To the Editor—We read with interest the case report by Sala-Blanch et al.1 The authors describe an unorthodox but interesting treatment for patients undergoing continuous sciatic nerve block that raises several concerns. In short, using computed tomographic imaging without clear clinical indication, the authors documented that nerve stimulator-guided needle placement during sciatic nerve block through the anterior approach resulted in an intraneural needle placement. The authors then inserted the catheter and administered local anesthetics. Conventional wisdom suggests that intraneural needle placement and catheter insertion should be avoided because intraneural application of local anesthetics has been shown to result in neurologic injury in animal models.2-6 However, despite the documented intraneural needle and catheter placement—although it is not clear whether the stimulating needle lies between fascia and epineurium or between epineurium and perineurium—the patients did not have neurologic injury. Therefore, this case report suggests that not all intraneural injections lead to neurologic injury. It also suggests that nerve stimulators may not be reliable in avoiding intraneural needle or catheter placement. Finally, a better definition of what constitutes an intraneural versus an intraepineural sheath injection during blockade of peripheral nerves and plexuses is needed for more meaningful discussion of this matter. Some experts may view the patient treatment in report by Sala-Blanch et al. unusual or even potentially hazardous. However, their findings should be welcomed because they clearly pose some important questions. At the least, they suggest that future research should continue to focus on developing more reliable and objective tools of nerve localization and injection monitoring techniques to help avoid intraneural injection and reduce the risk of consequent neurologic injury.7 In any case, it is recommended to withdraw the needle or the catheter if one has any doubt that its position is too close to the nerve, for the safety of regional anesthesia.

Stephan Blumenthal, M.D., Maud Lambert, M.D., Alain Borget, M.D.* ∗Balgrist University Hospital, Zurich, Switzerland.
aborget@balgrist.unizh.ch

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epineurium is a slack collagen and fatty tissue, which contains the vasa and nervi nervorum.5–8 Most of the nerve section (between 30–75%) is occupied by nonnerve structures.5–9 The sciatic nerve produces abundant epineurium, which covers between 72% and 78% of the nerve section.10

Intraepineurial injection of anesthetic would not necessarily lead to nerve damage.11 On the contrary, intrafascicular injection is more likely to induce nerve lesions.12 However, because of their elastic properties, the fascicles probably separate from each other and get out of the way if a needle penetrates the nerve trunk. Although direct neural tissue lesion is unlikely, it should be taken into account that intraepineurial injection of a substance can cause an increase in neural pressure and secondary damage because of compression or a vascular lesion.

Clinical experience supports neurostimulation as a safe and effective technique with minimal incidence of nerve lesions. However, a large number of unresolved questions stemming out from our observations remain: How often does intraepineurial injection occur in routine practice? Does it occur preferentially in certain nerves? What are the clinical and radiologic signs suggesting intraneural puncture? What is the safe threshold for electrical near-nerve stimulation?

Xavier Sala-Blanch, M.D.,* Jaume Pomes, M.D., Purificación Matute, M.D., Josep Valls-Solé, M.D., Anna Carrera, M.D., Xavier Tomás, M.D., Anna L. García-Diez, M.D. * Hospital Clinic, Barcelona, Spain. xsala@clinic.ub.es

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To the Editor.—It was interesting to read the recent case report by Schummet et al.1 regarding management of anaphylactic shock. The use of vasopressin in anaphylactic shock is commendable.

However, before the use of vasopressin, two steps in the management of anaphylactic shock must be addressed. First, the simple measure of elevating the lower limbs could have helped to increase the venous return with vasodilatation, thereby contributing to an increase in blood pressure. This measure is well recommended in the management of anaphylactic shock.2–4

Second, all colloids have been shown to produce clinical anaphylaxis. The overall incidence has been estimated to range between 0.033 and 0.22%.3 The last French survey of anaphylaxis during anesthesia demonstrated that 2.95% of anaphylactic cases were due to colloids.3,5 The incidence of anaphylaxis with succinylated gelatin solution (Gelufusin®; Serumwerk Bernburg AG, Bernburg, Germany) is 0.34%, whereas with hydroxyethyl starch (HES), it is one sixth of this, i.e., 0.06%.6 HES seems to be the safest colloid, and the incidence of immunoglobulin G antibodies against HES is rare in the general population.6 However, this does not betoken the absolute safety of HES.

