Is the Administration of Ketorolac Associated with Preemptive Analgesia?

To the Editor:—Norman et al.1 presented evidence supporting the concept of possible preemptive analgesic effects based on differences in VAS pain at 2 and 4 h after tourniquet inflation in patients receiving the same dose of ketorolac intravenously before and immediately after tourniquet inflation. Their experimental design was carefully conceived, randomized and placebo controlled to minimize the difference between the prestimulus and poststimulus ketorolac plasma concentration. Although they did not measure ketorolac plasma concentration, they probably achieved their goal.

Katz and Siffert2 demonstrated that the concentration of antibiotics under the tourniquet was lower when injected intravenously immediately after tourniquet inflation than before inflation. These findings have dictated the way antibiotics are administered in orthopedics. Indeed, to maximize the concentration at the effector site, antibiotics are injected, preferably before tourniquet inflation, or if this is not done, after tourniquet deflation but not immediately after tourniquet inflation.

The effector site of nonsteroidal antiinflammatory drugs, including ketorolac as one of the antibiotics, is the surgical site. Therefore, Katz and Siffert2 finding suggests that tissue concentrations of ketorolac below the tourniquet would be significantly lower when the drug is intravenously administered after tourniquet inflation. In these conditions, the difference in VAS pain reported by Norman et al.1 may have reflected the difference in ketorolac concentrations at the effector site, rather than evidence of the preemptive properties of nonsteroidal antiinflammatory drugs. Indeed, VAS pain was higher in patients who received ketorolac immediately after tourniquet inflation, as compared with those who received ketorolac before tourniquet inflation.

Although, like the authors, we intuitively believe that nonsteroidal antiinflammatory drugs have preemptive analgesic effects, Norman et al. may want to modulate their conclusions.1

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References


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the target limb in any significant amount. This was a major point of our study design. To quote, “because ketorolac exerts its analgesic effects primarily at the peripheral level, the latter dose would function as a poststimulus dose, since it would not reach the site of action until after the tourniquet was deflated at the end of surgery” (emphasis added).

The tourniquets in Katz and Siffert’s study were never deflated because this study was undertaken to determine if cefazolin administered after tourniquet inflation reached the tissues in significant amounts. This study did not examine tissue drug levels after tourniquet deflation, hence we see no need to “modulate” our conclusions.

Drs. Manoir and Fletcher suggest that our results cannot be attributed to preemptive analgesia because they are not long-lived. We disagree. The definition of preemptive analgesia is the phenomenon by which analgesia administered before a painful stimulus decreases the intensity of the subsequent pain. There is no requirement that these effects must persist for a certain period of time. Our paper presents several possible explanations for the relatively short duration of the observed preemptive analgesic effects. Particularly important are the effects of ongoing tissue inflammation caused by surgical trauma. This inflammation, like repeat injury, will obscure preemptive effects, as discussed by Wooll and Chong. Optimal preemptive analgesia may necessitate continuing therapy into the postoperative period.

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References

To the Editor:—I read with great interest the article by Drs. Liu and McDonald published in the May 2001 issue of ANESTHESIOLOGY. They report that the incidence of cardiac arrest during spinal anesthesia is from 1/10,000 to 1/250,000 cases. Their own references do not support these incidences. They reference Auroy et al. who reported 26 cardiac arrests in a prospective study of 40,640 spinal anesthetics for an incidence of 1/16000 cases. The other reference cited is also problematic because it was a study of insurance claims for damages following anesthesia. Since only a small fraction of adverse events lead to claims for damages, this greatly underestimates the true incidence of these arrests. Other studies confirm a cardiac arrest rate during spinal anesthesia of close to 1/1000 cases.

Having more accurate information about the incidence of cardiac arrest during spinal anesthesia is important because it makes it clear that this problem is common enough to warrant more attention. Further proof of the immediacy of this problem comes from the observation that there are now more claims in the American Society of Anesthesiologists (ASA) Closed Claims Database for cardiac arrest during spinal or epidural anesthesia (170 cases), than there are for aspiration related injury. The severity of injury has remained high with death or brain damage in approximately 90% of these claims.

