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In Reply:—We have been using continuous low-dose epidural morphine infusion for treatment of postoperative pain since 1983. With this technique, due to the lack of sedation, some patients require additional low doses of tranquilizers to fall asleep at night.[1,2] Even with an increased epidural morphine dose, sedation could not be achieved.[3] For this reason, the mild degree of sedation with epidural fentanyl or alfentanil infusion has proved desirable for the patients.[4] On a scale of 0–4 (0 = no sedation, 1 = mild sedation, 2 = moderate sedation, 3 = severe sedation, asleep but arousable, 4 = asleep, not arousable), none of these patients had a score exceeding 1. They have been cooperative throughout the treatment, showing no signs of any tension. A greater degree of sedation would certainly not be of advantage for the reasons Bledsoe and Ready state in their letter.

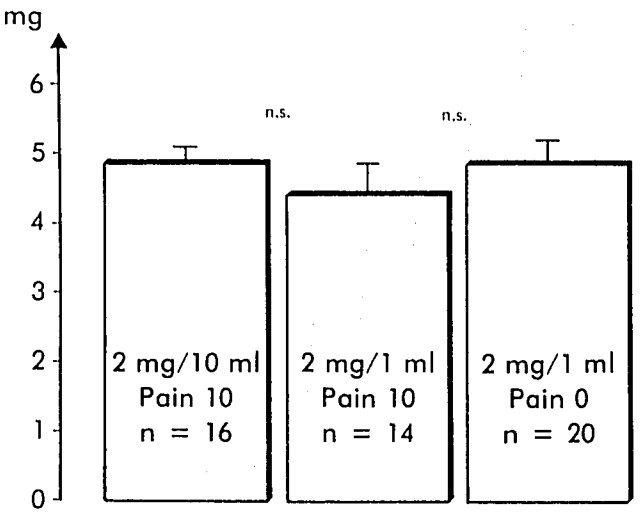

FIG. 1. Mean morphine usage (−SEM) until 8 A.M. on the first postoperative day using continuous, on-demand epidural infusion of morphine following an initial bolus epidural injection of 2 mg morphine in 10 ml or 1 ml saline when the patients complained of severe pain, or of 2 mg morphine in 1 ml saline immediately after the operation.

In a randomized single blind study, we established that there was no significant difference in the morphine usage up to 8 A.M. on the first postoperative day regardless of whether the pain treatment commenced immediately after the operation, or whether we waited until the patients complained of severe pain. Nor was there a difference when the initial bolus of 2 mg morphine was administered in a volume of 10 ml or a volume of 1 ml prior to continuous on-demand epidural infusion of morphine for continuous pain relief (fig. 1).[5] We therefore agree that postoperative treatment should commence as early as possible to spare the patients pain. However, the circumstances in our hospital make it difficult to initiate postoperative pain treatment in the operating theater. For this reason, it might be desirable to begin with a faster-acting analgesic when the patients arrive on the ward and complain of severe pain.

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Underdosage with Succinylcholine May Lead to Incorrect Diagnosis of Masseter Spasm in Children

To the Editor:—In their paper on the effects of succinylcholine on mouth opening in children, Van der Spek *et al.*[1] showed that there was an increase in tone in the jaw muscles following succinylcholine at a time when the twitch response in muscles of the upper limb was absent. They suggested that this indicated a fundamental difference in response to succinylcholine between jaw muscles and limb muscles, but as tension measurements were performed only on jaw muscles this conclusion is invalid. Data from our recent study on the actions of succinylcholine in pediatric patients provide evidence of a corresponding increase in tone occurring in muscles of the thumb.[2]

Figure 1 shows a typical train-of-four tension recording obtained from muscles of the thenar eminence in an infant during succinylcholine

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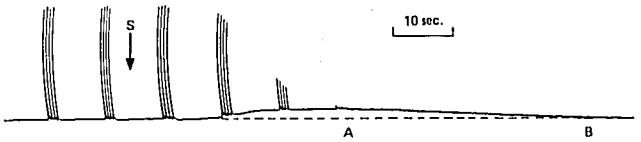

FIG. 1. Train-of-four tension recording obtained from the adductor pollicis muscle of the thumb of an infant, during administration of 2 mg/kg of succinylcholine. The broken line indicates control muscle tension. Injection of succinylcholine (S) was followed by an increase in muscle tone, indicated by elevation of the baseline, which persisted after abolition of the train-of-four response (*A*) for 30–40 s (*B*).

administration. It will be seen that following succinylcholine 2 mg/kg there was an increase in muscle tone, indicated by elevation of the baseline, which persisted for 30–40 s after the abolition of the train-of-four response. This transient increase in muscle tension is presumably due to the agonal effects of succinylcholine.