Isotonic crystalloids are the recommended fluids during anaphylaxis, and rapid infusion of 1–4 l may be required to compensate for the peripheral vasodilatation that often accompanies anaphylaxis. The American Heart Association (Dallas, Texas), in collaboration with the International Liaison Committee on Resuscitation (Antwerp, Belgium), has endorsed the use of isotonic crystalloids in anaphylactic shock.3,4

Although immediate discontinuation of the offending drug, Gelufusin®, was justified,2,5 we are skeptical of its replacement with other colloids with the potential, albeit low, for anaphylaxis. Patients might have developed anaphylaxis to HES, thereby obviating the response to the conventional drugs, such as enzyme-linked immunoabsorbent assay for reactive antibodies to HES was performed to rule out its allergy.7

Last but not least, minimal invasive direct coronary artery bypass grafting does not preclude the possible use of cardiopulmonary bypass. Considering the recent warning by the US Food and Drug Administration (Rockville, Maryland) regarding use of HES for cardiopulmonary bypass, its use over crystalloids does not seem to be justified in this case.

Rajesh Mahajan, M.D.,* Rahul Gupta, M.B.B.S. * Acharya Shri Chander College of Medical Sciences, Jammu, India. drmahajanreddiffmail.com, drmahajan@yahoo.com

Management of Anaphylactic Shock


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In Reply.—We thank Drs. Mahajan and Gupta for their interest in our case report and appreciate the opportunity to reply.

Drs. Mahajan and Gupta obviously believe in algorithms. However, the complexity and severity of anaphylaxis is such that no single algorithm can adequately treat all cases. Anaphylaxis is generally an unanticipated severe allergic reaction, often rapid in onset, and starts within seconds to minutes after exposure to the allergen. Symptoms progress rapidly, can affect most organ systems, and can lead to cardiovascular collapse and death, even when appropriately treated. The management of anaphylaxis consists of withdrawing the offending drug, interrupting the effects of the preformed mediators that were released in response to the antigen, and preventing more mediator release.

The intention of our case report was to adjoin a practicable method to the management of anaphylaxis by considering the application of vasopressin to standard therapy as an approach for mediator-induced vasodilatory shock and not to add on a discussion about the right colloid.1

Yes, effective fluid therapy is a mainstay of treating critically ill patients. The ideal kind of volume replacement in this situation remains a matter of debate. Despite an immense number of contributions to this problem, there is no answer yet. This topic is often discussed emotionally rather than scientifically. The ideal solution should not only maintain gross hemodynamics, but organ perfusion and microcirculation should also be guaranteed or even improved. To treat hypovolemia, in Germany, colloids are used more often than crystalloids. The lack of acceptance of synthetic colloids such as hydroxyethyl starch (HES) as a solution for volume replacement is most likely due to reports on abnormal coagulation function. This cannot be used as an argument when new, modern HES preparations with low molecular weights (70,000 or 200,000 Da) and a low degree of substitution (0.5) are used. This is the commonly used priming solution of the cardiovascular bypass machine at our institution.2,5

In clinical practice, with the given situation of a high-risk patient with cardiovascular disease, being placed on the operation table for minimally invasive direct coronary artery bypass grafting, elevation of the legs and head-down tilt is not a suitable therapeutic option. Infusion of up to 4 l of a crystalloid is time-consuming and might end in fluid overload.

There was no need for skin testing of HES because further infusions did not provoke anaphylactic reactions. Furthermore, the cause of adverse reactions due to HES is not yet clear. Major histamine release is not known to occur.5 In a multicenter, prospective trial, 200,906 infusions of colloid volume substitute have been examined. The frequency of severe reactions (shock, cardiac and/or respiratory arrest) was 0.006% for HES.5 HES-reactive antibodies are extremely rare, and they do not necessarily induce anaphylaxis.5 Also, there is no known cross-reactivity between the different colloids, so a particular allergy to one should not preclude the use of a different colloid.7

Tachycardia, a common symptom in anaphylaxis, may have devastating consequences, especially in patients with cardiovascular disease, and should be terminated as soon as possible. High-dose epinephrine, administered with the intention to stabilize hemodynamics, may cause cardiac fibrillation, whereas vasopressin increases perfusion pressures and has an antitachycardic effect.6

Since the publication of our case report, we have gained more experience with the use of vasopressin in the management of anaphylactic shock beyond standard therapy. Administration of vasopressin, regardless of the causing agent (e.g., antibiotics, nonsteroidal anti-inflammatory drugs), always stabilized hemodynamics quickly: The need for epinephrine reduced dramatically, and the heart rate normalized.

