

## Hepatotoxicity after Desflurane Anesthesia in a 15-month-old Child with Mobius Syndrome after Previous Exposure to Isoflurane

Geneviève Côté, M.D., M.Sc., F.R.C.P.(C),\* Sarah Bouchard, M.D., F.R.C.S.(C), F.A.C.S.†

HALOGENATED anesthetic agents have been associated with perioperative liver dysfunction and injury. Most cases of hepatotoxicity reported involve halothane exposure. Halothane produces two types of hepatotoxicity. A mild, transient syndrome with modest increases in transaminase enzymes occurs in 20% of patients given halothane.<sup>1</sup> A more severe and fulminant hepatitis with marked liver dysfunction, jaundice, encephalopathy, and even death is observed in 1 in 20,000 anesthetic exposures to halothane.<sup>2</sup> Halothane toxicity occurs in children as well as in adults.<sup>3,4</sup> Cross-sensitization between halothane and other volatile agents has been previously reported with isoflurane,<sup>5</sup> enflurane,<sup>6-8</sup> and desflurane.<sup>9,10</sup>

To date, only four cases of hepatotoxicity in adults were related to desflurane exposure.<sup>9-12</sup> We report the case of a young child who developed acute hepatotoxicity after desflurane anesthesia in the context of two previous isoflurane exposures.

### Case Report

A 15-month-old boy with Mobius syndrome (also known as Mobius congenital oculofacial paralysis, a nonprogressive birth defect caused by the absence or underdevelopment of the cranial nerves VI and VII) presenting with symptomatic gastric regurgitation and slow gastric emptying was scheduled to undergo a Nissen fundoplication, pyloroplasty, and gastrostomy. He also had multiple vertebral abnormalities and cerebellar hypoplasia. Medication before surgery consisted of cisapride and omeprazole. No known allergies and no family history of anesthetic disease were reported.

Previous surgical procedures included: tracheoesophageal fistula repair that was performed on his first day of life and a gastrostomy at 10 months of age. For both procedures, general anesthesia was maintained with isoflurane and remifentanyl infusion. Both anesthetics were uneventful. For his third surgical intervention, elective Nissen fundoplication, general anesthesia was induced using propofol, fentanyl, and rocuronium followed by desflurane and remifentanyl infusion for maintenance. The procedure was completed without any specific surgical or anesthetic problems.

On the second postoperative day, gastrointestinal bleeding was observed. One hundred milliliters fresh blood was suctioned from the nasogastric tube. The patient's vital signs remained stable, but he

eventually required a blood transfusion. On investigation, he was found to have a coagulopathy with an elevated international normalized ratio, and increased liver enzymes (table 1). Preoperative liver biochemistry was unremarkable. The patient was given vitamin K. Cardiopulmonary examination was normal. Abdominal examination did not reveal an increase in liver size or liver tenderness. No evidence of asterix or encephalopathy was noted. An ultrasound of the upper abdomen revealed a normal liver with no ascites.

The patient was not previously transfused. Serologies for viral hepatitis were all negative, as well as laboratory tests for various autoantibodies, hemochromatosis, and Wilson disease. The acetaminophen level was in the therapeutic range. A liver biopsy was refused by the parents. Enzyme-linked immunosorbent assay for antibodies that react with one or more liver trifluoroacetylated microsomal proteins was not performed. This test is not available at our institution. Therefore, an exclusionary diagnosis of desflurane-induced hepatitis after previous exposure to inhalation anesthetic agent was established. The patient was discharged on postoperative day 9 without clinical sequelae.

This patient returned at 22 months of age for an eye procedure. He received total intravenous anesthesia consisting of propofol and remifentanyl. The anesthetic machine was previously flushed with 100% O<sub>2</sub> at 10 l/min for 10 min. Preoperative liver enzymes were in the expected ranges, 21 U/l alanine aminotransaminase and 34 U/l aspartate aminotransaminase.

### Discussion

To our knowledge, we report the first pediatric case of desflurane hepatotoxicity. Only four previous reports have been published in the adult anesthetic literature.<sup>9-12</sup> The temporal relation between exposure and liver injury is consistent with desflurane hepatotoxicity as an exclusionary diagnosis. Other possible perioperative etiologies were excluded, such as preexisting liver disease, new onset of biliary obstruction and cholangitis, coexisting obesity, systemic viral infection, septicemia, drug abuse, adverse reactions to other medications given in the perioperative period, and various metabolic and immunogenic diseases.

