

## All Roads Do Not Lead to Rome

REGIONAL anesthesia has evolved in recent years from a technique into an art, practiced by some pioneering anesthesiologists. As with any art form, regional anesthesia has obtained a level of complexity not seen before. Until recently, regional anesthesia was used by a select group, using sophisticated wording and techniques that prohibited its widespread implementation. However, with the development of more reliable equipment and introduction of more effective local anesthetics, as well as improved techniques, this time is over. Any society starts with a theocracy and finishes in a democracy, as stated by Victor Hugo 150 yr ago. The democratization of regional anesthesia needs well-defined guidelines to ensure its safe, effective practice. As the number of anesthesiologists performing regional blocks increases, so may the number of serious complications. In this issue of ANESTHESIOLOGY, Sardesai *et al.*<sup>1</sup> makes a substantial contribution in regard to improving the safety of regional anesthesia.

The authors investigate the challenging question of whether the technical approach chosen to perform an interscalene block could influence the possibility of entering the spinal canal. The different angles necessary to gain access to the intervertebral foramen of the sixth cervical vertebra between the high and the classic lateral modified approach and the Winnie technique were compared in 10 healthy volunteers undergoing magnetic resonance imaging of their necks. The results of this investigation demonstrated that the Winnie approach had a shorter skin-intervertebral foramen distance and the smallest discrepancy angle. The latter finding is compelling because the smaller the discrepancy angle is, the easier it is to penetrate into the spinal canal through the intervertebral foramen. That is, the approach vector (direction of the needle) and the exit vector (angle at which the nerves are exiting the intervertebral foramen) have a higher degree of alignment with the Winnie technique.

William Halsted performed the first brachial plexus block in 1885. The technique involved surgical exposure of the brachial plexus with direct application of cocaine.<sup>2</sup> The first recorded interscalene plexus block was most likely performed by July Etienne in 1925.<sup>2</sup> He inserted a needle at

the level of the cricothyroid membrane halfway between the lateral border of the sternocleidomastoid and the anterior border of the trapezius muscle to block the plexus.

The next improvement was made by Winnie.<sup>3</sup> He was the first to recommend placing the needle within the interscalene groove after manual identification. The direction of the needle was perpendicular to the skin in every plane, pointing in a direction that is mostly medial, but slightly posterior and slightly caudad. The interscalene catheter is nowadays the accepted standard for perioperative and postoperative pain therapy after shoulder surgery. It offers better pain control, fewer side effects, earlier mobilization, and higher patient satisfaction.<sup>4-6</sup> However, the use of the Winnie approach does not offer the best conditions for the placement of a catheter, because the direction of the needle is perpendicular to the trunks. Therefore, new approaches making its placement easier have been developed.<sup>7,8</sup> Interestingly, changing the direction in a more caudad direction—within the tridimensional plane of the interscalene groove—was initially driven more to help catheter placement than to increase the safety of the technique.

Severe complications have been reported after interscalene block, including spinal<sup>9,10</sup> or epidural anesthesia<sup>11,12</sup> and even permanent loss of cervical cord function.<sup>13</sup> Sardesai *et al.*<sup>1</sup> provides substantial new information to have a better understanding of the occurrence of these complications. Although it seems obvious that directing the needle medially during performance of an interscalene block carries some risk, Sardesai *et al.*<sup>1</sup> were able to demonstrate that the angle of approach for performing an interscalene block using the Winnie technique almost matches the angle made by the exiting nerve and neural foramen. These findings are clinically relevant because they clearly show that this technique has the greatest degree of alignment, making epidural, spinal, and intramedullary local anesthetic application a likely occurrence. It also enhances the risks of drug administration into the extraforaminal space. It is known that the neural sheath may extend to the paravertebral space. From there, the local anesthetic may diffuse from the paravertebral gutter through the intervertebral foramina to the epidural space and then reach the cerebrospinal fluid.<sup>14</sup> This mechanism may explain some of the complications occurring with the Winnie approach.

Another important aspect of the investigation by Sardesai *et al.*<sup>1</sup> is the demonstration that the shortest skin-intervertebral foramen was found with the Winnie technique. "Regionalists" already surmised this issue, because one recommendation is not to use a needle longer than 2.5 cm. This may be a safe precaution, but certainly not a panacea. The needle may be too short in patients with a thick neck or a generous fat layer. For the latter, it is often quite hazardous to try to anticipate the depth at which to expect to encounter the trunks. Moreover, the use of a short needle in this context will not be a definitive

◆ This Editorial View accompanies the following article: Sardesai AM, Patel R, Denny NM, Menon DK, Dixon AK, Herrick MJ, Harrop-Griffiths AW: Interscalene brachial plexus block: Can the risk of entering the spinal canal be reduced? A study of needle angles in volunteers undergoing magnetic resonance imaging. ANESTHESIOLOGY 2006; 105:9-13.

Accepted for publication December 20, 2005. The author is not supported by, nor maintains any financial interest in, any commercial activity that may be associated with the topic of this article.

precaution to avoid drug administration within the extraforaminal space.

This investigation has some weaknesses. Detractors will still criticize the current study because of its small sample size, because the body mass index was not taken into account, and because the experimental conditions do not match the clinical reality. The latter point is certainly true with the new approaches,<sup>7,8</sup> but is not far from the clinical reality when using the Winnie technique. This further highlights the potential danger associated with the classic Winnie technique.

