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PCA Is Effective for Older Patients—But Are There Limits?

PATIENT-CONTROLLED analgesia (PCA) has proven to be an important concept and therapeutic tool in the quest to improve acute pain management. The pharmacologic and nonpharmacologic benefits of PCA have been studied extensively and discussed widely. Despite these benefits, some acute pain therapists are reluctant to offer PCA to older patients, having seen that some members of this group are unwilling or unable to use PCA effectively. Explanations for this failure of therapy have included lack of understanding of the technique by older patients, different attitudes among older patients with regard to pain relief, and patient roles and fears of complications associated with analgesics or PCA equipment. The possibility of differences in pain perception or pain reporting with advancing age has also been considered. In this issue of ANESTHESIOLOGY, Gagliese *et al.*[1] have provided further insight into the influence of patient age on PCA therapy. The authors have systematically compared postoperative PCA use in two adult populations with mean ages of 39 and 67 yr. In addition to the anticipated findings related to opioid consumption and pain scores, this work includes evaluation of the effects of age on preoperative psychologic factors, concerns regarding PCA therapy, and treatment satisfaction.

Gagliese *et al.*[1] observed that, on the first postoperative day, PCA morphine consumption averaged 66.6 mg in the younger group, compared with 39.1 mg in the older group (see table 5). These values are remarkably similar to the morphine requirements predicted by Macintyre and Jarvis in 1996.[2] Those authors recommended the following formula for estimating average morphine requirements based on patient age:

$$\text{Average postoperative 24-h morphine use (mg)} = 100 - \text{age (years)}$$

In the current study, it was further shown that older patients did not self-administer less opioid than did younger patients on the basis of their concerns about pain relief, adverse drug effects (including opioid addiction), or PCA equipment use or malfunction. In fact, with the lower doses the older patients chose to use, they reported levels of analgesia at rest and with movement that were similar to those of their younger counterparts. By contrast, lower pain scores after surgery in older patients has also been reported.[3] Another finding in the current study was that older patients preferred less information about and less direct involvement in their health care, but, compared with the younger group, they had similar attitudes toward PCA, similar confidence in their ability to use it successfully, and similar satisfaction with the technique.

Overall success in using PCA is a function of the inherent benefits of the technique, in combination with the expertise and knowledge of the supervising therapists. It can be argued that, because older patients tend to be medically more complex and more vulnerable to complications, they may benefit more from such expertise. All patients in this study received medical supervision from an anesthesiology-based acute pain service. One wonders whether outcomes would have differed more in the two study groups if they had less expert medical and nursing supervision.

It should be remembered that all patients in this study were screened and selected on the basis of absence of confusion and an ability to understand and participate in their own care. It would be interesting to know how many octogenarians were excluded from the study because they did not meet those criteria.

Finally, as the authors have emphasized, this study compared groups with average ages of 39 and 67 yr. Although it is reassuring to see that the older group was as successful as the younger group in effectively using

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PCA, we must await further studies to learn how effective PCA would be in a group with an average age of 80 yr or older. To this reader, the age of 67 does not seem nearly as "old" as it once did.

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Infant Cardiac Surgery

Keeping a Cool Head

DURING the past 15 yr, the survival rate of children born with congenital heart disease has improved dramatically. With the improved survival rate, attention is being directed to morbidity, especially neurologic impairment, which affects 5–25% of the survivors.[1] As with the survival rate, improvements in neurologic outcome probably will result from many factors. The observations in the article by Bissonnette *et al.*[2] in this issue of ANESTHESIOLOGY have the potential to make an impact on neurologic outcome.

Neurologic impairment in congenital heart disease clearly is multifactorial in origin. It may originate before, during, or after surgery, as a result of genetic defects or hypoxia–ischemia. The brain contains several types of cells that vary in sensitivity to hypoxic–ischemic death. In the immature brain, neurons and oligodendricytes in the neocortex, hippocampus, and striatum are most vulnerable.[3]

Ischemia and reperfusion injure these cells through several biochemical reactions. An example is glutamate binding to the *N*-methyl-D-aspartate receptor, which increases the amount of intracellular calcium and subsequently activates proteases, phospholipases, DNAases, free radical generation, and so forth. These reactions, in turn, injure the organelles. At this point, the injured cells are dysfunctional but not yet dead. Organelle injury subsequently triggers a host of biochemical and genetic reactions, which ultimately decides their fate: death or recovery. With recovery, organelle repair occurs and neurologic function returns to normal. Cell death may occur as a result of apoptosis or necrosis.[4] In apoptosis, cell death is orchestrated, involving the activation of specific genes and enzymes through which cells neatly break up into membrane-packaged bits for removal by resident macrophages. Cell death by necrosis, in contrast, is uncontrolled, involving catalysis and membrane rupture, spilling cellular contents that cause inflammation and secondary injury. Whether a cell undergoes apoptosis or necrosis after ischemia depends on many factors. For example, mild to moderate ischemia tends to cause apoptosis, whereas severe ischemia causes necrosis.

