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Distinguishing Endotracheal and Esophageal Intubation

To the Editor:—I enjoyed the article of Raphael[1] that described a new instrument for distinguishing endotracheal and esophageal intubation by acoustic reflectometry. I agree with his conclusion that such a device has a place in emergency airway management because it is independent of carbon dioxide exhalation, it is not subject to operator bias, and it does not necessitate ventilation into a possibly misplaced tube. However, I was surprised that Raphael missed some of the previous literature on this subject.

First, Mansfield *et al.*,[2] who in 1993 were the first to describe this application of acoustic reflectometry, must be mentioned. Second, the related technique of Akerson,[3,4] which used resonant sound, was patented in the US, and was presented at the American Society of Anesthesiologists meeting in 1994, should be mentioned. From the Akerson patent, the Sonomatic Confirmation of Tracheal Intubation (SCOTI) device was developed, which was sold in Europe by Penlon (Abingdon, Oxon, UK) from April 1995 until 1996 and then was withdrawn.[5] Several papers about the SCOTI device were published in Medline-indexed journals, most of which described problems.[6–11] Although the algorithm used by Raphael[1] is much more sophisticated than that used by the SCOTI device, each of the problems described with the SCOTI device should be discussed to clarify whether they can be excluded with the device of Raphael.

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In Reply:—I thank Dr. Maleck for giving me the opportunity to discuss other acoustical approaches for distinguishing endotracheal from esophageal intubation.

The intent of my article[1] was to focus on the imaging of the upper airway and esophagus, as made possible by the development of the Hood Labs acoustic reflectometer (Pembroke, MA), which generates a "one-dimensional" image in the form of an area–distance profile. These area–distance profiles are intuitive in that the operator is able to assess the total cross-sectional area at any given distance into the cavity of interest, whether the trachea or esophagus. For a breathing tube in the trachea, the cross-sectional area is constant within the endotracheal tube (ETT), and increases further with deeper penetration into the lung. By comparison, if the esophagus is intubated, the area amplitude goes essentially to zero immediately beyond the ETT tip as a result of the nonrigid esophagus collapsing around the distal ETT.

Mansfield *et al.*[2] conducted a similar and earlier investigation in dogs. The approach used the delivery of a series of sonic impulses into the airway, with a miniature microphone placed in the endotracheal tube wall to monitor sound pressure. The key to the Mansfield system is the following: When the incident impulse encounters a boundary where there is a sudden increase in area (*e.g.*, endotracheal intubation), the reflected wave approaches the absolute amplitude but is inverted in an amplitude-*versus*-time delay (A-TD) plot. In contrast, if the incident wave encounters a large decrease in amplitude (*e.g.*, esophageal intubation), the reflection is large but is not inverted in the resulting A-TD plot. The presence of this deflection at the ETT tip allowed discrimination between esophageal intubation (upward deflection) and endotracheal intubation (downward deflection). Endotracheal intubation was confirmed by the presence of additional negative airway deflections in an A-TD plot. My concern, however, is that this approach is not intuitive and user-friendly, because the operator must look for the presence or absence of a key reflection amidst a series of undulations in a plot. By comparison, when a Hood Labs reflectometer area–distance profile is used, even an inexperienced operator can, in an instant, distinguish easily between endotracheal and esophageal intubation. Nonetheless, the Mansfield *et al.*[2] system, in principle, is workable and, with appropriate modifications and operator training, could lead to a useful device.

Akerson[3] developed a technique that used resonant sound as the basis for distinguishing between endotracheal and esophageal intubation. In complex asymmetric branching structures, such as the lung, there is not one resonant frequency but multiple resonant peaks caused by the clumping of eigenvalues into clusters in the low-frequency range.[4] In earlier publications,[5,6] I developed explicit, closed-form mathematical solutions to calculate the expected resonance frequencies for symmetrical and asymmetrical branching structures exhibiting an arbitrary number of bifurcations in which the branch areas and lengths were known *a priori*. The inverse problem, that of predicting cavity volumes and branch characteristics from the observed resonant frequencies, is a considerably more difficult mathematical problem that has not been solved. However, in the low-frequency range, the fundamental resonance frequency for such structures can be shown to be approximately inversely proportional to the cavity volume.

