THE Bispectral Index (BIS) is increasingly used to monitor the level of (un)consciousness during surgical anesthesia and conscious sedation.1 Generally, an intraoperative BIS of 40–60 is considered sufficient to maintain adequate hypnosis for surgery.2,3 Recently, a new version of the BIS® monitor has been introduced: the BIS-XP® (Aspect Medical Systems, Newton, MA). The BIS-XP® is said to exhibit improved resistance to artifacts from electrocautery devices and to detect and filter interference from electromyographic activity and other conditions commonly encountered during monitored anesthesia care sedation that may cause artifacts.#

We report three cases in which volunteers receiving combinations of propofol and midazolam as part of a pharmacokinetic–dynamic interaction study remained responsive to verbal command, although the BIS-XP® values were at, or just above, 40. A Web enhancement is provided with an MPEG1 digital video file displaying the responsiveness of one of the volunteers in relation to the BIS-XP® values.

Case Reports

With the approval of the Leiden University Medical Center ethics committee and informed consent from the subjects, a study on the pharmacokinetic–dynamic interaction between propofol and midazolam at varying concentration combinations was performed. For each subject, the electroencephalogram was recorded continuously using the BIS® Quatro sensor (Aspect Medical Systems), placed as prescribed on the left side of the skull and connected to the BIS-XP®. For all subjects, the impedance was low (on the order of 2–4 kΩ), and the signal quality index was high (0–100; well above 50) at the times of sedation assessments. The processed electroencephalogram variables were stored on a disk for off-line analysis. In addition, the electrocorticogram, transcutaneous arterial oxygen saturation, end-tidal carbon dioxide concentration, respiratory rate, and arterial blood pressure were monitored continuously throughout the study.

The volunteers breathed spontaneously through a mask with an inspiratory oxygen fraction of 40%. All three volunteers maintained adequate spontaneous respiration and were hemodynamically stable throughout the study. After a 10-min baseline recording period, a target-controlled infusion of propofol was started using the Diprifusor® (AstraZeneca, Macclesfield, United Kingdom) with maintenance of a constant target propofol concentration for 455 min. Fifteen minutes after the start of the target-controlled infusion of propofol, midazolam was given as a rapid infusion for 1 min, followed by a slower continuous infusion for 59 min. At regular intervals, when blood samples were taken from the arterial line for analysis of blood midazolam and propofol concentrations, the level of sedation was assessed by verbal command and/or mild prodding.

**Case 1**

The first subject was a 27-yr-old man who weighed 85 kg and was 185 cm tall. The target propofol concentration for this subject was 0.6 μg/ml, and the initial and secondary midazolam infusion rates were 0.05 mg · kg⁻¹ · min⁻¹ and 0.05 mg · kg⁻¹ · h⁻¹, respectively (total midazolam dose in 60 min, 8.5 mg). For the awake volunteer, the BIS exceeded 95 in the absence of any medication. Then, with the target propofol concentration of 0.6 μg/ml, the BIS was maintained at 97 after blood-effect site equilibration (fig. 1). Thereafter, during the first 40 min after the start of the midazolam administration, the BIS decreased to ∼60 during unstimulated periods and increased to 98 after verbal stimulation. Forty minutes after the start of the midazolam infusion, the BIS gradually decreased further to 40 at the end of the midazolam infusion. Unexpectedly, throughout the study period the volunteer remained responsive to verbal commands and/or mild prodding at the shoulder to a degree equivalent to an Observer’s Assessment of Alertness/Sedation score between 2 and 4, even at BIS levels of 40–45.

