To the Editor.—We read with interest the study by Gautier et al.¹ that compared bolus administration of epidural bupivacaine 0.125% wt/vol and epidural ropivacaine 0.125% wt/vol in combination with 7.5 μg sufentanil for labor analgesia in 90 patients. Given the relatively high concentrations used, it is not surprising that both groups experienced effective analgesia. After the third epidural injection, the ropivacaine group demonstrated significantly less motor block. Recognizing that the two initial study groups may not have received equipotent concentrations, an additional 40 patients were randomized to receive bupivacaine 0.125% wt/vol with 7.5 μg sufentanil or ropivacaine 0.100% wt/vol with 7.5 μg sufentanil. The authors are to be commended in attempting to adjust their methodology to consider possible potency differences. We assume the aim was to determine if ropivacaine 0.125% wt/vol appeared clinically similar to 0.100% wt/vol bupivacaine in combination with sufentanil.

Bupivacaine concentrations of 0.125% wt/vol are at the top of the analgesic concentration-response curve,² and the addition of sufentanil results in significant reductions in bupivacaine requirements.³ All four of the tested combinations are likely in the flat upper portion of the curve, where differences in analgesic potency are obscured. The authors concluded that patients in the 0.100% wt/vol bupivacaine–sufentanil group experienced inferior analgesia. This conclusion seems to be based on patient feedback elicited during the postpartum period and the greater number of epidural injections, because there was no significant difference in prospective visual analog pain scores. It should also be noted that this group was studied closer to the time of delivery,³ and because bupivacaine requirements increase with progression of labor,⁴ this may account for the more frequent bolus doses.

In addition, the authors state that “most clinical studies suggest that ropivacaine is approximately 20% less potent than bupivacaine” and reference our work.⁵ In fact, our study demonstrated that ropivacaine was approximately 40% less potent than bupivacaine with a potency ratio of 0.6. Therefore, in the study by Gautier et al., the 0.125% wt/vol ropivacaine group would be approximately equipotent with a 0.075% wt/vol additional bupivacaine group. We fully agree with the authors that equipotent concentrations must be determined before making comparisons of local anesthetic side effects.

Linda S. Polley, M.D.
Assistant Professor
Department of Anesthesiology
Women’s Hospital
University of Michigan Health System
Ann Arbor, Michigan
lpolley@umich.edu
Malachy O. Columb, F.R.C.A.
Consultant in Anaesthesia and Intensive Medicine
South Manchester University Hospital
Withington, United Kingdom

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In Reply.—We appreciate the interest in our work and the comments by Drs. Polley and Columb. The exact determination of the relative potency of ropivacaine when compared with bupivacaine is an important issue. It directly questions the advantages associated with the use of this new local anesthetic, i.e., reduced incidence of motor blockade and lower central nervous system or cardiac toxicity.

We agree with the comments of Drs. Polley and Columb. Our study design was clearly in favor of ropivacaine: relatively important doses were used, sufentanil was added, and early stage of labor was considered. Nevertheless, even in these conditions, our conclusion was clear: ropivacaine 0.125% with sufentanil affords reliable analgesia with minimal motor blockade when compared with bupivacaine. However, this benefit is not of major clinical importance because it becomes obvious only after the third epidural injection and has no influence on the rate of instrumental delivery.

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With regard to the relative potency of ropivacaine when compared with bupivacaine, when the study was designed (end of 1997/beginning of 1998), there were not many studies that clearly quantified the lower potency of ropivacaine. This is obvious when considering studies performed in obstetrics1,2 or using intrathecal ropivacaine in human volunteers.3 We also studied intrathecal ropivacaine in a clinical situation4 and were able to confirm the 50% difference in potency reported by McDonnald et al.3

Philippe Gautier, M.D.
Staff Anesthesiologist
Department of Anesthesiology
Clinique Ste Anne-St Remi
Brussels, Belgium

Marc De Kock, M.D., Ph.D.
Staff Anesthesiologist
Department of Anesthesiology
Cliniques Universitaires St Luc
Brussels, Belgium

DeKock@anes.ucl.ac.be.

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To the Editor:—The animal study on postischemic spinal cord cooling by Kakinohana et al.1 touches on important therapeutic implications concerning ischemic injury to the spinal cord. Their study indicated that a very narrow window of 0–10 min exists after aortic balloon occlusion for hypothermia to be efficacious. A clinical study by Davison et al.2 on epidural cooling during thoracoabdominal aneurysm repair indicated the feasibility of using this method during the surgical procedure.