The rationale behind the Akerson device was to exploit the differences between the higher-valued fundamental resonant frequency associated with an esophageal intubation (smaller cavity) *versus* the lower-valued fundamental resonant frequency associated with an endotracheal intubation (larger lung cavity). For configuration purposes, attention was paid to the resonant frequency characteristics of the ETT, considered as a cavity in itself. It was followed by the Sonomatic Confirmation of Tracheal Intubation (SCOTI) device, which required configuration to the individual ETT before its use. The device generated a series of numbers that were used to decide on proper ETT

configuration and to determine correct endotracheal *versus* esophageal placement. Although previous studies were promising in that the SCOTI device could determine most esophageal intubations, the device encountered several difficulties in that it could not determine tracheal intubations with the same level of success.[7] The SCOTI device could not be configured reliably for ETTs with a diameter smaller than 6.0 mm and gave inconsistent results in cut ETTs.[8] In patients who had been intubated already, the ETT position could not be checked without first removing the ETT from the patient for the required configuration of the device. The conclusions of several studies indicated caution in the use of the device, and, ultimately, because of disappointing sales, the device was withdrawn from the market. As Dr. Maleck points out, the SCOTI device merits mention as another acoustic device that aimed to distinguish between endotracheal and esophageal intubation.

Unlike the SCOTI device, the acoustic reflectometer can be used in an already-intubated patient, regardless of whether the ETT is cut. The present acoustic reflectometer is intended for use in adults and is capable of reproducing accurate longitudinal area changes in adult ETTs with internal diameters as small as 6.0 mm without difficulty. A large-scale validation study is currently in progress to determine the specificity and sensitivity of the acoustic reflectometer in the detection of endotracheal and esophageal intubations.

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Perioperative Myocardial Infarction (PMI): A Never-ending Story

To the Editor:—We read with great interest the article on analysis of risk factors for myocardial infarction and cardiac mortality by Sprung *et al.*[1] Some statements however, need clarification.

The reported overall incidence of perioperative myocardial infarction (PMI) in this high-risk population is extremely low (1.54%). In contrast, Badner *et al.*[2] found the incidence of PMI to be more than 3.5 times higher (5.6%). Sprung identified PMI by clinical symptoms, creatine kinase–myocardial band increases, or diagnosis of new Q waves on the electrocardiogram. Badner *et al.*[2] additionally determined troponin concentrations. He also found that PMI was an early event only occasionally associated with chest pain and usually non–Q wave in nature. This clearly shows that the incidence of PMI reported by Sprung *et al.*[1] was underestimated, and that the results should be interpreted with caution.

General anesthesia was associated with a significantly greater risk of PMI.[1] Does this mean that regional anesthesia in these high-risk patients resulted in significantly lower incidence of PMI? Was there any difference in postoperative pain therapy between groups? For more than a decade, there has been an ongoing discussion of whether general or regional anesthesia is more beneficial in patients at increased cardiac risk. In 1996, an editorial stated that no more studies were needed to answer this question[3] because the largest study at the time showed no difference in outcome.[4] A retrospective analysis comparing the effects of general and regional anesthesia on outcome in patients with hip fracture repair also showed no significant difference in PMI. However, use of general anesthesia decreased from 95% in 1981 to 47% in 1993.[5] The reasons for enhanced employment of regional anesthesia could not be determined, but it was shown that "sicker" patients were allocated to the regional group. It would be interesting if Dr. Sprung *et al.*[1] could add valuable data to this debate.

Patients with β-blocker therapy were more likely to experience PMI.[1] The authors speculated that this surprising finding was because intraoperative extremes of heart rate did not differ between groups. β Blockers have been shown to prevent PMI and improve long-term survival after noncardiac surgery.[6] However, it should be stressed that intraoperative and postoperative lower heart rates (below 80 beats/min) are the key to successful treatment with β blockers. This has been shown by Poldermans *et al.*[7] who evaluated the effect of bisoprolol in a group of patients with positive dobutamine echocardiography results who were scheduled to undergo major vascular surgery. In the bisoprolol group, 3.4% of patients died of cardiac causes, compared with 17% of patients in the standard care group ($P = 0.02$). Nonfatal myocardial infarction occurred in 17% of patients given standard care only and in none of those to whom bisoprolol was administered ($P = 0.001$). Mean heart rates in the bisoprolol group were significantly lower than in standard care patients. An accompanying editorial stated that it seems likely that the cumulative morbidity resulting from three sequential procedures (angiography, revascularization, major vascular procedure) would be higher than the 3.4% rate of major cardiac complications in bisoprolol patients.[8]