It is important to emphasize that the worst outcomes may be avoided if patients receive appropriate treatment. Often this treatment should include use of a strong vagolytic agent. Liu and McDonald acknowledge that there can be a shift in cardiac autonomic balance toward the parasympathetic system related to a sudden decrease in volume. They also allude to increases in baroreflex activity but there is no mention of the potential benefits of vagolytic therapy during spinal anesthesia. The first two prophylactic measures that they discussed can help decrease vagal tone partially, but may be inadequate by when used alone. Gratadour et al. reported that neither volume loading nor infusion of a mixed α- and β-agonist during spinal anesthesia was sufficient to prevent three study patients from experiencing bradycardia and hypotension associated with an increase in baroreflex activity. To help prevent an overwhelming vagal response, prophylactic treatment with atropine should also be considered for patients at risk for severe bradycardia and cardiac arrest during spinal anesthesia. With the popularity of spinal anesthesia and the reported frequency of these arrests, increased use of all three of these interventions to decrease vagal tone could enhance the safety of spinal anesthesia.

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References

To the Editor:—In my years as an editor of ANESTHESIOLOGY and a reviewer for numerous journals, I frequently encountered submissions in which figures contained fitted curves (linear regression or other nonlinear functions) extrapolated beyond the range of measured X
values. Authors were often resistant to changing these figures to limit
their predictions to the observed range of X values. The folly of
extrapolating beyond the observed range is seen in figure 4 in a recent
review article in Anesthesiology. The fitted function indicates that a
decrease in blood volume increases cardiac output, certainly an unex-
pected and unphysiologic finding (and not supported by the data).
Fortunately, the authors limited their extrapolation; had they extended
their curve further, it would suggest that profound hypovolemia was
desirable, certainly not the authors’ intention.

In Reply.— We appreciate the comments by Drs. Pollard and Fisher.
Dr. Pollard points out the wide variability in estimated incidence of
cardiac arrest in spinal anesthesia. Depending on the data examined,
interpretation of data, and definition of cardiac arrest, the incidence
could certainly be as common as 1:1,000. We agree with Dr. Pollard
that clinicians must maintain vigilance during spinal anesthesia. Dr.
Pollard also recommends prophylactic treatment with atropine to
reduce sudden baroreflex activity and prevent cardiac arrest in high-
risk patients. Although a reasonable suggestion, this intervention has
not been definitively studied, and efficacy is unclear. In fact, the cited
reference (8) concluded that it was very difficult to prospectively
identify patients at risk for sudden increase in baroreflex activity
associated with sudden bradycardia and hypotension.

Dr. Fisher’s comments on data presentation are appreciated. The
figure was reproduced with permission from a previous publication in
Anesthesiology by other investigators. Because we were not involved
in the statistical analysis and editorial process for the original publica-
tion, we have no additional insight to offer.

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(Accepted for publication September 10, 2001).

To the Editor—Drs. Heier et al. are to be congratulated on a coura-
geous and convincing study. Their conclusion that “spontaneous
recovery from succinylcholine-induced apnea may not occur suf-
ciently quickly to prevent hemoglobin desaturation in subjects whose
ventilation is not assisted” is well taken. I was somewhat disappointed,
however, that the authors did not take the next logical step in the
discussion of their findings. They state, “a smaller dose of succiny-
choline would have decreased the duration of muscle paralysis but the
results would have been less clinically relevant.” I think it is time to
question the correctness of the assumption that the intubating dose of
succinylcholine must be 1.0 mg/kg. I was taught as a resident back in
the early 1960s that if you never exceeded a dose of 40 mg of
succinylcholine (to an adult) that most patients would likely survive
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succinylcholine (to an adult) that most patients would likely survive
the experience. In the subsequent four decades I have rarely found it
necessary to exceed a dose of 0.50 to 0.60 mg/kg for routine
intubations.

A few facts are worth reviewing:

- The ED_{95} of succinylcholine is less than 0.30 mg/kg. Thus,1
  1.0 mg/kg of succinylcholine represents between 3.5 and 4.0 times
  the drug’s ED_{95}.
- Even when administered in subparalyzing doses, 90% of the blocking
effect of succinylcholine (at the adductor pollicis) is still evident
within 75 s.2 The time to peak effect at sites more relevant to the
adequacy of conditions for intubation such as the masseter, dia-
phragm, and laryngeal adductors is even more rapid.
- As the intrinsic speed of onset of a neuromuscular blocker becomes
  more rapid, smaller multiples of the ED_{95} are required to assure
timely ease of intubation. The usually recommended “intubating
dose” of cisatracurium (a slow onset drug) is 0.15 to 0.20 mg/kg
(3–4×ED95). The commonly cited intubation-dose of 0.60 mg/kg for
rocuronium represents less than 2×ED95, yet the drug’s onset profile
is slower that that of succinylcholine.