It is important to point out that the therapeutic window for experimental traumatic spinal cord injury, on the other hand, is considerably longer. In 1968, Albin et al.3 demonstrated functional recovery after spinal cord injury (weight-drop technique) in the subhuman primate when localized cooling (to 10.0°C in spinal cord) was instituted 4 h after trauma and conducted for 3 h. All of the controls without hypothermia developed permanent hind leg paraplegia. Kakinohana et al. noted the positive correlation between the paravertebral muscle temperature and that of the lumbar intrathecal space at the L3 level. Because the anatomic target of both ischemia and hypothermia is the spinal cord itself, do the authors have any data indicating the order of magnitude of the decrease in spinal cord temperature per se? The decrease in spinal cord temperature seems to be inferred because the authors measured temperature in the lumbar intrathecal space, reflecting cerebrospinal fluid temperature and not that of intrinsic spinal cord. Might it be possible that the therapeutic window could be enlarged if intrinsic spinal cord temperature were reduced to lower levels? Our studies in various species4,5 indicate that localized spinal cord perfusion cooling has no deleterious effect functionally or histologically even when cord temperature was decreased to < 10°C for at least 3 h.

Maurice S. Albin, M.D., M.Sc.(Anes.)
Professor of Anesthesiology and Neurosurgery
Department of Anesthesiology
University of Texas Health Science Center at San Antonio
San Antonio, Texas 78284
albin@uthscsa.dcci.com

Robert J. White, M.D., Ph.D.
Professor of Neurosurgery
Case Western Reserve University School of Medicine
Cleveland, Ohio 44120

References
CORRESPONDENCE

In Reply—We read carefully the comments of Drs. Albin and White on our recent article. As indicated by these authors, induction of spinal cord hypothermia (10°C) after traumatic spinal injury is still effective in providing protection if initiated 4 h after injury. In contrast, in our study, we observed a significant protection only if spinal hypothermia (27°C) was initiated immediately (5 min) after ischemia. Although the differences between the therapeutic efficacy of spinal hypothermia after spinal trauma and spinal ischemia are not clear, there are several points that need to be addressed.

After injurious intervals of spinal ischemia, the development of irreversible neuronal degeneration shows very rapid onset. Using Nauta silver impregnation technique in rat, rabbit, or dog spinal ischemia models, we have found that irreversible neuronal degeneration is clearly developed as soon as 60–120 min after reflow following injurious intervals of ischemia. These data indicate that the process of irreversible neuronal degeneration is likely initiated during the early period (0–30 min) of reflow. Importantly, based on the data from our study, this process shows high temperature sensitivity. The lack of hypothermic protection after a longer period of normothermic reflow thus likely reflects the fact that the process of irreversible degeneration was already initiated or completed.

In our study, intrathecal temperature was measured during and after a period of subcutaneous cooling. It is possible that the magnitude of the decrease in spinal parenchymal temperature does not completely reflect measured intrathecal temperature. However, in our previous methodologic study describing this cooling technique, we showed that there is a 2–3°C temperature gradient if measured on dorsal versus ventral spinal cord surface. Based on these data, we believe that spinal temperature is in the range of temperatures measured between dorsal and ventral spinal cord surface. Accordingly, measurement of spinal somatosensory evoked potentials showed a temperature-dependent increase in the N3 (postsynaptic) component during the period of cooling and a return to baseline after rewarming.

Based on our previous data describing a very rapid onset of irreversible neuronal degeneration after spinal ischemia, the possibility that the therapeutic window could be enlarged if intrinsic spinal cord temperature were reduced to lower levels is uncertain. However, it is possible that the fraction of neurons undergo prolonged or delayed degeneration and that rescue of these neurons will eventually improve outcome. In this case, it is possible that deeper hypothermia (10–20°C) will be more effective in protecting this neuronal pool than temperatures (27°C) tested in our study. Accordingly, it would be important, using localized epidural or intrathecal cooling techniques, to determine the efficacy of such hypothermic treatment.