The management of anaphylactic shock must be immediate because time is running against the patient. Restoring cerebral and coronary perfusion quickly plays a pivotal role; therefore, one should consider the early addition of vasopressin complementary to standard therapy.

Wolfram Schummer, M.D., D.E.A.A., E.D.I.C.,* Claudia Schummer, M.D., Jens Wippermann, M.D., Juergen Fuchs, M.D. *Friedrich-Schiller-University Jena, Jena, Germany. cwsm.schummer@gmx.de

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To the Editor.—I was excited when I read the title ‘Severe Neurological Complications after Central Neuraxial Blockades in Sweden 1990–1999.’1 I expected that the article would broaden my knowledge in this important aspect of anesthesiology. Although the article was very good and I commend the authors for their work, I was disappointed in one respect. The most surprising and new observation was that the authors uncovered 11 cases of cauda equina syndrome (CES) associated with bupivacaine spinal anesthesia. This finding is both eye opening and discouraging. It is discouraging because of all the local anesthetics used for spinal anesthesia, bupivacaine seems to be the safest. This is supported by the fact that a literature search of the terms cauda equina syndrome and bupivacaine turned up only four case reports. Furthermore, in vivo and in vitro studies show limited bupivacaine neuronal toxicity. However, in the report by Moen et al.3 bupivacaine caused the greatest number of cases of CES associated with spinal anesthesia. This could be due to the fact that bupivacaine is indeed neurotoxic in certain clinical situations and surfaces as neurotoxic in this report by Moen et al.1 because bupivacaine is more widely used for spinal anesthesia than are any of the other local

Bupivacaine Spinal Block Cauda Equina Syndrome: Why Did It Happen?

The above letter was sent to the author of the referenced Editorial View. The authors did not feel that a response was required.—Michael M. Todd, Editor-in-Chief
To the Editor:—The authors should be congratulated on their comprehensive work “Severe Neurological Complications after Central Neuraxial Blockades in Sweden 1990—1999.” A variety of biases, which usually are inherent in such study designs, were successfully controlled. Considering previous data from the 1990s, the current incidence of complications is alarmingly high, but it stands in line with most recent respective surveys.

One question, however, deserves discussion. The authors described significantly higher incidence of spinal hematomas in the population of orthopedic patients, but the underlying causes remain partially unexplained. The authors presume that the high incidence may be related to low-molecular-weight heparin administration, which was introduced for thromboembolism prophylaxis during the study period.

In an 8-yr survey regarding serious complications after regional anesthesia at our institution, we observed three spinal epidural hematomas in 28,933 central neuraxial blocks, of which two occurred in the subgroup of orthopedic patients (n = 4,205), indicating similar incidences and risk factors as reported by Moen et al. One of the hematomas was previously reported elsewhere, and the second one occurred after spinal anesthesia in a patient treated with unfractionated heparin. Both patients were concomitantly treated with nonsteroidal antiinflammatory drugs (NSAIDs), which were not considered a risk factor at that time. The third hematoma developed in a patient with heparin-induced thrombocytopenia during postoperative epidural pain therapy after hemiepatectomy. No hematoma occurred in urologic (n = 10,817) or obstetric (n = 4,250) patients.

The discussion about the risk of spinal hematoma in patients with NSAIDs (antiplatelet) therapy remains controversial. In orthopedic patients treated with aspirin, Horlocker et al. did not observe an increased risk of spinal hematoma during spinal anesthesia. However, in 1984, Cronberg et al. reported on the effects of NSAIDs on the second wave of aggregation, which was considered a key issue in our patient. In urologic patients (no hematoma in 10,817 neuraxial blocks), the risk profile regarding comorbidity and comedication is comparable to that of orthopedic patients, with the exception of concomitant therapy with NSAIDs.

We believe that the combination of heparin and NSAIDs in orthopedic patients is responsible for the higher risk of spinal hematoma in this group as compared with obstetric patients. Because pain therapy with NSAIDs is widespread in patients in need of (orthopedic) hip or knee joint replacement surgery, it would be of interest if the authors could provide any information regarding the concomitant use of NSAIDs in their cohort of orthopedic patients.