Recent percutaneous transluminal coronary angioplasty (PTCA) was not cardioprotective in regard to reinfarction rate; however, it significantly prevented death after PMI evolved.[1] The only patient with PTCA who died had undergone PTCA more than 12 months before surgery. These data are not in accordance with a study from Posner *et al.*,[9] who demonstrated a significantly higher incidence of PMI after noncardiac surgery if PTCA was performed less than 3 months before surgery. Therefore, it would be interesting if Dr. Sprung *et al.*[1] could determine the exact time when PTCA was undertaken.

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In Reply:—We thank Dr. Mollhoff *et al.* for addressing our article.[1] We wish to offer clarification on their questions.

We agree that the rate of perioperative myocardial infarction (PMI) may be underestimated in our study, as we acknowledged in our study limitations section. In the article, we listed four reasons: (1) perioperative surveillance for PMI was not done prospectively; (2) not all patients were monitored for PMI; (3) monitoring sometimes was not performed in a systematic manner; and (4) troponin concentrations were not assessed in any of the patients. As we have stated, we are confident that we reported all cases of clinically significant PMI, and it is possible that a certain number of silent PMIs were not diagnosed. It should be noted that, for another variable we studied, mortality after PMI (death is an unmistakable complication), the rate we reported (17%) is similar to the one reported by Badner *et al.*[2] (20%).

In our study, general anesthesia carried a higher risk of PMI than did neuraxial anesthesia (odds ratio, 3.25, $P < 0.003$). However, in the multivariate analysis, type of anesthesia did not play a significant role in PMI or cardiac mortality. We cannot comment about possible differences in perioperative pain management between the two groups because we did not analyze this variable.

In our discussion, we suggested some of the explanations for why we think β blockade did not reduce the PMI rate in our population. First, patients to whom β blockers are administered are not absolutely protected against perioperative myocardial ischemia, and Slogoff and Keats[3] demonstrated that patients with coronary artery disease who use β blockers may experience myocardial ischemia intraoperatively, despite better hemodynamic control. Second, β blockade in our patients was probably inadequate because we found no difference between the heart rates in the two groups. Third, β blockade may have been administered preferentially to the patients with more severe coronary artery disease, and it may be that the increased severity of disease was the reason why these particular patients had a higher incidence of PMI.

The patients who had undergone percutaneous transluminal coronary angioplasty (PTCA) and subsequently experienced PMI had lower odds of cardiac death than did patients with PMI who had not undergone PTCA (the odds ratio of 0.32, however, was not statistically significant). Because we did not know the preoperative coronary artery anatomy (sites of stenoses) of our patients, we may postulate the following: PTCA is typically used to dilate stenotic lesions in larger coronary vessels, and, therefore, occlusions on smaller coronary artery branches may persist even after successful PTCA, but PMI that results from occlusion of these areas affects less cardiac muscle mass, with consequently lower mortality. Therefore, our finding has some logical explanation but needs to be further studied. We collected data on PTCA and categorized the procedures in four intervals: less than 3 months before surgery, 3–6 months previously, 7–12 months previously, and more than 12 months previously. In the PMI group, we had 12 patients: 3 underwent PTCA within 3 months before surgery, 4 underwent it 4–6 months before surgery, and 5 underwent it more than 12 months before surgery (1 of these 5 patients died). Although we have representative subjects in three of four categories, this is a small number of patients to make a definitive conclusion about the optimal timing of surgery after PTCA. In our previous (retrospective) study, we found very low cardiac morbidity in patients undergoing vascular surgery who underwent PTCA up to 18 months preoperatively.[4] The retrospective study by Postner *et al.*[5] did not provide one important piece of information for the patients who had PTCA and subsequent PMI: Did PMI develop in the area that was treated with PTCA or in some other area that was not originally amenable to treatment? It is moot to discuss the value of PTCA without this information. This issue may be resolved only by analyzing coronary angiograms in patients who underwent PTCA and consequently experienced PMI, and, to the best of our knowledge, this type of study does not exist.

