Implantable Cardiovertor/Defibrillator Placement in a Patient with Amiodarone Pulmonary Toxicity under Thoracic Epidural Anesthesia

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Placement of the implantable cardioverter/defibrillator (ICD) has become standard treatment for refractory malignant dysrhythmias, such as ventricular tachycardia (VT) and ventricular fibrillation. Since the introduction of first-generation devices in 1980, more than 15,000 ICDs have been inserted, substantially reducing long-term mortality from episodes of sudden cardiac death.1 While each previous discussion of perioperative management of ICD patients has described general anesthesia,2 3 we report a case of ICD implantation under regional anesthesia in a patient with refractory VT and severe amiodarone pulmonary toxicity. Using an extrapleural surgical approach, we successfully implanted and fully tested the ICD in this patient entirely under midthoracic level epidural anesthesia.

Case Report

A 68-year-old man presented for elective ICD placement. Significant past medical history included separate anterior transmural and subendocardial myocardial infarctions, 9 yr and 2 yr prior to admission, respectively. Recurrent paroxysmal VT developed following the first MI, and initially responded to mexiletine. Treatment with amiodarone was initiated after the recrudescence of sustained VT following his second MI and continued throughout the 18 months prior to admission. Pertinent history also included intermittent congestive heart failure, pericarditis, chronic anemia, and distant history of drainage of a left-sided empyema and smoking.

Subjectively, he was dyspneic at rest and with minimal exertion. Pulmonary function tests revealed a forced vital capacity of 2.21 (53% predicted), forced expiratory volume of 1.611 l/s (57% predicted), peak expiratory flow of 2.21 l/s (28% predicted), and diffusion of carbon monoxide of 7.6 ml·min⁻¹·mmHg⁻¹ (42% predicted). Severe bilateral upper-lobe atelectasis was noted on chest radiography, computerized tomographic scanning, and bronchoscopy (under local anesthesia). Electron microscopy of bronchial effluent noted lipid-laden, foamy macrophages in alveolar spaces and septal walls, consistent with amiodarone-induced interstitial fibrosis/toxicity. Amiodarone and des-methylamiodarone levels were 979 ng/ml (normal level 500–2,500) and 1,845 ng/ml, respectively, prior to discontinuation 1 month before ICD placement. Cardiovascular workup included a resting electrocardiogram with first-degree atrioventricular block, left ventricular hypertrophy, and a nonspecific intraventricular conduction delay; cardiac catheterization with 100% occurrence of the left anterior descending artery and a large apical left ventricular aneurysm; transesophageal echocardiography with severe global hypokinesia, aneurysmatic and septal akinesia, and an estimated ejection fraction of less than 20%. Radionuclide ventriculography corroborated an ejection fraction of 16%. Following meticulous management of fluids, nutrition, and pulmonary toilet with antibiotics, corticosteroids, diuretics, and digoxin, cardiopulmonary function was considered to be optimal as documented by decreased dyspnea, a roughly 10% improvement in the aforementioned PFTs, and no clinical evidence of congestive heart failure. Oral doses of ranitidine (150 mg) and sodium bicarbonate (30 ml) were given preoperatively.

In the cardiac catheterization laboratory, routine noninvasive monitoring and oxygen administration by nasal cannula were begun. Under local anesthesia (1% lidocaine) and incremental sedation with midazolam (2 mg intravenously, total), a low right atrial unipolar electrode (superior vena cava [SVC] coil) and a right ventricular endocardial bipolar pacing lead were placed via a left subclavian cut-down. Hemoglobin oxygen saturation ranged between 88–95%. An oxymetric pulmonary artery catheter was placed through the right internal jugular vein under fluoroscopic guidance to prevent intra-cardiac entanglement with the previously placed leads. Brief but nonsustained VT occurred during catheter insertion and required no treatment. Initial values noted were a cardiac output of 2.2 l/min, a pulmonary arterial pressure of 30/13 mmHg, a pulmonary capillary occlusion pressure of 11 mmHg, and a mixed-venous saturation of 60–64%.

In the operative suite, a left radial arterial cannula was inserted (baseline mean arterial pressure, 90–100 mmHg). With the patient in a sitting position, an epidural catheter was inserted 2 cm the T6 interspace. A T1 level of anesthesia was obtained by administering incremental doses of 7 ml 1.5% plain lidocaine with 100 μg fentanyl citrate added. For the remainder of the procedure, the patient was

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kept in a supine position with a head elevation of 15°. Ephedrine (5 mg aliquots) was required during the first 90 min following epi-
dural induction to maintain a mean arterial pressure within 20% of
baseline. The patient was comfortable and showed no signs of dys-
pnea. Light sedation with 0.25 mg aliquots of midazolam (total dose
used was 1.25 mg) was added without significant respiratory depres-
sion. No supplementation with local anesthetics was necessary.

