CORRESPONDENCE

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In Reply:—Sood *et al.* point out that the negative pressure generated by the Ellick's evacuator or the self-inflating bulb, as opposed to gentle aspiration with a 60-ml syringe, is more likely to cause airway collapse when the device is connected to endotracheal tubes in patients with increased airway resistance. Although this is conceivably true, a direct comparison between the two devices has not been reported. As has been shown previously, both the syringe and the self-inflating bulb occasionally fail to confirm tracheal intubation[1–5]: in infants, in whom the tracheal wall is not held upon by rigid cartilaginous rings[2]; if the tube is obstructed[1–3]; in patients with high airway resistance[3]; when the tube is at the carina or in the right main bronchus[1]; in morbidly obese patients[4,5]; and in other patients who have marked reduction in their functional residual capacity.[4,5]

The performance of the self-inflating bulb when connected to tracheally or esophageally placed patent tubes depends on several factors, including the material and size of the bulb, the presence of kinking or obstruction of the tube, the technique used, the location of the distal end of the tube in relation to the anatomy of the tracheobronchial tree, the presence of airway disease, and the patient's functional residual capacity. The negative pressures generated by the plastic self-inflating bulbs used in the United States (and used in our studies) seem to be less than those of comparable size used in Europe, which are made of thick rubber material.[6] Because of its smaller radius, a smaller self-inflating bulb (capacity 20 ml) can generate greater negative pressure (−82 mmHg) as compared to the larger self-inflating bulb (capacity 75 ml).[7] A higher incidence of false-positive results is encountered when the smaller self-inflating bulbs are compressed after connection to tubes placed in the esophagus and, hence, are not recommended.[7]

Two techniques have been used with the self-inflating bulb to differentiate esophageal from tracheal intubation. In one advocated by Nunn[8] and Williams and Nunn,[9] the self-inflating bulb is compressed after it is connected to the endotracheal tube. In the other, the self-inflating bulb is compressed before connection to the tube.[5–7] Recently, we studied the incidence and demography of false-negative results (no reinflation or delayed reinflation >4 s) in 2,140 consecutive anesthetized adult patients comparing the two techniques.[5] The incidence of false-negatives was 4.6% when the self-inflating bulb was compressed before connection to the endotracheal tube, whereas it was 2.4% when the self-inflating bulb was compressed after attachment to the endotracheal tube ($P < 0.01$).[5] Eighty-five percent of patients in whom false-negative results occurred were morbidly obese, and the rest had severe bronchospasm, chronic obstructive pulmonary disease, mainstem intubation, or pulmonary secretions or edema.[5] We surmise that this phenomenon could be related to several factors. The severe reduction in functional residual capacity, especially after anesthetic induction and muscular relaxation, could lead to reduced caliber of intrathoracic airways, inducing terminal airway closure and contributing to the collapsibility of the trachea upon the application of subatmospheric pressure. When the structural integrity of the airway is compromised, as in patients with chronic obstructive pulmonary disease, subatmospheric pressure readily would produce collapse of these airways. Other phenomena that may contribute to false-negative results observed in these patients include invagination or collapse of the posterior tracheal wall and larger airways and mediastinal compression. Compression of the self-inflating bulb after rather than before connecting it to the tube simply can avoid 50% of these apparently false-negative results by introducing a volume of gas into the airway before the subatmospheric pressure is generated.

The advantages of the self-inflating bulb over the Wee[10] original esophageal detector device are simplicity and speed (<4 s).[9] Although we initially employed the technique of compressing the self-inflating bulb before connection to the endotracheal tube,[6] we now believe that compressing the self-inflating after its connection to the tube is the superior technique, because it obviates approximately one-half of the apparently false-negative results.

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The History of Immediate Hypersensitivity Reactions

To the Editor:—An unreferenced article by Ovary on the history of immediate hypersensitivity reactions describing the first recorded death from anaphylaxis[1] is referenced in major anesthetic textbooks.[2,3] This event is said to be recorded in hieroglyphics on an Egyptian ebony plate from 2600 BC and to tell the story of "Menes, son of Sargon the Great, who came to Conquer Egypt but died following a bee sting."

Many tales from ancient Egypt become embellished over the years. The curse of Tutankhamen for example, was fictitious. This tale of anaphylaxis is no exception.

Menes is the name given to the first Egyptian pharaoh (the first king of the first dynasty). Although semilegendary, this figure was based on a native Egyptian who, modern chronologic studies would suggest, ruled about 3100 BC.

The ebony plate found at the entrance to his otherwise empty tomb appears to show a wasp or hornet and was translated by Waddell 1930[4] to suggest that Menes died from a wasp sting. This has been disputed by many eminent Egyptologists, however, and other interpretations are perhaps more likely.[5] The earliest "contemporary" account from a Greek historian states that Menes was killed by a hippopotamus!

As for the rest of the story, Sargon the Great, ruler of Akkad (in modern Iraq), lived in the 27th century BC. His son and successor was Narim-Sin. They were a warlike family, but there is no evidence that there was any contact with Egypt at this time, warlike or otherwise. 2600 BC is a period of demonstrable stability and prosperity in Egypt, and there was no evidence of war with anyone during this period. There were no foreign invasions of Egypt until the Syropalestinian Hykos rulers of the 17th century BC. (None of these had names like Menes or Sargon.)

The true fate of Menes may never be known. The consensus would seem to be that, in the absence of more factual data, the tale of Menes dying from a bee sting must be considered a myth, and the remainder of the story as told by Ovary is, at best, inaccurate.

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