What about the interscalene catheter? The results of this investigation let us fear that inadvertent catheterization of the epidural or spinal space can (too) easily occur. The catheter will go toward the direction of the needle. Sardesai *et al.*<sup>1</sup> showed that the use of the Winnie technique gives the catheter good conditions to go through the intervertebral foramen.

Another issue is to consider whether the new approaches (direction of the needle more caudad) will create new complications, such as pneumothorax. It is still too premature to give a definitive response, but initial studies of interscalene single-shot and catheter have reported only one case of pneumothorax occurring in a patient with Marfan syndrome.<sup>7,15,16</sup>

Should the Winnie technique be avoided for interscalene block? When considering the results of Sardesai *et al.*<sup>1</sup> and the safety of regional anesthesia, the answer is yes. First, alternatives do exist, because approaches that likely have a wider margin of safety have been described. Second, usual precautions, like the use of a short needle for performing this block, are not sufficient for all patients. Last, the safety margin is very small, an important issue for nonexperienced anesthetists. What about experienced anesthesiologists? Compared with nonexperienced colleagues, competent anesthetists have good tires to drive on an unsalted icy road, but the road is

nevertheless still icy. It is therefore still recommended for all drivers to use the salted icy road! *Primum non nocere.*

**Alain Borgeat, M.D.**, Department of Anesthesiology, Orthopedic University Hospital Balgrist, Zurich, Switzerland. [alain.borgeat@balgrist.ch](mailto:alain.borgeat@balgrist.ch)

## References

1. Sardesai AM, Patel R, Denny NM, Menon DK, Dixon AK, Herrick MJ, Harrop-Griffiths AW: Interscalene brachial plexus block: Can the risk of entering the spinal canal be reduced? A study of needle angles in volunteers undergoing magnetic resonance imaging. *ANESTHESIOLOGY* 2006; 105:9-13
2. Winnie AP: *Plexus anesthesia, Perivascular Techniques of Brachial Plexus Block*. Philadelphia, WB Saunders, 1983
3. Winnie AP: Interscalene brachial plexus block. *Anesth Analg* 1970; 49:455-66
4. Borgeat A, Schappi B, Biasca N, Gerber C: Patient-controlled analgesia after major shoulder surgery: Patient-controlled interscalene analgesia *versus* patient-controlled analgesia. *ANESTHESIOLOGY* 1997; 87:1343-7
5. Borgeat A, Tewes E, Biasca N, Gerber C: Patient-controlled interscalene analgesia with ropivacaine after major shoulder surgery: PCIA *versus* PCA. *Br J Anaesth* 1998; 81:603-5
6. Singelyn FJ, Seguy S, Gouverneur JM: Interscalene brachial plexus analgesia after open shoulder surgery: Continuous *versus* patient-controlled infusion. *Anesth Analg* 1999; 89:1216-20
7. Meier G, Bauereis C, Heinrich C: Interscalene brachial plexus catheter for anesthesia and postoperative pain therapy: Experience with a modified technique [in German]. *Anaesthesist* 1997; 46:715-9
8. Borgeat A, Ekatothramis G: Anaesthesia for shoulder surgery. *Best Pract Res Clin Anaesthesiol* 2002; 16:211-25
9. Dutton RP, Eckhardt WF 3rd, Sunder N: Total spinal anesthesia after interscalene blockade of the brachial plexus. *ANESTHESIOLOGY* 1994; 80:939-41
10. Passannante AN: Spinal anesthesia and permanent neurologic deficit after interscalene block. *Anesth Analg* 1996; 82:873-4
11. Kumar A, Battit GE, Froese AB, Long MC: Bilateral cervical and thoracic epidural blockade complicating interscalene brachial plexus block: Report of two cases. *ANESTHESIOLOGY* 1971; 35:650-2
12. Mahoudeau G, Gaertner E, Launoy A, Ocquidant P, Loewenthal A: Interscalenic block: Accidental catheterization of the epidural space [in French]. *Ann Fr Anesth Reanim* 1995; 14:438-41
13. Benumof JL: Permanent loss of cervical spinal cord function associated with interscalene block performed under general anesthesia. *ANESTHESIOLOGY* 2000; 93:1541-4
14. Purcell-Jones G, Pither CE, Justins DM: Paravertebral somatic nerve block: A clinical, radiographic, and computed tomographic study in chronic pain patients. *Anesth Analg* 1989; 68:32-9
15. Borgeat A, Ekatothramis G, Kalberer F, Benz C: Acute and nonacute complications associated with interscalene block and shoulder surgery: A prospective study. *ANESTHESIOLOGY* 2001; 95:875-80
16. Borgeat A, Dullenkopf A, Ekatothramis G, Nagy L: Evaluation of the lateral modified approach for continuous interscalene block after shoulder surgery. *ANESTHESIOLOGY* 2003; 99:436-42

*Anesthesiology* 2006; 105:2-5

© 2006 American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

## Postpneumonectomy Pulmonary Edema

### Good News, Bad News

IN this issue of *ANESTHESIOLOGY*, Drs. Fernández-Pérez *et*

This Editorial View is accompanied by the following article: Fernández-Pérez ER, Keegan MT, Brown DR, Hubmayr RD, Gajic O: Intraoperative tidal volume as a risk factor for respiratory failure after pneumonectomy. *ANESTHESIOLOGY* 2006; 105:14-8.

