

Which Troponin I Peak Concentration?

To the Editor:—We read with interest the article of Conzen *et al.*¹ demonstrating a reduction of cardiac troponin I release after coronary artery bypass grafting on the beating heart when sevoflurane was compared to propofol. Most previous studies identified peak postoperative troponin I as the major endpoint.^{2,3} It is not clear from the article of Conzen *et al.* which was the peak postoperative troponin I value in the two groups and whether this value was significantly different.

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In Reply:—We appreciate the interest of Drs. Zangrillo, Landoni, and Putzu in our article.¹ The authors state that we did no statistical analysis on the peak troponin I values found in the patients of our study, whereas other authors based their conclusions on such data.

The main reason for us to refrain from comparing “peak” troponin I was because of the discontinuous character of this parameter, hence the time intervals that elapsed until the next blood sample was collected. All parameters that cannot be determined continuously have an immanent risk that the true peak may be missed. In the early postoperative period of our study, blood samples were obtained every 3 h. Thereafter, even longer intervals were allowed. Considering these intervals, we cannot exclude the possibility that the true troponin I peak value in our patients was missed. This is supported by the finding that the individual maxima were obtained at different measuring

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points, and that would render a direct comparison problematic. We therefore compared concentrations found at the specific measuring point as well as the changes over time. This was accomplished by two-way analysis of variance.

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Cerebrospinal Fluid Physiology and Cerebrospinal Fluid Drainage

To the Editor:—It was interesting to read the case report by Fleck *et al.*¹ suggesting an appropriate duration of cerebrospinal fluid drainage after thoracoabdominal aneurysm repair. However, toward the end of their discussion, the authors mention that draining 1,700 ml cerebrospinal fluid over 24 h is seven times greater than the normal 250 ml cerebrospinal fluid that is produced in the same period. Although it may be a minor point, cerebrospinal fluid is actually formed at the rate of 500–550 ml/24 h and not 250 ml, as mentioned by the authors.^{2–5}

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In Reply:—We thank Drs. Mahajan and Gupta for their comment. Our data were based on a neuroanatomy text written by the head of the anatomy department of the Medical University of Vienna.¹

However, we wish to stress that the main purpose of our case report is to discuss the clinical value of a longer period of cerebrospinal fluid drainage. The details of normal cerebrospinal fluid physiology are beyond the scope of our article.

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Using the Visual Analog Scale

To the Editor:—I read with interest the report of Woehlck *et al.*¹ on the use of acetazolamide in the treatment of referred pain after laparoscopic surgery. To assess postoperative pain, the authors report using a visual analog scale but state that some patients were unable to respond with a number, whereas others reported a range (*e.g.*, 2–3). The visual analog scale is a continuous line or band along which a subject selects a point corresponding to the intensity of pain experienced.² From the authors' description, one may conclude that patients were verbally rating their pain on a scale from 1 to 10. This method, although acceptable, is neither a visual analog nor a continuous measurement. Instead, the scores obtained are ordinal and, as such, should not be reported as means or analyzed using an unpaired *t* test, as was done in this article. How this misunderstanding may have affected the interpretation of the results and the conclusions drawn is unclear.

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In Reply:—Dr. Cohen raises a multitude of interesting points that may apply to many studies of pain.

Is a verbal report of the value represented on a visual analog scale (VAS) equivalent to pointing to the same number drawn on a line or marking a line mechanically? Studies have found absolute differences but good correlation between different methods in these rating scales.¹ In our study, we accepted verbal responses because of the ease of obtaining such a verbal response at the necessary time, which was immediately after anesthesia and surgery. The potential difficulty in accurately pointing to or physically marking the intended number when postsurgical pain and residual anesthetic effects were present may have made this maneuver impractical.

The rationale for accepting a positive response when a patient could not respond with a number, either verbally or by pointing, is logical because some patients were in too much pain to care about the study while just emerging from anesthesia or were still too groggy to give anything except a positive response regarding the presence of pain. For this reason, we performed a sensitivity analysis substituting mid-range (5), high (7), or low (3) values for positive responses, and recalculated the statistical analysis. For each of these values, the acetazolamide group had lower initial referred pain scores than the placebo group, demonstrating that the actual value of the positive responses was not important.

The question of how to statistically handle the VAS in a general sense, *i.e.*, whether to treat it as an ordinal value or a continuous variable, is important. Fortunately, this question of whether VAS scores can be evaluated using parametric statistical analyses, such as a *t* test or analysis of variance test, or should only be subjected to nonparametric analyses, such as the Wilcoxon rank sum test (nonparametric two sample *t* test) or Kruskal-Wallis test (nonparametric analysis of variance), has already been studied. Parametric tests compare means of data, and these data should ideally be normally distributed. The methods of analysis have been controversial, and early manuscripts from the 1970s and 1980s emphasized using nonparametric tests to avoid erroneous claims of difference when none existed.² However, more recently, Dexter and Chestnut³ determined that the *t* or analysis of variance tests had slightly greater power than nonparametric tests to detect differences between groups and are good choices to compare VAS scores. Using actual data and computer simulations, Dexter and Chestnut further demonstrated that parametric analysis of as few as five categories of VAS data had power similar to that of a continuous VAS, although the use of fewer than five categories had reduced