Martin Marsala, M.D.
Department of Anesthesiology
University of California, San Diego
La Jolla, California 92030-0818
mmarsala@ucsd.edu

References


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Relative Potencies of Ropivacaine and Bupivacaine

To the Editor:—The April 1999 issue of Anesthesiology contained two articles that relate to the relative potencies of bupivacaine and ropivacaine in the respective settings of epidural analgesia in obstetrics and spinal anesthesia in volunteers. Comparing potencies of local anesthetic agents in the intact subject is difficult, and the findings in the article on obstetric epidural analgesia are qualified by an appropriate accompanying Editorial View.

A comparison of block (defined as tolerance to transcutaneous electrical stimulation of sensation [TES]), and duration of sensory and motor block. A comparison of effects of the two drugs, including height of block, intensity of block (defined as tolerance to transcutaneous electrical stimulation [TES]), and duration of sensory and motor block. A comparison of potencies can be found in the data listed in our table 3, which gives the characteristics of the two spinal anesthetic drugs. For example, 4 mg bupivacaine produces tolerance to TES at T10 and T12 dermatomes, but 4 mg ropivacaine does not (duration not a consideration). Although the strictest definition of potency may be found by testing an isolated nerve in a controlled laboratory setting, to the clinician, who deals with “intact subjects,” duration is an important measure of potency.

Surely the data in their figure 1 shows that it is. Ropivacaine 12 mg produced the same mean height of block as bupivacaine 12 mg, but its time to complete regression was little more than half. I have concerns about several aspects, but this result is very interesting.

Professor and Head
Department of Anaesthesia
Ninewells Hospital and Medical School
University of Dundee
Dundee DD1 9SY, United Kingdom
j.a.w.wildsmith@dundee.ac.uk

References


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involves only one anesthetic.\textsuperscript{2,3} We chose to use volunteers for two reasons: (1) asking patients to receive an anesthetic in doses not yet proven to produce surgical anesthesia is unethical; and (2) our crossover method of control is unsuitable for patients who require only one anesthetic.

The association of back pain and ropivacaine in our study was not statistically significant (as stated, \(P = 0.098\)), but the trend is concerning. Our conclusion was that “the possibility of side effects . . . warrants further investigation.” We apologize if the study design was not clearly stated, but the order of drug injection was randomized as well. In our interpretation of the results, we commented that there was no association whether ropivacaine was given first or second and, in addition, that all three doses were represented.

Finally, Dr. Wildsmith suggested that our figure 1, which depicted block height to pin prick, was encouraging evidence that ropivacaine may be a suitable spinal drug for outpatients. However, sensory block to pinprick does not equal surgical anesthesia. Our results regarding TES, which was designed to simulate surgical incision, were far from encouraging.\textsuperscript{4} Again, referring to our table 3, the 12 mg dose of ropivacaine produced tolerance to TES at the ankle for 48 ± 40 min \textit{versus} 153 ± 54 min for 12 mg bupivacaine or 46 ± 30 min for 8 mg bupivacaine. Ropivacaine 8 mg did not produce any tolerance to TES. This data coupled with its unfavorable recovery profile brought us to the conclusion that ropivacaine is unlikely to offer an advantage over presently available drugs such as bupivacaine. Other researchers have also come to a similar conclusion for patients undergoing outpatient surgery.\textsuperscript{5}

\textbf{Susan B. McDonald, M.D.}

Staff Anesthesiologist

\textbf{Dan J. Kopacz, M.D.}

Staff Anesthesiologist

\textbf{References}


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\textbf{Anemia and Arterial Partial Pressure of Oxygen}

\textit{To the Editor}—We have considerable interest in the study by Nielsen \textit{et al}.\textsuperscript{1} Their article provides additional insights into the incredible physiologic tolerance to severe isovolemic anemia. We were particularly struck by the arterial blood gas data, which revealed an increase in arterial partial pressure of oxygen (\(P_{\text{aO}}\)) with hemodilution and an apparent strong inverse correlation between \(P_{\text{aO}}\) and hematocrit. Even the relatively small increase in \(P_{\text{aO}}\) in the control group was associated with a decrease in hematocrit from 35% to 28%. However, despite rather large increases in \(P_{\text{aO}}\) with hemodilution (mean increases of 50, 98, and 72 mmHg in the three groups), the authors suggest that the changes were not statistically significant. We realize that this was not a primary outcome measure of focus of the study, but an improvement in \(P_{\text{aO}}\) with hemodilution has been noted by us\textsuperscript{2} and other investigators. Perhaps a more robust test of significance involving this type of protocol would be repeated-measures analysis of variance between treatment (hemodilution) and control animals. The use of one-way analysis of variance to test for differences at individual points will result in considerable loss of power to detect differences between groups over time because of the between-subject variability at each time point.