**Case 2**

The second subject was a 25-yr-old man who weighed 100 kg and was 195 cm tall. The target propofol concentration for this subject was also 0.6 μg/ml, and the initial and secondary midazolam infusion rates were 0.05 mg · kg⁻¹ · min⁻¹ and 0.05 mg · kg⁻¹ · h⁻¹, respectively (total midazolam dose in 60 min, 10 mg). For the awake volunteer, the BIS exceeded 95 in the absence of any medication. Then, with propofol at a target concentration of 0.6 μg/ml, the BIS was maintained at 97 after blood-effect site equilibration (fig. 2). Thereafter, within 3 min after the start of the midazolam administration, the BIS decreased to 67 and gradually decreased further to as low as 40 at the end of, and just after termination of, the midazolam infusion. Again, throughout the study period the volunteer remained responsive to verbal commands and/or mild prodding at the shoulder to a degree equivalent to an
Command and/or mild prodding at all times within the grid lines. The volunteer was responsive to verbal commands while receiving a combination of propofol and midazolam. In our hospital, we tend to administer propofol infusion regimens during propofol–opioid anesthesia on the basis of the BIS level. Based on the current literature, we advise our residents to maintain the BIS level between 40 and 60 intraoperatively. Most of our patients receive midazolam for premedication. The observations described herein therefore raise various questions that are relevant to our daily clinical practice.

Two issues must be considered when interpreting our observations in relation to data from the current literature. First, most data in the literature were determined using earlier versions of the BIS® monitor. Second, few data exist from careful evaluation of the effect of combinations of agents on the BIS.

Regarding the first issue, it may well be that the BIS-XP® provides lower BIS values than previous versions at similar midazolam infusion for the awake volunteer, the average BIS was 96 in the absence of any medication. With a target-controlled infusion of propofol of 1 µg/ml, the BIS decreased to a mean level of 92 after blood–effect site equilibration (fig. 3). Then, within 3 min after the start of midazolam administration, the BIS decreased to values as low as 44. During midazolam administration, the BIS varied between 40 and 60. Again, throughout the study period the volunteer remained responsive to verbal commands and/or mild prodding to a degree equivalent to an Observer’s Assessment of Alertness/Sedation score of 2 and 4, even with BIS levels of 40–45. The videotape, displayed as a Web enhancement, furthermore provides data on the responsiveness of this volunteer at low BIS levels. Additional information regarding this case is available on the ANESTHESIOLOGY Web site at http://www.anesthesiology.org.

**Case 3**

The third subject was a 25-yr-old man who weighed 87 kg and was 187 cm tall. The target propofol concentration for this subject was 1 µg/ml, and the initial and secondary midazolam infusion rates were 0.035 mg · kg⁻¹ · h⁻¹ and 0.035 mg · kg⁻¹ · h⁻¹, respectively (total midazolam dose in 60 min, 6 mg). With written informed consent and Leiden University Medical Center Ethics Committee approval, we gathered digital video data from this session until the termination of the

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**Fig. 1.** The Bispectral Index (BIS) versus time in case 1. Time 0–5 min displays the last 5 min (i.e., the time after blood–effect site equilibration) of the 15-min period, when only propofol was given by a target-controlled infusion with a target concentration of 0.6 µg/ml. At time 5 min, midazolam was added at 0.05 mg/kg in 1 min, followed by a continuous infusion of midazolam of 0.05 mg · kg⁻¹ · h⁻¹ for 59 min. All three volunteers maintained adequate spontaneous respiration and hemodynamic stability throughout the study period. At time 65 min, the midazolam infusion was terminated, but the target-controlled infusion of propofol was still continued. The area considered to be associated with adequate hypnosis for surgery (BIS, 40–60) is shown within the grid lines. The volunteer was responsive to verbal command and/or mild prodding at all times.

**Fig. 2.** The Bispectral Index (BIS) versus time in case 2. Time 0–5 min displays the last 5 min (i.e., the time after blood–effect site equilibration) of the 15-min period, when only propofol was given by a target-controlled infusion with a target concentration of 0.6 µg/ml. At time 5 min, midazolam was added at 0.05 mg/kg in 1 min, followed by a continuous infusion of midazolam of 0.05 mg · kg⁻¹ · h⁻¹ for 59 min. At time 65 min, the midazolam infusion was terminated, but the target-controlled infusion of propofol was still continued. The area considered to be associated with adequate hypnosis for surgery (BIS, 40–60) is shown within the grid lines. The volunteer was responsive to verbal command and/or mild prodding at all times.