Hepatotoxicity with halothane inhalation has been studied extensively. Risk factors include obesity, female sex, a history of drug allergies, and multiple exposures to anesthetic agents.<sup>13-15</sup> Anesthetic agents including halothane, enflurane, isoflurane, and desflurane can produce metabolic hepatocellular injury in humans to a variable extent. The likelihood of suffering postoperative immune hepatitis depends on the amount of the anesthetic metabolized and is thereby considerably less with enflurane, isoflurane, or desflurane as compared with halothane.<sup>16</sup> The extent of cytochrome P-450 2E1-mediated metabolism of fluorogenated anesthetic agents halothane, sevoflurane, isoflurane, and desflurane is reported

\* Staff Anesthesiologist, † Assistant Professor of Pediatric Surgery.

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Address correspondence to Dr. Côté: Department of Anesthesiology, Centre Hospitalier Universitaire Sainte-Justine, 3175 Chemin de la Côte Sainte-Catherine, Montréal, Québec, Canada, H3T 1C5. Information on purchasing reprints may be found at [www.anesthesiology.org](http://www.anesthesiology.org) or on the masthead page at the beginning of this issue. ANESTHESIOLOGY's articles are made freely accessible to all readers, for personal use only, 6 months from the cover date of the issue.

**Table 1. Perioperative Liver Function Tests and Drug Level**

Test	Reference Range	Preoperative	Postoperative Day							
			2	3	4	5	7	8	9	
AST	15–55 U/l	30		6,080	1,757		371	90	57	49
ALT	5–45 U/l	20		6,180	3,008		2,559	1,378	932	662
GGT	5–32 U/l	10		24	24		29	44	47	
PT	11–15 s		24.7	24.7	16.8					
aPTT	23–35 s		34.6	38.2	36.4					
INR			2.7	2.16	1.32*			0.94		
Acetaminophen	μm/l		210							

\* Subcutaneous vitamin K received.

aPTT = activated partial thromboplastin time; ALT = alanine aminotransaminase; AST = aspartate aminotransaminase; GGT =  $\gamma$ -glutamyltranspeptidase; INR = international normalized ratio; PT = prothrombin time.

to be 20%, 2–5%, 0.2–0.6%, and 0.02%, respectively.<sup>17</sup> In the current case, our patient was exposed to isoflurane on two previous occasions. Although desflurane and isoflurane produce low levels of trifluoroacetylated product formation, this small amount is sufficient to induce hepatotoxicity, particularly in the sensitized patient.<sup>9</sup> Medications known to induce cytochrome P-450-mediated metabolism increase the degree of haptenic protein labeling after anesthetic exposure. This phenomenon was established in rats exposed to isoniazid and etha-

no.<sup>18–22</sup> Our patient received cisapride and omeprazole, both known inhibitors of cytochrome P-450 3A4 and cytochrome P-450 2C19, respectively. The patient's immunologic susceptibility could explain immune reaction and cross-reactivity to halogenated agents.

Although there is no reported difference in halothane metabolism in pediatric and adult patients,<sup>18</sup> the incidence of halothane hepatitis is much lower in the pediatric age group.<sup>18–21</sup> There have been two case series reporting halothane-induced acute liver failure. Seven

**Table 2. Previous Reports of Desflurane Hepatotoxicity<sup>9–12</sup>**

Reference, Year	Patient Sex, Age	Procedure	Symptoms and Day of Appearance
Tung <i>et al.</i> , <sup>12</sup> 2005	F, 81 yr	Resection of colonic cancer	POD 6
Chung <i>et al.</i> , <sup>11</sup> 2002	M, 54 y	Radical thyroidectomy	POD 26: jaundice, chill, fever
Berghaus <i>et al.</i> , <sup>10</sup> 1999	F, 37 y	Fixation of a tibial shaft fracture	POD 14: jaundice, epigastric pain, nausea, malaise, dark urine, macular rash
Martin <i>et al.</i> , <sup>9</sup> 1995	F, 65 y	Left hemithyroidectomy	POD 12: jaundice, epigastric pain, fever, malaise, macular rash
Current case	M, 15 mo	Nissen fundoplicature	POD 2

