review of 96 consecutive ICD implantations was evaluated by dividing patients into 3 groups: (1) abdominal implant with GA (n=22), (2) pectoral implant with GA (n=40), and (3) pectoral implant using PDNA deep sedation with fentanyl and versed (n=34). As compared to Groups 1 and 2, Group 3 demonstrated reduced implant and total procedure times, a shorter recovery time, reduced requirement for postprocedure analgesia, and lower total costs.⁴⁴ Though not powered to show a difference in complication rates, there were no reported sedation-related adverse events. A nonrandomized prospective study of patients undergoing ICD implant in the beginning of the pectoral implant era compared GA (n=40) versus brief PDNA deep sedation (n=50) at the time of defibrillator threshold testing. Two respiratory complications (pulmonary edema and respiratory depression delaying extubation) occurred, both in the GA group. Also, the sedation group had shorter procedure times (2.1±0.5 hours versus 2.9±0.5 hours, $P<0.0001$).⁴⁵ A 2007 prospective observational study of 118 ICD implants with defibrillator threshold testing compared “minimal sedation” (sublingual midazolam and intravenous opioid) to GA. There were no significant differences in postprocedure pain scores regardless of sedation method and no adverse events were reported.⁴⁶

In contrast to the above studies, Sayfo et al³ performed a single-center retrospective review of PDNA sedation with propofol for 582 ICD implants. They reported a 10% rate of 1 or more serious events, including 1 requiring intubation for excess sedation, 4 requiring unplanned intensive-care unit admission for hypoxemia, 9 requiring PPV, and 47 needing significant hemodynamic support. It is noteworthy that the majority of patients were administered propofol by continuous infusion and use of continuous infusions was conspicuously higher in the group that had more serious adverse events (91% versus 73%, $P=0.001$). Greater total doses of propofol were also associated with higher adverse event rates ($P=0.001$).³ A second prospective observational study using propofol in 269 consecutive patients undergoing a cardiovascular implantable electronic devices procedure, including lead

extraction, reported a respiratory complication rate of 19% (comprising hypoxia 86%, apnea 30%, and aspiration 2%). Details regarding airway interventions were not given. The majority of cases (92%) were performed with anesthesia-administered deep sedation (bolus propofol and nalbuphine); the remaining 8% received GA.⁴⁷ A 2009 retrospective study of 197 ICD/CRT implants used propofol and midazolam administered by an anesthesiologist with a target of moderate sedation. In this study, hypotension was common (25% in ICD patients, 56% in CRT) and was associated with a higher New York Heart Association class, decreased left ventricular function, impaired renal function, and longer procedure times. Vasoactive drugs were used in 10.3% of ICD and 24% of CRT implants, but respiratory complications were unreported.⁴⁸

Summary: pacemaker and cardioverter-defibrillator implant

Based upon the limited observational studies, even for patients undergoing a relatively minor procedure such as cardiovascular implantable electronic devices insertion, and even when administered by an anesthesia provider, propofol causes significant rates of respiratory depression and hypotension. The majority of cardiovascular implantable electronic devices implantations appear safe when PDNA moderate sedation is targeted, but if deep sedation or use of propofol is required (ie, for subcutaneous ICD implantation), anesthesia services may be more suitable.

Approach to Sedation

A comprehensive discussion of the preanesthesia evaluation is beyond the scope of this review. However, there are a number of excellent references on this topic including the most recent American Society of Anesthesiologists practice advisory.^{49,50} In brief, this evaluation should include a review of readily accessible medical records, a patient interview, a directed examination, and preoperative tests and additional consultations when indicated. The patient’s fitness for undergoing the planned procedure is determined and the patient is medically optimized to the extent possible. For EP procedures important details include the following: fasting status, projected case duration, anticipated degree of stimulation and pain, how well the patient can tolerate lying supine for an extended time, sedation preferences of the patient and proceduralist, potential for procedural complications and hemodynamic instability, and significant comorbid medical conditions and other factors that might impact anesthetic management.

