Superior vena cava syndrome (SVC) is caused by obstruction of the thoracic vascular bed by either external compression or occlusion. This syndrome and its symptoms, including oropharyngeal edema, cough, hoarseness, stridor, vocal cord edema, and vocal cord paralysis, can pose an anesthetic challenge. A case report presented herein describes a case of SVC stent placement in the interventional radiology (IR) suite and the subsequent difficulties related to the anesthetic management outside the operating room (OR).

Case Summary
A 46-year-old woman was scheduled for an SVC stent placement in the IR suite after presenting to the emergency department (ED) with a sudden onset of upper extremity edema, head and neck swelling, and difficulty breathing. Her medical history included past metastatic rectal carcinoma, with metastases found in the lungs, liver, and peritoneum. Treatment of this disease continued approximately 2.5 years. Comorbidities included type 2 (noninsulin-dependent) diabetes mellitus, hypertension, Hodgkin lymphoma, chronic back pain, and peripheral neuropathy. Her home medication regimen consisted of metformin, lisinopril, and oxycodone.

Initial treatment of the rectal cancer included a low anterior resection with a coloanal anastomosis and a diverting ileostomy with concurrent placement of a right SVC port (Mediport). Partial liver resection and left thoracotomy with wedge resection were required in the ensuing year. Development of a lower extremity deep vein thrombosis required placement of an inferior vena cava filter that was subsequently removed after 3 months. Most recently, cancer recurrence was treated with a FOLFOX (leucovorin, calcium, fluorouracil, and oxaliplatin) combination regimen, with completion of therapy 2 weeks before the onset of these new congestive symptoms.

One week before this ED admission, the patient was brought to an outside hospital after experiencing a witnessed seizure with postseizure loss of consciousness. Another seizure occurred in the ED. Magnetic resonance imaging (MRI) of the brain proved normal; however, levetiracetam (Keppra) therapy was started as a precaution. A tilt-table examination was performed for complaints of syncopal symptoms before the onset of seizures, and results of this examination were also normal. The patient’s complaints on this most recent ED visit included swelling of her upper extremities and head and neck, with reported difficulty breathing. Computed tomography (CT) examination was performed, and the images demonstrated no pulmonary embolus but did show collateralization of upper thoracic veins consistent with SVC syndrome without evidence of external venous compression. Differential diagnosis was determined as rule out thrombus. The patient was immediately started on treatment with enoxaparin sodium (Lovenox). A consult with the head and neck surgeon secondary to her reports of difficulty breathing revealed edema of oropharyngeal soft tissue, but normally visualized vocal cords and larynx. Both the vocal cords and larynx were without evidence of swelling and demonstrated full mobility. Stent placement with or without thrombolytic therapy in...
the IR suite was then scheduled. Enoxaparin therapy was changed to a heparin infusion in anticipation of the procedure.

After discontinuation of anticoagulation therapy, the patient was transferred to the IR procedure room. Results of laboratory studies including coagulation studies were within normal range at the start of the procedure. The anesthesia team administered very light sedation using only intravenous (IV) midazolam with as-needed dosing. Patient positioning for the procedure was supine on the fluoroscopy table with both arms tucked at her sides using sheets. Standard monitoring included continuous electrocardiography (ECG), noninvasive blood pressure measurement, and peripheral capillary oxygen saturation measured by pulse oximetry (SpO₂). Supplemental oxygen therapy was given via nasal cannula at 3 L/min. Simultaneous monitoring included continuous end-tidal carbon dioxide (ETCO₂) was also performed. Midazolam, 2 mg IV, was given along with a fluid infusion of normal saline via a previously placed 22-gauge IV catheter in the left antecubital vein.

The patient was prepared and draped with only her neck and right anterior aspect of the chest exposed. As the radiologist began to attempt fluoroscopy, the patient became agitated and unable to remain still despite 4 mg of IV midazolam in divided doses of 2 mg each. Because of patient movement interfering with the procedure and imaging, general anesthesia was initiated via a size 4 laryngeal mask airway (LMA) device without further sedation. Appropriate tidal volume and positive ETCO₂ concentration was achieved, and maintenance of general anesthesia included 2.5% sevoflurane with a mixture of oxygen and air at a 50% fraction of inspired oxygen (FIO₂).