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A Normal aPTT Does Not Guarantee Adequate Coagulation Factor Levels

To the Editor:—Spahn and Casutt[1] reviewed the strategies of how to reduce allogeneic erythrocyte transfusions. They briefly discussed the importance of transfusion algorithms based on coagulation monitoring. We would like to mention that a single coagulation factor deficiency may not be detected by routine coagulation assays. Recently, we observed a patient with an *F11* deficiency who bled more than expected during surgery. Results of routine presurgical tests, which include prothrombin time, activated partial thromboplastin time (aPTT), fibrinogen, and platelet count, were normal. *F11* was later found to be low at 11%.

Although hemostasis is complex *in vivo* and laboratory testing does not always predict intraoperative bleeding, prothrombin time, aPTT, and fibrinogen data usually are ordered preoperatively. When prothrombin time and aPTT are within the normal range, it is assumed that each coagulation factor concentration is adequate for surgery (*i.e.*, 30% of plasma factor concentration).[2] However, factor sensitivity for aPTT depends on the coagulation factor, the aPTT reagent, and the instrument. Our normal range for aPTT is 25.0–35.0 s, verified by analyzing 15 healthy men and 15 healthy women (mean ± 2 SD). Table 1 shows the factor sensitivity for intrinsic factors using Synthasil® (Lexington, MA) performed on the CS6000 coagulation analyzer (Sysmex, Long Grove, IL). Even when *F8* and *F11* concentrations are 30%, aPTT was normal. Especially for *F12*, in which congenital deficiency is not uncommon, aPTT was normal when the concentration was down to 9%.

Therefore, it should be emphasized that obtaining bleeding history, especially after surgery or tooth extraction, is important to suspect a bleeding diathesis. Clinicians should be aware of the factor sensitivity of the current aPTT in their hospital when considering plasma transfusion.

Table 1. Effects of Intrinsic Coagulation Factor Deficiencies on aPTT Results

	F5	*F8*	*F9*	*F10*	*F11*	*F12*
aPTT at 30% concentration (s)	40.8	32.7	36.3	27.1	29.2	29.4
Abnormal aPTT (> 35.0 s; %)	55	13	35	3	9	10

Normal range = 25.0–35.0 s. Normal reference plasma (FACT 06; George King Biomedical, Inc., Overland Park, KS) was diluted to the desired concentrations with plasmas deficient for each factor shown (Dade Behring, Marburg, Germany). When each factor concentration was 30%, the activated partial thromboplastin time (aPTT) was measured. When the aPTT was prolonged to more than the normal upper limit, *i.e.*, 35.0 s, each factor concentration was measured. Values are mean of 3 measurements.

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Eliminating Blood Transfusions: What about Hypotensive Anesthesia?

To the Editor:—In their recent review, 'Eliminating Blood Transfusions: New Aspects and Perspectives,' Spahn and Casutt[1] devote only five sentences and cite only two references to the method of controlled hypotension, and they create the impression that this method has limited safety, efficacy, and applicability. This contradicts the many articles, book chapters, and books that have been written on behalf of this subject,[2–5] and it contradicts the experience of many experts in the field. Of course, skill, experience, and vigilance are essential for the conduct of safe and effective controlled hypotension.

We recognize that a review article on such a broad topic cannot be totally comprehensive; however, we believe that controlled hypotension warrants a more in-depth discussion. Our concern is that a lack of attention to the science and art of controlled hypotension will result in the technique's not being passed on to the next generation of anesthesiologists. This would deprive these practitioners and, more importantly, their patients of a useful and safe method of blood conservation.

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In Reply:—We appreciate the commitment of all with a particular interest in avoiding allogeneic blood transfusions.[1] In the first letter, Drs. Teruya *et al.* show that a substantial deficiency of one coagulation factor may go undetected by standard preoperative activated partial thromboplastin time (aPTT) measurement. Indeed, the sensitivity of commercial aPTT kits to detect isolated coagulation factor deficiency may be extremely variable.[2] In widely used aPTT kits, such as the Actin® FS (Dade Behring, Marburg, Germany), abnormal results are obtained at the following coagulation factors thresholds: *F8* < 52%, *F9* < 41%, *F11* < 29%, and *F12* < 30%, thus at significantly higher concentrations, as with the kits studied by Drs. Teruya *et al.* Nevertheless, knowing the coagulation factor sensitivity of the aPTT kit used at the local hospital is important.