The Intubating Dose of Succinylcholine

To the Editor—Drs. Heier et al. are to be congratulated on a coura-
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rocuronium represents less than 2×ED95, yet the drug’s onset profile
is slower that that of succinylcholine.
To the Editor:—We read with interest the article by Heier et al. entitled ‘Hemoglobin Desaturation After Succinylcholine-induced Apnea.”1 In our practice, we are often called on to perform emergency airway management, and after reading this article, it appears that the safety of rapid sequence induction (RSI) has been called into question. The study by Heier et al.; demonstrated that there is a potential risk of desaturation with the RSI sequence. We believe, however, that the methodology used by the authors does not permit formulation of a definitive answer in this regard. In the authors’ study, the facemask was removed after induction and remained off throughout the period of apnea. In emergency airway management, when difficult intubation occurs, the bag mask must be applied against the face of the patient with continuous oxygen infusion, but without active ventilation, until oxygen saturation is greater than 90%. Several studies have demonstrated that oxygenation may be maintained for more than 30 min with passive oxygenation in the absence of active ventilation.2 The true clinical relevance of the author’s study is apparently in the situation of complete airway obstruction or in the “cannot intubate, cannot ventilate” situation. However, this latter situation is very rare (about 0.01–0.2 per 10,000).3 In short, we believe that the degree of hemoglobin desaturation during apnea from the RSI procedure may be more appropriately assessed with a bag-mask connected to a high-flow oxygen source applied to the face of the patient. The RSI technique was designed to reduce pulmonary aspiration, and, to our knowledge, the combination of a rapid neuromuscular blocker (succinylcholine) and a hypoxic remains the anesthetic procedure of choice in the case of emergency invasive airway management.4

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In Reply.—We appreciate the responses by Dr. Kopman and by Dr. Adnet et al. to our article.1 Dr. Kopman raised the pertinent clinical question about what is the correct dose of succinylcholine necessary to facilitate tracheal intubation. We used 1 mg/kg of succinylcholine because that is the standard recommended dose.5 We did not devote much discussion to the question of dose because it was not a focus of our study. Dr. Kopman argues that smaller doses than 1 mg/kg are adequate for intubation because the ID 95 of succinylcholine is less than 0.5 mg/kg.5 This estimate of potency was obtained using the dorsal interosseous muscle of the hand, a muscle which is not as relevant to quality of tracheal intubation as are the laryngeal adductor muscles. In particular, the duration of action of succinylcholine at the laryngeal adductor muscles is several minutes shorter than that at the intrinsic muscles of the hand.6 As a consequence, with small doses of succinylcholine recovery of the laryngeal adductors may occur so rapidly that the “window of opportunity” for tracheal intubation is very short. Consequently, delaying intubation may, in fact, lead to a diminished, not increased, chance of successful intubation. This may explain why a dose of 0.5 mg/kg results in worse conditions for intubation than a dose of 1.5 mg/kg.7 Dr. Kopman eloquently identifies the need for a definitive study on the relationship of dose of succinylcholine to timing and quality of tracheal intubation; we look forward to the results of such a study.

Regarding the comments by Dr. Adnet et al. on rapid-sequence intubation (RSI), our study did not address RSI nor call its safety into question. Our intent was solely to investigate the validity of a clinical belief, namely that recovery from succinylcholine occurs sufficiently rapidly to permit a margin of safety in airway management. We sought to replicate conditions in the “can’t intubate and can’t ventilate” situation, and while the incidence of this problem is low, the potential for a catastrophic outcome elevates its importance. In this situation, passive transfer of oxygen into the lungs will not take place, therefore, to approximate this circumstance we removed the oxygen mask from the subject’s face. The argument by Adnet et al. that in the absence of ventilation, passive oxygenation will maintain hemoglobin saturation for 30 min is misleading. In the study they cite subjects had an endotracheal tube placed, and their lungs were ventilated with 100% oxygen for 30 min before apnea was induced.8 This does not resemble in any meaningful way the clinical situation of managing a difficult airway with a face mask in a paralyzed patient whose lungs cannot be effectively ventilated. In addition, in that study, during the time of passive oxygenation PaCO₂ increased to between 130 and 250 mmHg and pH decreased to between 6.72 and 6.97.9 We believe we have provided important new information that clinicians can use in their decision-making processes, whether that is for an elective and uncumpliated or an emergent rapid-sequence intubation.