Fluoroscopy identified a large thrombus emanating from the right atrium and continuing up the wall of the SVC to the azygos vein. An attempt at stent placement was made, and after multiple efforts over the course of an hour, the intervention was abandoned and thrombolytic therapy was initiated. Catheter-directed thrombolytic therapy using low-dose tissue plasminogen factor was administered; however, this treatment also failed. Heparin, 3000 U, was then given directly into the SVC, and the decision was made to stop further interven-
tion at this point. General anesthesia was discontinued, and efforts were made to transfer the patient to a stretcher so the head of the bed could be elevated in anticipation of emergence and LMA removal.

Secondary to procedure length and requisite positioning, the patient's upper body engorgement and vasodilation worsened. The entire procedure time, including positioning, preparing, and draping the patient, was greater than 2 hours as demonstrated in the case timeline depicted in Figure 1. Before the patient could be transferred to a stretcher, she rapidly began vomiting serosanguinous fluid through and around the LMA. The LMA was immediately removed and the patient's oropharynx was suctioned. Obstruction of her airway led to a precipitous fall of the SpO2 concentration to 54%. Sinus tachycardia at 147/min was noted on the ECG, and her blood pressure became increasingly elevated. Chin lift was maintained by the anesthesia provider with administration of 100% oxygen via face mask while the circulating nurse performed manual bag ventilation. Immediate assistance was summoned, and approximately 7 minutes later, 3 anesthesia providers and 2 nurses arrived. A head and neck surgeon was called to the procedure room and a surgical tracheostomy tray was brought to the bedside in anticipation of possible emergent airway access. Oral airway placement was attempted, causing a large amount of bleeding from the patient's oral and nasopharyngeal cavities, further compromising the airway.

The patient was transferred to a stretcher with the head of the bed elevated to sitting position while the staff maintained mask ventilation. The SpO2 concentration rose to 95%, and effective spontaneous respiration was noted. Her respiratory effort, however, once again decompensated, with laborious breathing and inspiratory and expiratory stridor emanating from the upper airway. An 18-gauge IV catheter was placed in the patient's right foot for further resuscitation. Dexamethasone, 10 mg IV, was administered along with a racemic epinephrine nebulizer. The patient once again became increasingly agitated, decreasing the effectiveness of the oxygen therapy via mask. To protect the patient’s airway, a decision for oral endotracheal intubation was made.

Topicalization of the oropharynx was achieved with a 4% lidocaine atomizer and a 2-mg dose of midazolam given through the IV catheter in the right foot. Once the patient was sedated, a fiberoptic bronchoscope was passed through the patient’s oral cavity. Vocal cords were easily visualized and noted to be normal, with no signs of edema or blood. A 7.0-mm endotracheal tube was placed into the trachea with confirmation of placement via fiberoptic bronchoscope and ETCO2 monitoring. Inhalational 1.7% sevoflurane was administered via the endotracheal tube, with spontaneous ventilation. The patient was transferred to the intensive care unit and was extubated within 24 hours.

Review of Literature

A PubMed search was conducted using the keywords: superior vena cava syndrome, superior vena cava thrombus, interventional radiology anesthesia, interventional radiology sedation, SVC syndrome anesthesia, and catheter-directed thrombolytic therapy. This review included 18 articles pertaining to treatment of SVC syndrome in IR with mention of anesthetic or airway management. Also included were articles relating to anesthetic management and patient safety concerns in procedure areas located a distance from OR zones. Most of the literature reviewed describes SVC syndrome, its etiology, presenting signs and symptoms, and morbidity and mortality. Two of the articles reviewed describe anesthetic considerations for patients presenting to the OR with SVC syndrome caused by mediastinal mass. Koizumi et al3 illuminate acute SVC syndrome caused by aortic dissection worsened by the induction of general anesthesia. Amundson et al4 detail an acute onset of SVC syndrome caused by intraoperative retractor placement, along with mention of the anesthetic management of this case. Dumantepe et al5 report cases in which SVC syndrome caused by thrombus are treated by catheter-directed thrombolytic therapy and anticoagulation therapy. The necessity of IV access in a lower extremity is emphasized by Birch et al.6 Most articles referring to stent placement for SVC syndrome in IR call for these procedures to be performed with an awake patient and the use of local anesthetics. Dee et al12 describe securing the airway with an endotracheal tube.
tube for catheter-directed thrombolysis and cases in which airway edema is present. Bedini et al.\textsuperscript{13} present a case in which local anesthesia was used for a patient with SVC syndrome during a transsternal biopsy.