If the purpose of Van der Spek *et al.* was to explain the unduly high rate of diagnosis of masseter spasm in children,[3] it is arguable that their most important finding was that 1.5 mg/kg of succinylcholine failed to achieve 100% suppression of twitch in three children. These observations are in agreement with the results of our study,[2] which suggests that children require at least 2 mg/kg and infants 3–4 mg/kg of succinylcholine to produce clinical effects comparable to those obtained in adults. Since these doses are approximately double those currently used in pediatric patients,[4] it seems likely that many of the difficulties experienced while intubating children with succinylcholine are the result of inadequate neuromuscular blockade.

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In Reply:—Masseter spasm or masseter muscle rigidity has been suggested to be the result of inadequate dosage of succinylcholine.[1] An adequate dose of succinylcholine for intubation means the interruption of neuromuscular transmission as evidenced by cessation of neurally evoked muscle twitches. Thus, one criterion advanced for the diagnosis of masseter spasm is the inability to open the mouth or difficulty intubating the trachea, although the neurally evoked muscle twitch is completely ablated.[1] In our study, however, an increased resistance to mouth opening occurred in all patients in whom the twitch was ablated.[2] In three patients, the twitch was greatly diminished yet not fully ablated after 1.5 mg/kg of intravenous (iv) succinylcholine. Resistance to mouth opening in these patients was also increased, but in only one of these three patients was intubation hindered by the reduced mouth opening. The tracheas of an additional two patients were difficult to intubate, despite complete abolition of the twitch response. These patients were "adequately dosed," fulfilling one criterion for "masseter spasm."[1]

In their abstract, Meakin et al. determined the ED_{95} for the suppression of the adductor pollicis force developed upon supramaximal ulnar nerve stimulation to be 0.423 mg/kg (no standard deviation given) of iv succinylcholine in children, age 1–5 yr.[3] Thus, our dose of 1.5 mg/kg, administered iv in the two 2-yr-old children and one 16-yr-old child who did not fully lose their muscle twitch, was at least three times the ED_{95} for twitch suppression,[2] and should have been sufficient to abolish the indirectly evoked adductor pollicis twitch, even by Dr. Meakin's (and others') standards. Indeed, we think that *neuromuscular transmission* in our study was completely inhibited in all patients, even in those in whom the twitch was greatly diminished but not completely lost. The twitching of the fingers observed was most likely due to improper stimulation technique rather than an inadequate dose of succinylcholine. The resulting direct muscle stimulation was probably responsible for the minimal twitch. In subsequent study, care was taken to avoid direct muscle stimulation, and the adductor pollicis twitch was fully ablated after 1.5 mg/kg iv succinylcholine in all patients (age 2–13 yr).[4] This was also the case in two subjects whose tracheas were difficult to intubate due to an increased resistance to mouth opening.

Quite unlike the 30–40 s of baseline tension increase observed by Dr. Meakin, our subsequent study showed an increased resistance to mouth opening in six of our patients for as long as 10 min.[4] It should be pointed out, however, that the isometric tension measurement provided by Dr. Meakin does not equate with measurements of joint stiffness. Such measurements in the hand (during anesthesia) have yet to be carried out, although clinically an increase in joint stiffness has not usually been apparent beyond the period of twitch ablation and fasciculation. The elucidation of the mechanism(s) responsible for these observations may help to establish the true significance of "masseter spasm." We hope that continued debate on the merit of these studies promotes and encourages further research to establish the mechanism(s) involved. We thank Dr. Meakin for his interest.

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