An improvement in arterial partial pressure of carbon dioxide with anemia may occur because of changes in pulmonary blood flow distribution and improved ventilation-perfusion distribution.\textsuperscript{2} Given the general interest in this subject, we would welcome any further information the authors could provide.

\textbf{Steven Deem, M.D.}

Assistant Professor

Departments of Anesthesiology and Medicine

Harborview Medical Center

sdeem@u.washington.edu

\textbf{Erik R. Swenson, M.D.}

Associate Professor

Department of Medicine

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In Reply:—We appreciate the interest expressed by Drs. Deem and Swenson with regard to our recent article.1 Our experimental design involved four experimental groups, three of which underwent four serial hemodilutions with concurrent measurement of several physiologic and biochemical parameters. Because one of the groups was sham-operated, it was acceptable to have used one-way analysis of variance with a rigorous post hoc test to determine if there were any significant differences between the groups.

Of further interest, Drs. Deem and Swenson suggest that hemodilution may increase arterial partial pressure of oxygen (PaO2) by improving ventilation-perfusion distribution. They commented that there was "an apparent strong inverse correlation between PaO2 and hematocrit." When the hematocrit and PaO2 data from all four groups of our study1 are analyzed by linear regression, an inverse relationship is observed (R2 = 0.14; P < 0.0001). Although highly significant, the relationship between PaO2 and hematocrit in this model is weak, with only 14% of the change in PaO2 associated with the change in hematocrit. To test the hypothesis that hemodilution per se is associated with an increase in PaO2, we analyzed the combined PaO2 data from all three hemodilution groups (n = 25) of our study, using one-way repeated-measures analysis of variance with a post hoc Tukey test. The PaO2 values (mean ± SD) are as follows: 445 ± 54 mmHg before hemodilution, 472 ± 47 mmHg after the first hemodilution, 475 ± 51 mmHg after the second hemodilution, 484 ± 38 mmHg after the third hemodilution, and 519 ± 37 mmHg after the fourth hemodilution. The PaO2 values observed after each hemodilution were significantly (P < 0.05) greater than the PaO2 values observed before hemodilution. Furthermore, the PaO2 values observed after the fourth hemodilution (hematocrit = 5%) were significantly (P < 0.01) greater than those observed after all preceding hemodilutions. Finally, a comparison of the sham-operated group (n = 8) with the hemodilution group with repeated-measures analysis of variance did not demonstrate a significant difference between the groups (P = 0.3). The relative similarity of mean values between the sham-operated and hemodilution groups (at best 8% different) after most of the hemodilutions combined with the observed variability (SD of 5–10% of mean value) are the most likely reasons for the inability to demonstrate statistical significance. Although our study was not specifically designed to test the hypothesis that hemodilution is associated with an increase in PaO2, our data are, in general, similar to that of Deem et al.2

Vance G. Nielsen, M.D.
Associate Professor
Department of Anesthesiology
The University of Alabama at Birmingham
Birmingham, Alabama 35249-6810
vance.nielsen@ccc.uab.edu

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Ropivacaine: Drug of Choice? Or Not?

To the Editor—I read with interest the case report by Autore et al.1 that described anesthetic management of three parturients with hypertrophic cardiomyopathy receiving epidural anesthesia for cesarean delivery. After describing two cases wherein epidural anesthesia with lidocaine was provided and a third in which ropivacaine was used, the authors concluded that ropivacaine should be the “drug of choice” in this setting because of less cardiotoxicity versus bupivacaine and slower onset of block versus lidocaine.

This conclusion does not follow from the cases as presented nor from the available evidence currently published. Ropivacaine may be less cardiotoxic, but potency issues need to be settled before this can be concluded with certainty.2 It is not at all clear that patients with hypertrophic cardiomyopathy are more sensitive to local anesthetic–induced cardiotoxicity than patients with normal hearts. The toxicity of both ropivacaine and bupivacaine is not enhanced by pregnancy.3 Local anesthetic–induced toxicity would only occur with an unintentional large intravascular dose of drug, because proper epidural administration of either drug in doses used for cesarean delivery would not result in toxic blood levels.4 If one has a patient with hypertrophic cardiomyopathy, presumably one will be dosing the epidural slowly and carefully; therefore, large, rapid, intravascular drug administration should not occur. Most importantly, without regard to how the issue of cardiotoxicity is settled, certainly all would agree that lidocaine is less toxic than either, and thus this drug remains, in my opinion, the drug of choice for epidural use for any cesarean delivery unless highly unusual circumstances preclude its use.