Reference, Year	Maximum Value			TFA	Outcome	Previous Surgical Procedures	Anesthetic Agent
	ALT, U/l	AST, U/l	INR				
Tung <i>et al.</i> , <sup>12</sup> 2005	2,188	425	2.29		Good	Appendectomy Cystocele repair	NA NA
Chung <i>et al.</i> , <sup>11</sup> 2002	212	249	NA	NA	Good	Resection of a mandibular soft tissue tumor Radical neck dissection and radical flap reconstruction	Isoflurane Sevoflurane*
Berghaus <i>et al.</i> , <sup>10</sup> 1999	1,776	1,258		+	Good	Appendectomy Tonsillectomy Cesarean delivery Salpingectomy	NA NA Isoflurane Halothane
Martin <i>et al.</i> , <sup>9</sup> 1995	1,886	1,280	2.0	+	Good	Tonsillectomy Tubal ligation Total abdominal hysterectomy Laparotomy Cholecystectomy	NA Cyclopropane Cyclopropane Halothane Halothane
Current case	6,180	3,008	2.7	NA	Good	Repair of esophageal fistula type III Gastrostomy	Isoflurane Isoflurane

\* After sevoflurane exposure, increase in liver enzymes was noted on postoperative day (POD) 9. Aspartate aminotransaminase (AST) and alanine aminotransaminase (ALT) peaked at 287 and 543 U/l, respectively.

INR = international normalized ratio; NA = not available; TFA = trifluoroacetylated liver microsomal protein antibodies measured by enzyme-linked immunosorbent assay.

children out of 48 patients<sup>3</sup> experienced halothane-induced hepatitis between 1965 and 1984, whereas 2 children out of 18 patients experienced injury from halothane between 1985 and 1995.<sup>4</sup> Several trifluoroacetylated proteins purified from rat liver microsomes are recognized by serum antibodies of patients with halothane hepatitis.<sup>22</sup> Cross-reactivity between volatile anesthetic agents was previously reported. Desflurane-induced hepatic dysfunction has been reported with previous exposure to halothane,<sup>9,10</sup> isoflurane,<sup>10,11</sup> and sevoflurane.<sup>11</sup> Antibodies against trifluoroacetylated proteins were found on two occasions with desflurane-induced hepatitis<sup>9,10</sup> (table 2). Previous patients with desflurane hepatotoxicity were diagnosed between 6 and 26 days after exposure. All 5 subjects, including our patient, recovered from their liver dysfunction.

It is our view that the hepatic injury observed here resulted from an immunologic process associated with desflurane exposure in a previously sensitized patient by two isoflurane anesthetics. The use of the Naranjo probability scale indicated a probable relation between liver enzyme increase and desflurane in our patient.<sup>23</sup> Patients with multiple sclerosis are at no higher risk for liver injury after exposure to inhaled fluorinated anesthetic agents than the general pediatric population.

## Conclusion

We believe this to be a case of hepatotoxicity related to desflurane exposure after previous exposure to isoflurane. Postoperative laboratory tests have excluded obvious viral, metabolic, and organic etiologies. Serology against trifluoroacetylated labeled proteins would have confirmed this diagnosis. In the future, this patient will receive total intravenous anesthesia.

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## Subacute Spinal Subarachnoid Hematoma after Spinal Anesthesia That Causes Mild Neurologic Deterioration

Joon-Hee Park, M.D.,\* Keun-Mann Shin, M.D., Ph.D.,† Sung-Joon Hong, M.D.,\* Il-Suk Kim, M.D.,\*  
Sung-Keun Nam, M.D.‡

SPINAL subarachnoid hematoma after lumbar puncture has been reported to occur in the presence of preexisting coagulopathy or anticoagulant therapy.<sup>1-4</sup> Although occult spinal subarachnoid hematoma may be possible, symptom onset generally is acute and dramatic, and it also represents serious complications for hemostatically compromised patients.<sup>5-7</sup>

We report a patient who developed spinal subarachnoid hematoma after lumbar puncture. However, she had normal blood coagulation profiles and no spinal disorders such as spinal stenosis or spinal fractures. The onset of her symptoms was gradual, and neurologic deterioration was mild. To our knowledge, this is the first report about spinal subarachnoid hematoma with mild and vague symptoms in a hemostatically normal patient.