OSA is of critical importance and merits particular attention. Patients who are either at risk for OSA or have a documented history of the disease are at high risk for

sedation-related adverse events such as upper airway obstruction, often making them unsuitable for moderate or deep sedation. Use of the “STOP-BANG” screening tool is a simplified method to identify those with OSA. The score consists of 8 yes–no questions (Snoring, Tiredness, Observed apnea, high blood Pressure, Body mass index ≥ 35 kg/m², Age older than 50 years, Neck circumference < 40 cm, and male Gender); a score of ≥ 3 identifies patients at high risk for OSA and ≥ 5 indicates possible moderate–severe OSA.⁵¹ Both mask ventilation and endotracheal intubation can be very challenging in the OSA patient. If sedation is planned, strategies for managing the airway include judicious use of respiratory depressants to maintain spontaneous ventilation, placing an oral or nasal pharyngeal airway, and intra- and postprocedure continuous positive airway pressure (CPAP). Even when these strategies are employed, progressive hypoventilation and hypercapnia can occur: conditions that are often poorly tolerated in these patients because of concomitant pulmonary hypertension and/or right-sided coronary disease, as hypercarbia and hypoxemia both increase pulmonary vascular resistance and might precipitate acute right heart failure. In the OSA or non-OSA patient, capnography should ideally be employed throughout the sedation period. Capnography depicts the flow of CO₂ throughout the respiratory cycle, which is termed “End-tidal CO₂.” End-tidal CO₂ is sampled as proximal as possible to the

expired gases (end of endotracheal tube if intubated, from the nasal cannula or face mask if nonintubated). While all sedative agents engender some degree of dose-related respiratory depression, capnography changes (a surrogate for sedation level) provides a means to monitor the depth of sedation. Capnography appears to be underutilized in the EP lab, given its omission from EP literature documenting safety of sedation by nonanesthesiologists. There are several valuable types of information that can be assessed from capnographic wave forms (Figure 1A through 1C).

Sedation Delivery, Guidelines, and Evidence

Systems of Sedation Delivery

A 2011 national survey evaluating approaches to sedation in EP labs cited several reasons for selecting PDNA rather than the anesthesia-provider administered model, including ease of scheduling, faster turnover times, and lower costs.² These potential advantages must be balanced against the safety concerns of a PDNA approach, especially for complex patients and if deep sedation is planned or might be needed. Mayr et al⁵² reported that in 726 various EP procedures conducted with moderate–deep sedation by a dedicated cardiac anesthesiologist involved in each case, 44 (6.1%) required PPV and 16 (2.2%) required emergency intubation with 3 (0.4%)

