

■ CASE REPORTS

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Sulfhemoglobinemia Presenting as Pulse Oximetry Desaturation

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SULFHEMOGLOBIN is a rare cause of cyanosis that is usually drug induced. The effects of sulfhemoglobin on pulse oximetry have not been reported widely. We present the case of a 48-yr-old woman who was scheduled to undergo palliative surgery. Before anesthesia, she had an oxygen saturation, measured by pulse oximetry (SpO_2), of 85%. This apparent desaturation subsequently was discovered to be the result of a blood sulfhemoglobin concentration of 28%. She had been prescribed metoclopramide daily for more than 1 yr. Long-term ingestion of metoclopramide is a cause of drug-induced sulfhemoglobinemia.

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The patient, a 48-yr-old woman with locally advanced carcinoma of the cervix, presented for palliative surgery for a rectovaginal fistula. At admission, she was being administered 100 mg slow-release morphine (MST) twice a day and 25 mg amitriptyline at night. For the previous year, she had also been prescribed 10 mg metoclopramide three times a day for persistent nausea and vomiting. At preoperative assessment, her cardiorespiratory function was normal, although she was anemic, with a hemoglobin concentration of 8.2 g/dl.

The patient was premedicated with 2 mg oral lorazepam 2 h preoperatively. At arrival in the operating room, a pulse oximeter (Hewlett Packard 54S; Hewlett Packard, Avondale, CA) was connected to her. Her SpO_2 while breathing room air was recorded as 85%, with the sensor on her index finger. At close observation, the patient was cyanotic but not dyspneic. A radial arterial cannula was inserted, and arterial blood was analyzed. Blood gas analysis (CIBA-Corning 288; CIBA-Corning Diagnostics, Medfield, MA) showed a pH of 7.41, a partial pressure of carbon dioxide (P_{CO_2}) of 38 mmHg, and a partial

pressure of oxygen (P_{O_2}) of 99 mmHg, with the patient breathing room air. A diagnosis of methemoglobinemia was presumed, so the sample was sent for analysis by hemoximeter (Chiron Rapidlab 865; Bayer Diagnostics, Tarrytown, NY). This showed a methemoglobin concentration of 0.3% and a carboxyhemoglobin concentration of 0.3%, but sulfhemoglobin concentration greater than 1.5%. Because the hemoximeter was able to indicate only the presence of sulfhemoglobin rather than an absolute concentration, anesthesia was postponed for further investigation.

A sample of arterial blood was analyzed by use of a manual assay with a variable wavelength spectrophotometer (Cecil Series 2; Cecil Instruments, Cambridge, UK); this showed a sulfhemoglobin concentration of 28%.¹ The patient's surgery was planned for the week after blood transfusion. However, the patient decided against operative treatment. She was discharged to outpatient hospice care and declined further investigation of her sulfhemoglobinemia.

Discussion

In the case described, the presence of significant pulse oximetry desaturation associated with a normal arterial oxygen tension (Pa_{O_2}) alerted us to the possibility of an abnormal hemoglobin species interfering with the pulse oximeter. Pulse oximeters use two light wavelengths (660 and 940 nm) to determine the ratio of pulse-added absorbencies.² This ratio is associated with SpO_2 by means of a table derived from data from healthy volunteers. Dyshemoglobin molecules that have light absorbance peaks at 660 or 940 nm affect the ratio of light absorbencies at these wavelengths and lead to spurious SpO_2 readings. Methemoglobin has significant absorbencies at both of these wavelengths and has been reported widely to interfere with pulse oximetry.^{3,4} Sulfhemoglobin has a greater absorbance at 660 nm than do oxyhemoglobin, deoxyhemoglobin, and methemoglobin.⁵ We could not find data relating to the absorbance of sulfhemoglobin at 940 nm. Therefore, it is difficult to predict how increasing concentrations of sulfhemoglobin might interfere with pulse oximetry. The effects of increasing concentrations of methemoglobin on pulse oximetry have been determined experimentally in dogs.⁴ There is no similar report available for sulfhemoglobin, possibly because of the difficulty of inducing sulfhemoglobinemia *in vivo*. In a literature search, we could find only two cases of sulfhemoglobinemia in

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which the use of pulse oximetry was reported. The first report described an SpO_2 of 88%, with a sulfhemoglobin concentration of more than 1.5%, as indicated by hemoximetry.⁷ Unfortunately, although the presence of sulfhemoglobin was proven by gas chromatography, no absolute concentration was reported. The second report described an SpO_2 of 92–94%, with a sulfhemoglobin concentration of 16%, as measured by spectrophotometry.⁸ This compares with an SpO_2 of 85% associated with a sulfhemoglobin concentration of 28%, as seen in the current patient.

Hemoximeters use multiple wavelengths to determine concentrations of oxyhemoglobin, deoxyhemoglobin, carboxyhemoglobin, and methemoglobin. Some machines do not distinguish between methemoglobin and sulfhemoglobin because of their similar absorption peaks.⁹ This has caused inappropriate treatment of sulfhemoglobinemia with methylene blue.⁷ Some machines, such as the Chiron Rapidlab 865, indicate the presence of sulfhemoglobin but cannot quantify the concentration. The laboratory measurement of sulfhemoglobin relies on an absorption peak at 620 nm, which, unlike methemoglobin, persists after the addition of cyanide or dithionate.¹

