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A Study of Hand Hygiene in the Postanesthesia Care Unit—It's about Time!

AS a hospital epidemiologist, my inclination when I reviewed the article by Pittet *et al.* entitled "Hand-cleansing during Postanesthesia Care"[1] was to say, "It's about time someone addressed this issue in the PACU"! In contrast, readers of the Journal may be asking, "What's the big deal? These authors haven't demonstrated that poor compliance with hand-cleansing, or hand hygiene, in the PACU is linked to subsequent nosocomial infections." If that is how you responded, you are correct when you say the authors did not prove that poor compliance with hand hygiene caused nosocomial infections. In fact, their goal was simply to evaluate compliance with hand hygiene in their PACU. They accomplished this goal and documented that compliance with this basic infection-control measure was as low or lower than that previously reported from intensive care units (ICUs).

Before I go further, I want to congratulate the authors for addressing this difficult topic. I also want to congratulate nurses and physicians working in PACUs who take hand hygiene seriously. That said, I'd like to address skeptics in the reading audience. I also would like to address the issue of time— because it is not only about time someone did a study on this topic, but it is also time that makes this a difficult topic to study, and time (or lack thereof) that may prevent some staff from practicing good hand hygiene.

Given the brief time that patients are in the PACU, it will be difficult to prove that specific nosocomial infections occurring while the patient is in the surgical ICU or on the surgical ward were caused by the PACU staff's failure to perform hand hygiene appropriately. However, data from ICUs indicate that patients typically acquire pathogens from the hands of healthcare workers and that hand hygiene decreases the transmission of these organisms and prevents nosocomial infections.[2,3] I cannot envision a universe in which rules that apply in ICUs do not apply in PACUs. Thus, a Gram-negative organism may be carried on a PACU nurses' hands from the Foley catheter to the hub of the central venous catheter and from there into the bloodstream of a patient. When signs and symptoms of bloodstream infection are manifest, the patient will be in the surgical ICU. The infection-control program will report the infection to surgical ICU staff, and PACU staff will never receive feedback about that or any other infection.

PACU staff members are extremely busy caring for patients who are unstable, in pain, have numerous invasive devices, and require substantial nursing care. Obviously, if the choice is between performing hand hygiene and performing a task that will save the patient's life, staff members should save the patients' life. However, this author suspects that staff members infrequently must choose between performing hand hygiene and saving the patient's life. Instead, I believe that PACU staff and other staff neglect to cleanse their hands because they have not been trained to identify all situations in which hand hygiene should be performed or because the culture in the unit is such that staff members do not put a high priority on this practice.

The argument that PACU staff members do not have time for hand hygiene is mitigated in part by the alcohol-based hand-hygiene products available in many hospitals. These products can be placed at the bedside so that staff members do not even need to cross the room to cleanse their hands. Moreover, Voss and Widmer documented that these products reduce by 50–75% the time needed for hand hygiene in an ICU.[4]

Two recently published studies are pertinent to the study by Pittet *et al.* Rogues *et al.* documented that 33% and 41% of patients carried pathogenic organisms in their nares or on skin adjacent to their surgical sites when they were admitted to the PACU and when they were discharged, respectively.[5] Nineteen percent of staff also carried pathogenic organisms. These investigators concluded that cross-contamination could occur in PACUs and that staff needed education regarding hand hygiene, isolation precautions, and environmental cleaning. Hajjar and Girard conducted surveillance for nosocomial infections related to anesthesia, which they defined as infections occurring within 72 h of a general or regional anesthetic procedure.[6] They identified 25 infections—12 respiratory, 9 vascular catheter–associated, 2 eye, and 2 mouth—for a rate of 3.4 infections/1,000 patients. The infections could have been acquired in the operating room, PACU, or surgical ICU. Although we can't prove that they originated from errors in the PACU, we also can't prove that they didn't.

The PACU is usually an open ward without barriers, such as walls, between patients to remind staff members that they need to cleanse their hands when moving from one patient to another. Also, patients usually are not

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alert enough to ask their caregivers whether they have cleansed their hands. In addition, numerous articles have outlined the infectious hazards that PACU staff encounter in their routine work.[7] Hand hygiene and other basic infection-control precautions protect not only patients but also healthcare workers. Thus, it would behoove PACU staff to understand the risks they face and to use infection-control precautions to prevent exposures. The old adage, an ounce of prevention is worth a pound of cure, applies in this setting. Infection-control staff members are often amazed when healthcare workers who have been exposed to infectious agents reply "I didn't have time" when they are asked why they did not put on personal protective equipment. Yet they have time to demand prophylaxis after the exposure.

