D-Tubocurarine and Upper Airway Obstruction: A Historical Perspective

To the Editor—A study by Pavlin et al. recently published in Anesthesiology 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. 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 wall as recently pointed out again by Pavlin et al. They clearly recognized that the single most important hazard was upper airway obstruction due to relaxation of the jaw muscles and/or tongue. They also knew that such obstruction and its sequelae could be readily prevented or counteracted by manual support of the jaw and/or insertion of an airway. 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 mishandling of obstructive breathing."

As curare was introduced into anesthesia and gradually abandoned by psychiatrists as the sole adjunct for convulsive therapy, appreciation of this important clinical action seems to have waned—as far as can be inferred from anesthetic publications. Although anesthetists 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 anesthetists, 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, even quantified in terms of changes in upper airways resistance. 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. 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.


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REFERENCES


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