Sulfhemoglobinemia is recognized as a rare cause of cyanosis, including intraoperative cyanosis.¹⁰ Sulfhemoglobin is a stable, green-pigmented molecule that lasts the lifetime of the erythrocyte. Certain drugs and chemicals cause oxidation of hemoglobin, which, with the addition of a sulfur atom, forms sulfhemoglobin. The most common drugs known to cause sulfhemoglobinemia are phenacetin, dapsone, and the sulfonamides. It has also been described with occupational exposure to sulfur compounds and with drug abuse.¹¹ Long-term metoclopramide ingestion has been reported as a cause of sulfhemoglobinemia.¹² Short-term high-dose metoclopramide therapy combined with *N*-acetylcysteine has also caused sulfhemoglobinemia.^{7,8} Our patient had been prescribed 30 mg/day metoclopramide for more than 1 yr. She was not administered any other drugs known to cause sulfhemoglobinemia. Metoclopramide is structurally related to aniline dyes and prilocaine, both of which cause methemoglobinemia. Why the same drugs can cause methemoglobinemia in some patients and sulfhemoglobinemia in others is not clear.

Central cyanosis is surprisingly difficult to detect, especially if, as in this case, the patient is asymptomatic.

Only 0.5 g/dl sulfhemoglobin is needed to cause clinically detectable cyanosis, as compared with 1.5 g/dl methemoglobin and 5 g/dl deoxygenated hemoglobin.⁵ Sulfhemoglobin cannot carry oxygen; however, high concentrations of sulfhemoglobin are well-tolerated, despite the resulting physiologic anemia. This is caused by a right shift in the hemoglobin-oxygen dissociation curve of the normal heme in the presence of sulfhemoglobin, thus facilitating tissue oxygenation. This is in contrast to methemoglobin, which causes a left shift in the curve. This can result in severely impaired tissue oxygenation at higher concentrations. There is no specific treatment for sulfhemoglobinemia, other than removing the suspected cause. The concentration of sulfhemoglobin decreases as erythrocytes are destroyed and replaced.

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Neuropathic Pain following Cervical Epidural Steroid Injection

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CERVICAL epidural steroid injections (CESI) are used commonly for the treatment of cervical radiculopathy. Significant subjective patient satisfaction has been reported, although controlled trials have not yet delineated the effectiveness of this procedure.¹⁻³ Complications of CESI are uncommon, the most frequent being dural puncture and transient paresthesia.^{1,3} Two recent reports of more serious sequelae associated with CESI bring into question the risk-benefit ratio of attempting this technique in conservative management of cervical pain.^{3,4} We report three cases of severe pain consistent with nerve injury immediately after CESI.

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Case 1

A 33-yr-old man with a history of worsening neck pain that radiated to the left second and third fingers was referred for consideration of CESI. The patient reported no weakness or sensory change in the arms

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Key words: Analgesia; epidural; spinal cord injuries; spinal nerve roots.

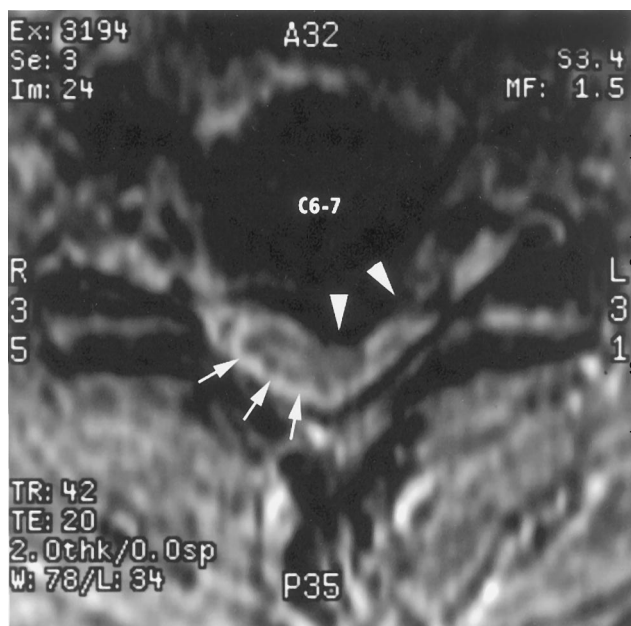


Fig. 1. Axial T₁-weighted magnetic resonance image at the C6-C7 level of the patient described in case 1. There is a large central herniated intervertebral disk that lateralizes to the left (arrow heads). The spinal cord is displaced toward the right posterior portion of the spinal canal (arrows).

or legs. Results of physical examination were normal, except for slight decrease in sensation to light touch over the dorsal side of the left second finger and absence of the left triceps deep tendon reflex. Magnetic resonance imaging (MRI) showed a large central herniated nucleus pulposus at the C6-C7 interspace, lateralizing to the left, with displacement of the spinal cord toward the right (fig. 1). Posterior displacement of the spinal cord and effacement of the cerebrospinal fluid signal that is normally anterior to the spinal cord was evident on the sagittal image (fig. 2).