It is time that anesthesia and PACU staff members—both physicians and nurses–view themselves as links in the infection-control chain. A chain is only as strong as its weakest link. PACU staff members do not want to be the weakest link in the infection-control chain, but their compliance with hand hygiene places them in this position. To upgrade the strength of their link, PACU staff members must change the culture of their units such that good hand hygiene is considered an essential part of the job. Physicians, in particular, must become good examples. A recent study in Germany found that 70% of healthcare workers attending an infection-control meeting thought that physicians and other supervisory staff were poor role models.[8] Infection-control specialists may be able to suggest changes that improve practice, but they cannot design the best solutions because they do not work in PACUs. PACU staff, who know how the work is done and know the limitations of staffing, space, and time, are the only ones who can develop effective strategies. In addition, administrators must provide adequate staffing levels and training. The latter are important not only to improve infection control but also to maintain or improve the overall quality of care and to provide a safe work environment for staff.

Pittet and colleagues have laid down the gauntlet to PACU staff and to hospital administrators. It remains to be seen whether those staff and administrators will pick it up, own the problem, and find creative solutions, or whether they will refuse to acknowledge their place in the great chain of infection control.

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Allergic to Anesthetics

THE article by Mertes *et al.*[1] in this issue contributes important new knowledge to the anesthesia community on the epidemiology of perioperative anaphylactoid and anaphylactic (immunologically mediated) reactions in France. Although cutaneous reactions to drugs are common, life-threatening reactions to anesthetic drugs and adjuvants are unusual. These reactions occur approximately once in every 5,000–10,000 anesthetics. Over half of these serious reactions are immunologically mediated; the remainder are chemically mediated. Because most anesthesiologists witness only a few such reactions in a lifetime, mechanistic and epidemiologic studies such as this provide insights that can contribute directly to clinical practice. Mertes *et al.*[1] give clinicians new information about the prevention, recognition, and identification of these life-threatening reactions. Their observations confirm and extend those made by the Nancy group in five previous reports over the past 20 yr.[2–6] Taken as a whole, these six studies document the epidemiology of more than 4,500 life-threatening reactions during anesthesia. This report also documents emerging trends in the reactions, provides objective evidence that their etiology can be detected, and offers associations that can directly improve patient care.

Although muscle relaxants remain the primary etiologic agents of immunologically mediated reactions (58.2%, [n = 306]), emerging trends of relaxant use demonstrate that rocuronium (43.1% [n = 132]) has surpassed succinylcholine (22.6% [n = 69]) as the drug most frequently implicated in these reactions. Mertes *et al.* confirm the frequently observed clinical predominance of such reactions in female subjects, thought to be due to a common epitope relaxants share with many cosmetics.[7,8] Such an explanation is consistent with the observation that many patients manifest an allergic reaction to muscle relaxants on first exposure. Their data also remind us that drugs that do not elicit the chemical release of histamine can and do cause allergic reactions. Latex is the second most frequent cause (16.7% [n = 79]) of reactions, but this has not increased significantly since the last survey. Perhaps because of the growing recognition by clinicians and preoperative screening for patients at risk for this syndrome by the radioallergosorbent test and other methods, the twentyfold increase in latex allergy in the early 1990s seems to have stabilized. As the third most common cause (15%), reactions to antibiotics have increased eightfold since 1989. The etiology of this relative and absolute increase in reactions to antibiotics is unclear, but it may be due to a more widespread use of antibiotics in the community. Reports of reactions to opiates and local anesthetics still remain uncommon, despite their frequent identification as allergens by patients. Given the trend toward polypharmacy and the complexity of the surgical setting, it often takes considerable detective work to identify the responsible agent. In a recently reported case, the aprotinin in fibrin glue was implicated as the cause of a fatal anaphylactic reaction.[9] In other instances, an allergy to latex was apparent only after deflation of the tourniquet or as a component of disinfectant sprays used to sterilize anesthesia and surgical equipment.[10,11] However, that so few cases in this series remain without etiology suggests that the tools for a thorough investigation do exist.