A left subcostal incision was performed, followed by an extrapleural
dissection of the attachment of the diaphragm near the xiphoid and
placement of the defibrillator patch on the pericardium. The leads
from the previously made subclavian incision were tunneled sub-
cutanetiously beneath the left rectus muscle for connection to the
ICD. R-wave amplitude, pacing thresholds and defibrillation thresh-
olds (DFT) were determined. VT then was induced to test the anti-
tachycardia feature of the ICD. During each of the 15 testing episodes
of induced VT (each lasting 30–60 s), mean arterial pressure de-
creased to 20–30 mmHg and mixed venous saturation decreased to
40–50%, while hemoglobin oxygen saturation remained greater than
88%. The patient maintained spontaneous respiration throughout
each dysrhythmic induction but became moderately tachypneic with
a perceptible decrease in tidal volume. Unable to communicate, he
appeared unconscious following each induction of dysrhythmias. After
either spontaneous or induced electric cardioversion, he would regain
consciousness and communicate appropriately; a return to baseline
respiratory rate and depth, mixed-venous saturation, and blood pres-
sure occurred within the 5 min periods between testing.

The thoracic epidural required incremental doses of 1 ml of 1.5%
lidocaine every 15 min to maintain a T1–T2 level. One brief episode
of desaturation to 83% occurred when tachypnea prompted place-
ment of a left midaxillary (fourth intercostal space) thoracostomy
tube to exclude collapse from pneumothorax; however, no appreci-
able amount of air was aspirated. Following ICD implantation in the
subcostal abdominal pouch, the subcostal and SVC lead/coil incisions
were closed. Following the 4.5 hr procedure, the ICD was
activated, and the patient was transferred to the cardiac recovery arca
with hemodynamic parameters similar to preinduction levels: blood
pressure, 110/50 mmHg; mean arterial pressure 85 mmHg; pul-
monary arterial pressure, 31/17 mmHg; central venous pressure, 7
mmHg; cardiac output, 3.5 l/min; hemoglobin oxygen saturation,
96%; and mixed venous saturation, 57%. A postoperative electro-
cardiogram showed no change from that taken preoperatively. Chest
radiography confirmed appropriate positioning of the ICD, extra-
pericardial and intravascular leads, and the left-sided thoracostomy
tube without appreciable pneumothorax. Pain management consisted
of a 10 ml/hr infusion of 0.1% bupivacaine with 5 μg/ml fentanyl
citrate for 36 hr. Recovery was uneventful. Although able to recall
portions of the operative experience in fair detail, he was able to
remember only three or four cardioversions, which he characterized as
"insignificantly" painful with respect to the "excruciating" trans-
 thoracic cardioversions that he had previously experienced.

Discussion

Amiodarone therapy produces substantial pulmonary
function in up to 10% of patients treated chronically.1 Two other antiarrhythmics, mexiletine5 and toca-
indomethacin, also have been associated with pulmonary fi-
brosis. Although his initial exposure to mexiletine was
without apparent pulmonary sequelae, the patient's
chronic use of amiodarone resulted in increasing dys-
pnea, malaise, and nonproductive cough. Confirmatory
drug levels, radiologic findings, and open lung biopsy
implicated amiodarone as the predominant cause of
toxicity.

ICD placement offers an attractive, nonpharmacologic alternative treatment for malignant dysrhythmias,
avoiding the potential for drug-induced pulmonary toxicity. The anesthetic management for ICD placement
relies on the approach required for insertion of the sensing, pacing, and defibrillating leads and for for-

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this patient requires minimal cardiac exposure through
an extrapleural approach for extracardiac lead implantation.\textsuperscript{13} By avoiding the pleural space, spontaneous
respiration can be maintained. Transvenously placed
SVC coils and endocardial pacing leads obviate the need
for multiple epicardially accessed leads and can be
placed during local anesthesia. Tunneling of the trans-
venously placed lead and formation of the abdominal
pouch require blockade of levels T1–T9 and T8 to L1,
respectively. Even though these two initial steps could
be performed during local block, the large volumes of
local anesthetic (particularly amides) can interfere
with appropriate testing of the device and may yield
an unacceptable quality of anesthesia. The plasma level
of lidocaine associated with antiarrhythmic efficacy
ranges from 1.5 \( \mu \)g/ml to 5.0 \( \mu \)g/ml\textsuperscript{14} and can be
achieved with absorption either from a local block or
the epidural space. Block of thoracic sympathetic fibers
may have an adverse effect on the induction of VT or
DFTs. The systemic effect of lidocaine on elevating
DFTs has not been consistent and may depend on factors
other than plasma concentration, such as plasma pH,
defibrillation technique, and coanesthetics.\textsuperscript{15} In our
patient, the use of low doses of epidurally administered
lidocaine, combined with the avoidance of significant
respiratory acidosis and minimal use of intravenous an-
esthetics, was believed to affect intraoperative DFTs
the least. Although general anesthetics may be expected
to reduce the cerebral metabolic requirements for oxy-
gen and provide a cerebral protective effect during
periods of hypotension, the ability to reassess function
repeatedly after each dysrhythmic episode enhanced our
monitoring for neurologic deterioration.