**Fig. 3.** The Bispectral Index (BIS) versus time in case 3. Time 0–5 min displays the last 5 min (i.e., the time after blood–effect site equilibration) of the 15-min period, when only propofol was given by a target-controlled infusion with a target concentration of 1 µg/ml. At time 5 min, midazolam was added at 0.035 mg/kg in 1 min followed by a continuous infusion of midazolam of 0.035 mg · kg⁻¹ · h⁻¹ for 59 min. At time 65 min, the midazolam infusion was terminated, but the target-controlled infusion of propofol was still continued. The area considered to be associated with adequate hypnosis for surgery (BIS, 40–60) is shown within the grid lines. The volunteer was responsive to verbal command and/or mild prodding at all times.

Observer’s Assessment of Alertness/Sedation score between 2 and 4, even at BIS levels of 40–45. We then decided to monitor the next volunteer even more closely and to record the next session on videotape.

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**Discussion**

We describe three cases in which the BIS-XP® provided BIS values of 40–50 for volunteers who were responsive to verbal commands while receiving a combination of propofol and midazolam. In our hospital, we tend to administer propofol infusion regimens during propofol–opioid anesthesia on the basis of the BIS level. Based on the current literature, we advise our residents to maintain the BIS level between 40 and 60 intraoperatively. Most of our patients receive midazolam for premedication. The observations described herein therefore raise various questions that are relevant to our daily clinical practice.

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ilar hypnotic levels in similar subjects. As stated earlier, the BIS-XP® is claimed to be less sensitive to artifacts of the electromyographic activity than earlier versions of the BIS® monitor. Previously, it was reported that electromyographic activity falsely elevates the BIS.5 Introducing a version that is less sensitive to this may thus result in lower BIS levels in the absence of full muscle relaxation (as occurs in most patients).

Regarding the second issue, we note that the electroencephalographic activation induced by both propofol and midazolam has been difficult to interpret and model in the past.6,7 It may well be that the particular combination of propofol and midazolam at these low concentrations is not part of the BIS-behavioral database on which the BIS calculation is based. As a result, the electroencephalographic pattern induced by this combination may well be misinterpreted by the BIS® monitor as an electroencephalograph pattern associated with a patient experiencing a surgical hypnotic sedation level instead of actually being responsive to verbal commands. However, to our knowledge, no controlled studies have been done to examine hypnotic drug interactions and their effect on BIS, especially not using the BIS-XP®.

In conclusion, we report on the responsiveness of three volunteers with BIS-XP® values of 40–50 while receiving a combination of propofol and midazolam. The case reports draw attention to the relationship between the BIS and the responsiveness of patients as derived by the BIS-XP® in the presence of a combination of two hypnotic agents. The BIS user should be aware that the BIS is a measure of drug effect, not an independent measure of brain function. Consequently, the clinical anesthesiologist has no guarantee that a particular BIS will relate to the desired effect when a particular drug, or combination of drugs, is not part of the data file used to train the algorithm of the BIS calculation. As such, the case reports stress the need for further investigation of both the BIS-XP® itself and the effect of combinations of hypnotic agents on the BIS-XP®. Furthermore, the case reports stress the need for careful interpretation by the anesthesiologist of the BIS-XP® values in the clinical setting as long as the scientific basis for the clinical application of the BIS-XP® is not yet completely clear.

References


CASE REPORTS

Transient Cardiovascular Toxicity with Unintentional Intravascular Injection of 3% 2-Chloroprocaine in a 2-month-old Infant

Franklyn P. Cladis, M.D.,* Ronald S. Litman, D.O.†

THE caudal approach to epidural anesthesia is a safe and effective analgesic technique for neonates and infants. However, amide local anesthetics may cause serious cardiovascular complications such as arrhythmias and life-threatening depression of myocardial contractility.1–3 We describe transient cardiovascular toxicity in a 2-month-old infant after unintentional intravascular injection of 3% 2-chloroprocaine through an epidural catheter. To our knowledge, this is the first report of cardiovascular toxicity after accidental intravenous injection of an ester local anesthetic.