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In the brain, the amount of time from hypoxic–ischemic injury to death ranges from days to weeks.[4,5] This gives ample time to initiate neuroprotective therapies but also necessitates that they continue well after the ischemic injury. Hypothermia is the only proven neuroprotective therapy for infant heart surgery. Hypothermia confers neurologic protection in large part because it slows the activity of the enzymes and receptors involved in the biochemical reactions that injure the cell.[6] Although the effect of a few degrees of hypothermia on a single enzyme or receptor is minimal, the cumulative inhibition on the many enzymes and receptors involved with cell injury yields a profound effect. Conversely, a few degrees of hyperthermia can markedly increase hypoxic–ischemic cell death.[7]

Many reports have shown that neurologic impairment can occur during infant heart surgery as a result of ischemia related to cardiopulmonary bypass (CPB) and total circulatory arrest. However, in clinical studies, the correlation between neurologic impairment and circulatory arrest duration is weak, and, in animal studies, neurologic impairment does not occur until circulatory arrest is prolonged ($>$ 60 min) outside the duration of customary clinical practice.[1,3] These observations point to the importance of factors other than CPB and circulatory arrest in neurologic impairment, suggesting a role for the vulnerable early postoperative period, in which hemodynamics tend to deteriorate, as well.

Although hypothermia represents the mainstay of cerebral protection during infant cardiac surgery, brain temperature is not measured but inferred from other core body temperatures, typically from the nasopharynx, rectum, and esophagus. Normally, brain temperature is 0.5–1°C more than core temperature, and, within the brain, the temperature of deep regions (*e.g.*, the caudate) is 0.5–1°C more than that of superficial regions (*e.g.*, the neocortex).[8,9] These temperature differentials increase by several degrees during CPB cooling— up to 5°C in some subjects.[9,10] Because brain temperature differences of 3–4°C influence neurologic outcome after ischemia, it is hypothesized that subjects with large temperature differences are at greatest risk of neurologic impairment, either from insufficient brain cooling or from excessive brain rewarming. To test this hypothesis, a clinically practical method to measure brain temperature is needed, as well as a description of brain temperature postoperatively, when cellular ischemic injury is evolving and treatable.

Bissonnette *et al.*[2] have provided this needed information. These authors threaded a thermocouple through a catheter, for which the tip was located in the jugular bulb, in children undergoing cardiac surgery using hypothermic CPB. They recorded jugular bulb venous temperature and core body temperatures during surgery and for 6 h postoperatively. They observed jugular bulb venous temperature at the end of CPB as 36.9 $\pm$ 1.4°C, increasing at 6 h postoperatively to 39.6 $\pm$ 0.8°C. Although the study was ended after 6 h, jugular bulb venous temperature in all subjects had not reached a plateau, indicating the possibility of higher cerebral hyperthermia. The jugular bulb venous temperature was similar to core temperatures at the end of CPB and increased after surgery 2 or 3°C more than core temperatures. These observations suggest that cerebral hyperthermia of sufficient magnitude and duration to influence outcome after ischemic injury develops regularly after infant cardiac surgery. Hemodynamic deterioration also occurs at 6 h postoperatively, rendering the injured brain cells vulnerable to further ischemic injury.

This observation raises many questions. First, does jugular bulb venous temperature reflect brain temperature? No study directly addresses this. However, the authors make a strong case that, if anything, it would underestimate brain tissue temperature. Second, did the authors' perioperative temperature management contribute to the development of cerebral hyperthermia? At their institution, management revolved around rectal temperature. Some institutions use this strategy, whereas others measure temperatures from a combination of sites because rectal temperature lags behind these temperature sites, rendering its estimation of core temperature inaccurately low. Thus, the use of rectal temperature alone to guide rewarming may predispose patients to hyperthermia. Yet, in the study by Bissonnette *et al.*,[2] hyperthermia did not develop at the core sites during CPB rewarming, but rather hours afterward, despite surface cooling in those patients with core hyperthermia, making this possibility unlikely.

The observations of Bissonnette *et al.*[2] will hopefully spur clinical studies to address the role of brain temperature and postoperative care in neurologic impairment after infant heart surgery.

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