Although this patient had severe, coexisting cardiac
dysfunction, the greater risk of prolonged postoperative
ventilation from his marginal pulmonary function was
considered to outweigh other concurrent diseases in
dictating anesthetic technique. Life-threatening post-
operative pulmonary complications—including death—have occurred in patients with previous amio-
darone pulmonary toxicity who have undergone cardio-
 thoracic operations, including ICD placement.\textsuperscript{16}
Despite the documentation of moderate obstruction
and severely decreased diffusion capacity in this pa-
tient, routine pulmonary function tests alone have not
been useful in predicting pulmonary side effects of
amiodarone.\textsuperscript{17}

The risk of aspiration during dysrhythmia-induced
hypotension and impaired consciousness was decreased
only partially by the use of histamine type-2 blockers,
antacids, and slight head elevation. The awake and
spontaneously respiring patient who requires signifi-
cant head elevation to alleviate dyspnea has a poten-
tially increased risk of air entrainment through any
portion of the surgical field located above right atrial
level (i.e., the subclavian cutdown site). This patient's
history of previous empyema and surgical drainage
(with an attendant likelihood of pleural adhesions) was
believed to decrease the risk of left-sided pneumothorax.
Nonetheless, a heightened vigilance for intra-
operative desaturation lead to the discovery of a small
pleural rent and expedient thoracostomy tube place-
ment for presumed pneumothorax. Pneumothorax may
be less likely to cause tension in a spontaneously re-
spiring patient than in one undergoing positive-pressure
ventilation. Blockade of multiple levels of intercostal
nerves, regardless of technique employed, may impair
accessory muscles of respiration. Although multiple-
level intercostal nerve blocks were considered, the
attendant risk of pneumothorax proscribed their use in
this patient. Because roughly 25\% of tidal volume is
contributed by rib cage displacement in a normal, su-
pine patient,\textsuperscript{18} it is unknown to what extent this patient
depended on intercostal muscle excursion for ven-
tilation. Although preoperatively he used accessory
muscles of respiration minimally, incremental dosing
of the epidural anesthetic was performed to assess the
progressive loss of intercostal muscle function.

The use of a thoracic epidural anesthetic in this pa-
tient obviated the need for tracheal intubation or me-
chanical ventilation, afforded acceptable hemodynamic
stability, allowed repeated assessment of neurological
status, and provided an effective mode of both intra-
operative and postoperative analgesia. Although ben-
eficial for this patient, the use of a regional anesthetic
during ICD placement and testing must be weighed
against many risks: impaired respiratory function,
pneumothorax, hypotension, aspiration, venous air
embolism, and awareness of electrical cardioversion.
In addition, DFTs and ICD testing may be impaired by
absorption of local anesthetics or blockade of cardiac
sympathetic tracts. The specific surgical approach cho-
sen for device placement may dictate the choice of
anesthetic technique.

References

1. Kelly PA, Cannon DS, Garan H, Mirabel GS: The automatic
implantable cardioverter/defibrillator (AICD): Efficacy, complica-
tions, and survival in patients with malignant ventricular arrhythmias.
J Am Coll Cardiol 11:1278–1286, 1988
2. Gaba DM, Wyner J, Fish KJ: Anesthesia and the automatic im-

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Effect of Subarachnoid Catheter Position on the Efficacy of Intrathecal Baclofen for Spinal Spasticity

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Several studies have demonstrated that intrathecal baclofen reduces spasticity secondary to spinal cord injury.1–4 Long-term control of spasticity is achieved by continuously infusing baclofen using a surgically implanted infusion pump and subarachnoid catheter. Pharmacologic agents that are administered intrathecally migrate in a rostral direction, following cerebrospinal fluid (CSF) flow patterns. However, it has been unclear whether the position of the spinal catheter orifice (site of drug delivery) has any influence on the degree of spasticity reduction. Previously, most experience with intrathecal baclofen has been limited to delivery of drug caudal to the spinal cord injury. Theoretically, delivery of intrathecal baclofen cephalad to the spinal cord injury should also be associated with significant spasticity reduction, because suprasegmental (above the spinal cord injury) excitatory reflexes

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