Case Report

A 4-kg 2-month-old girl with biliary atresia presented for a liver biopsy and Kasai procedure. Continuous epidural analgesia (using the caudal approach to epidural anesthesia) was planned to supplement intraoperative anesthesia and provide postoperative pain relief. After induction of general anesthesia and tracheal intubation, an 18-gauge angiocatheter inserted into the epidural space via the sacrococcygeal ligament was used to facilitate insertion of an epidural catheter equipped with a stylette (Portex, Keene, NH) to a presumed T7 level. Aspiration of the catheter was performed and failed to produce cerebrospinal fluid or blood. Test...
catheter was then removed. Postoperatively, the infant was well with-
CASE REPORTS

Anesthesia Management of Orthotopic Liver Transplantation in a Patient with Mustard Repair of Transposition of Great Arteries and Superior Vena Caval Obstruction

Charles Boucek, M.D.*, Prema Krishnamurthy, M.D.,† James Wallis Marsh, M.D.,‡ Jennifer Lee, M.D.§

AS an increasing number of patients with surgically repaired congenital heart disease survive into adulthood, more of these patients may require complex, lifesaving surgical procedures. We report our experience with providing anesthesia for liver transplantation in a patient with surgically corrected D-transposition of the great arteries.

Case Report

Our patient was born with D-transposition of the great arteries and underwent Mustard repair in infancy. Transfusion resulted in hepatitis B and subsequently progressive liver failure. By 29 yr of age, he was experiencing fatigue, encephalopathy, and refractory ascites and was referred to our center for liver transplantation. Aggressive diuretic therapy was limited by hyponatremia. His exercise tolerance was poor, but a single-lumen infusion catheter, REF product No. SC-14701, Arrow International (Reading, PA) and venous cannulae (MAC International) in the right internal jugular and right subclavian veins. Attempts at placement of a pulmonary artery catheter were unsuccessful, but a single-lumen infusion catheter, REF product No. SC-14701, Arrow International) was advanced via the internal jugular vein and displayed a waveform with a, v, and c waves consistent with central venous pressure. Transesophageal echocardiography confirmed the transit of systemic venous blood from the caval system to the left atrium, ventricle, and, ultimately, the pulmonary artery via the atrial baffle (fig. 2).

Removal of the native liver required 6 h and was performed by use of the piggyback technique.1 Oxygen saturation remained between 98% and 100% throughout the operation, with PaCO2 between 86 and 98%, but a single-lumen infusion catheter, REF product No. SC-14701, Arrow International) was advanced via the internal jugular vein and displayed a waveform with a, v, and c waves consistent with central venous pressure. Transesophageal echocardiography confirmed the transit of systemic venous blood from the caval system to the left atrium, ventricle, and, ultimately, the pulmonary artery via the atrial baffle (fig. 2).

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Tris(hydroxymethyl)aminomethane (Abbott Laboratories, Abbott Park, IL) and sodium bicarbonate to prevent rapid changes in serum sodium and osmolality. Furosemide and insulin were given to reduce serum potassium in anticipation of graft reperfusion. Serum potassium declined from 5.2 mEq/l to 3.8 mEq/l immediately before reperfusion. Thirty seconds after reperfusion, it increased to 5.3 mEq/l. Reperfusion of the donor liver was accompanied by a brief episode of sinus bradycardia that responded to a small bolus of epinephrine. Hepatic arterial and duct-to-duct biliary reconstruction proceeded uneventfully. Four units of erythrocytes, 2 units of plasma, and 6 units of platelets were infused, resulting in a final hematocrit of 51%. Blood component therapy was guided by serial thromboelastography (Hemoscope Corporation, Skokie, IL). At the end of surgery, the TEE probe was removed. Norepinephrine was discontinued in the early postoperative period, and the trachea was extubated on the second postoperative day. Lack of TEE made assessment of the adequacy of ventricular filling difficult in the intensive care unit. A pulmonary artery catheter was placed under fluoroscopic guidance via the right femoral vein. Mean pressure in the superior vena cava was 28 mmHg; the atrial pressure was 25 mmHg at end expiration and 0–5 mmHg on inspiration. Pulmonary artery systolic pressure was 45–50 mmHg. Computer tomography was obtained to evaluate the biliary system. This demonstrated that the superior vena cava was interrupted. Collateral flow through the azygous system carried blood to the atrial baffle and anatomic left ventricle. The patient subsequently returned to the operating room for a revision of the biliary anastomosis and was eventually discharged to home in stable condition.