Three articles were found pertaining to anesthesia considerations in IR.\textsuperscript{14-16} Youn et al.\textsuperscript{17} discuss general anesthesia considerations outside the OR, referring to it as “nonoperating room anesthesia (NORA)”. Ferrari\textsuperscript{18} also examines anesthesia concerns outside the OR.

### Discussion

Superior vena cava syndrome is a collection of symptoms caused by the occlusion of the SVC at the junction of the right atrium (Figure 2).\textsuperscript{1,2,5,6,8,9-11} These symptoms include swelling of the face, head, neck, upper aspect of the trunk and upper extremities; dyspnea and/or cough; hoarseness; dysphagia; and hemoptysis (Figure 3).\textsuperscript{1-10} Congestion of thoracic collateral veins may be seen on chest radiogram or CT scan, as well as distended jugular veins and edema of the oropharynx and head and neck on physical examination (Figure 4).\textsuperscript{1,2,4,6-10} Signaling a poor prognosis, extreme cases may cause cyanosis, cerebral edema, and laryngeal edema.\textsuperscript{2,4,6,8}

Most cases of SVC syndrome, reported in some sources at upward of 97% of all occurrences, develop in patients with bronchogenic carcinoma or non-Hodgkin lymphoma secondary to external compression of the thin-walled SVC.\textsuperscript{1,4,5,8,10,11} Superior vena cava syndrome is described in 3% to 20% of patients presenting with these masses, estimated in the United States at 15,000 cases in 2013.\textsuperscript{7,8,11} A smaller but increasing number of cases, with recent estimates at up to 40%, are caused by thrombus in the SVC secondary to the increased use of intracaval catheters or implantable pacer wires.\textsuperscript{2,3,10}

A rapid onset of symptoms occurs in cases in which there is partial or total occlusion of the SVC and the collateral thoracic veins (ie, azygos and brachiocephalic) are not recruited because the blockage develops suddenly or the collateral vessels also are occluded.\textsuperscript{2,6,8,10} If collateral veins are open to absorb congestion, symptoms will be very slow and insidious in onset, and the prognosis is much better.\textsuperscript{2,3,8,10} Should the azygos vein be occluded and collateral veins unable to bear the congestion, onset of symptoms will be rapid, requiring emergent treatment.\textsuperscript{3,6,8,10}

In the case presented herein, the patient had clear symptoms of SVC syndrome. Her head, upper aspect of the trunk, and upper extremities were severely edematous, so much so that peripheral IV access was limited to a 22-gauge IV catheter located in a left antecubital vein, which was placed by the ED staff. To prevent additional upper body venous congestion and to facilitate drug circulation, lower extremity peripheral IV access is necessary in these cases.\textsuperscript{2,4,6} Without lower extremity IV access in these circumstances, the venous congestion will delay onset of any IV medications given in the upper torso, and the administration of IV fluid in the already blocked upper venous system will add to venous congestion and exacerbate symptoms.\textsuperscript{2,4,6} In this patient, however, no IV access was placed in the lower extremities.