*al.*<sup>1</sup> present a retrospective analysis of perioperative risk factors related to the development of respiratory failure necessitating mechanical ventilatory assistance beyond 48 h after pneumonectomy. Of 170 pneumonectomy patients studied during a 4-yr period at one institution, 30 developed postoperative respiratory failure. Half (15) of these respiratory failure cases were due to complications common to all major intrathoracic (and many non-thoracic) surgeries such as cardiogenic pulmonary edema, pneumonia, and pulmonary emboli. The other 15 cases (9% of pneumonectomies) were due to acute

Accepted for publication February 14, 2006. The author is not supported by, nor maintains any financial interest in, any commercial activity that may be associated with the topic of this article.

lung injury (ALI). This latter 9% represent a dreaded complication both for anesthesiologists and thoracic surgeons because this “postpneumonectomy pulmonary edema,”<sup>1</sup> unrelated to other identifiable etiologies of respiratory failure, has a case-fatality rate of more than 50% in most reports.<sup>2</sup> The most significant perioperative factor that Fernández-Pérez *et al.* found to be associated with postpneumonectomy respiratory failure was larger intraoperative tidal volumes (median 8.3 ml/kg predicted body weight in failure patients *vs.* 6.7 ml/kg in nonfailure pneumonectomy controls). The other significant factor was larger amounts of intraoperative fluids administered (median 2.2 l for cases *vs.* 1.3 l for controls). Patients who developed respiratory failure had a higher 60-day mortality than controls (23% *vs.* 4%) and a longer hospital stay (22 *vs.* 6 days).

Before we conclude that large tidal volumes and intravenous fluids contribute to postpneumonectomy pulmonary edema, there are some caveats that we must place on the analysis and that the authors largely acknowledge. In their retrospective analysis, the authors were not able to get clear data on the exact tidal volumes or duration of one-lung ventilation. They could only document the largest intraoperative tidal volume; this could represent one- or two-lung ventilation. However, because it has been a common clinical practice to use the same tidal volume for one- and two-lung ventilation,<sup>3</sup> I believe these data are probably valid. Also, the authors do not provide a between-subgroup comparison of the associations with tidal volume and fluids for the ALI *versus* the non-ALI respiratory failure cases. It seems plausible that tidal volumes might impact ALI but would not have an effect on non-ALI cases, whereas fluids could impact both cardiogenic and noncardiogenic pulmonary edema.

The original description of postpneumonectomy pulmonary edema as a specific entity seems to have been in a series of 10 cases published in 1984 by Zeldin *et al.*<sup>4</sup> After retrospective comparison with controls, they identified three significant risk factors: right pneumonectomy (9 of 10 cases), increased perioperative intravenous fluids, and increased postoperative urine output. Zeldin *et al.* further demonstrated their thesis that this was an anesthetic complication caused by overhydration by producing postpneumonectomy pulmonary edema in a dog model with fluid overload. In their recommendations, they wrote, “. . . the most important thing that we can do in terms of recognizing this problem is to watch our anesthetists as they start loading the patient up with fluid.” In the 20+ yr since the article of Zeldin *et al.* was published, there have been at least a dozen similar case-series reviews of this topic, with varied conclusions about the role of fluid administration as a cause of this complication. Also, a variety of other associated and potentially causative factors have been proposed, such as the administration of fresh frozen plasma, mediastinal

lymphatic damage,<sup>5</sup> serum cytokines, and oxygen toxicity.<sup>6</sup>

The largest study of postpneumonectomy pulmonary edema was by Turnage and Lunn.<sup>7</sup> In a retrospective survey of 806 pneumonectomies published in 1993 (from the same institution as Fernández-Pérez *et al.*), they found 21 cases (2.5%) of postpneumonectomy pulmonary edema, one of the lowest incidences reported of this complication. They found no differences in any measure of perioperative fluid balance between postpneumonectomy pulmonary edema cases (mean positive fluid balance at 24 h = 10 ml/kg) *versus* uncomplicated pneumonectomy controls (24-h positive balance = 13 ml/kg). However, the routine practice at their institution was rigorous fluid restriction, compared with many other reports where the 24-h fluid balance often exceeds 20 ml/kg.<sup>8</sup> This suggests that by limiting fluids the incidence of postpneumonectomy, ALI can be decreased but not eliminated.

Traditional teaching has been to use large tidal volumes, 10–12 ml/kg, during one-lung ventilation to prevent atelectasis in the dependent lung and to avoid hypoxemia.<sup>3</sup> However, the incidence of hypoxemia during one-lung ventilation has declined from 20–25% in the 1970s<sup>9</sup> to less than 1% currently.<sup>10</sup> This decrease can be attributed to several advances in thoracic anesthesia, including the use of fiberoptic bronchoscopy for positioning double-lumen endobronchial tubes and bronchial blockers and the use of newer volatile anesthetics<sup>11</sup> (isoflurane, sevoflurane, desflurane) that cause less inhibition of hypoxic pulmonary vasoconstriction and less shunt during one-lung ventilation than older volatile agents.<sup>12</sup> Before this study of Fernández-Pérez *et al.*, several other reports have also suggested that the use of large tidal volumes and pressures during one-lung ventilation may contribute to post-lung resection ALI. Van der Werff *et al.*<sup>13</sup> found ALI, diagnosed radiographically, in 42% of pneumonectomy patients who were ventilated with peak airway pressures greater than 40 cm H<sub>2</sub>O. Licker *et al.*<sup>14</sup> found that the most significant predictor of ALI was the product of the airway pressure and the duration of one-lung ventilation. Also, bronchial lavage levels of some inflammatory markers were higher after one-lung ventilation with 10 ml/kg tidal volumes *versus* 5 ml/kg.<sup>15</sup>