Axel R. Heller, M.D., Ph.D., D.E.A.A.* Rainer J. Litz, M.D.
*University Hospital Carl Gustav Carus, Dresden, Germany.

donaldam@fastmail.us

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To the Editor.—We strongly support reporting systems and therefore read with interest the Editorial View by Auroy et al.3 However, before these methods become a standard audit or educational tool, some of their limitations must be considered further.

Clinicians can often select the type of adverse incident or outcome they will record. These tend to be those that are more severe, or those in accordance with individual perspectives of safety.2,3 For example, prolonged paralysis after a regional block is more likely to be reported than transitory paresthesia. Temporary complications are often ignored, despite their potential educational value. Sometimes, reporters select those incidents most likely to carry a message to the organization’s management.4 Incidents over inappropriate waiting times for patients or surgeons are not exceptional in anesthetic incident–reporting systems. Such selection and reporting biases may seriously distort perception of safety problems in anesthesia.

When reporting systems focus on near misses (prevented or mitigated adverse events), another difficulty arises, one familiar to aviation safety experts: information overload.4 A progressively larger amount of data is collected and stored to be further analyzed. It can become increasingly difficult and costly to classify and retrieve meaningful events in such an extensive system analysis.5 Gradually limited by resources and complexity, experts may end up fixing near misses instead of addressing system errors concealed behind the data overload. This may jeopardize the didactic value of such events.

Finally, anesthetic and medical practices in general are largely controlled by a professional body of knowledge.6 Organizational guidelines and standards are much less the norm than, for example, in chemical or nuclear industries.7 Variability in local practices, professional culture, and political context seriously challenge the generalizability of organizational analysis.

To address these problems, suggested approaches could include the use of international standardized definitions of incidents and the development of guided reporting through generic adverse event indicators. The specificities of the healthcare organization analyzed could also be more systematically described and addressed.

If limitations such as these are not well understood and properly addressed, case reports and root cause analyses of adverse incidents and near misses are likely to remain largely narrative and of limited educational value within the broader anesthetic community.

Guy Haller, M.D.* Paul S. Myles, M.B., B.S., M.P.H., M.D., F.C.A.R.C.S.I., F.A.N.Z.C.A. * Alfred Hospital, Melbourne, Australia. guy.haller@med.monash.edu.au

In Reply.—We thank Dr. Lambert for his interest in our study, which was performed primarily to investigate the incidence of severe complications after central neuraxial blockades.1 The strength of our study is the comprehensive study design and the large number of cases recorded. The limitation, common to most retrospective studies, is the relative lack of detail. Before attempting to answer the ultimate question of “why does it happen,” we set out to answer the question of “does it happen,” because the causal relation between central blockades and complications has been questioned, even recently.2 Our data were subsequently analyzed to answer the question “to whom does it happen.” This is a straightforward epidemiologic approach that can produce some but not all of the answers.

We share Dr. Lambert’s preoccupation regarding the high number of patients with cauda equina syndrome.

This patient group is heterogeneous, regarding both type of blockade and patient characteristics and, most importantly, probably also regarding pathophysiology of the complication. To attempt a better understanding of the different pathogenetic processes, we grouped nine of the patients with cauda equina syndrome and the four patients with paraparesis, because they were all found to have spinal stenosis (table 5 in the article).3 Most of these patients were older than 70 yr, and nine patients had received epidural blockade or combined spinal epidural blockade. Compression was thought to have an important role in the development of the complication in these patients. The proposed pathogenetic process is demonstrated by a recent case report of transient paraplegia during epidural infusion in an elderly woman with severe kyphosis.5 This case report included a magnetic resonance image showing cord compression caused by epidural infusion: Remarkably, the neurologic deficit spontaneously receded shortly after the infusion was stopped, thus illustrating the possibility of creating high epidural pressure with epidural pump infusion in subjects with restriction of the spinal canal and with outflow obstruction. Also, during spinal anesthesia in a patient with spinal stenosis, maldistribution of local anesthetic in the subarachnoidal space could cause higher concentration, thus favoring neurotoxic processes.

In our study, a remaining 25 patients experienced cauda equina syndrome in the absence of spinal stenosis. Single-shot spinal blockade had been given to 15 of these patients, and in 1 additional patient, a spinal catheter was placed. These patients were younger, half of them being younger than 55 yr. Lidocaine had been used in seven cases, bupivacaine had been used in five cases, and a combination of both drugs had been used in one case. No information was obtained in three cases and complications has been questioned, even recently.2,3 For example, prolonged paralysis after a regional block is more likely to be reported than transitory paresthesia. Temporary complications are often ignored, despite their potential educational value. Sometimes, reporters select those incidents most likely to carry a message to the organization’s management.4 Incidents over inappropriate waiting times for patients or surgeons are not exceptional in anesthetic incident–reporting systems. Such selection and reporting biases may seriously distort perception of safety problems in anesthesia.

