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Endotracheal tube connector fracture - an avoidable hazard

To the Editor:

I was called to see a two-year-old patient who was nasally intubated with a #4.0 mm Portex endotracheal tube. The 15 mm connector had fractured at a point where the connector narrows to a cone for insertion into the polyethylene tube (Figure). The patient required hyperventilation and was therefore receiving neuromuscular blocking drugs. Careful apposition of the broken connector ends enabled ventilation to continue prior to re-intubation of the child.

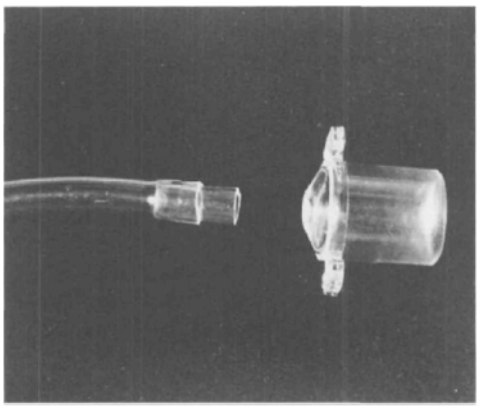

FIGURE Photograph of fractured endotracheal tube connector.

The cause of this breakage was initially a mystery as no unusual stresses were thought to have been placed on the connector. Since the patient was receiving neuromuscular blocking drugs, sudden head movement was not responsible. Careful examination of the tube revealed imprints of the jaws of forceps in the polyethylene tube. The endotracheal tube had been shortened two days earlier and forceps were used to aid the insertion of a new connector. Excessive stress at this time was presumably responsible for weakening of the connector. Minor stresses since that time had resulted in its ultimate breakage.

Defective tracheal tube connectors have been reported[1,2] but testing of a batch of these connectors revealed no weakness. Microscopic examination of the broken connector showed no evidence of moulding faults. The practice of cutting an endotracheal tube whilst the tube is *in situ* is felt therefore to be potentially hazardous. If required, re-intubation with an endotracheal tube of correct length is recommended.

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Haemodynamic reactions to succinylcholine - an alternative hypothesis

To the Editor:

I read with great interest the elegant hypothesis proposed by Nigrovic concerning the mechanisms of adverse reactions to succinylcholine (SCh).[1] The hypothesis postulates that SCh modulates norepinephrine (NE) release from the sympathetic nerve terminals by acting on the presynaptic nicotinic (N) and muscarinic (M) cholinergic receptors. Activation of the N receptors increases the release of NE, while activation of the M receptors attenuates the release. Due to the mutually opposing effects, the net response to SCh is small, variable, and of little clinical significance. The initial activation is followed by a period of desensitization. However, desensitization of the N receptors may outlast that of the M receptors leading to an unbalanced response which can result in adverse haemodynamic reactions.

Our alternative hypothesis attributes the adverse cardiovascular effects of SCh to its postsynaptic muscarinic action on the sinoatrial node (SAN), which is modulated by its nicotinic action on both the vagal and sympathetic systems.

Succinylcholine, consisting as it does of two acetylcholine molecules linked together, appears to display all the stimulant actions which acetylcholine can excercise on both the N and M cholinergic receptors.[2] Goat has confirmed the direct acetylcholine-like action of SCh on the isolated heart.[3] This effect is a muscarinic response and can be blocked by atropine. The direct effect of SCh on the postsynaptic muscarinic receptors around the SAN can be modulated by the sympathetic and vagal balance controlling the SAN. Succinycholine can activate the nicotinic cholinoceptors responsible for transmission through vagal ganglia, and hence potentiates the direct muscarinic effect of SCh on the SAN. However, SCh, similar to other nicotinic agonists can also stimulate the ganglionic sympathetic cells, as well as the nicotinic receptors on the postganglionic sympathetic terminals,[4] resulting in release of NE,[4,5] which can counteract the vagotonic and direct muscarinic effect of SCh, and may even result in tachycardia and hypertension. This release following the initial bolus of nicotinic agonists may transiently deplete the stores of NE, and hence repeating the injection of SCh within a certain period may not release NE in a concentration that can counteract its vagotonic and direct muscarinic effects. This alternative hypothesis can explain the absence of bradycardia following a single bolus of SCh, and the frequent occurrence of bradycardia following repeated doses.

In contrast to the intermittent administration of SCh, no serious haemodynamic changes are usually associated with the continuous infusion of SCh in normal patients, or with a bolus injection in patients with the atypical pseudocholinesterase. The prolonged presence of SCh may produce an initial stimulation followed by gradual desensitization of both the nicotinic and muscarinic receptors, and hence may induce a mutually opposing effect on the sympathetic and the vagal control of the heart.

In conclusion, it appears that the direct postsynaptic muscarinic action of SCh on the SAN is modulated by its nicotinic effect on the cardiac vagal/sympathetic balance. Whenever the cardiac vagal tone predominates, the injection of SCh can produce bradycardia and hypotension. In contrast, whenever the sympathetic tone predominates, SCh can produce tachycardia and hypertension.

Nigrovic suggested in his hypothesis that "clinical situations most frequently associated with early haemodynamic crises after the administration of SCh share an altered state of the sympathetic nervous system as their common denominator"[1]. However, these haemodynamic crises can also occur secondary to an abnormal neuromuscular response to SCh, such as malignant hyperthermia, or excessive K^+ release from denervated muscles.

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