Central to our current understanding of postpneumonectomy ALI is the appreciation that the patients develop a low-pressure, high-protein-content pulmonary edema, which indicates an endothelial injury.<sup>7</sup> It has been demonstrated that the nonoperated lung develops a capillary-leak injury after a pneumonectomy but not a lobectomy.<sup>16</sup> There is no single mechanism that can fully explain ALI after lung resection, and its etiology is likely multifactorial; it may represent one end of a spectrum of lung injury that occurs with all pulmonary resections and is proportional to the amount of lung tissue resected. Changes in plasma makers of oxidative damage after

pulmonary resection were found to be largest in pneumonectomy patients, less in lobectomy, and not significant in wedge resection or abdominal surgery.<sup>17</sup>

Understanding that lung endothelial injury occurs after major lung resection supports management principles similar to other conditions associated with ALI and acute respiratory distress syndrome.<sup>18</sup> As a general principle, it seems that the lung is least injured when a pattern of ventilation as close as possible to normal spontaneous ventilation can be followed: fraction of inspired oxygen as low as acceptable, variable tidal volumes, beginning inspiration at functional residual capacity, and avoiding atelectasis<sup>19</sup> with frequent recruitment maneuvers.<sup>20</sup> Studies in acute respiratory distress syndrome demonstrate that ALI is exacerbated by the use of large tidal volumes and that lung-protective ventilation strategies with low tidal volumes and positive end-expiratory pressure are less injurious.<sup>21</sup> The most important factor in the etiology of ventilator-induced lung injury is the end-inspiratory lung volume.<sup>22</sup> Many patients, particularly those with emphysema, develop auto-positive end-expiratory pressure during one-lung ventilation,<sup>23</sup> thus beginning inspiration at a lung volume above functional residual capacity. It is conceivable that routine use of large tidal volumes (10–12 ml/kg) during one-lung ventilation in such patients produces end-inspiratory lung volumes close to levels that contribute to ALI, particularly in the smaller left lung.

Based on our current appreciation of post-lung resection ALI, several management principles for pneumonectomy (and potential pneumonectomy) patients seem evident. Overinflation of the nonoperated (ventilated) lung should be avoided using lung-protective ventilation (tidal volumes 5–6 ml/kg), adding positive end-expiratory pressure to those patients without auto-positive end-expiratory pressure and limiting plateau and peak inspiratory pressures to less than 25 cm H<sub>2</sub>O and less than 35 cm H<sub>2</sub>O, respectively.<sup>24</sup> Minimizing pulmonary capillary pressures by avoiding overhydration for patients undergoing pneumonectomy is reasonable, while acknowledging that not all increases in pulmonary artery pressures perioperatively are due to intravascular volume replacement. Other factors, such as hypercapnia, hypoxemia, and pain, can all increase pulmonary pressures and must be treated.

It should be appreciated that not all hyperinflation of the residual lung occurs in the operating room. Overexpansion of the remaining lung after pneumonectomy may occur postoperatively either with or without a chest drain in place. This prolonged hyperinflation during the period of increased endothelial permeability may be one of the major causes of postpneumonectomy pulmonary edema. There is currently no consensus among thoracic surgeons on the best method to manage the postpneumonectomy chest cavity. There are at least four methods: chest closure without a chest drain, attachment of a

chest drain to underwater seal, repeated unclamping of a chest drain, and use of a balanced chest drainage system to maintain the mediastinum in a neutral position.<sup>25</sup> Use of a balanced chest drainage system has been suggested to contribute to a marked decline in postpneumonectomy pulmonary edema in one center.<sup>26</sup> A sheep study (University of Western Australia, Perth, Australia) found a significant reduction in postpneumonectomy pulmonary edema with the use of a balanced chest drainage system compared with no drain or the other methods of chest drain management (personal communication, John M. Alvarez, M.B., B.S., F.R.A.C.S., Clinical Associate Professor, Department of Cardiothoracic Surgery, January 2006).

In summary, there is good news and bad news about postpneumonectomy ALI. The good news is that as the etiology begins to become clearer and we understand that there is a postresection lung endothelial injury, we can begin to use ventilation strategies that have been shown to improve survival in patients with other forms of ALI. Also good news is that we, as anesthesiologists, do not cause the injury with intravenous fluids (we can make it worse, but we do not cause it). And also good news is that the mortality of postpneumonectomy pulmonary edema seems to be decreasing. Fernández-Pérez *et al.* found that greater than 75% of patients survived. This compares to less than 50% survival in previous reports. However, this may be more related to better intensive care of established cases than to anesthetic management.<sup>27</sup>

The bad news is that the incidence of postpneumonectomy pulmonary edema does not seem to be decreasing. The incidence in the current study is 9%, compared with an incidence of less than 3% at the same institution 10 yr ago.<sup>7</sup> This could be due heightened awareness and more aggressive treatment. Also, few anesthesiologists have yet adopted lung-protective ventilation in thoracic anesthesia, so it may be too early to expect an improvement. Also bad news is that fluid restriction does seem to be indicated for anesthetic management of pneumonectomy patients. This complicates perioperative management in patients who often receive thoracic epidural analgesia and tend to be hypotensive. And finally, bad news is that much of the etiology of post-lung resection ALI may be related to the extent of the surgical resection and the postoperative chest drain management and thus may be out of the control of the anesthesiologist.