The patient presented with complaints of dyspnea,
indicating an edematous airway secondary to the blockage of blood return from the upper aspect of the torso, the limbs, and the head to the right atrium.\textsuperscript{1,2} A head and neck surgeon examined the patient on her initial presentation to the ED. The patient’s airway was assessed by flexible endoscopy and deemed normal at that time, although edema was noted in the oropharynx. No premedication was given that could have minimized her oropharyngeal edema before she went to the IR suite. Although its onset is slow, approximately 8 hours, the use of IV dexamethasone has been shown to decrease airway edema in cases of SVC syndrome.\textsuperscript{1,10,13,15,17} Prolonged use of IV corticosteroids, however, can aggravate the swelling of the head and increase fluid retention.\textsuperscript{13} Diuretics, such as mannitol, have also been given for the successful relief of airway and cerebral edema.\textsuperscript{1,2}

An MRI of the brain, obtained to establish the cause of the patient’s seizures and syncope, was normal. After CT was performed to rule out pulmonary embolus, this test aided in the diagnosis of SVC syndrome, because the images revealed engorged collateral thoracic veins. In patients with presenting symptoms of SVC occlusion, CT imaging is the best means by which to identify evidence of collateral vein development and engorgement.\textsuperscript{1,6,11} Seizures and syncope are worrying signs of amplified mortality risk in SVC syndrome.\textsuperscript{6} Increased intracranial pressure is a symptom of progressive SVC syndrome and is a very poor prognostic indicator.\textsuperscript{10} Edema of the brain can also be identified with CT imaging.\textsuperscript{6}

The patient was positioned supine on a fluoroscopy table for greater than 2 hours, adding to her head and neck edema and venous engorgement. It is recommended in the literature that these patients remain in a semi-Fowler “head-up” position to avoid supine positioning for any length of time.\textsuperscript{1,3,4,8,11,13} This venous congestion greatly affected the degree of edema in the patient’s airway and impeded respiration on emergence from anesthesia.\textsuperscript{1,2} The immobile fluoroscopy table also increased the risk at emergence, because it was impossible to elevate the head to any degree.\textsuperscript{6,11}

The patient was given thrombolytic therapy in addition to heparin. Patients with SVC syndrome are at increased risk of airway bleeding secondary to engorgement and edema.\textsuperscript{1} The addition of thrombolytic therapy in these cases creates an increased bleeding risk with any manipulation of the airway.\textsuperscript{5,10} Because thrombolytic therapy was one of the preplanned treatment options, a plan for safely securing the airway should have been implemented.\textsuperscript{1,8} Despite evidence demonstrating the avoidance of airway manipulation as the treatment goal, in a high-risk scenario a secured airway is the safest option.\textsuperscript{2} This is tempered by the understanding that the induction of general anesthesia in these patients may cause profound hypotension and cardiovascular collapse because of the venous outflow obstruction from the heart to the body.\textsuperscript{3}

The anesthetic management of patients presenting to the IR suite for stent placement, thrombolytic therapy, or both requires thorough planning and collaboration with the radiology team.\textsuperscript{13,16} In procedure areas other than the OR, many factors other than the patient’s comorbidities contribute to potential difficulty in a patient’s anesthetic care.\textsuperscript{15-18} Not only are these treatment areas typically foreign to the anesthetist, the procedures themselves are often outside their customary experience.\textsuperscript{15-18} The available equipment and resources frequently are not typical of those in the anesthetist’s more familiar environment, the OR.\textsuperscript{15-18} Because of the required procedure-related equipment in locations such as IR, access to the patient during treatment may be impossible.\textsuperscript{15,16} Depth of anesthesia and choice of agents and techniques may interfere with imaging or the ability to perform the procedure successfully.\textsuperscript{9} In light of these potential pitfalls, a discussion with the interventional radiologist about the patient’s condition, procedure plan and goals, and anesthetic strategy and concerns is of the utmost importance.\textsuperscript{15-18}

As demonstrated in the review of the literature, there is little reported regarding the anesthetic management of patients who are presenting to an IR procedure area (or any other non-OR procedure location) with SVC syndrome. Many of the intraoperative anesthetic management essentials broadly described for patients with mediastinal mass compression presenting to the OR, therefore, can be applied to cases of SVC syndrome caused by thrombotic occlusion that end up in a non-OR procedure site.\textsuperscript{1,2}