### Case Report

A previously healthy 47-yr-old woman underwent an elective sling operation for stress incontinence by our urologic department. At our hospital, coagulation profiles, including platelet counts, prothrombin time, activated prothrombin time, and bleeding time used to be routinely checked in all patients, 1 week before surgery. Laboratory examinations such as platelet counts ( $254,000/\text{mm}^3$ ), prothrombin time (119.4%), activated prothrombin time (27.9 s), and bleeding time by the method of Duke (3 min) were within normal limits. The patient had no clinical history of bleeding tendency, and she also had not taken any medication.

The patient's height and weight were 1.57 m and 46.6 kg, respectively. Before spinal anesthesia, the anesthetist explained the procedure and its possible complications. The patient wanted spinal anesthesia.

The patient was placed in the left lateral position. Under sterile conditions, a 26-gauge Quincke spinal needle (viola) (Sato, Kitamoto, Japan) was inserted into the L3-L4 interspaces, but accidental paresthesia occurred through the patient's left leg. The needle was removed, and a second attempt at L4-L5 with a 26-gauge Quincke spinal needle (viola) successfully yielded clear cerebrospinal fluid. Two milliliters heavy bupivacaine, 0.5%, was injected slowly through the spinal needle. In the aspiration after injection, there was no bleeding, and the cerebrospinal fluid was clear. The spinal anesthetics produced a sensory block to T10, with motor block to L1 or more. After the



**Fig. 1.** Magnetic resonance imaging demonstrating spinal subarachnoid hematoma. T1-weighted sagittal image showing heterogeneous increased signal intensity consistent with subarachnoid hematoma from L4 to S1.

operation, the patient's estimated blood loss was 50 ml, and the operation time was 60 min. Therefore, no blood product or colloid solution was infused. Three hundred milliliters Hartmann solution was infused during the operation.

Five days after the spinal anesthesia, the patient noted intermittent low back pain and bilateral lower extremity pain in both thighs and the gluteal areas. She also reported gait discomfort because of her pain. However, she had no voiding difficulty. Although these symptoms increased, muscle power and sensory functions were intact. Plain radiographs of the spine did not reveal any abnormalities. A sagittal T2-weighted magnetic resonance image of the spine disclosed a mass-like lesion of high signal intensity in the intradural space from the L4 lower endplate level to the S1 lower endplate. It was located between the nerve roots of the cauda equina (fig. 1). The official magnetic resonance image reading indicated a suspected spinal subarachnoid hematoma in the L4-S1 area.

Before the patient was transferred to the orthopaedic department, we asked her again meticulously about previous bleeding tendency, but no history could be found out. Investigations at this time included a normal coagulation screen. Platelet counts ( $252,000/\text{mm}^3$ ), prothrombin time (118.4%), activated prothrombin time (27.2 s), and bleeding time by the method of Duke (3 min) were within normal limits. In addition, we performed thromboelastography. Normal findings of the thromboelastography were obtained (reaction time, 18.3

\* Lecturer, † Professor, ‡ Resident.

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Address correspondence to Dr. Park: Department of Anesthesiology and Pain Medicine, Kang-Dong Sacred Heart Hospital, Hallym University, Seoul, Korea, Republic of Korea. anesjhpark@yahoo.co.kr. Information on purchasing reprints may be found at [www.anesthesiology.org](http://www.anesthesiology.org) or on the masthead page at the beginning of this issue. ANESTHESIOLOGY's articles are made freely accessible to all readers, for personal use only, 6 months from the cover date of the issue.



min; k time, 7.2 min;  $\alpha$  angle, 27.6°; maximum amplitude, 50.0 mm). Despite conservative treatments such as medication and physical therapy, the patient's radiating pain was getting worse. Therefore, she decided to undergo surgery. A laminectomy was performed at L4 and L5. There was no evidence of bleeding in the epidural space. On opening the dural sac, there was clotted blood between the cauda equina in the subarachnoid space. The hematoma was removed completely from the dural sac. After no hematoma in the subdural space was confirmed, the dura was sutured. After surgical decompression, the patient's bilateral sciatica improved considerably, and only minimal low back pain remained.