The rationale of transfusion algorithms guided by coagulation monitoring is not to predict high or low blood loss in an individual patient based on a preoperative coagulation test but to monitor blood coagulation throughout surgery. This enables one to detect a coagulation deficit early and to characterize its cause—low platelet number *versus* compromised platelet function *versus* low coagulation factors. In turn, this enables a specific treatment to avoid unnecessary blood loss and allogeneic blood transfusion caused by a significant coagulation deficit. Bedside coagulation monitoring, such as Thrombelastograph® analysis[3] (Thrombelastograph® Coagulation Analyser; Haemoscope, Morton Grove, IL) or hemoSTATUS[4] (Medtronic Blood Management, Parker, CO) may be particularly helpful because of the short turn-around time and high sensitivity.

In the second letter, Drs. Klowden *et al.* suggest that controlled hypotension warranted more recognition as an anesthesia technique to reduce the need for allogeneic blood transfusions. To document efficacy, four book chapters from 1979–1996 are referenced. To our knowledge, there is only one prospective randomized study with defined transfusion criteria that has shown efficacy,[5] and this study was discussed in our review article.[1] Although hypotensive anesthesia seems safe at relatively high hemoglobin concentrations, efficacy has been challenged recently,[6] and this is conceivable because of the fact that a majority of surgical bleeding is venous bleeding. By which mechanism should a lower arterial pressure thus reduce venous bleeding? At least to us, this is unclear. In contrast, low central venous pressure may reduce blood loss and transfusion requirement substantially in liver surgery.[7–9] Therefore, anesthesia techniques do have an impact on surgical blood loss and transfusion requirements, but attention probably should focus more on central venous pressure than on arterial blood pressure.

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Compliance Compensation of the Narkomed 6000 Explained

To the Editor:—The recent study by Stevenson *et al.*[1] compared the performance of the AV2+ ventilator of the Narkomed GS anesthesia workstation (North American Draeger, Telford, PA) with the Divan ventilator of the Narkomed 6000 workstation (North American Draeger) using an infant lung model. Both of these ventilators are designed to work with a circle anesthesia circuit. Among its many features, the Divan ventilator is designed to ensure more accurate delivery of set tidal volume to the airway of the patient than a traditional anesthesia ventilator by compensating for the compliance of the breathing system and patient circuit. In their article, Stevenson *et al.*[1] state that '. . . the compensation for a decrease in lung compliance by the 6000 ventilator system is incomplete' and that 'the clinical advantage of this compliance feature needs further evaluation.' Their findings and the clinical implications of compliance compensation as implemented on the Narkomed 6000 can be understood by examining the manner in which the compliance compensation feature is designed.

During setup, the Divan ventilator is designed to determine an external compliance compensation factor (C_{ext}) for those components external to the patient, which include the machine components of the breathing system (C_{bs}) and the hoses of the breathing circuit (C_{circ}).

$$C_{ext} = C_{bs} + C_{circ} \quad (1)$$

Therefore, the total compliance (C_{Tot}), including the compliance of the patient's lungs, (C_L) is as follows:

$$C_{Tot} = C_L + C_{ext} = C_L + C_{bs} + C_{circ} \quad (2)$$

For the set tidal volume (V_T) to be delivered to the airway of the patient, the volume the ventilator must deliver (V_{vent}) is the tidal volume plus sufficient volume to compensate for the compliance of the system external to the patient (V_{ext}).

$$V_{vent} = V_T + V_{ext} \quad (3)$$

During mechanical ventilation, the volume delivered is the product of peak inspiratory pressure (P_{insp}) and compliance:

$$V = P_{insp} \cdot C \quad (4)$$

Therefore, equation 3 can be rewritten in the following manner:

$$V_{vent} = V_T + (P_{insp} \cdot C_{ext}) \quad (5)$$

During each mechanical breath, the ventilator continuously measures inspiratory pressure and uses the known external compliance compensation factor (C_{ext}) and the set tidal volume to find the total volume that must be delivered to ensure that the set tidal volume is delivered to the airway.

