Mean Arterial Pressure and Intracranial Pressure

To the Editor:—We read with great interest the article by Petersen et al., which demonstrated the expected clinical response to the three anesthetic agents studied. An increase in arterial pressure may result in an increase in the cerebral perfusion pressure and reduce reflex cerebral vasodilatation, potentially resulting in reduced intracranial pressure. We note that the mean arterial pressures in the propofol group were substantially higher than in the two inhalation groups, both before and after hyperventilation. The propofol group was shown to have both a lower measured intracranial pressure and subjective surgical estimation of brain swelling at craniotomy. The influence of cerebral vasodilatation, potentially resulting in reduced intracranial pressure, was associated with significant vascular trauma, because “blood could be steadily aspirated.” Those who perform continuous lumbar plexus blocks on a regular basis are certainly familiar with this complication. Although there is little doubt that the administration of enoxaparin thromboprophylactic therapy were developed because a large number of patients (>50 and, to date, probably around 80 patients) developed epidural hematoma when receiving therapeutic doses of low molecular weight heparin. The majority of those patients suffered serious neuro-

In Reply:—Thank you for your interest in our study on intracranial pressure and cerebral hemodynamic in patients with cerebral tumors. We performed an analysis of the relationship between mean arterial blood pressure and intracranial pressure (ICP) both before and during hyperventilation but found no significant correlation. The correlation coefficients, however, was negative in the propofol/fentanyl group but positive in the isoflurane/fentanyl and sevoflurane/fentanyl groups. These differences might be caused by difference in autoregulatory capacity between propofol/fentanyl and the other two groups, suggesting that cerebral autoregulation was better preserved in the propofol/fentanyl group compared with isoflurane/fentanyl and sevoflurane/fentanyl anesthetized patients, but this does not explain that ICP was significantly lower during propofol/fentanyl anesthesia. On the contrary, one would expect a low ICP in the isoflurane and sevoflurane groups, because the mean arterial blood pressures were significant lower. We also analyzed the correlation coefficients for the relationship between cerebral perfusion pressure and ICP. In all groups, the correlation coefficients were negative but insignificant. The negative correlations were not surprising, considering that cerebral perfusion pressure was calculated as the difference between mean arterial blood pressure and ICP. Because cerebral perfusion pressure and ICP are not independent variables, we did not include these data in the results.

Georg Emil Cold, M.D. Aarhus University Hospital, Århus, Denmark. gcold@akh.aaa.dk.

What Has Happened to Evidence-based Medicine?

To the Editor:—We read with interest the two cases of retroperitoneal hematoma following lumbar plexus block reported by Weller et al. These cases and the authors’ conclusions prompt a number of comments. In the first case, it is clear that the introduction of the catheter was associated with significant vascular trauma, because ‘blood could be steadily aspirated.’ Those who perform continuous lumbar plexus blocks on a regular basis are certainly familiar with this complication. Although there is little doubt that the administration of enoxaparin contributed to the development of the observed retroperitoneal hematoma, it is surprising that the authors did not consider the initial vascular trauma as a major risk factor, and furthermore, that they did not at that time abort the placement of the perineural catheter. As far as the second case is concerned, the authors describe a patient who had been grossly overdosed on heparin therapy (activated partial thromboplastin time > 100) and had developed a “moderate-sized retroperitoneal hematoma.” Although the authors acknowledge that spontaneous retroperitoneal bleeds are possible in anticoagulated patients, it is the introduction of a 21-gauge needle 3 days earlier that is primarily incriminated in the etiology of the hematoma.

What is particularly unfortunate is that, on the basis of these two cases, the authors go on to recommend that we apply the same guidelines for continuous lumbar plexus blocks as those proposed for neuraxial blocks, thus insinuating that performing lumbar plexus blocks in patients scheduled to be anticoagulated might be considered malpractice. Although no quantitative information is available, our group performs many of these blocks; to date, none of us has any recollection of the development of a retroperitoneal hematoma in any of our patients. We acknowledge that a perineural or retroperitoneal hematoma is a potential and rare complication, but it is also important to remember that the guidelines for neuraxial blocks in the presence of thromboprophylactic therapy were developed because a large number of patients (>50 and, to date, probably around 80 patients) developed epidural hematoma while receiving therapeutic doses of low molecular weight heparin. The majority of those patients suffered serious neuro-
logic injury. Of note, neither of the two patients reported by Weller et al. suffered neurologic injury, despite one being massively overdosed with intravenous heparin.

