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D-Tubocurarine and Upper Airway Obstruction: A Historical Perspective

To the Editor:—A study by Pavlin *et al.* recently published in ANESTHESIOLOGY¹ shows that d-tubocurarine in humans produces marked weakness of muscles maintaining the upper airway at doses that do not seriously compromise the ventilatory muscles of the chest wall. Although not stated explicitly in the article, these findings mean that the most potent life-threatening action of d-tubocurarine is on the muscles of the upper airway. It is clear that progressive paralysis with this agent, if left unattended, would lead to respiratory failure from upper airway obstruction (or perhaps aspiration) before failure from weakness of the intercostals and diaphragm.

While not acknowledged in the report, this basic observation is not original. Indeed, it has a history dating back almost 50 yr. This history is interesting and, I believe, instructive.

Prior to its introduction into anesthesia in 1942, curare was administered by psychiatrists just before convulsive therapy in order to reduce the risk of traumatic fractures.²⁻⁵ Typically, the awake patient was "curarized" by slow iv injection until he or she was barely able to lift the head and legs. At this level of paralysis, a convulsion was induced.

The psychiatrists who promoted curare for this purpose, first A. E. Bennett and then P. T. Cash, accurately described its relative effects on the muscles of the upper airway, neck, limbs, and chest wall²⁻⁴—as recently pointed out again by Pavlin *et al.*¹ They clearly recognized that the single most important hazard was upper airway obstruction due to relaxation of the jaw muscles and/or tongue.^{3,4} They also knew that such obstruction and its sequellae could be readily prevented or counteracted by manual support of the jaw and/or insertion of an airway.^{3,4} By 1941, an estimated 30,000 psychiatric patients had been given curare alone for convulsive therapy and the two reported deaths were both attributed to "the (mis)handling of obstructive breathing."⁵

As curare was introduced into anesthesia and gradually abandoned by psychiatrists as the sole adjunct for convulsive therapy, appreciation of this important critical action seems to have waned—as far as can be inferred from anesthetic publications. Although anesthesiologists found that d-tubocurarine could abort laryngospasm and facilitate endotracheal intubation during the course of anesthesia, the fact that it could precipitate upper airway narrowing or collapse before substantial chest wall muscle weakness seems not to have been clearly recognized. (One wonders if this was because anesthesiologists, unlike psychiatrists, nearly always administered curare during general anesthesia in which case its independent effect on the airway would be difficult to differentiate). From that period until the present, this effect of d-tubocurarine on the upper airway has been observed, inferred, or experienced many times,^{6-9*} even quantified in terms of changes in upper airways resis-

tance.* Nonetheless, it does not seem to have regained the notoriety that it once had.

It is to be hoped that the report of Pavlin *et al.*¹ will lead to a clearer recognition of this most potent critical action of d-tubocurarine among anesthesiologists—a recognition long overdue. It is also to be hoped that it will direct greater attention to the functions of the upper airway when assessing recovery from nondepolarizing block, as recommended by our psychiatric colleagues many years ago.^{3,4}

* 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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In Reply:—We thank Dr. Knill for his interest in our article¹ and for his discussion of older psychiatric literature. While the clinical observations made were commendable for psychiatrists five decades ago,^{2,3} they are somewhat inadequate for modern anesthesiology. The articles mentioned cite an order of appearance of weakness of skeletal muscle with weakness of the throat and tongue appearing before that of breathing. Yet, while enhancement of breathing was affected in some patients by mandibular support, we don't know the degree of obstruction, its incidence, or its quantitative relationship to ventilation. Since

arterial blood gases were not clinically available, and the articles cited give no measurements of tidal volume or vital capacity, the adequacy of respiration in the face of curare relaxation cannot be judged. Indeed, the authors mention the occasional appearance of apnea and of the need for manual ventilation. Thus, these older observations are not helpful in determining the relative sensitivities of these different groups of muscles. Other articles have, in passing, alluded to airway obstruction on some subjects while breathing was documented as adequate.⁴ Other than our study, one other communication (published only in abstract

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 data¹ allow the prediction of post-extubation 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 study¹ clearly

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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.

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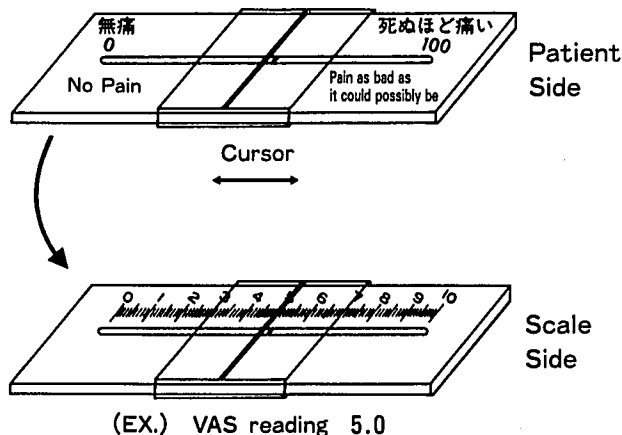


FIG. 1. Analogue Pain scale showing patient side and corresponding digital equivalent on the reverse sides.

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