**Discussion**

This patient with complex congenital heart disease developed cirrhosis secondary to transfusion-related viral hepatitis. Liver transplantation is a potentially lifesaving operation for patients with end-stage liver disease, but it is a complex procedure, made more so by residual cardiovascular abnormalities.

In patients with transposition of the great arteries, the right ventricle supplies blood to the aorta. The left ventricle supplies blood to the pulmonary artery. The systemic and pulmonary circuits coexist in parallel rather than intersecting pathways. Survival after delivery depends on intracardiac mixing of blood through atrial or ventricular septal defects or a patent ductus arteriosus. A variety of surgical techniques have been developed for the correction of this condition, all of which cross the pulmonary and systemic circulation at the level of the atria, the ventricles, or the great arteries themselves. The Mustard and Senning operations are atrial repairs. The Rastelli procedure is a ventricular repair using a ventricular septal defect and a valved conduit from the right ventricle to the aorta. The arterial switch procedure reimplants the aorta and pulmonary artery. Atrial repairs such as the Mustard operation retain the right ventricle as the systemic ventricle. With the Mustard operation, the atrial septum is excised, creating a common atrium. An atrial baffle is created with pericardium. The baffle isolates blood returning from the venae cavae and redirects it through the atrium and into the left ventricle. Oxygenated blood returning from the pulmonary veins passes into the atrium on the other side of the baffle. The oxygenated blood then passes through the tricuspid valve into the right ventricle and from there into the aorta. The systemic circulation remains dependent on the right atrium; LV = left ventricle; RA = right atrium; RV = right ventricle.

![Fig. 1. Transesophageal echocardiographic image showing a portion of the atrial baffle. LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle.](image1)

![Fig. 2. Transesophageal echocardiographic image showing spontaneous saline contrast in atrium and left ventricle (with illustrated chamber outline superimposed). LV = left ventricle; RA = right atrium; RV = right ventricle.](image2)
ventricle. Survival into adulthood is common after a suc-
cessful repair, but patients continue to be at risk for pre-
mature death, heart failure, and dysrhythmias. Conduc-
tion defects and attenuated augmentation of cardiac output 
with exercise because of flow restriction through the atrial 
bafile are not uncommon in these patients. We are un-
aware of any previous reports of liver transplantation in 
patients with this condition.

Common problems encountered during liver trans-
plantation include massive blood loss, vasodilatation, 
progressive acidosis, and coagulopathy. Because our pa-
tient’s femoral pulses were not palpable, a radial artery 
catheter was placed for blood pressure monitoring; a 
second radial arterial catheter was placed to permit fre-
muuent arterial sampling. Venous access for rapid infusion 
was established, but fluid management was complicated 
by the limitations of monitoring. It was anticipated that 
measurement of central venous pressure would reflect pre-
load. The anesthesia team anticipated that navigating a 
pulmonary artery catheter through the atrial baffle 
would be difficult, although not necessarily impossible; 
pacemaker placement via this route has been performed in 
other patients. Attempts at placement of a pulmonary 
artery catheter through the jugular and subclavian can-
nulae failed. Fluoroscopy was not used but would not 
have resulted in successful placement. Obstruction at the 
level of the superior vena cava, a recognized com-
plication of the Mustard operation, was present but was 
not adequately appreciated preoperatively in our pa-
tient. The report of his cardiac catheterization, which 
had been performed 20 yr previously, was not available 
at the time of the operation. Subsequent computed to-
mography clarified the residual anatomy and confirmed 
that the superior vena cava was interrupted. Preopera-
tive placement of a pulmonary artery catheter through 
the inferior vena cava was not attempted because liver 
transplantation requires surgical manipulation of both 
the hepatic vein and the inferior vena cava. A catheter in 
this location would have been at risk for surgical entrap-
ment during the operation. A pulmonary artery catheter 
placed from the femoral route postoperatively demon-
strated that the mean superior vena cava pressure ex-
ceeded atiral pressure.