Because compliance compensation involves increasing the volume delivered by the ventilator, there are safety features incorporated into the ventilator design that limit the amount of volume that can be added in an attempt to compensate for compliance. For example, it is possible that a user could place a low-compliance circuit, such as a pediatric circuit, on the machine without executing the compliance test. If the last circuit tested had a greater compliance, the ventilator could deliver excessive volumes to the patient. To prevent this from occurring, when tidal volumes of less than 200 ml are selected, the Divan will only use the measured circuit compliance if it is 0.8 ml/cm H_2O or less. If the measured circuit compliance exceeds 0.8 ml/cm H_2O, a default value of 0.6 ml/cm H_2O is used as the circuit compliance compensation factor.

In their study, Stevenson *et al.*[1] tested the ability of the Divan ventilator and the AV2+ to deliver tidal volumes of 50, 100, and 200 ml to an infant test lung when lung compliance was changed from 3 ml/cm H_2O (normal) to 1 ml/cm H_2O (low). Their results showed more accurate volume delivery by the Divan ventilator because a greater minute ventilation was delivered by the Divan than by the AV2+ ventilator, which does not compensate for compliance. However, the Divan did not compensate completely when lung compliance was reduced because the minute ventilation delivered to the test lung was less than the initial value.

The relevance of these findings to patient care can be understood by putting the test conditions into a clinical perspective. At the low lung-compliance setting (1 ml/cm H_2O), the tidal volumes of 100 and 200 ml would generate respective peak pressures of 100 and 200 cm H_2O, which exceed clinical conditions. Therefore, the Divan ventilator is not designed to provide compliance compensation under those conditions.

For the 50-ml tidal volume at the low lung-compliance studied by Stevenson *et al.,*[1] the peak pressure would be 50 cm H_2O—within the clinically possible range. In this case, the Divan would need to deliver 50 ml plus enough additional volume to compensate for circuit and breathing system compliance. We obtained a sample of the Pediatric King breathing circuit (King Systems Corp., Noblesville, IN) used in the study and found the compliance to be approximately 1.6 ml/cm H_2O. As explained, the Divan would substitute a circuit compliance compensation factor of 0.6 ml/cm H_2O for the actual hose compliance of the circuit. The internal breathing system compliance of the Divan is about 2.2 ml/cm H_2O. Therefore, the external compliance compensation factor used by the ventilator would be 2.8 ml/cm H_2O, whereas the actual external compliance of the experimental setup would be 3.8 ml/cm H_2O. Because the actual external compliance was greater than the compensation factor used by the ventilator, the volume (V_{ext}) that was added to the set tidal volume would not compensate completely for the actual external compliance (see equation 5).

To obtain optimum performance from the Divan ventilator, users are instructed to execute the simple compliance test each time the circuit is changed. Users also are advised to use pediatric circle circuits when volume mode is used to deliver tidal volumes of less than 200 ml. North American Draeger does not describe the compliance compensation limits in the specifications published in the user's manual for the Narkomed 6000. Therefore, Stevenson *et al.*[1] were not aware of the manner in which this feature was designed. We congratulate these investigators on a thorough and well-presented study. We hope this additional information will provide the appropriate clinical perspective on their results. As a result of this study, North American Draeger is updating user manuals to include more detailed information on compliance compensation by the Divan ventilator. North American Draeger also is pleased to help customers to select circuits that will ensure the performance they require to meet their clinical needs.

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In Reply:—Draeger has provided additional information regarding compliance compensation in the Narkomed 6000. No effort was made by the authors to obtain or include this detailed information in our manuscript; the goal of our study was to present an independent evaluation of the Narkomed 6000 during simulated infant ventilation. We believe that our statements that "the compensation for a decrease in lung compliance by the 6000 ventilator system is incomplete" and that "the clinical advantage of this compliance feature needs further evaluation" are true. Some readers may find the additional detail provided by North American Draeger to be of interest.

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CO_2 *via* CPAP Mask? A Near Disaster

To the Editor:—We wish to describe an incident that recently occurred in our endoscopy suite. We consulted for a 55-yr-old obese woman (weight, 128 kg; height, 140 cm) with a history of obstructive sleep apnea that necessitated nasal continuous positive airway pressure at home. She was scheduled to undergo colonoscopy to evaluate rectal bleeding and cramping abdominal pain. Her baseline forced expiratory volume in 1 s was 0.67 l, and she was home oxygen dependent. Her room air oxygen saturation was 90–92%. When the anesthesia team was preparing the patient for the procedure, a respiratory therapist fit the patient with a continuous positive airway pressure mask.