The claim that ropivacaine has a slower onset than lidocaine is specious reasoning; it all depends on how you administer the drug. Previous data indicate that epidural ropivacaine, administered rapidly and in large doses,4 can produce very fast onset of surgical anesthesia. Likewise, lidocaine, administered slowly and carefully, as in the cases reported by Autore et al.,1 is perfectly compatible with stable intraoperative hemodynamics even in patients with severe cardiac disease. The authors are to be congratulated for fine anesthetic management of three patients with complex cardiac disease for cesarean delivery. Certainly, ropivacaine would be an acceptable drug to use in these patients; however, to claim that it is the “drug of choice” in this setting is just not supported by the authors own cases nor by the current evidence available in the literature.

William Camann, M.D.
Associate Professor
Department of Anesthesiology
Brigham and Women’s Hospital
Harvard Medical School
Boston, Massachusetts 02115
wcamann@bics.bwh.harvard.edu

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additional patients with hypertrophic cardiomyopathy with ropivacaine epidural anesthesia (unpublished data). Nevertheless, in light of comments by Polley et al.,5 regarding the therapeutic indices of bupivacaine and ropivacaine, it would not be unreasonable to reconsider using bupivacaine or, in the near future, l-bupivacaine in patients with hypertrophic cardiomyopathy.

Camillo Autore, M.D.
autore@axrma.uniromal.it
Fabrizio Apponi, M.D.
Stefano Brauneis, M.D.
Giovanni Pinto, M.D.
Department of Cardiovascular and Respiratory Sciences
Policlinico Umberto I
Rome, Italy

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In Reply—We appreciate the opportunity to comment on the case report by Autore et al.1 and the correspondence by Dr. Camann. We also have difficulty with the conclusion that ropivacaine should be the “drug of choice” for cesarean delivery in the setting of hypertrophic cardiomyopathy. The presumed lesser cardiotoxicity of ropivacaine is based on the assumption of equipotency with bupivacaine. Our recent minimum local analgesic concentration (MLAC) study of the relative analgesic potencies of ropivacaine and bupivacaine for epidural analgesia in labor found that ropivacaine was 40% less potent than bupivacaine.2 These results are in agreement with both a recent European study3 and a study comparing intrathecal administration of the two local anesthetics.4 Clearly, the cardiotoxic potential of local anesthetics can only be properly evaluated when comparing equipotent doses. The therapeutic index may favor bupivacaine. That said, we would like to emphasize that our studies determined analgesic potencies, and the results may not be generalizable to anesthetic potencies.

We agree with Dr. Camann that careful fractionated dosing of lidocaine allows for slow block onset and that it is the least toxic of the three local anesthetics. In addition, the shorter duration of lidocaine allows for a quicker return to preblock hemodynamics, which may be advantageous in hypertrophic cardiomyopathy.

Linda S. Polley, M.D.
Assistant Professor
Division of Obstetric Anesthesiology
University of Michigan Health System
Ann Arbor, Michigan
lpolley@umich.edu
Malachy O. Columb, F.R.C.A.
Consultant in Anaesthesia and Intensive Medicine
South Manchester University Hospital
Withington, United Kingdom

References

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Acidity and Particulate Characteristics of Aspirated Material May Affect the Severity of Pneumonitis

To the Editor:—The clinical spectrum of lung and systemic injury resulting from pulmonary aspiration of particulate material is broad. For instance, witnessed aspiration can have essentially no adverse clinical effects1 or can lead to acute lung injury/acute respiratory distress with multiple distant organ failures and even death.2 In addition to differing animal models (i.e., species-specific effects) or patient characteristics, several aspects of the aspirate per se may affect the severity of injury. As pointed out by O’Hare et al. in a recent article in Anesthesiology,3 these include the degree of acidity (pH), volume of the aspirate, the particle size, and concentration of the particles. However, another factor O’Hare et al. do not emphasize concerns the characteristics of the particulate aspirated. Thus, in some instances, it seems that with some particulate aspirates (e.g., human breast milk), there is no relation with pH and the severity of lung injury and systemic response.3 However, with other particulates, such as sucralfate,4 there can be a synergistic interaction between acidity and particulate-induced lung injury.4,5

Kenneth E. Shepherd, M.D.
Assistant Professor of Anaesthesia
Harvard Medical School
Assistant Anesthetist
Department of Anesthesia and Critical Care
Massachusetts General Hospital

References


(Accepted for publication August 24, 1999.)