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References

(Received for publication September 14, 2001)
To the Editor—We read with great interest the article by Kazama et al. They clarified the relationship between initial blood distribution volume and propofol induction dose using continuous infusion during the induction of anesthesia. The results are consistent with those of our study, and the study protocols differed in only two parameters: the infusion rate, and its basis. Kazama et al. administered propofol at 40 mg · kg⁻¹ · h⁻¹ as a function of lean body mass (LBM), whereas we administered it at 15 mg · kg⁻¹ · h⁻¹ as a function of total body weight. Using multiple linear regression analysis, Kazama et al. report that the induction dose can be determined from four variables, age, LBM, central blood volume (CBV), and hepatic blood flow (HBF), whereas we selected age, body weight, cardiac output (CO), and the plasma disappearance rate of indocyanine green (k) as our variables.

Multiple linear regression analysis in the study of physiologic systems provides a powerful statistical tool for sorting out the relationships among several variables in such experiments. However, there are many potential pitfalls when using regression analysis, including multicollinearity, which can lead to numerical problems in estimating the parameters in regression equations. Ideally, all selected predictive variables must be uncorrelated. When multiple regression analysis was applied to our results using the variables of Kazama et al., age, LBM, CO, BV, and HBF were identified as significant factors for predicting the induction dose of propofol (P < 0.01, table 1). However, our statistical analysis (NCSS2000, Kaysville, UT) detected possible multicollinearity among the selected predictive variables.

In our results, age did not demonstrate any correlation with BV or CBV, consistent with Kazama et al., whereas age showed marked correlation with HBF, and CBV was significantly correlated with LBM and BV. Age has been reported as a predictive variable for HBF. Normalization of accurate BV measurement using LBM has been recommended in a clinical setting, because LBM is an absolute predictive factor for CBV. These relationships might become sources of multicollinearity, and ordinary least squares multiple linear regression can give nonsensical results for the estimated parameter magnitude, sign, or standard error. Hepatic blood flow is a cross-product of BV and clearance slope (K̇); thus, it might become another source of artificial multicollinearity.

The results of Kazama et al. clearly demonstrate the predictive variables for propofol induction dose. Age and other circulatory variables play an important role in determining the dose, which is consistent with our results. The interpretation of the results using multiple regression analysis (including our study), however, might leave room for attention and discretion. Although multicollinearity can be avoided

<table>
<thead>
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<th>Variable Entered in Model</th>
<th>Regression Coefficient</th>
<th>SE</th>
<th>Standardized Regression Coefficient</th>
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<td>Age (yr)</td>
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<td>Sex (O M, 1 F)</td>
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<td>LBM (kg)</td>
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<td>Hemoglobin (mg dl⁻¹)</td>
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<td>Central blood volume (l)</td>
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<tr>
<td>Adjusted R²</td>
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</tr>
</tbody>
</table>

* Not selected as a predictor variable in the multiple linear regression model using the variables of Kazama et al.

P < 0.0001.

LBM = lean body mass; SE = standard error; M = male; F = female.

with good experimental design, not all interesting questions can be studied without encountering multicollinearity.

