form) was designed to compare the effects of muscle relaxants on the muscles of the airway to those of ventilation.* The latter study showed that resistance to breathing doubled if paralysis was sufficient to cause a 40% decrease in inspiratory pressure (and no decrease in vital capacity). There is no mention of complete obstruction. Hence, since a doubling of airway resistance can easily be managed by normal subjects, we know only that the upper airway muscles are presumably weakened, but we lack information regarding the point at which they become incompetent relative to ventilation.

Dr. Knill is correct when he states curare and its descendents are used differently than was the case for attenuating side effects of induced seizures. Patients are anesthetized and their tracheas often intubated before nondepolarizing muscle relaxants are administered. Thus, spontaneous ventilation may be present (and be adequate) while the intubated airway is still secure. Our data[1] allow the prediction of postextubation airway muscle strength based on maximum inspiratory pressure (MIP) and the ability to lift head and legs. Hopefully, this will decrease incidents of upper airway obstruction due to residual paralysis.

Although Dr. Knill alludes to the danger of postoperative aspiration, he cites no observation on the relative sensitivity to nondepolarizing muscle relaxants of muscles involved in swallowing and in glottic closure. Indeed, we are unaware of any, other than our own. Our study[1] clearly shows the inability to close the glottis and to swallow (thus removing oropharyngeal contents) at levels of paralysis at which muscles of ventilation are more than adequately recovered. It was clear to us that observations of the glottic elevation during attempted swallowing was not a sign that actual swallowing occurred. In most subjects, inability to swallow occurred at levels of paralysis before airway obstruction was experienced.

EDWARD G. PAVLIN, M.D.
Associate Professor of Anesthesiology
University of Washington
Seattle, Washington 98104

* Dodgson BG, Knill RL, Clement JL: Curare increases upper airway resistance while reducing ventilatory muscle strength (abstract). Can Anaesth Soc J 28:505–506, 1981.

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Visual Analogue Pain Scale with Convenient Digitizer

To the Editor:—Since the introduction of the visual analogue pain scale by Huskisson,[1,2] its reliability and reproducibility have been questioned.[3–5] However, when pain intensity is expressed numerically, it is important to digitize it precisely and quickly. For this purpose, we have combined a visual analogue pain scale with a numerical scale (fig. 1). The analogue scale on one side is for use by the patient. The reverse side contains the simultaneous digitizer.

After providing a standard explanation of the visual analogue scale, we ask the patients to estimate their experience of pain using the cursor, and we then read the corresponding number from the numerical scale on the other side. Some patients understand the concept of a visual analogue pain scale better when they see the present scale than when they are only given an explanation. An additional advantage with our scale is that we avoid the difficulties with understanding the meaning of unfamiliar foreign words stated by patients for whom Japanese is not the native language.

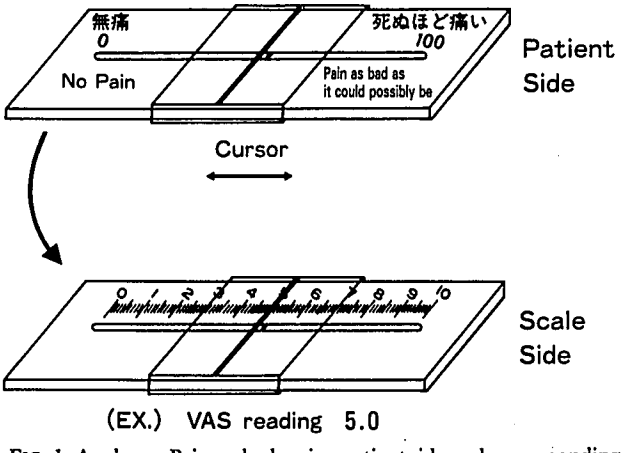

FIG. 1. Analogue Pain scale showing patient side and corresponding digital equivalent on the reverse sides.

SEIJI WATANABE, M.D., PH.D.
Anesthetist-in-Chief
KANJI KOYAMA, M.D.
Chief Resident
Deapartment of Anesthesia, Mito Saiseikai General Hospital
3–3–10, 1–choume, Futabadai, Mito, Ibaraki, 311–41,
Japan

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Editor's Comment

A recent publication by Lanier *et al.*[1] contained an unusually large number of typographical errors, the most important of which are included as errata in this issue of ANESTHESIOLOGY. It has been previously suggested[2] that three quarters of the errors that appear in print fall into the category of authors' errors and that one quarter are printers' errors. Furthermore, it was noted that printers' errors are also the responsibility of the author since they are published as a result of the author failing to meticulously correct the galley proofs.

In the case of the article by Lanier *et al.*, the authors were never offered an opportunity to review the galleys, and thus all the errors were translated directly to the Journal. This letter is meant both as a formal apology to the authors for this inexplicable oversight and as notification to our readers that the numerous and embarrassing typographical errors in this manuscript are not the result of author negligence.

LAWRENCE J. SAIDMAN, M.D.
Editor in Chief

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1. Lanier WL, Iaizzo PA, Milde JH: Cerebral function and muscle afferent activity following intravenous succinylcholine in dogs anesthetized with halothane: The effects of pretreatment with a defasciculating dose of pancuronium. ANESTHESIOLOGY 71: 87–95, 1989
2. Greene NM: Editor's Comments. Anesth Analg 63:90–91, 1984

Errata

In the May, 1989, issue a Laboratory Investigation (Serrao JM, Stubbs SC, Goodchild CS, Gent JP: Intrathecal midazolam and fentanyl in the rat: Evidence for different spinal antinociceptive effects. ANESTHESIOLOGY 70:780–786, 1989) contains an error. The dose range used 0.054–1.5 nmol for dose response curve and dose used in analgesic comparison with midazolam was 0.74 nmol and not 3.43 nmol.

In the July, 1989 issue, a Laboratory Investigation (Lanier WL, Iaizzo PA, Milde JH: Cerebral function and muscle afferent activity following intravenous succinylcholine in dogs anesthetized with halothane: The effects of pretreatment with a defasciculating dose of pancuronium. ANESTHESIOLOGY 71:87–95, 1989) contains the following errors.

On page 88, in the legend for figure 1, the second sentence should read: The raw MAA data in *this* group II dog consisted of a biphasic signal of approximately 16–24 μV amplitude superimposed upon a constant background noise signal of approximately 7 μV amplitude.

On page 90, table 2, under Treatment Groups, Group II, the dose for pancuronium is *0.01* mg/kg pretreatment.

On page 93, column 2, line 24 of the second paragraph should read: The ability of "defasciculating" doses of metocurine to prevent SCh-induced increases *in ICP* in lightly anesthetized patients . . . etc.

On page 94, in the legend for figure 6, the second to the last sentence should read: *We believe* these changes are due to cerebral stimulation from MAA increases accompanying endogenous muscle activation.