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cases. Roughly twice as many ampoules of bupivacaine were sold during the study period, indicating a higher incidence of cauda equina syndrome after spinal anesthesia with lidocaine. The fact that half of the spinal blockades were reported as technically difficult or resulting in unsatisfactory anesthesia could indicate uneven distribution of local anesthetic within the spinal canal, causing localized high concentrations resulting in neurotoxicity. The pathogenic processes, however, might vary: The diagnosis of arachnoiditis was made in two patients, and in one further patient, an elderly man, severe hypotension during surgery was believed to be at least partially responsible for the complication.

Cauda equina syndrome occurred after spinal blockade in roughly 1:100,000 of our patients. This is an unpleasant information, because our data do not permit us to provide a better understanding of the pathophysiological processes. However, we would like to point out that although case reports may yield important information, they should not be used as indication of incidence. One of our main results is in fact the demonstration of discrepancy between calculations of incidence based on case reports or research in larger but still limited sources and the incidences in our study, obtained with a more comprehensive research.

Drs. Heller and Litz raise the question of the role of nonsteroidal antiinflamatory drug (NSAIDs) in the development of spinal hematoma. In our study, only 4 of the 13 orthopedic patients with spinal hematoma were reported to have received NSAIDs, and a further 2 patients received low-dose acetyl salicylic acid, including one patient receiving steroid therapy. However, underreporting can be presumed. The respondents in the enquiry were asked to provide as much information as possible regarding the patients’ pathology and medication. Because the association of low-molecular-weight heparin and spinal hematoma was much debated, we can assume that the information regarding these drugs is more reliable than is the case with NSAIDs.

Drs. Heller and Litz partially misinterpret our conclusions regarding the higher incidence of spinal hematoma among the orthopedic patients. We do not propose that this is exclusively related to the administration of low-molecular-weight heparin, and on the contrary, we propose that spinal pathology is also an important factor, as well as bleeding disorders and medications interfering with coagulation. Also, in the aged spine, any space occupying mass causes cord compression at an earlier stage as compared with the younger spine.

The development of a symptomatic spinal hematoma is probably a multifactorial event, and NSAIDs, alone or in combination with thromboprophylaxis, might be one of these factors. Patients with spinal and vascular pathology might be more susceptible to alterations of platelet function. However, the opinion that NSAIDs are mainly responsible for the development of spinal hematoma in these patients could induce an ungrounded belief that epidural blockades are safe in orthopedic patients, permitted they refrain from taking NSAIDs. This solution seems too simplistic. Many data now indicate a high risk among orthopedic patients for spinal hematoma, particularly after epidural blockade. The use of epidural blockade for hip or knee replacement should be questioned because the risk seems out of proportion to the benefit.

Drs. Heller and Litz interestingly refer to three cases of spinal hematomas in their institution. However large and impressive, the total of 28,933 central neuraxial blockades does not permit reliable calculation of incidences, because spinal and obstetric anesthesia are included. The urologic patients, although of similar age to the orthopedic patients, include fewer females and usually, at least at our institutions, more often receive spinal blockades. These facts alone prevent any physiopathologic comparison between the two patient groups.

Vibeke Moen, M.D., Nils Dahlgren, M.D., Ph.D., Lars Irestedt, M.D., Ph.D. * County Hospital, Kalmar, Sweden. vibke@m@ltkalmar.se

References


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Stellate Ganglion Blockade for Acute Postoperative Upper Extremity Pain

To the Editor.—We read with interest the review article by Dr. Reuben entitled ‘Preventing the Development of Complex Regional Pain Syndrome after Surgery.’ The author mentioned that in his practice, he administers a stellate ganglion block to patients with a history of complex regional pain syndrome (CRPS) undergoing upper extremity surgical procedures in an effort to prevent its recurrence. Although the literature supports this practice, the efficacy of a stellate ganglion block has broader applications to patients without a history of CRPS. No study has examined its use in the acute postoperative setting. Nevertheless, we report the first case using a stellate ganglion block to treat acute postoperative pain successfully in a patient without a history of CRPS who underwent an open reduction and internal fixation of his left humerus.