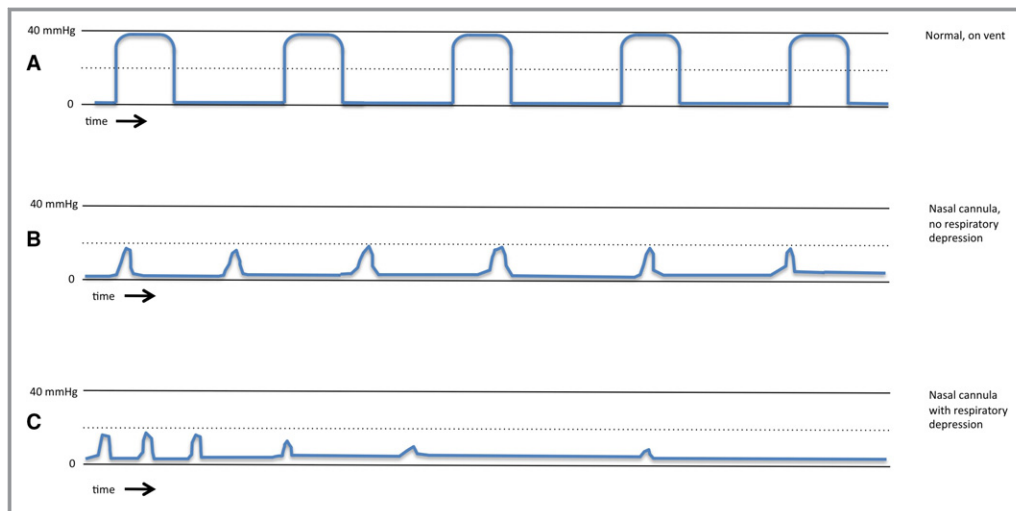


Figure 1. End-tidal CO₂ waveforms. A, Capnography on ventilated patient. Note that since the circuit is completely closed with a continuously sampled measurement, the tracing appears quite linear with each breath as carbon dioxide is exhaled. This characteristic shape is only seen with an invasive breathing device completely sealing the airway from room air entrainment. B, Capnography illustrating a regular breathing pattern similar to that sampled from a nasal cannula during moderate sedation with the patient spontaneously ventilating. Note that the tracing is slightly irregular with each breath due to the variable room air entrainment into the sampling line. This is particularly evident if the patient were to breathe primarily through their mouth, such that a continuously sampled measurement from the nasal cannula may not sample any exhaled CO₂. C, Capnography illustrating a progressively depressed respiratory rate with progressively smaller amounts of CO₂ measured by the sampling line. This would indicate impending respiratory depression.

requiring emergency fiberoptic intubation. Moreover, patients undergoing sedation outside of the operating room are at higher risk for airway-related complications.⁵³ A recent closed claims analysis of out-of-operating-room procedures found higher rates of inadequate oxygenation and ventilation (21% versus 3%, $P<0.001$), claims for death (54% versus 29%, $P<0.001$), and more frequent respiratory events (44% versus 20%, $P<0.001$).⁵³

Approaches to sedation in the EP lab are widely variable. Whether PDNA sedation, anesthesia provider administered sedation, or a blended approach is selected largely seems to depend on local culture. At Oregon Health & Science University and the Portland VA Hospital, 2 EP centers share the same campus, but use entirely different approaches: one (Oregon Health & Science University) almost entirely uses anesthesia-provider administered sedation (Figure 2) while the other (Portland VA Hospital) primarily uses a PDNA model. At the Portland VA, dexmedetomidine has become the sedative agent of choice for ablation procedures including AF. Since 2011, over 450 ablations have been performed using dexmedetomidine that is administered by specially trained nurses. When compared to the use of midazolam and fentanyl, this approach has reduced oxygen requirements and has not impacted SVT inducibility.⁵⁴

Professional Society and CMS Guidelines

In the United States, the Centers for Medicare and Medicaid Services (CMS) regulatory requirements often help determine local policy. A series of CMS revisions from 2009 to 2011 to the Interpretive Guidelines stipulate that individual hospitals

and anesthesiology service directors determine local policies with the expectation that these policies are underpinned by national guidelines (https://www.cms.gov/Regulations-and-Guidance/Guidance/Manuals/downloads/som107ap_a_hospitals.pdf). These changes have placed anesthesia directors in the position of overseeing practitioners who may have previously been performing deep sedation without involvement by anesthesia personnel. According to CMS, a proceduralist may not perform a procedure while concurrently administering deep sedation. This necessitates involvement by an appropriately credentialed second physician or an anesthesiology provider. The 2013 Heart Rhythm Society consensus statement on EP laboratory standards states that “it is desirable that anesthesia services be an integral part of the clinical practice in the EP laboratory.”⁵⁵ Nonetheless, it also allows for PDNA deep sedation if the trained sedation nurse has no responsibilities other than monitoring the patient.

Improving the Clinical Evidence Base for Deep Sedation Systems of Care

Professional practice guidelines and CMS regulatory requirements regarding deep sedation are based on little evidence. Given the criteria from the American Society of Anesthesiologists that require GA rescue skills by nonanesthesiologists administering deep sedation,¹ this is likely to lead to increases in the number of procedures for which anesthesiology services are required. For both economic and safety reasons, more evidence is needed to ensure that such a shift in care delivery is safe and efficient. While it is logical that the