Another Application of Dual-lung Capnography

To the Editor:—Dual-lung capnography allows carbon dioxide to be monitored from each lung individually or from both lungs simultaneously without interrupting two-lung ventilation. Dual-lung capnography has helped to detect major unilateral ventilation/perfusion mismatching during anesthesia.1 Here we describe a situation wherein information obtained via dual-lung capnography allowed the anesthesia team to render an informed opinion to the surgical team regarding the status of the flow in a major pulmonary artery after the surgeons expressed concern about accidentally stapling the vessel during a technically complex thoracic operation.

The case involved a 57-yr-old woman who had undergone a left upper lobectomy for adenocarcinoma 3 yr earlier and now presented with an anterior mediastinal mass (5 × 7 × 6 cm; adenocarcinoma). After induction of general anesthesia and placement of a left-sided double-lumen tube, we sampled end-tidal carbon dioxide partial pressure continuously using an adapter located between the double-lumen tube and the anesthesia circuit (standard approach). The surgical procedure was performed through a median sternotomy. Because the mass was adherent to the thymus and pericardium, the surgeons began their dissection from the right side of the thymus, excising the mass along with a portion of the thymus and a segment of pericardium. A wedge resection of the left lung was also required because the mass adhered tenaciously to portions of this lung. Despite our use of one-lung ventilation (right lung), the left pulmonary artery proved difficult to visualize, and the possibility arose that this vessel had been accidentally stapled (stapled) during the wedge resection.

Support was provided solely from institutional and/or departmental sources.
We hypothesized that if such an occlusion had indeed occurred, it would produce a major unilateral perfusion deficit characterized by a marked decrease in carbon dioxide delivery to the left lung and a correspondingly low end-tidal carbon dioxide partial pressure from the left lung that could be detected using dual-lung capnography. We therefore commenced double-lung ventilation using the set-up described by us previously to measure and record carbon dioxide waveforms from each lung during two-lung ventilation.1 The end-tidal carbon dioxide partial pressure values of the individual lungs were found to be similar (36 mmHg on the left vs. 38 mmHg on the right), and the waveforms from the two lumens of the double-lumen tube were similar. We interpreted these findings as an indication that there was no major blood flow limitation in left main pulmonary artery. Confirming our hypothesis was a subsequent intraoperative Doppler study that showed that the left pulmonary artery was patent with normal flow velocity.

Roger Russell, M.D.
Instructor

Kodali Bhavani Shankar, M.D.
Assistant Professor
bhavani@capnography.com

Phillip Mushlin, M.D., Ph.D.
Associate Professor
Department of Anesthesiology
Brigham and Women’s Hospital
Harvard Medical School
Boston, Massachusetts 02115

References
(Accepted for publication June 18, 1999.)

To the Editor:—Fournier’s gangrene is a necrotizing fasciitis of the genitalia and perineum that may extend to the neighboring soft tissues such as the anterior abdominal wall, buttocks, or lower extremities.1–3 Treatment of this syndrome requires adequate drainage and debridement as promptly as possible. We report a case of Fournier’s gangrene for which spinal anesthesia was considered to be contraindicated.

A 64-yr-old woman suffered from painful swelling in the perineal region. Her body temperature was 38°C for 1 week. She had a medical history of insulin for diabetes mellitus. On admission, the cellulitic activity extended to the anal and perineal regions and bilateral thigh walls with prominent subcutaneous crepitance. Her white blood cell count and blood sugar and C-reactive protein levels were 8,660/μl, 425 mg/dl, and 39.0 mg/dl, respectively. A surgeon requested spinal anesthesia for drainage because on physical examination, her back appeared normal. However, we found the existence of some gas in her back, including the paravertebral area on computed tomography, suggesting subcutaneous and paravertebral dissection of the infection (fig. 1). Therefore, we chose to use general anesthesia instead of spinal anesthesia.