A 46-year-old, 70kg male with a medical history significant for recent cocaine abuse underwent general anesthesia for an open reduction and internal fixation of a left humerus fracture. The 5h intraoperative course was uneventful during a 5% desflurane in 50% nitrous and oxygen anesthetic. Fentanyl, 500 μg, and 5 mg morphine were also given intraoperatively. In the postanesthesia care unit, an additional 20 mg morphine was titrated for pain relief, although to no avail. The patient’s pain score remained 10 out of 10. On further evaluation, his left upper extremity appeared cold, clammy, and edematous, whereas the pain characteristic was described as burning.

After informed consent, a left stellate ganglion block was performed using 10 mL bupivacaine, 0.25%, with a sterile technique. Pain relief (0 out of 10) was achieved 5 min after injection. Evidence of successful blockade included paresis and miosis, temperature increase in the ipsilateral extremity, and an increase in perfusion index.

Numerous publications in the literature support the use of a stellate ganglion block for chronic sympathetically mediated pain; however, to the best of our knowledge, we report the first case in which a stellate ganglion block was used in the acute postoperative setting. Furthermore, the success of the block in absence of a history of CRPS illustrates primary prevention—interventions to prevent a disease from...
occurring. Activation of the sympathetic pain pathways could have taken place before the patient’s operating room visit or even intraparatively. Nevertheless, the challenge is to recognize the clinical symptoms that differentiate acute CRPS from postsurgical pain, despite the close resemblance. Further review of the case revealed that the patient’s fracture was a week old, which may have contributed to an alteration of central nociceptive processing pathways, thus increasing his risk for development of postsurgical CRPS. Overall, we share the same enthusiasm of Dr. Ruben on regional anesthetic techniques to prevent the occurrence of CRPS. In our clinical practice, it is not only important to focus on CRPS prevention, but also to understand the pathophysiology so that we know which patients are at greatest risk.

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In Reply—I appreciate the comments by Drs. Kakazu and Julka. Although the role of the sympathetic nervous system in certain chronic pain states, including complex regional pain syndrome (CRPS), has been well documented, its role in acute pain and inflammation is still controversial. A coupling between the sensory afferent and sympathetic efferent system after peripheral nerve lesions has been previously described as a causative mechanism for neuropathic pain, including CRPS. However, the role of the sympathetic nervous system in acute pain still must be elucidated. The sympathetic nervous system may influence the nociceptive response to acute tissue injury in two ways. First, there is a potentiation of the chemical mediator cascade after trauma as well as augmentation of sensitization to substance P. Second, norepinephrine and α2 agonism in the dorsal horn neurons and in the locus ceruleus in the medulla play a major role in the descending medullospinal inhibition of nociception. Experimental studies in rats indicate that sympathetic postganglionic neurons may be involved in the generation of pain, hyperalgesia, and inflammation after tissue damage or trauma. Sensitization of nociceptive afferent fibers in the knee joints of rats involves a cascade of events in which the mast cell and the sympathetic terminal are sequentially activated, resulting in plasma extravasation in the synovium. Further, adrenal medullary-derived epinephrine can exacerbate and surgical or pharmacologic sympathectomy can decrease the severity of experimental arthritis. Although these behavioral studies in rats point to a potential role of the sympathetic nervous system in acute inflammatory pain, neurophysiologic studies of nociceptors in rats and psychophysical studies in humans have failed to provide confirmatory evidence for the role of sympathetic efferents in inflammatory pain and hyperalgesia. The use of a stellate ganglion block for acute postsurgical pain as described by Drs. Kakazu and Julka is intriguing. I agree with the authors that because this patient sustained his fracture 1 week before surgical correction, prolonged sensitization and alteration of nociceptive afferent pathways may have increased the risk for the development of postsurgical CRPS. However, the authors did not report whether this patient had any clinical symptoms consistent with CRPS before surgical intervention. Perhaps the surgical intervention exacerbated an underlying disease process that may have been initiated before surgery. Interestingly, since the success of our original report on the efficacy of stellate ganglion blocks for CRPS patients undergoing surgery, many of our surgeons now request that we routinely perform this sympathetic block for non-CRPS patients who are undergoing surgical procedures and are at increased risk for development of this disease. Although stellate ganglion block may be effective for treating acute postoperative neuropathic pain, it may also be useful in the management of acute nociceptive pain. Matsuura et al. describe the efficacy of stellate ganglion block in 29 patients undergoing ocular surgery. These authors report a significant reduction in postoperative pain and nonsteroidal antiinflammatory drugs with the use of stellate ganglion block. These case studies highlight the importance of moving from symptom control toward a mechanism-specific pharmacologic management of postsurgical pain. We have only begun to identify the multiple neurobiologic mechanisms responsible for different pain states. We hope to be able to develop diagnostic tools that will allow us to identify these mechanisms in individual patients and target them with appropriate analgesic interventions. Future research is needed in defining the clinical significance of the interaction of the sympathetic nervous system and the somatic afferent system in the development of postsurgical neuropathic pain. At that time, we may have available not only the resources for eliminating acute pain but also the tools necessary for preventing the occurrence of chronic postsurgical pain.