In our endoscopy suite, there are two carbon dioxide (CO_2) cylinders. CO_2 is used to insufflate the bowel, particularly in children who have had poor bowel preparation previous to the procedure. CO_2 is used to displace methane from the area where electrocautery is used, reducing the risk of fire or explosion. Recently, the flowmeter for these CO_2 cylinders had been mounted on the wall, in a conspicuous place.

This flowmeter was identical in appearance to standard oxygen flowmeters seen at most institutions, complete with a green oxygen nipple. The only noticeable difference was the labeling of this flowmeter with "CARBON DIOXIDE" in small print. When the anesthesia team was preparing the patient for the colonoscopist, the respiratory therapist connected the oxygen supply line from the continuous positive airway pressure mask to this flowmeter on the wall.

Just before the initiation of gas flow, one member of our anesthesia team noticed the oxygen supply line to the continuous positive airway pressure machine attached to the CO_2 source. The line was immediately removed from the CO_2 outlet and connected to an oxygen gas outlet, which also was mounted on the wall. CO_2 was not administered to the patient. The colonoscopy was performed uneventfully, with the patient undergoing conscious sedation by the anesthesia team and with an uneventful postprocedure course.

As soon as the postprocedure course was completed, our Medical Engineering and the Respiratory Therapy departments came to the unit to discuss this critical incident. Clearly, there are many problems with this treatment situation, but, in an attempt to address the broader issues as well as remedy this immediate concern, a placard was put up to label the flowmeter clearly.

This case shows that other gases are in use in endoscopy suites and are sometimes poorly or inadequately labeled. Without special care and diligence on the part of the anesthesiologist, a crisis may have occurred. Similar circumstances have the potential to occur in magnetic resonance imaging suites, in hyperbaric chambers, and even in "standard" operating rooms designed for specific surgical procedures. We hope that this letter calls attention to the potential for such occurrences and stimulates dialogue to reduce such risks in the future.

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A Method to Improve a Gas Leak on Mask Ventilation in the Patient with a Nasogastric Tube

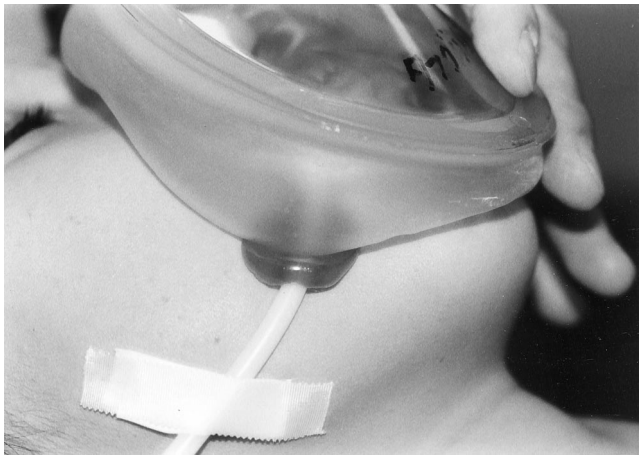

Fig. 1. The gas leak between the nasogastric tube and the cushion of the mask was covered with a denture adhesive while the tube came out of the right side of the mask.

To the Editor:—The presence of a nasogastric tube may make mask ventilation difficult. We found that the use of a denture adhesive (Tafugurippu®; Kobayashi Pharmaceutical Co., Ltd., Osaka, Japan) around the nasogastric tube reduced the gas leak (fig.1). We also conducted a brief study to evaluate the effectiveness of this technique during positive-pressure ventilation in volunteers.

After approval from the review board of Chikuho Rosai Hospital, Fukuoka, Japan, and informed consent were obtained, we studied 20 surgical patients (American Society of Anesthesiologists physical status I or II; aged between 51 and 83 yr) with normal airways. Anesthesia was induced with propofol, and the patient was paralyzed with vecuronium. An inflatable face mask (King Systems Co., Noblesville, IN) was applied on the face, and the airway was secured by triple-airway maneuver. The lungs were inflated with a ventilator (7800 ventilator; Ohmeda, Tokyo, Japan) in the volume control mode (10 ml/kg), with a frequency of 12/min and an inspiratory/expiratory ratio of 1:2. Expiratory tidal volumes were recorded from the 6th to the 10th breath. A 16-French-sized nasogastric tube (SF-GX1620; TERUMO, Tokyo, Japan) then was inserted into the right nostril, and the nasogastric tube was placed out, with the proximal end exiting under the right lateral part of the mask. The expiratory tidal volumes were measured in the same manner. Next, the gas leak between the nasogastric tube and the cushion of the mask was covered with the denture adhesive without changing tube position (fig.1). Tidal volumes then were measured in the same manner and expressed as a percentage of the volume measured before nasogastric tube insertion.