## Discussion

Spinal subarachnoid hematoma that arises from a spinal tap is usually associated with anticoagulants and underlying coagulopathy.<sup>1,2</sup> To our knowledge, only one case has reported that spinal subarachnoid hematoma may be the result of technical difficulties encountered in spinal anesthesia.<sup>3</sup>

Within the subarachnoid space at the level of the caudal equine, the only vessels of substantial size are the radiculomedullary artery of Adamkiewicz and its corresponding vein.<sup>4</sup> On occasion, this vessel may arise low and accompany L3, L4, or L5 nerve roots, where it could be jeopardized during lumbar puncture. Most authors believe such hemorrhages are caused by injury to these structures or to the smaller radicular vessels entering the subarachnoid space with each segmental nerve root.<sup>8,9</sup> Masdeu *et al.*<sup>9</sup> confirmed this mechanism at autopsy. Breuer *et al.*<sup>8</sup> estimated that the frequency of brushing a nerve root, with the associated risk of lacerating the radicular artery or vein on its surface, was more than 25%. They suggested that the frequency of occult spinal subarachnoid hemorrhage is much higher than currently suspected, but is usually clinically significant only in hemostatically compromised patients. Ruff and Dougherty<sup>2</sup> reported the results of the only large prospective study on the risks of lumbar puncture followed by anticoagulation *versus* lumbar puncture alone. They found a significantly higher incidence of complications from lumbar puncture in the group receiving anticoagulation therapy. Spinal subarachnoid hemorrhage rarely occurs in patients who have not been anticoagulated, and although spinal subarachnoid hemorrhage occurs, the development of a discrete hematoma causing symptoms of spinal cord or root compression is unusual. First, blood in the cerebrospinal fluid is rapidly diluted by diffusion, which is facilitated by spinal motion. It rarely reaches the concentration necessary for clot formation.<sup>10</sup> Second, the cerebrospinal fluid has an intrinsic fibrinolytic activity that increases after hemorrhage.<sup>11</sup> Third, the pulsatile motion of the dural sac aids this process. Kirkpatrick and Goodman<sup>12</sup> presumed that an anatomical block or relatively anatomical block to normal cerebrospinal fluid flow due to preexisting disease might contribute to hematoma formation.

In our case, there were significantly different points compared with previous reports. Our patient had no associated coagulopathy and took no anticoagulants. We confirmed this not only by the traditional clotting tests but also by thromboelastography, which provides a kinetic analysis of the entire clot formation and stabilization as well as clot dissolution by the fibrinolytic system. In the only previous report in which spinal subarachnoid hematoma was caused by technical difficulties of lumbar puncture, the patient was 81 yr old, so she was thought to have such a stenotic spinal canal due to natural degenerative changes that it might be a causative factor of spinal subarachnoid hematoma.<sup>13</sup> In our case, however, the patient had no preexisting anatomic block of cerebrospinal fluid flow such as spinal stenosis, fractures, or previous surgery, as her lumbar spine magnetic resonance image showed.

It has been reported that spinal subarachnoid hematoma causes compromise of the neurologic state and rapid deterioration.<sup>1,2,5-8,13,14</sup>

Furthermore, Kreppel *et al.*<sup>15</sup> reviewed all kinds of spinal hematoma, including epidural, subdural, and subarachnoid hematoma. In his review, the subarachnoid hemorrhage after lumbar puncture was also rare, and the patients' coagulation systems had been impaired. Although some cases had subacute clinical manifestation (5.6%), clinical symptoms were almost severe, including neurologic deficit. In our case, a hematoma was formed between the fourth and fifth lumbar segments. However, the patient had only mild back pain and discomfort of the thighs and gluteal areas 4 days after lumbar puncture. There was no neurologic deterioration, and it can be explained as follows. First, our patient did not have any disorders related to blood coagulation and had taken no anticoagulants; therefore, subarachnoid hemorrhage by lumbar puncture might be limited by the natural coagulation system. Second, although two attempts had been made during the spinal anesthesia and the patient felt paresthesia on the first attempt, it was never thought to be traumatic or a difficult lumbar puncture case. Because we used a 26-gauge spinal needle and no blood was found through the spinal needle, vessel injury by the lumbar puncture was not considered to be much.

Our case shows that spinal subarachnoid hematoma may occur in a patient who has a normal coagulation system and has taken no anticoagulants, through a non-difficult, nontraumatic lumbar puncture. Most of all, the clinical manifestation of our patient is different from previous reports in that she reported only mild and vague sciatica with no neurologic deficit several days after the lumbar puncture. In conclusion, although clinical features seem to be doubtful for spinal subarachnoid hematoma, early computed tomographic myelography or magnetic resonance imaging should be considered in case surgical decompression may be indicated for improving clinical symptoms and preventing complications

of the bowel or bladder that are significant sequelae of cauda equina syndrome.

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