To conclude, there is no doubt that caution should be exercised when placing a perineural catheter, especially a lumbar plexus catheter. In any patient for whom one has evidence of significant bleeding at the time of insertion of a perineural lumbar plexus catheter, we recommend removal of the catheter at the time of insertion and the use of an alternative approach. This practice seems more measured and reasonable than offering the radical suggestion that continuous lumbar plexus block should be banned in patients receiving anticoagulant postoperatively.

Anesthesiology 2003; 99:1029

In Reply:—We appreciate the opportunity to respond to Dr. Chelly and his expert coauthors to correct an apparent misinterpretation of our discussion of retroperitoneal hematoma following posterior lumbar plexus blockade (LPB). Most important, we would like the readers of ANESTHESIOLOGY to understand that our intent was to notify clinicians of the serious complication we observed, rather than to promulgate guidelines for clinical practice. Because the use of perineural catheters and lower extremity nerve block has increased substantially in recent years, we believe it is critical that complications of newer techniques are reported so that clinicians use appropriate caution as they introduce techniques novel to their practice. Because there are no data on the frequency of this complication, we believe no individual should suggest practice guidelines for others. To suggest we “insinuated malpractice” is ludicrous. We believe we were quite cautious in our discussion of retroperitoneal hematoma following LPB. We feel compelled to respond to Chelly et al.’s characterization of retroperitoneal hematoma as “potential and rare.” Certainly, retroperitoneal hematoma has been reported less frequently following LPB than spinal hematoma has been reported following epidural blockade, but none would debate that the number of continuous epidural anesthetics nationwide is substantially higher than the number of continuous LPB. Retroperitoneal hematoma following LPB remains a complication with unknown incidence. Although we were fortunate that neither of our patients suffered neurologic injury, we would like to reemphasize that retroperitoneal hematoma should be considered a significant complication—one that can result in major morbidity and death. In addition to retroperitoneal hematoma following LPB, Auroy et al. reported a rate of other serious complications following LPB that exceeds that of other peripheral nerve blocks. Finally, Chelly et al. may have collectively performed a large number of continuous LPBs, but they provide no evidence to support their “more measured” recommendation that the lumbar plexus approach should be aborted if “significant bleeding” occurs during placement. How is “significant bleeding” defined, and how is this recommendation any more measured than our current management? We stand by our conclusion that retroperitoneal hematoma following LPB is a serious complication whose incidence is unknown.

Robert Weller, M.D.,* J. C. Gerancher, M.D., James C. Crews, M.D., Kenneeth L. Wade, M.D. Wake Forest University School of Medicine, Winston-Salem, North Carolina. rweller@wfubmc.edu

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(Received for publication May 21, 2003.)

Anesthesiology, V 99, No 4, Oct 2005

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To the Editor—I read with interest the case report by Breitfeld et al., in which the use of clonidine to reduce symptoms of opioid withdrawal is mentioned. It should be pointed out that clonidine is a μ2-adrenoceptor agonist, not an antagonist as reported in this article.

The effectiveness of this technique for detoxification from opioid addiction remains controversial. Within the actively using opioid-addicted individual, this technique has been used to “reset” the opioid receptors in persons who have lost their sensitivity to illicit opioids, thus allowing them to use again. This results in a dramatic loss in opioid tolerance, which, in the context of illicit drug use can lead to fatal overdoses. Currently, detoxification of individuals addicted to opioids by this method is experimental and without a clearly favorable cost–benefit ratio.

Christa Breitfeld M.D., Matthias Elkermann M.D., Peter Kienbaum M.D., and Jürgen Peters, M.D. Klinik für Anästhesiologie und Intensivmedizin, Universitätsklinikum Essen, Essen, Germany. christa.breitfeld@uni-essen.de

References

In Reply—We appreciate Dr. Gourlay’s interest in our work. Of course, we are aware that clonidine, while diminishing sympathetic neural outflow both in volunteers and in opioid addicts, acts as an agonist on central α-receptors. We apologize for the typographical error that appears on page 571 of the original article.

We agree that antagonist supported detoxification from opioids during general anesthesia can restore opioid sensitivity. Accordingly, opioid receptor blockade, e.g., by oral naltrexone, should be maintained in formerly addicted patients as long as possible to prevent both relapses as well as potentially fatal effects of further opioid intake. However, our patient experienced chronic pain with tolerance to the analgesic effects of administered opioids. Thus, antagonist supported detoxification was intended to restore opioid sensitivity, allowing continuation of analgesic therapy by administration of much lower opioid dosages while again achieving sufficient analgesia.

With respect to cost–benefit ratios, we expect by this procedure high rates of successful detoxification with markedly shortened hospitalization. Accordingly, this procedure seems to be economically attractive. However, a decrease in the number of relapses may not be expected, although little data are currently available.