Cervical epidural steroid injection was performed with the patient sitting, with his neck flexed and his head resting on a cushion. A 17-gauge Weiss epidural needle was inserted through the C6-C7 interspace *via* a midline approach. The epidural space was identified using the hanging-drop technique, with saline administered at a depth of 5 cm from the skin. Immediately after entry of the needle into the epidural space, the patient experienced a shock-like sensation, extending to the right upper and lower extremities that resolved instantaneously. No blood or cerebrospinal fluid was aspirated from the needle. The transient paresthesia had completely resolved, and instillation of an additional 1 ml preservative-free saline produced no further

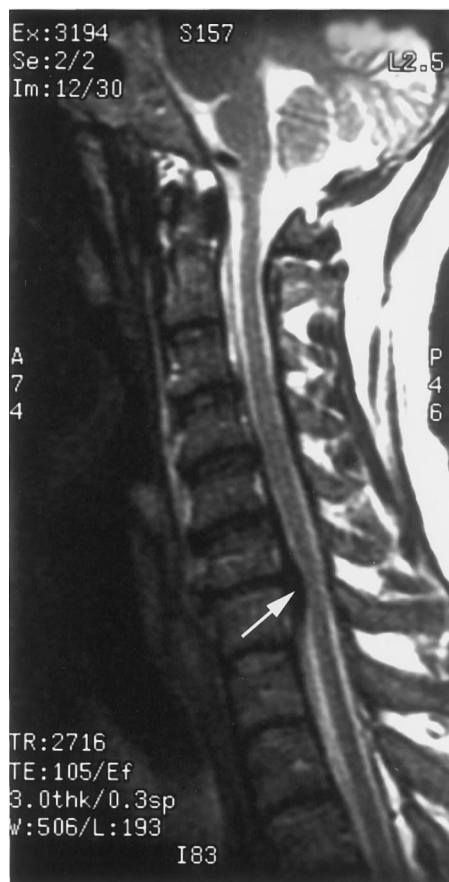


Fig. 2. Sagittal T₂-weighted image at the midline of the cervical spine of the patient described in case 1. There is a large disk herniation at the C6–C7 level, causing posterior displacement of the spinal cord (arrow) and effacement of the cerebrospinal fluid signal anterior to the spinal cord.

symptoms. A mixture of 80 mg methylprednisolone (2 ml) and 0.5% lidocaine (2 ml) was administered through the epidural needle during gravity flow, without further discomfort. Then, the needle was withdrawn. Several minutes after the procedure, the patient reported a dull ache in the web space between the right first and second fingers. Examination showed pronounced allodynia in the same area. The pain and allodynia in the right hand persisted through the patient's discharge from the clinic, 30 min later.

The day after CESI, the patient reported worsened pain and extreme sensitivity to touch in his right hand. Physical examination showed marked allodynia, warmth, and erythema in the web space between the right first and second fingers. Strength and deep tendon reflexes were normal in the right upper extremity. He was prescribed a tapering dose of oral steroids and oral oxycodone-acetaminophen, and his symptoms resolved over 3 days. Seven days after CESI, there was no further allodynia in the right hand. The original radicular symptoms in the left C7 distribution slowly resolved over 3 weeks. At the last follow-up, 6 months after CESI, the left C7 radicular symptoms continued to increase and decrease in intensity, but the patient experienced no further severe exacerbations.

Case 2

A 49-yr-old man with a 3-month history of worsening neck pain that radiated to both arms was referred for CESI. Five years previously, the patient had undergone anterior cervical discectomy and fusion at the C5–C6 and C6–C7 levels.

The patient reported no weakness or sensory change in the arms or legs. At physical examination, marked limitation of cervical range of motion in flexion and extension was noted. Sensory, motor, and deep tendon reflex examination in all extremities yielded normal results. MRI showed intact fusion at the C5–C6 and C6–C7 levels. An osteophytic ridge extended posteriorly into the anterior epidural space but caused no displacement of the spinal cord or effacement of the cerebrospinal fluid signal anterior to the spinal cord. There was no stenosis of the central spinal canal or the lateral recesses at any of the cervical levels.

Cervical epidural steroid injection was performed as described in the first case. Immediately after entry of the needle into the epidural space, the patient experienced an aching sensation that extended from the left shoulder to the left fifth finger. No blood or cerebrospinal fluid was aspirated from the needle. The transient paresthesia resolved after several seconds. A mixture of 120 mg methylprednisolone (3 ml) and 1% lidocaine (2 ml) was administered through the epidural needle, and the needle was withdrawn without further discomfort. The patient had ongoing discomfort in the left arm at the time of discharge from the clinic, 30 min later.

The day after CESI, the patient returned to the clinic, reporting pain that extended from the left forearm into the third through fifth fingers of the left hand, with burning and sensitivity to light touch, and swelling of the dorsum of the hand. Physical examination showed marked allodynia, warmth, erythema, and swelling of the left hand including the third through fifth fingers. There was decreased sensation to pin prick in the same area. He was prescribed a tapering dose of oral steroids and analgesics (hydrocodone-acetaminophen). He returned weekly during the next month, and his symptoms gradually resolved without further intervention. Sensitivity to cold that was not otherwise painful persisted in the left third through fifth fingers for more than 6 weeks after CESI. At the last follow-up, 3 months after CESI, the left-sided symptoms had resolved; however, the neck and right arm pain persisted.

Case 3

A 54-yr-old woman with a 10-yr history of pain and tingling in the left arm and hand, which was diagnosed as cervical radiculopathy, was referred for CESI. Her symptoms had responded to previous CESI and recurred 5 months before her presentation to the clinic. Her history was significant for stroke 3 months previously, which had initially caused left-sided weakness and decreased sensation. Physical examination showed weakness in all hand muscles and decreased sensation to pin prick in the entire left hand to the mid forearm, consistent with residual from the stroke. MRI of the cervical spine showed moderate C4–C5 disk herniation, with mild compression of the spinal cord at that level, and moderate disk protrusion at C6–C7, with facet joint hypertrophy, causing foraminal narrowing at the left C6–C7 level. CESI was performed at the C6–C7 level without complication and produced relief for approximately 2 weeks. The patient returned 1 month later for a subsequent CESI. The epidural space was located using the hanging-drop technique at C6–C7. A solution of 80 mg methylpred-