Obstruction of the superior vena cava complicated the 
anesthetic management, not only by preventing the intra-
operative use of a pulmonary artery catheter but also by 
making the central venous pressure unreliable, and it lim-
ited the options for venous return during removal of the 
native liver. In the absence of reliable conventional moni-
tors of preload, observation of right and left ventricular 
filling by TEE was especially helpful. Examination by TEE 
before attempts at pulmonary artery catheter placement 
might have been useful in this case. Other commercially 
available methods to measure cardiac output, such as bio-
impedance and Doppler interrogation of the aorta from the 
esophagus, may also be considered.

During liver transplantation, venovenous bypass is 
sometimes used to decompress the splanchnic and sys-
temic venous systems during native liver removal and graft 
insertion. Drainage cannulae are placed in the portal vein 
and inferior vena cava, and a pump returns blood to the 
superior vena cava through a cannula in an axillary or 
jugular vein. This preserves venous return during complete 
clamping of the inferior vena cava. Alternatively, the pig-
gyback technique permits continuous flow through the 
inferior vena cava, which is only partially clamped. Ven-
ovenous bypass can be cumbersome, requires additional 
cannulations, and is associated with complications such 
as air embolism and injury to the brachial plexus and vessels. 
Had it been used in our patient, venous return might have 
exceeded the capacity of the ayzygos collaterals that, in 
our patient, carried blood from the superior vena cava to the 
atrium. Fortuitously, blood loss was relatively small. Both 
the subclavian and jugular venous lines led to the superior 
vena cava and dilated ayzygos collaterals. Rapid replace-
ment of lost blood may have been limited by the capacity 
of this system.

We administered methylene blue, norepinephrine, and 
avasopressin to help maintain systemic vascular resis-
tance and contractility. Methylene blue may reduce hypo-
tension and inotropic requirements during liver transplan-
tation. These agents, used in combination, maintained 
 systemic arterial blood pressure without significant tachy-
cardia during periods of relative hypovolemia.

Candidacy for noncardiac organ transplantation need 
not be denied to well-compensated patients who have 
undergone repair of congenital heart defects. Careful 
preoperative evaluation is especially important in these 
patients. TEE can be extremely helpful intraoperatively.

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WE report an oxygen supply tank failure at our institution that occurred during the morning of a busy operating room schedule when medical center oxygen use was maximal.

Case Report

The bulk oxygen supply at our facility consists of three cryogenic oxygen storage tanks: a primary tank, A; a secondary tank, B; and a reserve tank, C (fig. 1). Tanks A and B are physically situated next to each other. The reserve tank (C) is located approximately 1 block (approximately 305 m) away and normally serves as the primary oxygen source for our hospital’s second large inpatient bed facility. Valves, piping, and regulators between the primary tank and primary reserve were installed in compliance with the National Fire Protection Association’s guidelines and commonly accepted installation practices.1,2 The tank failure and resulting major liquid oxygen spill were caused by the separation of a brazed joint between the stainless steel primary tank A and a brass pipe fitting. The resulting sudden release of approximately 8,000 gallons of liquid oxygen from tank A precluded the initial use of the adjacent secondary tank B. Tank B was inaccessible because of massive ice and vapor cloud formation, which initially made it impossible to determine whether the tank was stable and functional (fig. 2). As the pressure in the primary tank rapidly decreased, an automatic switch-over valve opened tank B to provide oxygen to our medical center. With uncertainty regarding the integrity of the reserve system (because of inability to immediately assess the secondary tank), an adjacent valve was immediately closed to isolate both tanks until the damage could be assessed.

Our hospital engineers reacted almost immediately, closing the valves to isolate both the primary supply within 10 h. At the same time, valves were opened to bring the reserve tank C online as the alternate supply source. Fire and police officials were notified of the situation, and personnel in critical areas (intensive care units, operating rooms, and chiefs-of-staff) also were simultaneously notified. Reserve oxygen E-cylinders were collected and distributed to critical areas within the medical center. Our bulk oxygen supplier was notified, and a supply tanker was dispatched to provide additional liquid oxygen. Fortunately, we never experienced total loss of pipeline supply pressure. However, in the operating rooms, the pipeline pressure was thought to be most likely a result of relatively high friction losses as the oxygen was routed through regulators near the distant reserve tank C to the main hospital and operating room facility. Forty-five minutes after the failure, the secondary tank B was determined to be functional and was put back into service. The valves to the reserve tank C were then closed.