Douglas Gourlay, M.D., F.R.C.P.C. University of Toronto, Wasser Pain Management Centre, Mount Sinai Hospital, Toronto, Ontario. doug.gourlay@camh.net

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(Received for publication May 22, 2003.)
To the Editor—Recently, some propofol was spilled on the floor (fig. 1) and—Halloween! I assure you that this white solution is propofol and that no actions were taken by staff to make it look like this. It just happened.

Alexander Avidan, M.D. Hebrew University Hadassah Medical School, Hadassah University Hospital, Jerusalem, Israel. avidan3@netvision.net.il

(Accepted for publication July 2, 2003.)

Support was provided solely from institutional and/or departmental sources.

Fig. 1. A propofol poltergeist?
To the Editor—Our transesophageal echo probes started to leave tenacious gray-green stains on the mouths of our cardiac surgery patients. The problem began when we were required to change our probe-disinfecting procedures. We had been using glutaraldehyde, which did not stain the probes (Philips Omniplane models I and II, Philips Medical Systems, Andover, MA). Unfortunately, glutaraldehyde is volatile, and its fumes were polluting the air of our probe-cleaning workroom. Therefore, we adopted less-volatile ortho-phthalaldehyde (OPA) as a disinfecting agent. Ugly OPA-derived oral stains were forthcoming and were not reliably prevented by copious rinsing of the probes with water (four times, 2 l). Staining is prominent when, as is common in cardiac anesthesia, an OPA-treated probe (but not a glutaraldehyde-treated probe) remains in prolonged contact with a patient on whom an endotracheal tube is secured with cloth adhesive tape. The stain first appears on the tape (both the Kendall Curity, Mansfield, MA, and 3M Health Care, St. Paul, MN, products) and can be transferred to the lip through routine manipulation of the probe. The lip stain is initially difficult to scrub away, but it does fade in a few hours with no apparent sequelae.

OPA is well-known to form deeply colored dyes with other molecules. Prolonged exposure to saliva and tape elicit visibly detectable dye from minute traces of OPA residues remaining on copiously water-rinsed echo probes. Although water rinsing does not reliably remove chromogenic residues from the probes, no stains are elicited from OPA-treated probes that are gently wiped clean with 3% hydrogen peroxide solution after the OPA protocol. Guidelines from one echo probe manufacturer mention that staining may occur if the standard OPA protocol is not performed "meticulously." Rather than breaching protocol, we have encountered especially favorable chromogenic conditions that unmask otherwise invisible trace residues.

It is theoretically troubling to discover that traces of OPA residue can cling to echo probes and then be transferred to patients. We have not established the identity of the staining material, nor have we determined what happens when it is inactivated by peroxide. The peroxide may remove the residues or it may merely render them nonchromogenic. We note that, like OPA, peroxide is federally approved for disinfecting endoscopy equipment. However, the common 3% solution is too dilute to be relied on for high-level disinfection.

Scott C. Streckenbach, M.D. Theodore A. Alston, M.D., Ph.D.*
*Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts. talston@partners.org

Reference


(Accepted for publication May 28, 2003.)

Interference Masquerading as Atrial Extrasystole

To the Editor.—We would like to report a source of potential electrocardiograph interference that may occur under anesthesia for laser treatment of capillary vascular malformation in children. During pulsed dye laser therapy (Scleroplus SPT-1 day; Candela, Wayland, MA) to a capillary vascular malformation that extended onto the chest, the electrocardiograph trace shown in Figure 1 was recorded. The initial diagnosis was atrial extrasystole, but there was no disturbance in the electrocardiograph trace shown in Figure 1 was recorded. The initial diagnosis was atrial extrasystole, but there was no disturbance in the electrocardiograph trace shown in Figure 1. Prolonged exposure to saliva and tape elicit visibly detectable dye from minute traces of OPA residues remaining on copiously water-rinsed echo probes. Although water rinsing does not reliably remove chromogenic residues from the probes, no stains are elicited from OPA-treated probes that are gently wiped clean with 3% hydrogen peroxide solution after the OPA protocol.

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Left Recurrent Laryngeal Nerve Paralysis after Interpleural Analgesia

To the Editor:—In patients with multiple rib fractures, interpleural analgesia has proved efficacious for improving the airway hygiene and reducing the need for ventilator support. Despite the ease and safety with the procedure, complications such as pneumothorax, pleural effusion, Horner syndrome, phrenic nerve paralysis, and unilateral bronchospasm are occasionally reported. We report a case with intermittent hoarseness due to recurrent laryngeal nerve block following each injection.

