A New Laryngoscope: The Combination Intubating Device

To the Editor---A new intubating device (prototype made by Achi Corporation, Fremont, CA; U.S. patents 4,828,725 and 5,261,592) combining a rigid laryngoscope blade and a flexible fiberscope has been designed and tested (fig. 1).

This device comprises a tubular, curved, bivalved, rigid blade portion and a flexible fiberscope portion. The blade portion has three detachable stainless steel parts: handle, main blade, and bivalve element (fig. 2). The handle is a cone-shaped tube that receives the fiberscope body. The main blade and bivalve element are arc-shaped, each having corresponding grooves that form a larger passageway for the endotracheal tube (ETT; ETT passageway) and a smaller passageway for the fiberscope insertion cord (fibercord passageway). An oxygen channel is alongside the fibercord passageway. The axes of the handle and blade are at an angle of 110°. By separate interlocking mechanisms, the bivalve element can be attached or released from the handle and main blade. The tip of the main blade is smoothly wedged to allow the blade to glide easily over the tongue, enter the vallecula, or lift the epiglottis. The vertical dimension (depth) of the tubular blade is 16–18 mm; a minimum mouth opening of 20–25 mm is, therefore, required for inserting and maneuvering this device.

The device is designed to facilitate orotracheal intubation with the patient's head in the neutral position (fig. 3, device in illustration shows only blade portion). When the larynx comes into view, a suction catheter through the ETT lumen is advanced to enter the glottis. Then, if necessary, the position of the distal main blade is adjusted accordingly to align the ETT with the glottis. The alignment usually is accomplished by moving the blade sideways or withdrawing, advancing, or lifting the device slightly. Once the ETT and the glottis are properly aligned, the ETT is advanced over the suction catheter into the glottis. The bivalve element is removed from the patient's mouth first; then the handle, main blade, and fiberscope are removed as one unit, leaving the ETT in place. By excluding the bivalve element, the device also may be used for nasotracheal intubation.

This combination device has many of the desirable features of existing rigid and flexible laryngoscopes such as the modified handle-to-blade angle for easier entry in obese patients; the fiberoptic imaging and oropharyngeal airway-shaped blade allowing smooth access to the larynx without the need for head extension, tongue lifting, or forceful jaw opening; and the ability to provide supplemental oxygen.

Fig. 1. The new device with the rigid and flexible portion fitted together and properly assembled with (a) an endotracheal tube (ETT) in the ETT passageway, (b) a suction catheter in the ETT lumen, and (c) oxygen tubing connected to the oxygen port.

References


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Fig. 2. Rear view of the three parts of the rigid blade portion of the device: (A) handle, (B) main-blade, and (C) bivalve element. a-locator on the handle that aligns with locator on the fiberscope; b-latch for the proximal interlocking mechanism, c-oxygen port, d-oxygen channel, e-fibercord passageway, f-endotracheal tube passageway, and g-slits for the distal interlocking mechanism.

and simple, effective suctioning. Although the Bullard laryngoscope1 (Circon-ACMI, Stamford, CT) may have similar functions, this new intubating device possesses two other features distinct from the Bullard. First, it has a tubular structured blade that protects the fiberoptic lens from blood, secretion, or redundant soft tissue and creates viewing and intubating space.2 Second, it has a built-in ETT passageway that provides an easy means by which to advance the ETT when the larynx comes into view; no forceps or stylet is needed.

We have used prototypes of the rigid blade portion of this device incorporated with the Olympus tracheal intubation fiberscope LF-2 or rhinolaryngoscope ENF-P3 (Olympus Corp., Lake Success, NY) and tested this device in clinical anesthesia. Over a 12-month period, after institutional approval and with informed consent obtained from all patients, my colleagues and I have used this device routinely for oral or nasal intubations. To date, the device has been used in more than 300 adult patients, including 48 patients with Mallampati airway classification 3 and 4. In 11 patients with such problems, receding jaw, short mandibular ramus, or caudal larynx,3 limited mobility of temporomandibular joint (TMJ), or small atlantooccipital gap, and in whom the trachea had been previously documented as being difficult to intubate by experienced anesthesiologists using conventional rigid laryngoscopes, tracheal intubation with this device was accomplished relatively easily. We have used this device successfully in various clinical situations, including emergency awake intubations in the emergency room and intensive care unit and in patients with obesity, kyphoscoliosis, active TMJ disease, pharyngeal obstruction, and cervical spinal lesion. Also, we have used this device as an emergency rescuer in four patients when use of the standard rigid laryn-
goscopes by other anesthesiologists failed to provide an adequate view of the larynx to permit intubation.