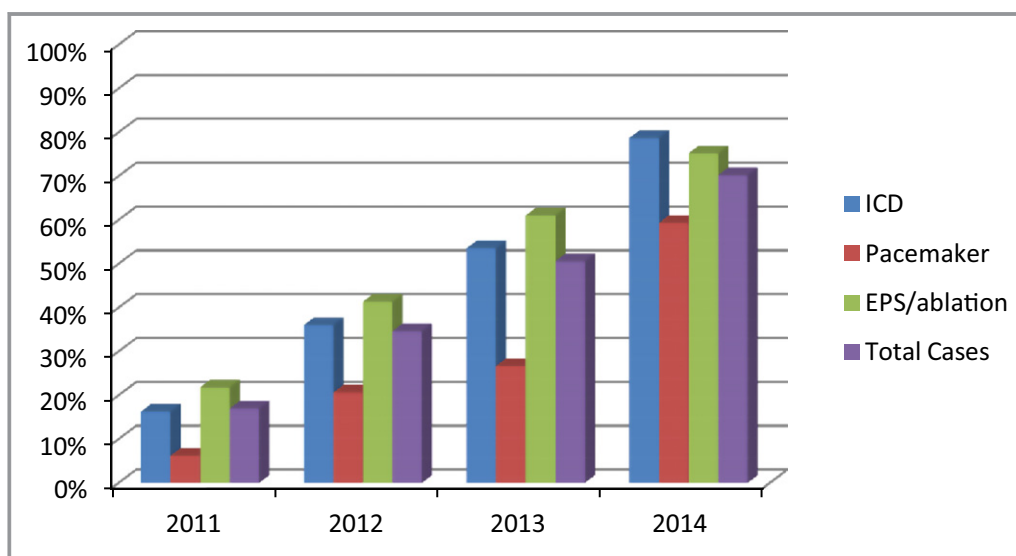


Figure 2. Oregon Health & Science University Anesthesiology use in electrophysiology lab 2011–2014. ICD, implantable cardioverter defibrillator; EPS, electrophysiology study.

addition of an anesthesia team for the care of sedated patients would improve safety, these benefits could be eroded if anesthesia care teams are inconsistently exposed to and unfamiliar with EP procedures or proceduralists. Absent clear improvements in safety, outcomes, or patient experience, the additional costs inherent to adding an anesthesia team for conscious sedation cases could be prohibitive.

One approach to resolve questions of safety, efficacy, and cost is creation of a national registry. Large anesthesia registries are already operational with more than 30 million cases entered in the National Anesthesia Care Outcomes Registry alone (<https://www.aqihq.org/>). CMS policymakers or individual hospitals could allow provision of deep sedation under the supervision of a procedural attending physician

contingent on registry tracking and outcome reporting. Cases could be propensity-matched for all characteristics aside from the team administering sedation, to define what the additional value of a dedicated anesthesia team is and in what situations it should be mandatory. Such an approach would rationalize policies requiring anesthesia services in contexts in which they had not been previously required and in which safety surveillance had not previously demonstrated risk.

Summary and Recommendations

There are numerous patient and institutional factors to consider when creating the optimal anesthetic plan (Figure 3).