The second contribution of the article by Mertes *et al.* is that it shows clinicians how best to identify the agents responsible for these reactions. Few anesthesiologists or allergists have experience with the methodology for skin testing described in detail by the authors. Further, because of the rapid catabolism of histamine and the technical difficulty in sample acquisition and measurement, histamine levels remain mostly a research rather than clinical tool. However, tryptase levels were significantly elevated in only 10.7% of chemically mediated reactions but in nearly two thirds of immune reactions, which gives clinicians a very practical tool for distinguishing between the two types of reactions. The use of tryptase to distinguish between chemical and immune reactions has been the source of debate in previous articles in ANESTHESIOLOGY. *In vitro* studies have suggested a generalized co-release of tryptase by high doses of chemical-releasing agents such as vancomycin.[12] However, in a clinical study of rapid administration of vancomycin, chemically mediated reactions did not cause tryptase release, although histamine levels increased fortyfold.[13] An Australian epidemiologic study suggested that the presence of an increased tryptase level highly favored an

This Editorial View accompanies the following articles: Mertes PM, Laxenaire M-C, Alla F, Groupe d-Etudes des Réactions Anaphylactoïdes Peranesthésiques: Anaphylactic and anaphylactoid reactions occurring during anesthesia in France in 1999–2000. ANESTHESIOLOGY 2003; 99:536–45; Licker M, Spiliopoulos A, Morel D, Chevalley C: Postextubation severe bronchospasm and hypotension triggered by exposure to a disinfectant spray. ANESTHESIOLOGY 2003; 99:739–41; Pirat P, Lopez S, Motais F, Bonnet M-C, Capdevila X: Latex anaphylaxis following tourniquet release during total knee arthroplasty. ANESTHESIOLOGY 2003; 99:741–3; Oswald A-M, Joly L-M, Gury C, Disdet M, Leduc V, Kanny G: Fatal intraoperative anaphylaxis related to aprotinin after local application of fibrin glue. ANESTHESIOLOGY 2003; 99:000–00.

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immunologic mechanism.[14] The present study settles the issue. It demonstrates a positive predictive value of 92.6% and a negative predictive value of 54.6% for tryptase as an indication of an immunologically mediated event. Therefore, the presence of a normal level does not exclude an immunologic reaction; markedly elevated tryptase levels are not found in almost a third of anaphylactic cases. However, a significantly elevated tryptase level (> 25 μg/l) strongly suggests an allergic mechanism. Although the authors appropriately caution that the diagnosis of anaphylaxis should not rely on a single test, the high positive predictive value of tryptase makes it important both medicolegally and for subsequent patient management. As a practical matter, because tryptase (a large tetrameric enzyme co-released with histamine) has a half-life of several hours and is unusually stable even at room temperature, it is possible to harvest samples during or even after urgent clinical situations.[15] A small number of surgical patients will have marginally elevated tryptase levels, so it is highly desirable for clinicians to collect serial samples over several hours.[16]

Another important observation is that although it may not be possible to distinguish between anaphylactic and anaphylactoid reactions in individual patients, cardiovascular and pulmonary events are more common in immunologically mediated reactions, and cutaneous manifestations are more common in chemically mediated reactions. Thus it is not surprising that immunologically mediated reactions were identified as more severe, although death from intraoperative latex anaphylaxis remains a rare event.

The current study also provides specific guidance for clinicians in managing atopic patients. Virtually one third of the patients seen in our preoperative clinic present with some history of hay fever, rhinitis, asthma, or food or drug allergy. Clinicians have long worried whether such patients are more likely to have an anaphylactic or anaphylactoid reaction during anesthesia. Such a correlation holds true for latex allergy. A history of generalized atopy or specific allergy to certain fruits (*e.g.,* kiwi, avocado, figs) are both recognized as significant risk factors for latex reactions. However, other than for latex, a generalized history of allergy seems to be of little consequence in predisposing to anaphylactic and anaphylactoid reactions to anesthetics. Although this finding was expected,[17–19] Mertes *et al.* furnish objective evidence that a history of generalized allergy need not preclude anesthetic choices. Specifically, clinicians should not be concerned about giving a histamine-releasing drug, such as morphine, to a patient with a generalized history of allergy. On the other hand, because there is significant cross-reactivity (as high as 80%) between anesthetic agents (*e.g.*, relaxants), a patient history of specific allergy to anesthetics *is* a cause for concern. These patients merit a more thorough preoperative evaluation and possible referral to a clinical allergist for skin testing. In urgent circumstances, using an alternate anesthetic technique (*e.g.,* regional anesthesia, avoidance of relaxants) may be the best clinical option. Although pretreatment with H1 or H2 antagonists will markedly attenuate chemically mediated reactions[13] and may even reduce the severity of immunologically mediated reactions,[20] this strategy is not a substitute for a comprehensive evaluation and anesthetic plan.