**Peter D. Slinger, M.D.**, Department of Anesthesia, University of Toronto, Toronto General Hospital, Toronto, Ontario, Canada. peter.slinger@uhn.on.ca

## References

1. Fernández-Pérez ER, Keegan MT, Brown DR, Hubmayr RD, Gajic O: Intraoperative tidal volume as a risk factor for respiratory failure after pneumonectomy. *ANESTHESIOLOGY* 2006; 105:14–8
2. Slinger P: Perioperative fluid management for thoracic surgery: The puzzle

of postpneumonectomy pulmonary edema. *J Cardiothorac Vasc Anesth* 1995; 9:442-51

3. Brodsky JB, Fitzmaurice B: Modern anesthetic techniques for thoracic operations. *World J Surg* 2001; 25:162-6
4. Zeldin RA, Normadin D, Landtwing BS, Peters RM: Postpneumonectomy pulmonary edema. *J Thorac Cardiovasc Surg* 1984; 87:359-65
5. Slinger P: Post-pneumonectomy pulmonary edema. *Curr Opin Anesthesiol* 1999; 12:49-54
6. Lases EC, Duurkens VAM, Gerristen WBM, Haas FJLM: Oxidative stress after lung resection surgery. *Chest* 2000; 117:999-1003
7. Turnage WS, Lunn JL: Postpneumonectomy pulmonary edema: A retrospective analysis of associated variables. *Chest* 1993; 103:1646-50
8. Waller DA, Gebitekin C, Saundres NR, Walker DR: Noncardiogenic pulmonary edema complicating lung resection. *Ann Thorac Surg* 1993; 55:140-3
9. Tarhan S, Lundborg RO: Carlens endobronchial catheter *versus* regular endotracheal tube during thoracic surgery: A comparison of blood gas tensions and pulmonary shunting. *Can Anaesth Soc J* 1970; 17:4-11
10. Brodsky J, Lemmens HJ: Left double-lumen tubes: Clinical experience with 1,170 patients. *J Cardiothorac Vasc Anesth* 2003; 17:289-98
11. Reid CW, Slinger P, Lenis S: Comparison of the effects of propofol-alfentanil *versus* isoflurane anesthesia on arterial oxygenation during one-lung anesthesia. *J Cardiothorac Vasc Anesth* 1997; 10:860-3
12. Slinger P, Scott WAC: Arterial oxygenation during one-lung anesthesia: A comparison of enflurane and isoflurane. *ANESTHESIOLOGY* 1995; 82:940
13. van der Werff YD, van der Houwen HK, Heilmans PJ, Duurkens VA, Leusink HA, van Heesewijk HP, de Boer A: Postpneumonectomy pulmonary edema: A retrospective analysis of incidence and possible risk factors. *Chest* 1997; 111:1278-8
14. Licker M, De Perrot M, Spiliopoulos A, Robert J, Diaper J, Chevalley C, Tschopp JM: Risk factors for acute lung injury after thoracic surgery for lung cancer. *Anesth Analg* 2003; 97:1558-65
15. Schilling T, Kozain A, Huth C, Buhling F, Kretzschmar M, Welte T,

Hachenb T: The pulmonary immune effects of mechanical ventilation in patients undergoing thoracic surgery. *Anesth Analg* 2005; 101:957-65

16. Waller DA, Keavey P, Woodfine L, Dark JH: Pulmonary endothelial permeability changes after major resection. *Ann Thorac Surg* 1996; 61:1435-40
17. Williams EA, Quinlan GJ, Goldstraw P, Gothard JW, Evans TW: Postoperative lung injury and oxidative damage in patients undergoing pulmonary resection. *Eur Respir J* 1998; 11:1028-34
18. The Acute Respiratory Distress Syndrome Network: Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. *N Engl J Med* 2000; 342:1301-8
19. Duggan M, Kavanagh B: Pulmonary atelectasis a pathogenic perioperative entity. *ANESTHESIOLOGY* 2005; 102:838-54
20. Mols G, Priebe H-J, Guttmann J: Alveolar recruitment in acute lung injury. *Br J Anaesth* 2006; 96:156-66
21. Boker A, Haberman C, Girling L, Guzman RP, Louridas G, Tanner JR, Cheang M, Maycher BW, Bell DD, Doak GJ: Variable ventilation improves perioperative lung function in patients undergoing abdominal aortic aneurysmectomy. *ANESTHESIOLOGY* 2004; 100:608-16
22. Dreyfuss D, Soler P, Basset G, Saumon G: High Inflation pressure pulmonary edema. *Am Rev Resp Dis* 1988; 137:1159-64
23. Slinger P, Hickey DR: The interaction between applied PEEP and auto-PEEP during one-lung ventilation. *J Cardiothorac Vasc Anesth* 1998; 12:133-6
24. Slinger P: Pro: Low tidal volume is indicated during one-lung ventilation. *Anesth Analg* 2006; 103:(in press)
25. Deslauriers J, Gregoire J: Techniques of pneumonectomy: Drainage after pneumonectomy. *Chest Surg Clin N Am* 1999; 9:337-48
26. Alvarez JM, Panda RK, Newman MA, Slinger P, Deslauriers J, Ferguson M: Postpneumonectomy pulmonary edema. *J Cardiothorac Vasc Anesth* 2003; 17:388-95
27. Keegan MT, Harrison BA, De Ruyter ML, Deschamps C: Post-pneumonectomy pulmonary edema: Are we making progress? (abstract). *ANESTHESIOLOGY* 2004; 101:A431

*Anesthesiology* 2006; 105:5-7

© 2006 American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

## Automated Anesthesia Charge Capture and Submission

### Wave of the Future, or Bridge to Nowhere?

**CME** This editorial accompanies the article selected for this month's *Anesthesiology* CME Program. After reading the article and editorial, go to <http://www.asahq.org/journal-cme> to take the test and apply for Category 1 credit. Complete instructions may be found in the CME section at the back of this issue.