A 39-yr-old man sustained a left lung contusion and fracture of left ribs two through six after a motorcycle accident. Examination showed patient tachypneic with respiratory rate of 30/min. The patient complained of severe pain on respiration, changing position, and breath holding. No paradoxical chest wall motion was observed. Radiologic findings showed, in addition to the fractures, mild parenchymal contusion and herniation of the left lung (fig. 1A).

Despite the use of intramuscular morphine and external bandaging, the pain was so severe that the patient became dyspneic and hypoxicemic (oxygen saturation measured by pulse oximetry: 82–85%). An interpleural block was performed. The patient was placed horizontally with the left side up. The point for injection was 5 cm medial to the posterior midaxillary line, i.e., 6 cm from the midline. The skin was infiltrated with lidocaine around the aortic arch near the ligamentum arteriosum. Then, it runs in the groove between the trachea and esophagus and proceeds superiorly to innervate the intrinsic muscles of the larynx. In close contact with various anatomic structures at the T4 level, the nerve is extremely vulnerable to pathologic conditions associated with these organs, e.g., dissecting aneurysm of the ascending aorta, and Pancoast tumor of the left lung. On the other hand, the right recurrent laryngeal nerve courses around the right subclavian artery that never enters the thoracic cavity.

In our case, contrast medium was accumulated near the sixth thoracic vertebral body and along the aortic arch at T4 level (fig. 1B). Because the analgesic effect was satisfactory, we withdrew the catheter for 5 cm under the fluoroscopy to avoid further blockade of the left recurrent laryngeal nerve. Bupivacaine was resumed on demand. There was no hoarseness after the subsequent injections of bupivacaine. Analgesia remained satisfactory for the following 3 days. The patient was discharged 5 days later.

Left recurrent laryngeal nerve arises from vagus nerve and courses around the aortic arch near the ligamentum arteriosum. Then, it runs in the groove between the trachea and esophagus and proceeds superiorly to innervate the intrinsic muscles of the larynx. In close contact with various anatomic structures at the T4 level, the nerve is extremely vulnerable to pathologic conditions associated with these organs, e.g., dissecting aneurysm of the ascending aorta, and Pancoast tumor of the left lung. On the other hand, the right recurrent laryngeal nerve courses around the right subclavian artery that never enters the thoracic cavity.

In our case, contrast medium was accumulated near the sixth thoracic vertebral body and along the aortic arch in the standing position. Because contrast medium injected via the interpleural catheter has been shown to diffusely distribute along the mediastinum, local accumulation of dye should not be visualized when 5 ml contrast medium is injected into the interpleural space. Accordingly, our catheter could be located in the subpleural space of the upper mediastinum and resulted in block of the adjacent left recurrent laryngeal nerve. It is possible that repeated injection and body movement may in part lead to migration of the catheter into the upper mediastinum.

Misplacement of the catheter either into the extrapleural space or into lung tissue has been previously reported. Identifying the pleural space by the ‘loss of resistance’ technique is associated with catheter displacement more frequently than by the traditional ‘detection of negative pressure’ technique. In our case, the swollen lateral chest wall and fractured ribs were extremely tender to touch. A more medial approach, i.e., 6 cm from the midline, was thus used to avoid inserting needle directly over the hematoma and the underlying fracture ribs. Therefore, the syringe had to be attached horizontally, and its entry into the interpleural space was identified by loss of resistance.

In summary, we demonstrated an intermittent blockade to the left recurrent laryngeal nerve following the inadvertent injection of 20 ml 0.5% bupivacaine into the left upper mediastinum. A sudden onset of hoarseness following the bolus injection and the local accumulation of contrast medium suggest a misplaced catheter in the subpleural space. Because subpleural block per se provides effective analgesia as well, the therapeutic benefit from replacing the catheter is outweighed by the potential risks with further procedures. In such situations, we recommend that the catheter be withdrawn immediately a few centimeters and replaced only if this intervention is insufficient.

Wei-Zen Sun, M.D.,* Yi Chang, M.D., Yung-Tai Chung, M.D. National Taiwan University Hospital and National Taiwan University College of Medicine, Taipei, Taiwan.

*Corresponding author.

Graham T Bell, F.R.C.A., Michael Bradnam, Ph.D., Iain McKay, F.R.C.S. Royal Hospital for Sick Children, Glasgow, United Kingdom.

Accepted for publication June 9, 2003.

Support was provided solely by institutional and/or departmental sources.
Fig. 1. Chest radiography in standing position after contrast medium injected through the catheter. (A) Posteroanterior view. (B) Lateral view. Accumulation of contrast medium near the sixth thoracic vertebral body and along the aortic arch is shown by the arrows.

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(Accepted for publication October 7, 2002.)