The failure of our primary oxygen supply tank could have caused complete oxygen pipeline system failure if the secondary tank had been damaged concurrently. The redundancy in our system with the remote reserve tank provided a continuous supply during the event. Very soon after the failure occurred, the low liquid oxygen volume alarms were activated at the engineering control center. Because of the design of our alarm system, however, these alarms alone would not have given enough advance warning to prevent loss of pipeline pressure. The inability of our alarm system to provide timely warning in this situation was due to the rapid rate at which the liquid oxygen was lost. These alarms were set to communicate when the level fell below a preset threshold value and were not designed to give engineers ongoing, quantitative information on the level of liquid oxygen within the tank. Typical alarm systems do not provide this quantitative information.2,3 Furthermore, if the main and reserve tanks had not been situated in a location that was readily visible, then engineers might have believed that the low oxygen level alarm was simply a result of normal use. The rapid response time to this system failure at our facility was at least partially because of easy visibility of the tanks and the quick reaction of our engineers, who were at the bulk oxygen storage facility at the time of the failure.

The failure of the liquid oxygen pipeline that caused this event was determined to be due to both electrolysis of the stainless steel-to-brass joint and thermal expansion damage. The primary tank A and piping were 12 yr old at the time of the event. After the event, the joint was replaced with a stainless steel-to-stainless steel welded joint. The repaired primary tank was subsequently tested and put back in service as the primary supply within 10 h.

Discussion

Our review of the literature uncovered instances of bulk oxygen system failures secondary to events such as pipeline crossover, filling with the wrong gas, faulty installation, modification of the pin index or diameter index safety systems, contamination, and other factors.4,5 We were unable to find a similar reported case of oxygen supply failure secondary to a scenario such as described above.

Cataclysmic bulk liquid oxygen supply failure is problematic not only for anesthesiologists but also for the entire medical center. At the time of this failure, oxygen use in our medical center was at peak. All 30 operating rooms (24 noncardiac, 6 cardiac) were in use. Several patients were in the postanesthesia care unit, and nine separate intensive care units were filled to capacity. The emergency department was busy, and many patients were receiving oxygen by facemask or nasal cannula in their hospital rooms throughout the medical center. Complete loss of oxygen supply could have been disastrous.

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In the operating room suite, several actions were taken as soon as a problem was identified. First, all anesthesia care providers were informed about the possibility of a bulk oxygen system failure. Additional E-cylinders were delivered to all anesthetizing locations. Low-flow anesthesia techniques were used where possible. Attempts were made to keep oxygen flowmeter flow below 1,000 ml/min. Mechanical ventilation was discontinued, and manual bag ventilation was instituted. Finally, all elective procedures were postponed until the problem was isolated.

When using gas-driven anesthesia ventilators such as the Datex-Ohmeda models 7800, 7810, 7100, or 7900 (Madison, WI), the drive gas used to compress the bellows is normally 100% oxygen, and the drive gas volume consumed equals the minute ventilation. Thus, in adult patients with a minute ventilation of 8–10 l, an equal quantity of drive gas can be conserved per minute by discontinuing mechanical ventilation and switching to manual ventilation. Interestingly, some newer ventilators may be switched between using either oxygen or air as their drive gas, and doing so would also conserve oxygen supplies. When one compares oxygen consumption in a low-flowmeter setting with manual ventilation and an intermediate-flowmeter setting with mechanical ventilation, the difference is dramatic. With fresh gas flow of 500 ml/min in conjunction with manual ventilation, a total of 500 ml/min oxygen is consumed. In contrast, when an intermediate-oxygen flowmeter setting of 2 l/min is used in conjunction with mechanical ventilation, providing 10 l/min of minute ventilation, the total oxygen utilization is 12,000 ml/min. In this scenario, manual ventilation with low oxygen flow would consume only 4.2% of the amount of oxygen that would be used during mechanical ventilation with intermediate flow. For anesthesia workstations that use a push drive (piston type) ventilator rather than a gas-driven one (e.g., Narkomed 6000, North American Dräger, Telford, PA), oxygen use would be comparable to the manual ventilation scenario described above.