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nisolone (2 ml) and 2 ml lidocaine, 0.5%, was administered through the epidural needle during gravity flow. The solution stopped flowing after half of the solution had been instilled. The needle was withdrawn and reinserted through the same interspace, this time using loss of resistance to air to locate the epidural space. During needle advancement, the patient reported a severe shooting pain to the left hand. The procedure was discontinued, and paresthesia resolved completely after several minutes. The patient returned 4 days later, reporting severe pain, swelling, and allodynia in the left hand. Examination results were not appreciably changed from baseline, except for hand swelling. She was administered oral dexamethasone and gabapentin. Symptoms gradually resolved over 3 months. She continued to experience neck pain that radiated into her left arm, and, 10 months later, she underwent anterior cervical discectomy at the C6–C7 level. The appearance of the C7 nerve root was unremarkable intraoperatively. Neck and left arm pain resolved after surgery.

Discussion

There have been two previous reports of neurologic sequelae similar to those we described.^{3,4} Siegfried⁴ reported a patient with lancinating pain in the right arm after CESI as the epidural needle was withdrawn. Allodynia occurred 6 h later in the same extremity, associated with symptoms of increased sympathetic activity (cool, swollen arm). CESI was performed without sedation using a midline approach and the hanging-drop technique at the level of spinal cord impingement shown by computed tomography (C5–C6). No cerebrospinal fluid or blood was aspirated during the procedure, but, because a local anesthetic test dose was not used, dural puncture could not be excluded.

Hodges *et al.*³ reported two cases of CESI with use of fluoroscopic guidance in sedated patients. For both, injections were performed at the level of disk herniation shown by MRI (C5–C6) and resulted in subarachnoid placement of the needle, with return of cerebrospinal fluid. Although neither patient reported symptoms during the procedure, both awoke with new neurologic symptoms and subsequently had spinal cord injury shown by MRI and permanent neurologic sequelae. The authors concluded that use of fluoroscopic guidance does not prevent dural puncture and that excessive sedation may mask warning signs of neural impingement during CESI.

We report three patients in whom signs and symptoms of nerve injury developed after CESI using a standard technique without sedation. The cases of nerve injury occurred with different practitioners in different practice locations. No patient had evidence of dural puncture or intraneural injection. A test dose of local anesthetic was not used in any of these cases. However, a small

dose of local anesthetic was mixed with the steroid in all cases, and none of the patients had evidence of spinal block after injection.

At least two mechanisms can explain the neuropathic pain experienced by these patients. Each patient reported painful paresthesia during CESI, suggesting that the needle tip was near an exiting nerve root. The symptoms were transient, and we administered methylprednisolone in each case. The methylprednisolone formulation used (Depo-Medrol; Pharmacia and Upjohn Companies, Kalamazoo, MI) is a particulate suspension of steroid in polyethylene glycol. This formulation has been implicated as a cause of neural toxicity. Perhaps deposition of the steroid near the exiting nerve root led to nerve root irritation.

Compromise of the spinal canal by a large herniated intervertebral disk or other anatomic abnormality that causes posterior displacement of the spinal cord toward the ligamentum flavum can result in spinal cord damage during epidural placement, without dural puncture.^{2,3} Imaging studies in case 1 showed marked posterolateral displacement of the spinal cord caused by a herniated disk (figs. 1 and 2); compression of the spinal cord was also shown by MRI in case 3. In case 2, there was no spinal cord effacement or displacement. However, this patient had undergone previous cervical fusion, and a small osteophytic ridge was present, extending from C5–C7 along the posterior part of the vertebral bodies. Loss of cervical mobility, combined with this osteophytic ridge, may have resulted in cervical cord displacement when the neck was flexed forward during CESI.

We hypothesize that neuropathic pain after CESI may result from either a direct nerve root irritation caused by the steroid solution or damage to the spinal cord or nerve roots without dural puncture by minor compression of neural elements. When a patient reports paresthesia during CESI, even if it is transient, the needle should be removed and placed at an adjacent level. This will avoid insertion of the steroid solution immediately adjacent to a nerve root and possibility of direct chemical neuritis. Clinicians should be fully aware of the nature of the disease, including any distortion of the spinal cord before performing CESI. Evaluation of imaging studies will allow rational decisions about where the injection should be positioned.

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Pulmonary Hemorrhage Associated with Negative-pressure Pulmonary Edema

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NEGATIVE-PRESSURE pulmonary edema (NPPE) is an infrequent complication of acute airway obstruction, such as what can occur with laryngospasm. Typically, this complication develops in young, healthy patients without underlying disease. We describe a case of severe endobronchial and alveolar hemorrhage that complicated postextubation laryngospasm.

Case Report

An otherwise healthy 33-yr-old woman underwent elective outpatient septoplasty at our institution for relief of chronic nasal obstructive symptoms. The patient quit smoking 2 yr before the surgery. She had previously undergone general anesthesia once, without complications. There was no family history of difficulties with anesthesia. The patient's height was 160 cm, she weighed 70 kg, and results of her physical examination were normal.