ELECTRONIC anesthesia records with anesthesia information management systems (AIMS) continue to grow in prevalence. AIMS have been of interest from the perspectives of anesthesiologists providing clinical care, such as implementing practice guidelines and patient safety, and from the perspective of hospitals or facilities,

This Editorial View accompanies the following article: Reich DL, Kahn RA, Wax D, Palvia T, Galati M, Krol M: Development of a module for point-of-care charge capture and submission using an anesthesia information management system. *ANESTHESIOLOGY* 2006; 105:179-86.

typically the "clients" actually purchasing the system and who strive for an improvement in cost and charge capture.<sup>1-4</sup> In contrast, ambulatory clinics have implemented electronic medical records with charge capture modules focused on the professional or physician services. In this month's *ANESTHESIOLOGY*, Dr. Reich *et al.*<sup>5</sup> have demonstrated the successful use AIMS to capture and submit charges for anesthesia professional services. The question for anesthesiology groups is no longer "Is it possible?" but rather "Is this something I need or want to pay for?"

The process of automatic charge capture and submission can be viewed as having four major components: (1) automatic charge capture from data in the AIMS; (2) point-of-care coding, using software implemented by the attending physician; (3) automatic submission of the charge if documentation is complete as determined by software; and (4) automatic notification (*via* e-mail) to the attending physician if documentation is incomplete as determined by software. Each component has advantages and disadvantages, as well as varying costs for implementation. Each can also be considered as an independent process that can be used in part or as a whole.

Accepted for publication February 13, 2006. The authors are not supported by, nor maintain any financial interest in, any commercial activity that may be associated with the topic of this article.

The first component, automatic charge capture, is not a new concept within medicine in general, but it has not been previously used for professional charges for anesthesia services. Its major advantage is that the charge is created without additional effort (paper or electronic) by the physician other than the clinical documentation. Automated charge capture also eliminates the need to reconcile the operating room schedule with the charges submitted, to be certain that none are missing. For academic departments, the system is attractive because it can be used to capture clinical services performed by residents but not currently billed as well as other potential revenue-producing activities. It may also enhance compliance because the charges cannot be created without requiring additional documentation. Also, anesthesia records for insurance appeals can be easily located within the AIMS.

Unfortunately, there are also distinct disadvantages of the automatic charge capture. First, because the AIMS does not have comprehensive software to capture charges automatically, the authors were required to develop their own. This additional software queried the existing AIMS and used a decision tree based on current billing rules. Therefore, IT support is critical to the system, and havoc could occur should such a critical individual depart. Also, billing rules change and system troubleshooting must be readily available. There is no "off-the-shelf" software for professional fees because the purchaser of the AIMS is typically the hospital, not the anesthesiologist or anesthesia group. It is no surprise that these charge capture packages developed for AIMS have centered around hospital billing, coding, and cost capture. However, because many hospitals must help to fund anesthesia staffing costs, any improvement in anesthesiology revenue should translate to an advantage for both the hospital and the anesthesiology group. Perhaps such off-the-shelf packages may become available in the future. Finally, the cost for interfacing with multiple information system platforms can be expensive and should not be underestimated. The system described by Reich *et al.* interfaces with many systems, including the AIMS, the hospital's financial system, the anesthesiology group's billing software, and the third party payer's software, which can be unique for each payer.

The second component of the AIMS is point-of-care coding, typically performed by the anesthesiologist with the assistance of drop-down menus and search engines. In a fully automatic system, the surgical procedure code could be automatically completed using the surgeon's entry. In the system described, the anesthesiologist completed this function, using the software's drop-down menus and electronic search of codes. Such a system can also be engineered to show the most common surgical procedures performed by a

surgeon. To make coding easier, several commercial products currently offer this technology using personal data applications. These allow for coding as well as electronic charge creation. Unlike the automatic charge capture noted above, the personal data application systems require the anesthesiologist to enter the elements of the charge slip, but they do so at the point of care.

The automatic charge submission is the logical next step. For physicians such as pediatricians and radiologists who produce many small charges, an AIMS coupled with automated charge capture is very attractive and fiscally efficient. In contrast, charges for anesthesia services are typically "big ticket" items, and the loss of even one per day can be substantial. The savings are less for anesthesia services because the volume of charges is smaller. The benefits of human oversight may still outweigh the advantages of automatic submission, and careful tracking of submitted charges is necessary to prevent their loss.

The automatic e-mail reminders for incomplete documentation are an attractive feature of the authors' system. However, it is unclear just where the anesthesiologists can actually access the AIMS to complete necessary documentation. Ideally, the anesthesiologist could do so from any terminal and would not need to be physically present in the operating room or hospital. The use of e-mail reminders could also be implemented with clerical reviews of documentation, but the advantage of automatic reminders may well be more timely.

The study by Reich *et al.* showed that this complete charge capture system is possible; however, the cost-benefit of its development and implementation are important. The authors found a one-time benefit from a reduction of "lag days" from 10 to 3. Applying this revenue to the initial costs of the system must be done with some caution. As noted by the authors, at the same time as the system was implemented, a physician incentive compensation system based on charges was also initiated. The incentive system itself could easily reduce lag days and increase charges. Further, if a group's lag days already range between 3 and 5, little of the one-time revenue gain would be realized.

