Correspondence

A Simple Connector for Transtracheal Ventilation

To the Editor:—It has been recommended that a large-bore needle be inserted through the cricothyroid membrane of a patient whose airway obstruction cannot be relieved by less drastic means.1 In an emergency situation, this maneuver may fail for lack of a way to connect the needle to a source of oxygen, preferably under positive pressure. The enclosed drawing shows a simple way of connecting a resuscitation bag or anesthesia machine to a standard needle or intravenous cannula. This connection can be made in seconds from items readily available in an operating room or on a resuscitation cart. A standard 15-mm adapter from an 8-mm endotracheal tube is fitted into the barrel of a 3-ml disposable plastic syringe (Monoject) from which the plunger has been removed. A very tight fit is obtained. Other brands of disposable plastic syringes may require different sizes of connectors; for example, a 7-mm endotracheal tube connector fits into the B-D "Plastipak" 3-ml syringe.

This device, when fitted to a 12-gauge, 2-inch cannula (Argyle) will allow gas flow of 10 to 12 l/min at pressures attainable with a standard resuscitation bag. Flow versus pressure measurements of this adapter and cannula combination yield a conductance of .2 l/min/cm H2O of applied pressure between 10 and 35 cm H2O. This is in agreement with the data of Attia et al.1 These flow rates can provide adequate ventilation for life support until a better airway can be established. When a 3-mm newborn endotracheal tube is available, its connector may be attached directly to the cannula without the syringe barrel, as suggested by Attia et al.

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REFERENCE

1. Attia RR, Battit GE, Murphy JD: Transtracheal ventilation. JAMA 234:1152–1153, 1975

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The Boston–Copenhagen Détente

To the Editor:—Everyone in the field of medicine acknowledges the many contributions of Dr. John W. Severinghaus. However, his report in Anesthesiology1 is causing distress among many of us in this country. Indeed, we are not accepting the "treaty" offered by Dr. Severinghaus, as the addition of the "arc" to the Siggaard-Andersen alignment nomogram does not solve the main difficulties in understanding acid–base balance created by the standard bicarbonate, base excess concept. Indeed, I consider the addition of the arc to analyze chronic hypercarbia a distortion of the Copenhagen nomogram to make it fit clinical situations, and it shows evidence that this in-vitro approach is not comprehensive.

Why is Dr. Severinghaus shunning the more direct approach of in-vivo titration to acid–base balance as conceived by William B. Schwartz? We would like to know his logic. The direct in-vivo titration is surely simpler in concept, easier to use at the bedside (no nomogram is necessary), and more representative of the actual state of acid–base balance in disease.2–4

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