After premedication with midazolam, anesthesia was induced with use of propofol, lidocaine, and fentanyl intravenously, followed by rocuronium to facilitate tracheal intubation. Anesthesia was maintained with sevoflurane in 70% nitrous oxide and 30% oxygen. The surgery was uneventful. As the procedure concluded, nasal packing

was inserted, neostigmine and glycopyrrolate were administered, and administration of inhalational agents was discontinued. The patient initiated spontaneous ventilation but began coughing vigorously. She reached for the endotracheal tube and was extubated subsequently. Her ventilatory efforts resulted in chest wall retraction, without apparent air movement for approximately 45 s. Continuous positive airway pressure with 100% oxygen was applied using a face mask. This failed to relieve the laryngospasm, and 30 mg succinylcholine was administered intravenously. The patient was reintubated without trauma to the trachea. Neither gastric contents nor blood was present in the oropharynx. Oxygen saturation measured by pulse oximetry (SpO_2), which had decreased to approximately 70% before administration of succinylcholine, improved after intubation; however, 8 cm H_2O positive end-expiratory pressure (PEEP) and 100% oxygen were needed to increase SpO_2 to 100%. Blood pressure decreased to 77/50 mmHg but returned to 100 mmHg systolic after administration of 5.0 mg intravenous ephedrine and a total of 900 ml crystalloid. Bilateral rhonchi were audible during auscultation and a frothy serosanguinous fluid was suctioned from the endotracheal tube. Within 5 min, this fluid became progressively more bloody. A chest roentgenogram obtained in the recovery room showed perihilar interstitial and alveolar opacification consistent with pulmonary edema, with a normal-sized heart (fig. 1). Peribronchial cuffing and air bronchograms were noted in the upper lung fields. The first arterial blood gas measurement obtained within 20 min of the initial event showed a pH of 7.34, an arterial carbon dioxide tension ($Paco_2$) of 39 mmHg, and an arterial oxygen tension (PaO_2) of 61 mmHg, with a fractional inspired oxygen tension (FIO_2) of 1.0. As the neuromuscular blockade regressed, the patient experienced paroxysmal coughing, which seemed to exacerbate pulmonary bleeding. She was administered 25-35 $\mu g \cdot kg^{-1} \cdot min^{-1}$ propofol by continuous infusion, 4 mg pancuronium bromide for neuromuscular blockade, and 10 mg furosemide. Mechanical ventilation was reinitiated. She was transferred subsequently to the intensive care unit with an FIO_2 of 0.6, a PEEP of 8.0 cm H_2O , and a respiratory rate of 10 breaths/min. With these settings, arterial blood gas measurements showed a pH of 7.35, a $Paco_2$ of 40 mmHg, and a PaO_2 of, again, 61 mmHg. During the next 4 h, respiratory and cardiovascular conditions worsened, necessitating increased FIO_2 to 1.0, a PEEP of 15 cm H_2O , and vasopressor support with use of 8.0 $\mu g \cdot kg^{-1} \cdot min^{-1}$ dopamine. The patient's condition and course were consistent with alveolar hemorrhage (fig. 2). Pulmonary and radial arterial monitoring were performed. Initial pulmonary

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Fig. 1. Chest roentgenogram obtained in the postanesthesia recovery room: bilateral interstitial and alveolar infiltrates with right upper-lung air bronchograms.

arterial readings included cardiac output, 4.9 l/min; pulmonary arterial pressure, 29/17 mmHg; pulmonary arterial occlusion pressure, 9 mmHg; and systemic vascular resistance, 950 dyne \cdot s \cdot cm⁻⁵. Transthoracic echocardiography showed an ejection fraction of 63% and normal chamber size. Empiric antibiotic coverage with alatrofloxacin was initiated. The patient continued to experience pulmonary hemorrhage. Her hemoglobin concentration decreased by 3.0 g/dl to 7.7 g/dl over 7 h. Bronchoscopy and open-lung biopsy were thought to be contraindicated because of the patient's respiratory status, which necessitated a PEEP of 15 cm H₂O and an F_{IO}₂ of 0.85 to yield a P_{aO}₂ of 61 mmHg. She continued to bleed extensively, resulting in transfusion of 2 units of packed erythrocytes. On the second day in the hospital, she continued to have extensive pulmonary bleeding. Decreasing arterial oxygenation necessitated an increase in F_{IO}₂ to 1.0. In addition to general supportive measures, administration of cyclophosphamide, corticosteroids, and plasmapheresis was started for presumed Wegener vasculitis, capillaritis, or Goodpasture syndrome. On the fourth postoperative day, the patient's condition began to show marked improvement. Oxygenation improved dramatically, and F_{IO}₂ was reduced to 0.4. Neuromuscular blockade was discontinued, sedation was decreased, and ventilatory support was reduced. On the sixth postoperative day, she was extubated. Tests for human immunodeficiency virus, lupus anticoagulant, antinuclear antibodies, antineutrophil cytoplasmic antibodies, and anti-glomerular basement membrane antibodies all yielded negative results, and multiple blood and urine

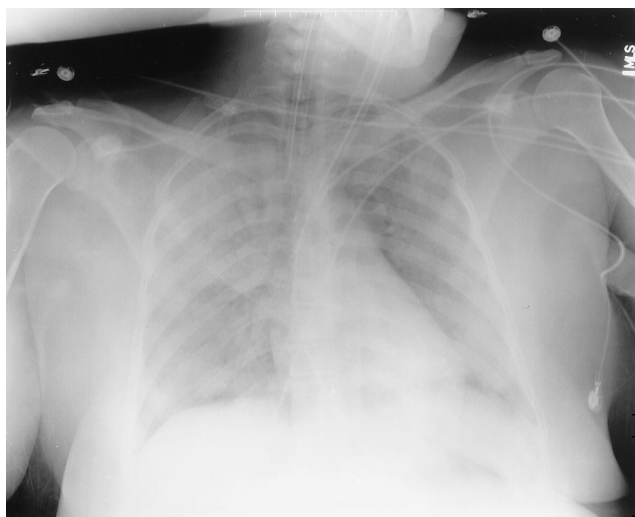


Fig. 2. Chest roentgenogram obtained in the intensive care unit 7 h after the roentgenogram obtained in the postanesthesia recovery room. Dense alveolar infiltrates are shown.

cultures had no microbial growth. Plasmapheresis and immunosuppressants were discontinued. She was discharged from the hospital after 2 weeks, with normal results of chest roentgenograph. Follow-up at 21 months showed no functional deficit, and, therefore, no pulmonary function tests were performed.