The costs of developing an automated information system from scratch, in addition to its ongoing maintenance, may well equal any potential savings from the reduction of clerical full-time equivalents. Indeed, the implementation of automated systems do not necessarily reduce labor costs, but rather shift them from clerical staff to staff dedicated to the automated system. If an AIMS is not available, an anesthesiology group can still take advantage of some of the processes studied by using other commercially available

products in conjunction with the billing office, perhaps at a fraction of the cost, using e-mail notification for incomplete documentation.

Finally, the strategic costs of having the hospital own the data, giving unfettered access to billing and revenue information, must be weighed carefully. Because the hospital typically owns the AIMS, the hospital, and not the anesthesiology group, owns the data. In our world today, this situation may be less than desirable.

**Amr E. Abouleish, M.D., M.B.A.,\* Lydia Conlay, M.D., Ph.D., M.B.A.†**  
\*Department of Anesthesiology, University of Texas Medical Branch, Galveston, Texas. aaboule@utmb.edu †Department of Anesthesiology, Baylor College of Medicine, Houston, Texas.

Anesthesiology 2006; 105:7-8

## References

1. Lubarsky DA, Sanderson IA, Gilbert WC, King KP, Ginsberg D, Dear GL, Coleman RL, Pafford TD, Reves JG: Using an anesthesia information management system as a cost containment tool: Description and validation. *ANESTHESIOLOGY* 1997; 86:1161-9
2. Junger A, Benson M, Quinzio L, Michel A, Sciuk G, Brammen D, Marquardt K, Hempelmann G: An anesthesia information management system (AIMS) as a tool for controlling resource management of operating rooms. *Methods Information Med* 2002; 41:81-5
3. Reich DL, Hossain S, Krol M, Baez B, Patel P, Bernstein A, Bodian C: Predictors of hypotension after induction of general anesthesia. *Anesth Analg* 2005; 101:622-8
4. Lesser JB, Sanborn KV, Valskys R, Kuroda M: Severe bradycardia during spinal and epidural anesthesia recorded by an anesthesia information management system. *ANESTHESIOLOGY* 2003; 99:859-66
5. Reich DL, Kahn RA, Wax D, Palvia T, Galati M, Krol M: Development of a module for point-of-care charge capture and submission using an anesthesia information management system. *ANESTHESIOLOGY* 2006; 105:179-86

© 2006 American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

# Lipid Infusion Resuscitation for Local Anesthetic Toxicity

## Proof of Clinical Efficacy

DR. Rosenblatt and colleagues at Mount Sinai Hospital in New York report in this issue of *Anesthesiology* the successful application of lipid emulsion infusion in the resuscitation of bupivacaine-induced cardiac arrest.<sup>1</sup> The patient was a 58-yr-old man with a history of coronary artery disease who presented for an elective shoulder procedure. Shortly after an interscalene block with bupivacaine (100 mg) and mepivacaine (300 mg), the patient experienced a brief seizure followed by asystole with intervals of ventricular tachycardia. These arrhythmias were refractory to multiple rounds of drugs and countershocks until a member of the staff recommended using intravenous lipid therapy. Soon after administering 100 ml of 20% Intralipid, a single heartbeat was observed, followed 20 s later by the return to a sinus mechanism with normal blood pressure. The patient was later extubated and recovered without neurologic deficit.

This remarkable case report is a watershed in the study of local anesthetic toxicity and might well mark the end of nearly 30 years of regional anesthesia practiced with-

out a specific antidote for its most dread complication. There is a well-defined conceptual framework linking this patient to one whose extreme sensitivity to bupivacaine was reported 9 yr ago.<sup>2</sup> That patient experienced ventricular arrhythmias and bradycardia after receiving 22 mg bupivacaine in a subcutaneous tumescence solution. These clinicians later learned that the patient was severely carnitine deficient and investigated a possible connection between this metabolic abnormality and her sensitivity to bupivacaine. The studies in isolated mitochondria revealed that bupivacaine interferes with carnitine-dependent mitochondrial lipid transport.<sup>3</sup> While attempting to understand the relation of lipid metabolism to bupivacaine toxicity, the unexpected finding in rats was that pretreatment with a lipid infusion increases the bupivacaine dose required to induce asystole.<sup>4</sup> Similarly, administering lipid during resuscitation reliably rescued rats from otherwise fatal doses of bupivacaine. Similar observations were made in dogs where the protocol included an interval of 10 min before treatment to mimic the clinical setting where a delay in administering lipid is likely.<sup>5</sup> None of the six controls receiving 10 mg/kg bupivacaine recovered with cardiac massage alone, whereas all lipid-treated dogs recovered normal hemodynamic profiles. Fortunately for the patient reported in this issue, the phenomenon of lipid rescue seems to work equally well in humans.

This case report might provide the impetus to establish a uniform, coherent, and rational approach to treating severe local anesthetic toxicity. A group from Wake Forest Medical Center (Winston-Salem, North Carolina)

This Editorial View accompanies the following article: Rosenblatt MA, Abel M, Fischer GW, Itzkovich CJ, Eisenkraft JB: Successful use of a 20% lipid emulsion to resuscitate a patient after a presumed bupivacaine-related cardiac arrest. *ANESTHESIOLOGY* 2006; 105:217-8.