With the nasogastric tube in place, expired tidal volume was 52 ± 13% of that recorded before tube insertion. When the denture adhesive was applied, expired tidal volume increased to 71 ± 12% of the control value ($P < 0.001$ *vs.* value obtained before application of the adhesive; paired *t* test). The use of denture adhesive clearly reduced the gas leak around the nasogastric tube and may offer some advantages in cases when anesthesia must be induced or managed in individuals with such devices *in situ*.

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Mask Tolerance and Preoxygenation: A Problem for Anesthesiologists but Not for Patients

To the Editor:—Preoxygenation is a simple but vitally important method to expand the time after induction of anesthesia and paralysis until the patient is endangered by hypoxia.[1] This additional time may be lifesaving in "cannot ventilate–cannot intubate" situations. Although it had been discussed by the American Society of Anesthesiologists Task Force on the Management of the Difficult Airway to include a statement on "routine" preoxygenation of every patient—if possible—before induction of general anesthesia (J. L. Benumof, M.D., written communication with W. S., September 2000), the current version of the difficult airway algorithm[2] contains no such statement. Anesthesiologists often argue that, for the patient, the mask represents a significant discomfort and that many patients find preoxygenation objectionable.

We recently have tested this hypothesis in 100 patients using an anonymous standard quality control inquiry with additional questions about oxygen mask tolerance. Simultaneously, 76 anesthesiologists were interviewed anonymously about their estimation of patient discomfort during preoxygenation.

Patients and anesthesiologists estimated the discomfort on a continuous scale from 1 (no discomfort) to 10 (maximum discomfort). The results are shown in figure 1. Preoperatively, the patients expected a moderate discomfort caused by the mask (median 2), whereas postoperatively, the patients mentioned no discomfort (median 1, $P < 0.01$ *vs.* preoperatively, Wilcoxon signed rank test). The anesthesiologists overestimated patient discomfort caused by the mask significantly, with a median of 5 ($P < 0.01$ *vs.* preoperative and postoperative estimation of patient discomfort, Krustal–Wallis one-way analysis of variance followed by the Dunn multiple comparison procedure). Patient discomfort during preoxygenation was not different from the discomfort during other established procedures, such as the placement of an intravenous line ($P = 0.3$).

Only very few patients experience significant discomfort caused by the mask during preoxygenation. A marked overestimation of the average patient discomfort by the anesthesiologist may contribute to the reluctance sometimes seen to use routine preoxygenation.

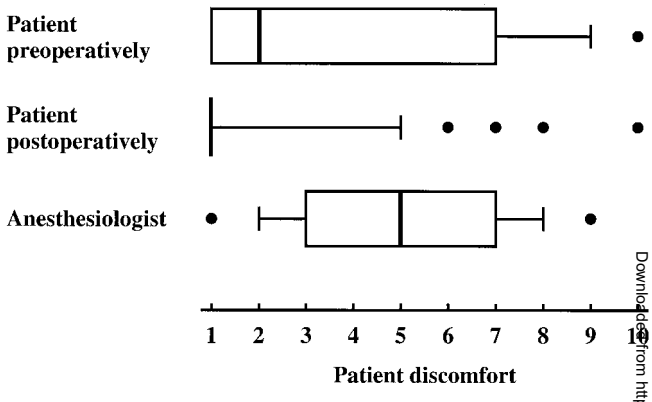

Fig. 1. Patients' and anesthesiologists' estimation of patient discomfort caused by the oxygen mask during preoxygenation on a continuous scale from 1 (no discomfort) to 10 (maximum discomfort). Shown are the median and 5th and 95th percentiles as box-and-whisker plot and outliers as single data points. The anesthesiologists markedly overestimated the discomfort of patients during preoxygenation.

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