Mertes *et al.* have done a great service to anesthesiologists and patients by continuing their survey and by careful analysis of the resultant data. Although life-threatening anaphylactic and anaphylactoid reactions are infrequent, they do contribute to patient morbidity and mortality. However, in many instances "allergy to anesthesia" is used as an explanation for poor outcome. In the interest of patient safety, it is important that clinicians identify those patients in whom allergy is the real cause of the event and determine which agents are responsible. The anesthesia community has done well with several other challenges to practice (*e.g.,* malignant hyperthermia, the difficult airway). It is hoped that this and other such studies will afford the basis for continued practice improvements.

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Thoracic Epidural Anesthesia

More than Just Anesthesia/Analgesia

SPLANCHNIC hypoperfusion following low systemic perfusion due to trauma, hemorrhage, or circulatory shock is thought to form part of the host response to these types of injury. At the level of the microcirculation, hypoperfusion may result either from redirection of blood flow away from the splanchnic organs, mediated by increased sympathetic activity, or from impaired blood flow distribution within the microvascular networks. Because splanchnic hypoperfusion is considered to be important in the development of increased mucosal permeability, endotoxemia, and organ failure, the adequacy of gastrointestinal perfusion has become a major concern in high-risk surgical and critically ill patients.[1] The importance of this concept is further emphasized by the observation that gastrointestinal hypoperfusion is also associated with increased mortality rates in such patients.[2,3]

In the current issue of ANESTHESIOLOGY, Adolphs *et al.* report the results of a carefully conducted experimental study on the effects of thoracic epidural block on gut microvascular blood flow in a hemorrhage model in rats.[4] The authors clearly demonstrate that thoracic epidural anesthesia (TEA) protected the gut from decreased microvascular perfusion and from increased leukocyte–endothelium interaction associated with insults due to hemorrhage/retransfusion. With regard to the effect of TEA on microvascular perfusion, most of the benefit was observed in the muscularis layer. Because sympathetic nerve fibers were detected in all layers of the gut except the mucosa, the authors argue that the favorable effects of TEA on the microvascular perfusion of the muscularis layer must be explained primarily by the effects of the sympathetic block.

One important issue in the effect of TEA on splanchnic perfusion is the location of the epidural block. A complete sympathetic block in the splanchnic region is achieved only if the spread of the local anesthetic includes the thoracic sympathetic nerve fibers, which extend from T5 to T10. On the other hand, the epidural blockade of lumbar segments results in increased sympathetic activity in the splanchnic nerves due to a baroreceptor drive.[5]

Others have performed studies in the area. Ai *et al.* measured intramucosal pH in the ileum of rabbits to determine the effects of TEA (catheter tip at T8–10) during progressive hypoxia to an inspired oxygen fraction of 0.1.[6] In their study, TEA slowed the progression of intestinal ischemia during hypoxia and conferred protection against an increase in portal endotoxin concentrations. Meissner *et al.* studied the effects of high thoracic epidural block (T1–5) on splanchnic blood flows using the microsphere technique in dogs.[7] The thoracic block did not alter blood flow to the splanchnic organs in the study, but the splanchnic sympathetic nerves were not included in the epidural block. In another study, by Sielenkämper *et al.*, intravital microscopy was used to measure gut mucosal blood flow in the ileum of rats during TEA (catheter tip at T7–9).[8] It was found that TEA increased mucosal blood flow and reduced irregular flow patterns such as stop-and-go flow in the capillary networks of the gut mucosa.

There is some supporting clinical information. In two studies, the effects of TEA in patients undergoing major abdominal surgery were determined using gastric tonometry.[9,10] Both studies found that TEA prevented a decrease in intramucosal pH during surgery; however, in one study the exact location of the epidural block was not given.[9] Mallinder *et al.* studied the effect of TEA (block T5–T11) on gastrointestinal blood flow in patients undergoing colorectal surgery.[11] These authors

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observed no beneficial effect of TEA on mucosal P_{CO_2}, intramucosal pH, or the P_{CO_2} gap. However, at all times, and independently of the type of anesthesia, parameters for mucosal perfusion were all within normal levels in most of the patients, thus suggesting that no mucosal hypoperfusion was present.[11] Two other studies reported the effects of epidural anesthesia on intramucosal gastric pH during aortic reconstruction surgery.[12,13] No beneficial effects of epidural anesthesia on intramucosal gastric pH were observed, but measurements were performed in the stomach, whereas the epidural catheters were placed at L3–4 and T9–10, respectively. Therefore, it seems that in these two studies the epidural block did not necessarily include the upper splanchnic organs in which the effect of the intervention was measured.[12,13]

In all of the experimental and clinical studies mentioned above, investigators who reported evidence of improved gastrointestinal perfusion associated with epidural anesthesia usually performed measurements within the region of the epidural block,[6,8,10] whereas those who did not observe beneficial effects most likely conducted the measurements outside this area.[7,12,13] It therefore seems that the current literature on this topic, although limited in extent, supports the view that a beneficial effect of epidural anesthesia on splanchnic blood flow can be expected only when care is taken to block the sympathetic nerve fibers that supply the splanchnic region.