Accepted for publication March 2, 2006. The author is not supported by, nor maintains any financial interest in, any commercial activity that may be associated with the topic of this article.

recently queried academic anesthesiology departments in the United States regarding their approach to treating local anesthetic toxicity.<sup>6</sup> Lamentably, it was clear from the 91 responding institutions that there is little uniformity in planning for this potentially catastrophic complication. Only a small fraction of respondents would consider using lipid to treat local anesthetic toxicity.

Substantial research on lipid rescue is still needed. Intravenous lipid emulsion has a long track record of safety as hyperalimentation and in formulations of propofol, but its safety is unknown when administered in the high doses used in lipid rescue. Although it is reassuring to note that the patient in the report of Rosenblatt *et al.*<sup>1</sup> was neurologically intact after the event, we must keep in mind that this represents a single case. Specific factors to study include defining the optimal lipid dose, rate, and duration of infusion as well as establishing a safe upper limit. It remains unanswered whether there is more benefit or harm in using epinephrine in local anesthetic cardiac toxicity, although currently, I would continue to recommend its use as part of the standard American Heart Association Advanced Cardiac Life Support protocol.

Physicians should be made aware that propofol is not a component of lipid rescue. I raise this issue because it is a common misconception that lipid rescue implies the use of propofol, which is formulated in a 10% lipid emulsion. Although small doses of propofol might be of benefit to control seizure activity in the early stages of a toxic event, propofol is contraindicated when there is any evidence of cardiac toxicity. The standard 1% formulation would require delivering gram quantities of propofol to provide the needed dose of lipid. This is unacceptable in the setting of cardiovascular collapse.

Until further studies identify an optimal regimen, lipid infusion should be used, as in this case report, only after standard resuscitative measures have proven ineffective. I believe the evidence in support of its use is now sufficient to warrant having 20% lipid emulsion available in all operating rooms, block rooms, obstetric units, and other sites where local anesthetics are used (including plastic surgery suites). It is worth noting that it has recently been found that bupivacaine delays the onset of myocardial acidosis during no-flow states, suggesting that bupivacaine may provide some degree of cardiac protection during cardiovascular collapse.<sup>7</sup> The point is

that lipid rescue should be considered before ceasing resuscitative efforts even if its use is contemplated after a significant delay in the setting of prolonged cardiac arrest.

The mechanisms underlying lipid rescue are still incompletely understood. Recent research found in isolated rat heart that lipid infusion accelerates the decline in bupivacaine myocardial content and speeds recovery from bupivacaine-induced asystole.<sup>8</sup> Lipid infusion might also provide a salutary metabolic effect to the heart<sup>9</sup> or some other, as yet unidentified benefit. Further research to delineate the mechanisms at play might lead to even more effective therapy.

Dr. Rosenblatt and her team are to be thoroughly congratulated for saving this patient's life. While proving the clinical efficacy of lipid rescue, they have also validated a contemporary model of academic anesthesiology. There are limits to the information one can draw from a single case, but in the scenario where prospective clinical trials are impossible, we can take heart from this reported experience. A once feared complication of regional anesthesia may have just become slightly less fearsome.

**Guy Weinberg, M.D.**, Department of Anesthesiology, University of Illinois at Chicago, Chicago, Illinois. [guyw@uic.edu](mailto:guyw@uic.edu)

## References

1. Rosenblatt MA, Abel M, Fischer GW, Itzkovich CJ, Eisenkraft JB: Successful use of a 20% lipid emulsion to resuscitate a patient after a presumed bupivacaine-related cardiac arrest. *ANESTHESIOLOGY* 2006; 105:217-8
2. Weinberg GL, Laurito CE, Geldner P, Pygon BH, Burton BK: Malignant ventricular dysrhythmias in a patient with isovaleric acidemia receiving general and local anesthesia for suction lipectomy. *J Clin Anesth* 1997; 9:668-70
3. Weinberg GL, Palmer JW, VadeBoncouer TR, Zuechner MB, Edelman G, Hoppel CL: Bupivacaine inhibits acylcarnitine exchange in cardiac mitochondria. *ANESTHESIOLOGY* 2000; 92:523-8
4. Weinberg GL, VadeBoncouer T, Ramaraju GA, Garcia-Amaro MF, Cwik MJ: Pretreatment or resuscitation with a lipid infusion shifts the dose-response to bupivacaine-induced asystole in rats. *ANESTHESIOLOGY* 1998; 88:1071-5
5. Weinberg G, Ripper R, Feinstein DL, Hoffman W: Lipid emulsion infusion rescues dogs from bupivacaine-induced cardiac toxicity. *Reg Anesth Pain Med* 2003; 28:198-202
6. Corcoran W, Beck C, Gerancher J, Butterworth J, Groban L: Local anesthetic-induced cardiac toxicity: A survey of contemporary practice strategies among academic anesthesiology departments (abstract). *Anesth Analg* 2006; 102:S-316
7. Weinberg G, Paisanthasan C, Feinstein D, Hoffman W: The effect of bupivacaine on myocardial tissue hypoxia and acidosis during ventricular fibrillation. *Anesth Analg* 2004; 98:790-5
8. Weinberg G, Murphy P, Edelman L, Hoffman W, Strichartz G, Feinstein D: Lipid infusion accelerates removal of bupivacaine and recovery from bupivacaine toxicity in the isolated rat heart. *Reg Anesth Pain Med* 2006; in press
9. Stehr S, Ziegeler J, Pexa A, Oertel R, Deussen A, Koch T, Hubler M: Lipid effects on myocardial function in L-bupivacaine induced toxicity in the isolated rat heart (abstract). *Reg Anesth Pain Med* 2005; 30:5