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Intra-Arterial Thiopental

To the Editor:—A solution of 2.5% thiopental recently was injected into a radial artery line. The total dosage of the drug that the patient received was 150–175 mg. There were no sequelae.

Gangrene of the hand, secondary to an intra-arterial injection of thiopental, is a well-known complication. This usually has occurred with a concentration of 5% or more.[1] In *Introduction to Anesthesia: The Principles of Safe Practice*, it is stated: ". . . to our knowledge, gangrene has not been reported following the use of 2.5 per cent thiopental."[2]

Communication with several anesthesiologists at other hospitals resulted in differing opinions. Most supported the view that there would be no sequelae. Two of these contacted stated that they had heard of gangrene occurring following an intra-arterial injection of 2.5% thiopental.

I have been unable to find, in the literature, a documented case of gangrene resulting after an intra arterial injection of 2.5% thiopental. The persisting question is: Does 2.5% thiopental cause gangrene?

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Safe Anesthetic Gas Pollution Control

To the Editor:—A cross connection of the scavenging circuit into the patient circuit has led repeatedly to near fatalities, both in reported cases and those unreported.[1–3] In such instances, the patient circuit is blocked. This problem was addressed specifically by American National Standard Z-79.11 which requires that the size of the scavenging port be incompatible with both the 15-mm and 22-mm anesthesia circuit end fittings or cuffs (respectively pediatric and adult circuits).*

Prudence suggests that all equipment not in compliance with the strictest ANS Z-79.11 recommendations, be replaced.

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* American National Standard Institute Z-79.11. Anesthesia Gas Pollution Control, New York, American National Standard Institute, 1982.

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Rapid Sequence Induction in Patients with a Full Stomach

To the Editor:—Rapid sequence induction of anesthesia using thiopental–succinylcholine is commonly performed in patients with a full stomach. Muravchick, Burkett, and Gold[1] have shown in humans that succinylcholine-induced abdominal fasciculations can be associated with a significant increase in the intragastric

pressure (IGP). Pretreatment with the nondepolarizing relaxant *d*-tubocurarine could block both the succinylcholine-induced muscle fasciculations and the associated rise of IGP. The authors suggested that succinylcholine-induced fasciculation and the associated increase of IGP introduces the risk of regurgitation, and advocated routine pretreatment with a nondepolarizing relaxant before administration of succinylcholine to patients with a full stomach.

Professor Smith[2] disputed these conclusions because the tendency to regurgitate the gastric contents is not dependent upon the magnitude of increase in IGP *per se*, but on the pressure gradient between the stomach and the lower esophageal sphincter (LOS). Succinylcholine fasciculations, although elevating the IGP, induce a correspondingly greater increase in LOS pressure so that the net effect on the barrier pressure (LOS pressure − IGP) is a small increase.[3] It was concluded that succinylcholine fasciculations do not increase the tendency to regurgitate in normal subjects with a full stomach, and that routine pretreatment with a nondepolarizing muscle relaxant is unnecessary.

Our experience during the tragic events of Lebanon supports the conclusions of Professor Smith. A rapid sequence induction of anesthesia was used in 400 casualties with full stomachs who were scheduled for emergency surgery. All patients were premedicated with iv atropine, and were induced in the supine position using a preoxygenation–thiopental or ketamine–succinylcholine–laryngoscopy–intubation sequence. In 200 patients, backward pressure was applied on the cricoid ring (Sellick's technique) as soon as the patient lost consciousness, while in the other 200 patients no cricoid pressure was applied. Artificial ventilation was not initiated until the trachea was intubated and the cuff inflated. In all patients, rapid sequence induction was followed by rapid and complete muscular paralysis providing optimal intubating conditions; also, no visible regurgitation was observed on inspection of the pharynx during laryngoscopy and intubation.[4]

These findings may not apply to patients with abnormal function of the gastroesophageal junction, such as hiatus hernia, which should be suspected in parturients and in obese patients over 40 years of age. In these patients, pretreatment with a nondepolarizing muscle relaxant is advisable, and backward cricoid pressure is mandatory. However, pretreatment can delay the onset and diminish the block of a subsequent dose of succinylcholine, and thereby may make endotracheal intubation more difficult and hazardous in the patient with a full stomach. This can be avoided by increasing the dose of succinylcholine.[5]

In patients with gastric or intestinal obstruction, the IGP usually increases and may overcome the gastroesophageal junction, and material can accumulate in the esophagus which can hold a surprisingly large volume of fluid without leakage into the pharynx because of the cricopharyngeal sphincter. A similar accumulation occurs in patients with esophageal lesions such as achalasia. In these high-risk patients, a rapid sequence induction, using depolarizing or nondepolarizing relaxants, can be followed by paralysis of the skeletal cricopharyngeal muscle which is "the last line of defense" against regurgitation. Any fluid accumulation in the esophagus escapes immediately into the pharynx.[6] Awake endotracheal intubation may be the technique of choice.

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