Discussion

We describe the case of a 33-yr-old woman whose routine surgery with general anesthesia was complicated by laryngospasm, followed by NPPE and alveolar hemorrhage. Aspiration, blood from the surgical site, fluid overload, and anaphylactoid reaction were ruled out as possible causes. The initial chest roentgenogram, arterial blood gas, and presentation after nasal surgery were characteristic of NPPE, except for the presence of pulmonary hemorrhage. Although a possible cause could have been disruption of bronchial vessels, which has been implicated as the cause of frank hemorrhage,¹ worsening oxygenation, continued hemorrhage with decreasing hematocrit concentration, and alveolar consolidation shown by chest roentgenography indicate alveolar hemorrhage.

Laryngospasm is a common complication after tracheal extubation, occurring in as many as 8–237 of every 1,000 anesthetic procedures.^{3–5} NPPE has been reported in 11% of healthy young patients who experience laryngospasm.⁶ These data suggest that the incidence of NPPE may be as high as 1 in 1,000 anesthetic cases. Despite these statistics, the presence of pulmonary hemorrhage complicating NPPE has not been extensively reported.

Frank pulmonary hemorrhage associated with NPPE is rare, with only one report in the English literature.⁷ Two other case reports describe bleeding from bronchial vessels.^{1,2} Goldenberg *et al.*⁸ reported the development of tricuspid and pulmonary insufficiency in two of six patients with postlaryngospasm NPPE. The current patient had only isolated, mild tricuspid regurgitation shown by echocardiographic examination. This may have been the result of higher pulmonary artery pressure development secondary to hypoxemia because she required increasingly higher levels of F_{iO_2} and PEEP.

Negative-pressure pulmonary edema is the result of three pathophysiologic processes: negative intrathoracic pressure,⁶ increase of systemic vascular and pulmonary capillary hydrostatic pressure, and mechanical stress at the alveolar-capillary level.⁹ First, negative intrathoracic pressure increases venous return by reduction in right atrial pressure, with a concomitant increase in pulmonary arterial and pulmonary capillary hydrostatic pressures, complicated by a reduction in perivascular interstitial hydrostatic pressure.⁹ This causes an increased transcapillary pressure gradient, favoring the transudation of fluid into the interstitial space. In addition, increased central venous pressures impede passive lymphatic blood flow.¹⁰ Second, pulmonary blood volume is augmented by increases in systemic pressure secondary to the release of norepinephrine in response to hypoxia, hypercapnia, and anxiety.⁷ The effect is distention of the right ventricle, leading to interventricular septal shift and a resultant reduction in cardiac output.⁷ The increase of systemic vascular resistance further increases left ventricular wall tension, which contributes to impediment of left ventricular ejection.¹¹ However, the classic description of NPPE includes serosanguinous or pink, frothy fluid, which implies some capillary leak of proteinaceous material. Therefore, the third invoked process indicates that this capillary leak could be a result of mechanical failure of the alveolar-capillary membrane, resulting in alveolar edema or frank hemorrhage.^{12,13} The actual negative-pressure threshold for pulmonary microvasculature disruption is not known in humans. In a rabbit model, this stress failure occurs at a microvasculature pressure of approximately 40 mmHg.¹⁴ Marked negative intrapleural pressures have caused increased capillary permeability in a rabbit model of reexpansion pulmonary edema.¹⁵

Laryngospasm after tracheal extubation was observed in the patient described, and the subsequent development of NPPE was not unexpected. However, the dramatic and prolonged pulmonary hemorrhage forced us to consider other causes. These included Wegener granulomatosis, Goodpasture syndrome, capillaritis, vasculitis, and infection. Each of these potential causes was treated empirically in this patient, although each was ruled out subsequently by studies that yielded negative results. This patient shows that frank pulmonary hemorrhage can complicate NPPE after laryngospasm.

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The shape of the preformed maxillofacial tube makes the use of a tube exchanger difficult. In the literature, there is a description of bypassing a tear with use of a smaller endotracheal tube inside the lumen of the severed regular tube,^{4,6} but there is no report of replacing or bypassing a damaged site of a special tube such as the one used in this case.

The cyanoacrylate glue used to seal the severed tube usually is indicated for use on live human tissue, such as in skin tears, to approximate wound edges or small surgical incisions. This topical skin adhesive is a sterile liquid that contains a monomeric (2-octyl cyanoacrylate) formulation. When applied, the liquid polymerizes within minutes. It is also used extensively in dental medicine as a tooth adhesive. The *in vitro* experiment we conducted prompted us to repeat it successfully *in vivo*. The glue should not be used on mucosal surfaces; therefore, we protected the nasal mucosa with gauze around the tube. The same experiment was performed on a clear polyvinyl chloride tube. However, in this case, 2 or 3 min were needed to achieve a complete seal of the tear.

Every anesthesiologist participating in the described

type of surgery should be aware of the possible complication of damage to the tube during a LeFort osteotomy. When replacing the damaged tube is not the solution of choice, sealing the partially severed tube with cyanoacrylate glue might be an acceptable option.