Scott S. Reuben, M.D., Baystate Medical Center and the Tufts University School of Medicine, Springfield, Massachusetts. Scott.reuben@bhs.org

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David C. Warltier, M.D., Ph.D., acted as Handling Editor for this Correspondence.
To the Editor—It is important to have suitable fluid warmers for pediatric anesthesia, especially in hypovolemic neonates and infants who require boluses of isotonic crystalloid, packed erythrocytes, or both given over 5–20 min. The purpose of this study was to evaluate the fluid warming and air venting capability of a new device (buddy fluid warmer; Belmont Instrument Corp., Billerica, MA) designed for use in pediatrics. With this device, fluids are heated to 38°C as they pass through a disposable set containing microporous membranes able to vent air. Air is released through the side vents of the set. The small heating unit and disposable set are placed in-line near the patient at the intravenous infusion site and can be used easily with volumetric infusion pumps.

Fluids tested were lactated Ringer’s solution, 1 l, at room temperature, and refrigerated, outdated erythrocytes diluted with 100 ml saline, 0.9%. The estimated hematocrit was 50%. Standard or Y-type blood solution sets were attached proximal to the commercial microheater disposable set (priming volume, 4 ml), which was connected to a 12.7 cm T connector (total volume, 0.4 ml). A rapid response thermocouple (Fluke 51 II; Fluke Corp., Everett, WA; accuracy, ±0.05%) was used to measure distal temperature at the point at which the T connector would be attached to the intravenous line. Temperature data were collected at 5 ml intervals for flows of 8 ml/min or greater and at 10 s intervals for slower flows. A volumetric infusion pump was used to regulate flow between 50 and 1,000 ml/h. For gravity-free flow, lactated Ringer’s solution was infused from a height of 1.8 m into a cylindrical scored beaker, and measurements were made every 50 ml. Pressure-driven flow was not used, per manufacturer guidelines.

A three-way stopcock with a 0.8 m extension was inserted proximal to the microheater, and a 22-gauge Angiocath (Becton Dickinson, Sandy, UT) was attached to the T connector distally. The Angiocath was submerged in a liquid-filled beaker for crystalloid infusion and attached to a cell salvage waste system for erythrocytes. Without the fluid warmer disposable set, injection of as little as 1 ml of air was readily visible in the liquid-filled beaker with submerged Angiocath. Aliquots of 5, 10, 20, 30, 40, 50, and 60 ml of air were rapidly injected into the stopcock toward the patient infusion site, followed by resumption of fluid flow. Visual inspection for air bubbles distal to the warmer was performed by two observers. Tests were repeated twice for each condition and fluid.

Distal temperatures are summarized in figure 1. Air bubbles were not seen in the T connector or in the liquid-filled beaker for any value of injected air.

The buddy fluid warmer was effective in delivering warm intravenous fluids at flows of 7 ml/min or greater. At the slowest flows, infusate temperature decreased, likely resulting from significant heat loss distal to the warmer. Venting of air by the fluid warmer is of great advantage to pediatric patients with congenital heart disease. Moreover, use of this warming device might theoretically reduce the risk of accidental infusion of air during crystalloid and blood resuscitation of children with hypovolemic shock. The manufacturer’s list price for the buddy fluid warmer is $1,599.00; the disposable set is $14.99.