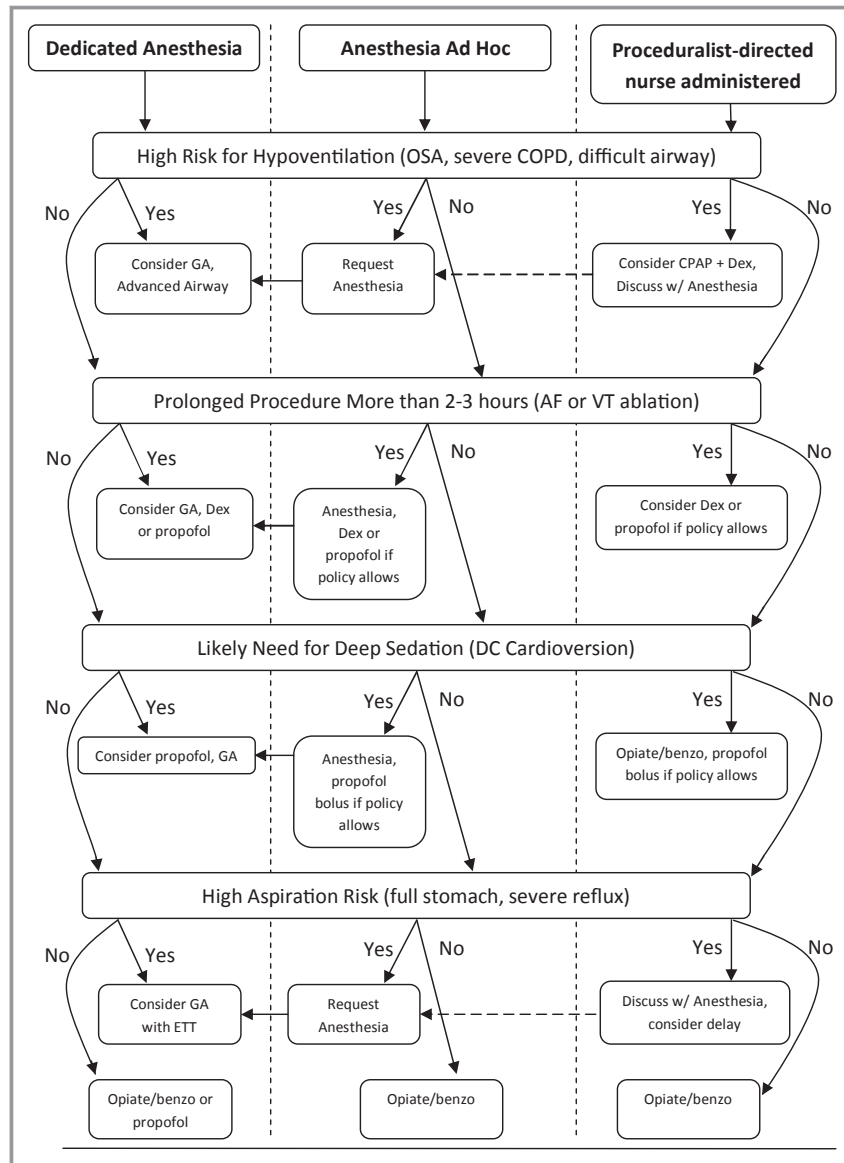


Figure 3. Systems of care flowsheet. AF, atrial fibrillation; COPD, chronic obstructive pulmonary disease; CPAP, continuous positive airway pressure; DC, direct current; ETT, endotracheal tube; GA, general anesthesia; OSA, obstructive sleep apnea.

Based on the available evidence, it is not possible to determine whether an anesthesia provider should be required for all EP procedures requiring deep sedation. Nonetheless, the literature does support that sedation-related complications are greater during longer procedures in patients who are elderly or have significant comorbidities; in these instances we recommended that anesthesiology services be considered for such cases. There are important potential benefits to routinely including anesthesia providers in EP procedures including the potential for an enhanced patient experience, a lower risk of sedation- or anesthesia-related complications, and the presence of medical professionals to help manage life-threatening EP-related complications. These must be balanced against increased costs, scheduling challenges, and the anesthesia care team's level of familiarity with EP procedures.

Regardless of the makeup of the team administering sedation, there is sufficient evidence to recommend the use of deep sedation by appropriately qualified clinicians for AF ablation procedures. Both propofol and dexmedetomidine have been safely used, with midazolam and fentanyl given to initiate sedation. Propofol and dexmedetomidine have intrinsic advantages and disadvantages, though both are suitable for EP lab sedation in appropriate circumstances. Aside from AF ablation procedures, there are limited data directly comparing the safety outcomes of the 3 basic models of sedation delivery (dedicated anesthesia team, ad hoc anesthesiology, or PDNA). In the absence of data, we feel systems of care should adhere closely to professional society guidelines, particularly those of the American Society of Anesthesiologists. It is also our opinion that as the volume of EP procedures continues to expand, a joint American Society of Anesthesiologists–HRS set of guidelines would be most welcome.

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Disclosures

None.

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