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Fatal Barotrauma Resulting from Misuse of a Resuscitation Bag

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MANUAL resuscitation bags may be assumed mistakenly to be simple devices, but they consist of multiple valves

to create positive pressure, permit exhalation, and segregate inspiratory and expiratory gases. Erroneous assembly or misuse can profoundly impact patient outcome. We wish to draw attention to such a case which had fatal consequences. Although the resuscitation bag was not used as intended by the manufacturer, we believe that a well-designed device should not permit a misassembly with such serious consequences. We wish to draw attention to the potential hazards of failing to monitor airway pressure during bag-mask ventilation.

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Key words: Case report; complication; equipment design; pulseless electrical activity.

Case Report

A 27-yr-old woman with stage 2B large-cell lymphoma was admitted to the hospital for treatment of spinal cord compression. The results of magnetic resonance imaging were consistent with thoracolumbar nodules, and the patient was treated using intrathecal cytosine arabinoside

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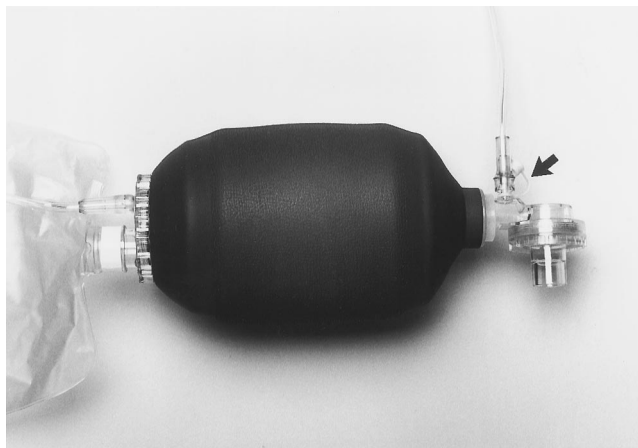


Fig. 1. FeatherFlex manual resuscitation bag with proximal manometry nipple (indicated by arrow) erroneously connected to oxygen tubing. Note the distal oxygen tubing, which is integral to the device.

and methotrexate, in addition to local irradiation. Her disease was complicated further by intermittent fevers, which yielded negative cultures. Three weeks before her death, bilateral perihilar infiltrates developed, along with increasing oxygen requirements, indicative of radiation pneumonitis or opportunistic infection. Empirical antibiotic treatment was initiated. Results of bronchoalveolar lavage were consistent with cytomegalovirus and *Pneumocystis carinii* pneumonia infection, and ganciclovir and γ -globulin were added.

Despite maximal oxygen therapy with use of a face mask, arterial oxygen saturation (SpO_2) decreased to 85%. The patient and her family had previously requested full resuscitation. After sedation with intravenous morphine and diazepam, tracheal intubation was attempted but was unsuccessful. Glottic visualization and tracheal intubation were finally achieved and confirmed by auscultation. Attempts to transfer the patient to the critical care unit were complicated by a decrease in SpO_2 to 70%, prompting a return to her room. Manual ventilation using a disposable self-inflating bag (FeatherFlex; Brathwaites-Olivier, Winnipeg, Canada) did not correct the hypoxemia. The reservoir bag was not filling despite high flow, and a decision was made to increase the oxygen supply by connecting a second flowmeter to a proximal nipple (fig. 1). The reservoir bag immediately became distended, and subcutaneous emphysema, abdominal distention, and pulseless electrical activity were noted. Nasogastric tube insertion and bilateral needle thoracostomies were performed with 14-gauge catheters. An air leak was noted, and chest tubes were inserted bilaterally. Incorrect endotracheal tube placement was suspected; the tube was withdrawn and replaced without difficulty. Chest compressions were commenced, and intravenous epinephrine was administered, resulting in restoration of a pulse. Profound hypoxemia persisted, and further resuscitative efforts were deemed futile. Consent for an autopsy could not be obtained.

Discussion

The resuscitation bag used was not available for subsequent evaluation. However, an identical device was connected to a test lung (Lung Simulator, Model LS122; Medishield, Harlow-Essex, UK) to recreate the problem. The initial failure to inflate the reservoir bag could not be reproduced. We do not have an adequate explanation for this event. Both flowmeters were functional. It is possible that there was a kink in the oxygen tubing.

We demonstrated that the addition of oxygen flow via the proximal nipple, intended for airway pressure monitoring, distorted the inspiratory duckbill valve and prevented exhalation. This is the probable cause of the fatal barotrauma experienced by this patient.

The device was not used as the manufacturer had intended. It is our opinion, however, that such a design is potentially dangerous. Airway-pressure monitoring is desirable to avoid exposing the lungs to excessive pressure. High airway pressures can inadvertently be caused with use of numerous manual resuscitator bags (manuscript in preparation). It is our belief that a manometry connection should be designed so that erroneous assembly will not result in valve malfunction. Ideally, the manometry nipple should also be incompatible with oxygen tubing.

It is not clear whether the effect on exhalation was design fault or a production fault. The device used is manufactured by Brathwaites but widely sold by other vendors, with various private labels. In Canada, Brathwaites-Olivier has voluntarily recalled and replaced all manual resuscitators with manometry ports (D. Olivier, president, Brathwaites-Olivier, written communication, April 1999). They have also made modifications to an expiratory valve to prevent excessive pressure production within the bag (D. Murdoch, representative, Brathwaites-Olivier, oral communication, December 1999). Such initiatives may not have taken place in other jurisdictions. The described modifications may reduce the likelihood of such an occurrence, but product familiarity and proper assembly are vital. The purpose of this report is to draw attention to the potential consequences of faulty use and to encourage proper use of manometry or the purchase of bag valve devices that do not have manometry nipples.