The failure of the oxygen supply system at our facility was caused by multiple factors. As is the case in many institutions, the main and reserve bulk oxygen storage tanks of our medical center are owned by the bulk oxygen supplier. The plumbing to the tanks and the rest of the oxygen supply system is owned and maintained by our medical center. This connection between the vendor-owned tank and the hospital-owned pipeline was the point of failure in our system. Maintenance and upkeep of all components should be coordinated and documented between all parties involved.

The problem resulting from the close proximity of the primary and secondary tanks would have been much more difficult to manage if we did not have a reserve tank. In our case, if there had been either a spatial or physical barrier between the primary and secondary tanks, engineers could have more easily and quickly assessed the status of the secondary tank. As it was, the liquid oxygen spill produced massive ice and vapor on and around the secondary tank, precluding inspection of its integrity. Usually, hospitals have only two liquid oxygen sources. If these two are somehow isolated from each other, a problem such as the one we encountered could be avoided. Separation of the primary tank and reserve tanks could be achieved simply by construction of a noncombustible barrier between the tanks and creation of a sloping surface away from the tanks and barrier wall for runoff. In addition, consideration should be given to providing enough room for runoff of the amount of liquid oxygen stored in the tanks.

Continuous measurement of the quantity of oxygen in the cryogenic tanks may provide an earlier warning of a massive system failure. This alarm monitor should commu-
nicate not only when the volume falls below a threshold level but also when the rate of volume loss is excessive.

Conclusion

We were fortunate that during this event, our cryogenic oxygen system failure was noted very soon after it occurred and that it was limited to only one tank. No adverse patient outcome or even significant delay in the elective surgery schedule resulted. Predetermined oxygen system failure protocols were immediately implemented. Careful planning and thoughtful consideration to the design of the bulk oxygen supply system most likely prevented a catastrophic failure of the oxygen supply system at our institution. Tying together bulk oxygen systems in large facilities that use more than one such system adds redundancy to the system that can allow continued use even when a major system failure has occurred.

To summarize, based on this case and review of the literature, we have several specific observations regarding possible bulk liquid oxygen supply system failure. (1) The events outlined in this case reinforce the importance of having a thoroughly prepared hospital-wide disaster plan available. It is critical to be able to identify key individuals in a timely manner to deal with the various responsibilities required to avoid making an already bad situation worse. (2) Anesthesia care providers who serve in leadership roles in the operating room are responsible for the safety of all patients within the surgical facility. As such, they should have a comprehensive understanding of their own hospital’s oxygen delivery system and associated disaster plans. Anesthesia care providers should know the locations of all major bulk oxygen supply components such as the tanks and associated shutoff valves. (3) Anesthesiology leaders must be involved in any new construction or remodeling planning. (4) Medical center leadership might consider separating the location of primary and secondary oxygen supply tanks or building a barrier between them to isolate one from the other in the event of a tank failure. (4) The National Fire Protection Association’s Standard for Bulk Oxygen Systems at Consumer Sites (NFPA-50) addresses both “Location of Bulk Oxygen Systems” (2.1-
2.1.5) and “Distances Between Bulk Oxygen Systems and Exposures” (2.2–2.2.14). However, it does not specifically address the placement of an isolation barrier between bulk storage tanks, which could have been helpful in our case. Furthermore, in addition to primary and secondary tanks, large medical centers with high oxygen use should consider having an additional reserve tank in the event that the primary and secondary tanks are simultaneously unusable, as in our case. (6) Finally, if catastrophic bulk oxygen supply failure does occur, oxygen consumption conservation techniques as described above should be instituted. Adequate oxygen cylinder supplies must be on-site, and a disaster plan that includes obtaining an additional supply of cylinders must be readily available in the event that the bulk oxygen supply system cannot be rapidly restored.

References


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