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Ramachandra R. Avula, M.D., Charles E. Smith, M.D., F.R.C.P.C.* 4 MetroHealth Medical Center, Case Western Reserve University, Cleveland, Ohio. cssmith@metrohealth.org
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Air Venting and In-line Intravenous Fluid Warming for Pediatrics

Fig. 1. Distal temperature (°C) of lactated Ringer’s solution (LR) and erythrocytes (RBCs) at the point where fluid would enter the patient. Proximal crystalloid temperature was (mean ± SD) 16.5 ± 2°C. Proximal crystalloid temperature was 19.2 ± 1.2°C. Gravity flow from a height of 6 feet was 68 ± 10 ml/min, and distal temperature was 37.8°C. A minimum of 40 measurements were made for each flow (total of 1,216 measures).

To the Editor—Several surgical procedures require single-lung ventilation. Because a double-lumen tube (DLT) allows for independent ventilation therapy (e.g., continuous positive airway pressure), suctioning, and bronchoscopy of each lung, the DLT is the accepted standard for the treatment of patients undergoing thoracic surgery.1 A difficult

intubation in this subset of patients is particularly challenging because tube insertion is impeded by the special shape of the DLT and the two separate cuffs, which can be damaged by the patient’s teeth and during repeated insertion maneuvers using intubation tools with sharp surfaces.

Recently, in two patients scheduled to undergo a minimally invasive direct coronary artery bypass procedure requiring DLT insertion, direct laryngoscopy failed. Because at our institution the Bonfils intubation fiberscope (Karl Storz GmbH, Tuttingen, Germany), a rigid fiber-
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Bayonet Artifact During Ultrasound-guided Transarterial Axillary Block

To the Editor—Ultrasound-guided regional anesthesia is an emerging field that potentially provides better block efficacy than other current techniques.\(^1\)\(^2\) In particular, ultrasound-guided axillary block had been described as an excellent technique for brachial plexus anesthesia.\(^3\)\(^4\) With this method, a short axis (transverse cross-sectional) view of the axillary artery and surrounding nerves is obtained with the block needle approaching from the lateral aspect of the arm in the plane of imaging. Although V-shaped redirection of the block needle can be used to place local anesthetic on the superficial and deep sides of the axillary artery, transarterial placement of the needle may occur during the procedure.

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

During an ultrasound-guided axillary block, we observed a bent echo spray of the 25-gauge 3.8-cm Quincke tip needle (Becton Dickinson and Company, Franklin Lakes, NJ). The needle shaft echo was bent toward the skin surface when the needle crossed the axillary artery (fig. 1). When examined after the procedure, the injection needle was per-rectly straight.

Described as a “bayonet artifact,” this ultrasound artifact causes apparent needle deformity and has been reported during breast biopsy in which a needle traverses a tumor surrounded by fat tissue.\(^5\) Bayonet artifact occurs when the ultrasound beam passes through tissues with different speeds of sound. Similar speed of sound artifacts have been described in the soft tissues of the kidney.\(^6\)\(^7\) Because all commercial ultrasound machines assume a uniform speed of sound of 1,540 m/s,\(^8\) actual differences among speeds of sound in tissue change the appar-
The speeds of sound among soft tissues may actually range from 1,450 m/s in fat to more than 1,600 m/s in muscle. Thus, clinical scanning can routinely produce speed of sound errors of approximately 5%.

This letter describes a bayonet artifact observed during regional anesthesia. The artifact occurred when the block needle passed through the axillary artery with the ultrasound beam nearly perpendicular to the needle. The speed of sound in whole blood (1,580 m/s) is higher than the average speed of sound in soft tissue (1,540 m/s). Therefore, bending of the needle echo toward the transducer was seen when the needle passed through the axillary artery.

Ultrasound guidance likely reduces the incidence of vascular puncture during regional block. However, inadvertent vascular puncture has been reported during peripheral nerve blocks despite use of ultrasound guidance. Therefore, bayonet artifacts from transarterial needle placement may occur during regional block even if vascular puncture was not intended.

Figure 1. “Bayonet” bending of the needle shaft echo as the block needle passes through soft tissue and the axillary artery during an ultrasound-guided axillary nerve block. The arrowhead indicates the part of the needle echo that bends toward the ultrasound probe on the skin surface. Tick marks are 10 mm apart.

The basic premise of the in-plane approach for ultrasound-guided regional blockade is that precise placement of the block needle tip near nerves is possible in real time. However, there are circumstances under which the actual needle position and perceived image do not agree. Here, we describe one of those circumstances that occurred during transarterial axillary block.

Andrew T. Gray, M.D., Ph.D.,* Ingeborg Schafhalter-Zoppoth, M.D. * San Francisco General Hospital, University of California, San Francisco, California. graya@anesthesia.ucsf.edu

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