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References


(Accepted for publication September 24, 2001.)
Sheath Placement over Transesophageal Probes: A Description of a “Self-Moving” Method

To the Editor—The use of transesophageal echocardiography (TEE) in the operating room, both for cardiac and noncardiac surgery, is increasing. In our hospital, we use disposable sheaths over the TEE-probe, to prevent nosocomial infections.1,2 Placing a Latex sheath over the TEE-probe in the operating room is not easy; it is time-consuming and the limited space can necessitate bending of the probe, which may decrease its longevity. Polyurethane sheaths are even more difficult to place over the probe. Potentially life-threatening allergic reactions to Latex have forced us to consider the use of polyurethane sheaths.3,4

In order to maximize the ease of sheath placement, we have designed a vacuum tube, mounted on the echo-machine. This consists of two Perspex or Polycarbonate tubes (Polycarbonate exhibits extremely high-impact strength); a smaller tube within a larger tube (Fig. 1). Holes are placed in the inner tube and vacuum is applied between the inner and outer tubes. The exact placement of the holes in the inner tube is crucial; they have been placed, after trial and error, at the section of the inner tube that coincides with the distal 60–80% of the probe, and also at the bottom of the inner tube. After placing the sheath and applying negative pressure, the sheath opens against the wall of the inner tube; starting proximally and extending distally. Once the distal part of the inner tube has been reached, the sheath closes the openings in the inner tube, and the suction force from the holes in the bottom of the inner tube now extends its length (the length of the probe extends a few cm from the last distal opening in the tube). Ultrasound gel is placed on the tip of the probe so that it can be effortlessly placed in the stretched sheath. After removing the negative pressure, the distal part of the sheath retracts first (the proximal side-holes are still occluded by the sheath), thereby fitting tightly around the tip of the probe. The remaining negative pressure is then reduced so that the rest of the sheath retracts around the shaft of the probe. Occasionally, minimal manipulation of the tip of the sheathed probe is needed in order to remove air (the fit of the polyurethane sheath is extremely tight and, in fact, much better). This procedure is quick, easy, and clean, and requires minimal manipulation of the probe. Readers may be interested in this “self-moving” method, which can quite easily be locally manufactured.

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Support was provided solely from institutional sources.

References


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Malfunction of Syringe Pump Caused by Fluid Infiltration

To the Editor—The breakdown or dysfunction of syringe pumps can present a serious threat to patient safety when they are used to administer high concentrations of narcotic, vasopressor, or depressor drugs. I report an experience in which the power supply circuit and syringe size sensory were damaged—apparently a result of leakage of intravenous infusion fluids into the housing of the pump.

My institute has had 19 anesthesia syringe pumps (Graseby 3500; Graseby Medical LTD, Watford, Hertfordshire, UK) in service for 4 yr or more, and these have been used exclusively for continuous infusion of Propofol. In recent periodic checks of all units, corrosion in the syringe size sensory springs was found in three syringe pumps, and damage to the power supply by short circuit was found in one. Figure 1 shows a corroded syringe size sensory spring. Because residual liquid or white crystal was found on the bottom of the housing between the syringe size sensor and the power supply circuit board, it is evident that intravenous infusion fluids had infiltrated into the housing from the narrow space between the main body and the syringe plunger, thus causing both corrosion of the spring and a short circuit of the power supply. Infusion fluid occasionally falls onto the syringe pumps because we usually attach them to the infusion stand during surgery. So, this kind of medical instrument should be waterproof by design. The instruction manual for this syringe pump does state in the Specifications section that its electrical safety category is “drip proof IPX1,” which guarantees protection against a 10-min exposure to artificial rain of 3–5 mm/min from a 50-cm height. Therefore, it is anomalous that dripping intravenous infusion fluids should result in the observed problems. Although short circuit of the power supply simply results in infusion interruption, corrosion in the syringe size sensory spring is a severe problem, because it may cause incorrect recognition of syringe size, resulting in an unpredictable drug infusion rate.

I suggest that the syringe size sensory spring be made from a material that is not corroded by the electrolytes included in the infusion fluids.

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References


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Fig. 1. The damaged spring of the syringe size sensor is shown. The hook was corroded, and the connection with the case has come off.

and that the seal between the main body and the syringe plunger be improved. In this case, the Japanese sales agent was unable to distinguish between the possibilities of a structural defect and an assembly problem after checking the faulty syringe pumps. To prevent recurrence of these problems, Graseby Medical and the Japanese sales agent suggested I install a syringe cover normally used with another of Graseby’s products with patient-controlled analgesia function that has a common housing. I accepted this suggestion at the time, but it is notable that such a modification potentially constitutes a warranty violation and exposure to legal problems. At any rate, the improved water resistance is expected to prevent this type of accident in the future.

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References


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