A careful assessment of the airway is essential.\textsuperscript{16-18} The nasal passages and the oropharynx are typically quite edematous and prone to obstruction and bleeding.\textsuperscript{1,2,6-10} Even though external compression of the SVC by tumor is not the concern in these cases, the supine position should be avoided to facilitate drainage of the thoracic bed, thereby enhancing respiratory effort.\textsuperscript{1,2,4,5,8,11,13} Because of the bleeding risk posed by venous engorgement in the head and oropharyngeal cavities, endotracheal intubation to secure the airway must be weighed based on the patient’s symptoms, potential length of procedure, hindrance to patient access, and the patient positioning required for the procedure.\textsuperscript{1,8} It is better to secure the airway with a fiberoptic bronchoscope-assisted intubation or a video laryngoscope than to risk emergent airway management and the possible need for a tracheostomy or cricothyroidotomy in a compromised patient who received anticoagulation therapy.\textsuperscript{1,2}

It is necessary to establish IV access in the lower extremities because the venous outflow of upper extremity veins may encounter obstruction at the right atrium impeding distribution of fluids and medications.\textsuperscript{1,2,4,6} The patient can be pretreated with IV dexamethasone and a racemic epinephrine nebulizer to abate potential airway edema.\textsuperscript{1,2,6,8,9,11,13} Emergency equipment such as a tracheo-
Aostomy tray and an immediately accessible head and neck surgeon should ideally be available during the procedure in a non-OR environment. The Table illustrates recommended anesthetic management for patients with SVC syndrome with the corresponding supportive literature.

In this case, a discussion with the IR team regarding patient positioning, treatment plans, and length of procedure may have changed the choice of anesthetic from light sedation to a general anesthetic with a secured airway. Oral tracheal intubation via a video laryngoscope or fiberoptic bronchoscope would have been a safe choice because of the patient’s condition, positioning, length of procedure, and potential use of antithrombolytics. A peripheral, large-bore IV inserted into the lower extremity before the procedure would have aided in the sedation and resuscitation needs of this patient.

The patient’s agitation during the case may have signaled increasing venous congestion caused by the supine positioning further compromising her respiratory efforts, and this behavior needed further investigation. Securing

<table>
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<tr>
<th>Anesthetic management recommendation</th>
<th>Source, year</th>
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<tr>
<td>Consider length of procedure, intraprocedural patient access, and positioning requirements for final airway management decisions</td>
<td>Lee et al, 2012&lt;br&gt;Wan &amp; Bezjak, 2010&lt;br&gt;Rubin, 2014&lt;br&gt;Eder &amp; Register, 2014</td>
</tr>
<tr>
<td>Discuss interventional and anesthetic needs and concerns with interventional radiology team</td>
<td>Chaudhary et al, 2012&lt;br&gt;Rubin, 2014</td>
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<tr>
<td>Secure airway with endotracheal tube if there is poor patient access, potentially lengthy procedure, or supine positioning</td>
<td>Chaudhary et al, 2012&lt;br&gt;Rubin, 2014&lt;br&gt;Eder &amp; Register, 2014</td>
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Table. Anesthetic Management Recommendations
the airway before the procedure in a controlled, safe environment is preferable to the loss of airway in an anticoagulated patient with head and neck edema.1,2

Conclusion
Superior vena cava syndrome and its exhibited symptoms create an anesthetic challenge, more so for procedures or interventions in areas other than the OR.1,4,14-18 Careful preoperative assessment, including physical examination, laboratory testing, chest radiography, CT, and MRI, is essential.1,3,6,11,16-18 Collaboration with all members of the healthcare team, in this case the interventional radiologists and IR staff, can help determine the appropriate anesthetic.14,15 Peripheral IV access in a lower extremity is necessary.1,2 The head of bed should be maintained in the sitting or semi-Fowler position, and pre-procedure treatment with corticosteroids and racemic epinephrine nebulizer may be beneficial.1,4,6,8,9,11,13 Treatment modalities, position, and length of intervention must be considered when the anesthesiology provider is deciding the anesthetic technique.1,8 Although the literature demonstrates that avoiding manipulation of the airway in these patients is preferable, the risk of respiratory obstruction and bleeding in an unsecured, edematous airway must be strongly weighed.1,2

REFERENCES

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DISCLOSURES
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