In summary, this new device has been well accepted and used successfully to facilitate tracheal intubations and, thus far, has not been associated with any complications. It appears sufficiently promising to justify further evaluations by other investigators.

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Interpreting Low-dose Anesthetic Effects on the Ventilatory Response to Hypoxemia: Facts, Findings, and Fanciful Formulations

To the Editor:—In a recent editorial, Robotham considers how low-dose halogenated anesthetics may affect the ventilatory response to hypoxemia. 1 In an admirable effort to reconcile “apparently conflicting reports” in the literature, he offers his own interpretations of research in this field. First, he concludes that, even though small doses of these agents may put a patient at risk for a depressed hypoxic response, a patient “who is otherwise healthy, responsive, and aroused by sensory stimulation, pain, or anxiety [as in anesthetic recovery] is much less likely to exhibit depression of respiratory drive to hypoxia.” Second, he infers that, “if [emphasis added] a behavioral state . . . ensues that results in the withdrawal of a central nervous system component [of ventilatory drive, the presence of . . . 0.1 MAC’ doses] may result in a reduced response. In my view, each of Robotham’s conclusions is open to serious question—as each rests upon a critical assumption that appears to be untenable.

The assumption underlying the first conclusion is that one can draw reliable inferences about a reflex of the “metabolic” ventilatory control system, such as the response to hypoxemia, in the presence of sensory stimulation that is known to incite the second ventilatory control system referred to as “behavioral” control. 2 In making this assumption, Robotham appears to have overlooked the distinctive anatomic and physiologic characteristics of each of these ventilatory control systems 2,3 and the considerable difficulty in identifying functional characteristics of one system when the other is simultaneously stimulated or uncontrolled. 4

The metabolic control system arises from chemoceptors that drive the brainstem controller to generate stable and automatic ventilation that defends metabolic homeostasis. The behavioral system, on the other hand, arises from multiple poorly defined neural inputs that activate a separate forebrain controller to superimpose highly irregular ventilation in conditions such as speech, sensory stimulation, anxiety, and REM sleep. 3,4 The assumption that one can obtain reliable information about metabolic control when the unstable behavioral system acts simultaneously and independently has been refuted by authorities and seems intuitively unlikely. For example, would it be possible to infer the effects of opioid on automatic, metabolically controlled ventilation by administering opioid to the point of marked respiratory depression and then activating behavioral control by periodically speaking to the subject and reminding them to breathe? More pertinent to the issue at hand, would it be possible to infer the effects of low-dose anesthetic on the metabolic response to hypoxemia when the behavioral system is activated simultaneously by the combination of repeated nudges, an exciting movie, and speech—as in the research to which Robotham refers? 3 Although behavioral control undoubtedly can be recruited in the clinical setting to stimulate ventilation and thereby protect a sedated patient from hypoxemia or a reduced response to hypoxemia, this does not imply that it modifies the ventilatory response to hypoxemia per se. Exactly how does Robotham reconcile his assumption with these well-founded concepts of ventilatory control?

The assumption underlying Robotham’s second conclusion is that the previously reported effect of low-dose halogenated anesthetic on the hypoxic response is conditional upon the development of a new central nervous system (CNS) state, described variously as “sleep,” “drowsiness,” “reduced arousal,” and “withdrawal of a CNS component of ventilatory drive.” 3,5 However, in making this assumption, Robotham appears to have overlooked the results of numerous investigations.

Several years ago, we observed that 0.1 MAC-equivalent states induced with methoxyflurane and with thiopental did not detectably

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