Inasmuch as TEA does not increase cardiac output, the question is whether the effects of sympathetic block on splanchnic blood flow are due to a redistribution of blood flow within the splanchnic organs, or to an effect of TEA to increase the proportion of flow directed to these organs. Adolphs *et al.* observed a preferential improvement of blood flow toward the muscularis layer in their preparation. Because the gut mucosa lacked sympathetic innervation, the authors postulate that the sympathetic block was followed by a redistribution of flow within the gut wall.[4] However, no direct evidence for this assumption is provided. In contrast to the work of the current authors, other investigators have reported an improvement in mucosal perfusion, both in experimental[6,8] and clinical[9,10] studies on TEA, thus arguing against a redistribution of blood flow away from the mucosa.

In an experimental study in rabbits, the epidural blockade of splanchnic sympathetic nerve fibers increased the diameter of venous capacitance vessels in the splanchnic region, whereas a lumbar epidural block was associated with reflex vasoconstriction of splanchnic vessels.[14] Although changes in organ blood flow were not measured in absolute values, these observations suggest that a segmental epidural block will probably result in blood flow redistribution in favor of those organs within the sympathetic block and away from regions in which reflex vasoconstriction occurs.

The possibility cannot be excluded that TEA induces quite complex hemodynamic changes—for example, by affecting the blood flow distribution both within and between organs. TEA also decreases endocrine metabolic responses and reduces the extent of sympathetic activity *via* a reduction in plasma catecholamine levels.[15,16] It is possible that in addition to the segmental block of sympathetic nerve fibers, the decrease in plasma catecholamine levels attenuates a stress-related distribution of flow away from the splanchnic region. In addition, as discussed by Adolphs *et al.*, the possible effects of absorbed local anesthetic must be considered.[4] The bottom line is that, currently, in view of the few published studies on this topic, the precise mechanisms underlying the effects of TEA on splanchnic blood flow remain unclear.

Another interesting finding of the study by the present authors is the observation that TEA prevented leukocyte–endothelium interactions caused by ischemia–reperfusion injury following hemorrhage and retransfusion. Although the possibility cannot be excluded that augmented hydrostatic forces, attributable to a higher microvascular flow rate, or antiinflammatory effects of absorbed local anesthetic, are partly responsible for the reduction in leukocyte adhesion, this finding could imply that TEA inhibited the inflammatory response to hemorrhage and retransfusion because of improved blood flow and a reduced extent of ischemia.

Other authors have reported that TEA protected against bacterial translocation when progressive hypoxia was used to establish splanchnic ischemia in rabbits.[6] In critically ill patients with peritonitis, epidural analgesia using bupivacaine improved gastric mucosal perfusion and gut function in comparison with a control group of patients in whom morphine was given to provide analgesia.[17] In view of these findings, it is tempting to postulate that TEA may not only be useful to prevent gut ischemia, but it could also be beneficial to protect against ischemia- or infection-related inflammatory responses originating from the splanchnic region. It is an open question whether TEA might be capable of being used as an added therapeutic approach to prevent the exacerbation of systemic inflammatory processes—for example, in sepsis or acute pancreatitis. In both sepsis and acute pancreatitis, gut mucosal hypoperfusion is a typical finding and is regarded as being important in the development of multiorgan failure.[18,19] Mesenteric vasodilation using TEA could be useful for preventing tissue injury, especially in conditions in which catecholamine therapy aggravates vasoconstriction in the splanchnic region.[20]

When discussing the effects of TEA on sympathetic activity, it must be emphasized that the effects of the sympathetic block are, of course, not restricted to the splanchnic region. Another important effect of TEA is the reduction of efferent sympathetic outflow to the heart—a factor claimed to be responsible for a reduction

in the incidence of myocardial infarction in patients anesthetized using TEA.[21]

In summary, the study by Adolphs *et al.* is one of the first to have shown evidence that segmental epidural blockade may be useful in providing protection against splanchnic hypoperfusion under the conditions of ischemia and reperfusion. The exact mechanisms underlying this protection, and the potential therapeutic uses of TEA beyond its use as an anesthetic or analgesic technique, are matters for further investigation.

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