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Paradoxical Vocal Cord Adduction

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UPPER airway obstruction in the immediate postoperative period necessitates expeditious diagnosis and treatment. The differential diagnosis of acute upper airway obstruction is distinguished by airway trauma, vocal cord paralysis, vocal cord edema, foreign body aspiration, and anaphylaxis. The differential diagnosis also is distinguished by the possibility of nonorganic upper airway obstruction associated with paradoxical vocal cord adduction (PVCA) during inspiration. Most patients with this syndrome have a long-standing history of upper airway obstruction that has been diagnosed as asthma. We present a case of upper airway obstruction immediately after extubation that was the result of paradoxical vocal cord motion in a previously asymptomatic patient.

Case Report

A 51-yr-old woman originally presented with hematuria and was scheduled to undergo transurethral resection of a bladder tumor. The patient's medical history included intermittent tobacco use but no cardiac or respiratory disease. Surgical history included cesarean section and appendectomy, both of which were performed with general anesthesia. The patient was not taking medication and indicated an allergy to sulfa drugs.

Physical examination showed normal vital signs. The patient was 1.63 m tall and weighed 82 kg. Auscultation showed that the patient's lungs were clear; her airway was normal, as was the rest of her physical examination. A preoperative electrocardiograph, chest radiograph, and laboratory data were all within normal limits.

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Key words: Airway obstruction; asthma; conversion reaction.

On the morning of surgery, the patient was sedated with intravenous midazolam (2 mg) and taken to the operating room. After application of standard monitors, the patient was preoxygenated, and general anesthesia was induced using thiopental and fentanyl. Rocuronium was administered, and the patient was intubated by direct laryngoscopy, without apparent trauma. The patient tolerated the procedure well, which was completed approximately 50 min after induction. The patient was administered neostigmine and glycopyrolate and was extubated after following commands and performing a sustained head lift.

Immediately after extubation, the patient exhibited stridor. She was administered 100% oxygen with use of a positive-pressure face mask and she maintained an oxygen saturation of 99-100% and hemodynamic stability. However, when positive pressure was released, the patient had increased stridor, and oxygen saturation decreased to 90-93%. The patient was not wheezing and was able to phonate. The patient showed a complete return of neuromuscular function; however, given the patient's stridor, an additional dose of neostigmine and glycopyrolate was administered and a nasal airway was placed. Neither of these measures resulted in change in the patient's clinical condition.

The patient was taken to the recovery room, where she was administered an aerosol treatment of racemic epinephrine, which did not significantly change her clinical condition. During this time, it was noted that the patient's stridor occurred primarily during inspiration and did not continue through expiration.

An otolaryngologist was consulted to perform indirect laryngoscopy, which revealed adduction of the vocal cords during inspiration. The vocal cords were not edematous and moved freely, openly, and completely with expiration. No evidence of vocal cord trauma or ligamentous injury was noted. The patient was treated with 1 mg midazolam and 25 mg meperidine. The patient's stridor began to subside within 10 or 2 min. She was allowed to rest undisturbed, and, within 10-15 min, her stridor subsided completely.

The patient was discharged to the hospital ward after an otherwise uneventful recovery period, and she was discharged from the hospital the next day, without further sequela.

Discussion

Paradoxical vocal cord adduction is a relatively uncommon cause of upper airway obstruction. Several reports have described patients with functional upper airway obstruction, including patients who have shown PVCA.¹⁻⁷ Patients presenting with PVCA have received a wide range of unnecessary treatments, from treatment for symptoms incorrectly diagnosed as asthma to intuba-

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tion and tracheotomy, and the diagnosis of PVCA usually is made only after a long period of ineffective treatment. The case presented is unusual in that it involves a patient without a history of respiratory disease who was diagnosed with PVCA during a short-term episode of upper airway obstruction.

Many patients presenting with PVCA are diagnosed initially with other forms of airway disease and undergo years of treatment. Additionally, there have been several reports of patients who have undergone repeated intubation for acute exacerbation of symptoms incorrectly diagnosed as asthma, only to have complete resolution of their symptoms after intubation.¹⁻³

Most patients with PVCA have a long history of airway or respiratory complaints. Usually, these patients are female, are younger than 40 yr of age, and have jobs in the healthcare field.⁴ Furthermore, the appearance of hypoxemia is considered to be uncommon in these patients.⁸ PVCA is considered to be an hysterical conversion reaction, and many patients have been treated using speech therapy that involves relaxation techniques, verbal support, placebo treatment, phonation therapy, and psychiatric or psychologic support.³

This case is unique in several respects. Although the patient was female, she did not have history of airway or respiratory disease, she was considerably older than most patients diagnosed with PVCA, and she was not involved in the healthcare industry. After extubation, the patient began to show signs of airway obstruction. The patient was oxygenated with supplemental oxygen, and treatments for possible causes of airway obstruction were instituted. During this time, otolaryngologic consultation was obtained, and the diagnosis was deter-

mined. This allowed treatment of the patient with use of sedatives and analgesics. Despite the continuation of the patient's symptoms, she was allowed to rest undisturbed, and, gradually, her symptoms subsided.

Paradoxical vocal cord adduction is an uncommon cause of upper airway obstruction, especially in a previously asymptomatic patient. However, the diagnosis must be considered when other causes of acute airway obstruction appear unlikely. Prompt and early diagnosis of PVCA could allow treatment of the patient with use of sedatives at a time when sedation would otherwise be considered inadvisable. This may allow appropriate treatment of the patient's condition and possibly spare the patient prolonged and repeated intubation.

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