In Fournier’s gangrene, emphysema in the subcutaneous and paravertebral area of the lower back have not been reported to date. Therefore, in most reports, either local or spinal anesthesia was selected for surgical debrideement. In our hospital, we have recently treated two patients with Fournier’s gangrene by surgical debridement. Local anesthesia was selected in one patient, and the other was administered spinal anesthesia; roentgenogram examination was not performed. These two operations were performed without any complication. Anesthesiologists generally know that spinal anesthesia should not be performed through infected tissues. However, sometimes infections are not clear on inspection or palpation. In this case, the extensive spread of the infection to the subcutaneous and paravertebral area was only visible with an imaging study. Therefore, computed tomography should be carefully checked to clarify the existence of gas in a patient’s back. Otherwise, general anesthesia should be selected in the drainage for Fournier’s gangrene to prevent intraspinal canal infections.

Fig. 1. Computed tomography at L3 level. Paravertebral gas formation (arrow) is present.

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A Case of Fournier’s Gangrene Contraindicating Spinal Anesthesia

Anesthesiology, V 92, No 1, Jan 2000
Was Chloroform Produced before 1831?

To the Editor.—In late 1831, Guthrie, Soubeiran, and Liebig independently reported their discovery of a compound obtained by distilling a mixture of concentrated ethyl alcohol and chloride of lime (CaCOCl₂).¹⁻³

In 1834, Dumas elicited the product’s chemical formula (CHCl₃) and named it chloroform.¹⁻³ Soubeiran and Liebig realized that they had discovered a new compound, but Guthrie thought he had found a simpler and cheaper method of producing chloric ether.² He learned of chloric ether and its potential medical use as a stimulant in the 1831 edition of Silliman’s Elements of Chemistry⁵ and was interested in the product’s commercial exploitation.⁴

Although the fall of 1831 is commonly accepted as the date of its discovery,¹⁻³ chloroform seems to have been produced at least 1 yr earlier, in mid-1830, by the German Moldenhawer.⁶⁻⁷ Moldenhawer, a pharmacist in Frankfurt/Oder, published in the early fall of 1830 a new process for removing the contaminant fusel oil from ethyl alcohol prepared from potatoes.⁷ In reviewing the various methods used to purify alcohol, Moldenhawer mentioned Zeise’s procedure⁸ of adding one-fourth loth (2.5 g) of chloride of lime to one quart (1 l) of ethyl alcohol, shaking the mixture frequently and vigorously for 20–24 h, and distilling out the alcohol (loth and quart were German measures of that period.) Moldenhawer judged Zeise’s method to be inadequate; he had found fusel oil in the distillate and none in the residue. He had also noticed that the vigorous shaking of the mixture released the strong odor of what he thought to be chloric ether (chloräther in German). Guthrie thus seems to have produced chloroform. Like Guthrie, he mistook the substance for chloric ether because of a similar smell.

In 1794, a group of Dutch chemists,⁹ mixing equal volumes of ethylene (C₂H₄) and chlorine (C₁₂), obtained an ethereal liquid initially called oil of the Dutch chemists (1,2-dichloroethane). In 1816, Robiquet and Colin¹⁰ analyzed the composition and properties of the Dutch liquid, named it hydrochloric ether, and suggested its use as a medicinal stimulant. Thomson, professor of chemistry at the University of Glasgow, described the compound in the 1820 edition of his System of Chemistry¹¹ and renamed it chloric ether. The name chloric ether was adopted by Silliman in the 1831 edition of his Elements of Chemistry.⁵ Chloräther (1,2-dichloroethane) was well known in the German chemical literature at the time of Moldenhawer’s publication.¹²⁻¹³ His article shows an extensive knowledge of the contemporary chemical literature. He also must have experimented with chloric ether because he thought that he had noticed its smell in the mixture of alcohol and chloride of lime he had tested. Had he investigated the compound whose odor he had detected, or at least reported his discovery more extensively, he may have been the first discoverer of chloroform. Zeise, who mixed alcohol and chloride of lime even before Moldenhawer, may also have fortuitously produced chloroform, but the details and the date of his experiments remain unknown.

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Ray J. Defalque, M.D.
Professor
Department of Anesthesiology
A. J. Wright, M.L.S.
Librarian
University of Alabama at Birmingham
Birmingham, Alabama 35249-6810
pat.thompson@ccc.uab.edu

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