Table 1. Initial Referred Pain Sensitivity Analysis and Statistical Significance with Inclusion of Nonparametric Analyses

Initial Referred Pain Sensitivity Analysis	Acetazolamide Group	P Value (t Test)	Placebo Group	P Value (Nonparametric)
+ Replaced by 3	0.78 ± 1.55	0.012	3.10 ± 3.42	0.021
+ Replaced by 5	1.00 ± 1.94	0.014	3.40 ± 3.48	0.020
+ Replaced by 7	1.22 ± 2.52	0.022	3.70 ± 3.70	0.020

statistical power to identify a real difference. However, if large numbers of patients (> 16%) cluster at the extremes of the data range, nonparametric analyses may be less prone to error.³

Applying this general question to our study,⁴ 45% (placebo group) and 78% (acetazolamide group) of patients had no referred pain at the initial postoperative measurement, and thus had a VAS of 0, representing no pain and hence a true, absolute zero. The VAS extended to 10, producing 11 categories, which exceeds the minimum of five found to be necessary by Dexter and Chestnut.³ In our acetazolamide study, the data clustered around the lower extreme but not both extremes, and the SDs were small. In addition to the *t* tests, we simultaneously performed a nonparametric analysis, and both methodologies had similar *P* values. Space limitations encouraged us to neither publish nor describe the details of the nonparametric analysis because the results were redundant. Table 1 in this letter is a revision of table 2 from the original study. It includes the *P* values derived nonparametrically for comparison with the parametric results originally published.⁴

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Increased Margin of Safety of Morphine-6-glucuronide Relative to Morphine

To the Editor:—With interest we read the Editorial View by Dr. Jeffrey Gross in the October 2003 issue of *ANESTHESIOLOGY*¹ commenting on our article in that same issue.² In this article, we estimated an effect site (*i.e.*, brain) concentration of morphine-6-glucuronide (M6G) to cause 25% reduction in breathing during hypercapnic and hypoxic stimulation (C_{25}) of 530 and 870 nM, respectively. Dr. Gross compares these values to plasma concentrations of 400 nM from a study by Lötsch *et al.*³ at which no analgesia was perceived. We believe that these numbers are incomparable. Our C_{25} values are the estimated brain concentrations, whereas the value of Lötsch *et al.* is a plasma concentration. Taking into account a blood effect site equilibration half-life of approximately 6 h for M6G,^{4,5} we calculated that the brain M6G concentration in the study of Lötsch *et al.* was maximally 144 nM, which is a factor of 4-6 less than our values. For morphine, the corresponding C_{25} values estimated by us were 30 and 20 nM for hypercapnic and hypoxic breathing, respectively.²

In fact, in a subsequent study in which we tested the analgesic properties of M6G, we observed that a steady state or brain M6G concentration (C_{25}) of 275 nM is needed for a 25% increase in electrical current (pain tolerance).⁵ The corresponding C_{25} value for morphine is 20-30 nM.⁶ This indicates that much greater M6G concentrations are needed to suppress respiration relative to the values needed to induce analgesia greater than placebo.⁵ In sharp contrast, for morphine, these values are of the same order of magnitude. Evidently, how our C_{25} and C_{25} values derived from an electrical pain model reflect concentrations needed for postoperative pain relief necessitates further study.

Finally, we were surprised to find the most important statement in the Editorial View in a footnote. In that footnote, Dr. Gross indicates the need for a respiratory model that adequately describes apnea at

relatively high opioid concentrations. In contrast to the other model described in the review, the model used by us (the power or Leiden model) is able to predict apnea at realistic opioid concentrations. For example, the Leiden model indicates that hypercapnic breathing is abolished at a morphine brain concentration of approximately 100 nM in a volunteer without pain or stress. This is a very realistic value.

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In Reply:—In their response to my editorial,¹ Romberg *et al.* question my conclusion that the jury is still out regarding the relative effects of morphine-6-glucuronide (M6G) and morphine sulfate on analgesia *vis-à-vis* respiratory depression. Clearly, the clinical question is, Does M6G cause less respiratory depression than an equianalgesic dose of morphine sulfate? Unfortunately, the study² addressed in the editorial did not directly answer this question. Although the authors determined

the relative effects of M6G and morphine sulfate on ventilatory drive, no data were presented to establish whether the doses (or brain concentrations) of the two compounds provided equivalent analgesia. For M6G, the brain concentration required to decrease the ventilatory response to hypercapnia by 25% was 528 nM (with *n* = 9, their reported SE of 88 nM translates to 95% confidence limits of 329-727 nM).

The data that Romberg *et al.* cite in their letter to establish the

analgesic potency of M6G come from an abstract that was presented at the October 2003 Annual Meeting of the American Society of Anesthesiologists (San Francisco, California)³ after my editorial had appeared in print. Furthermore, based on the data presented in the abstract, the confidence limits for the analgesic potency of M6G were even wider than those for respiratory depression. The mean brain concentration estimated to increase pain tolerance by 25% was 275 nM, with 95% confidence limits ranging from -7 to 557 nM; these values clearly overlap the 95% confidence range for respiratory depression. Thus, the combined data from the two studies of Romberg *et al.* do not demonstrate a statistically or clinically significant respiratory-sparing effect of M6G. Therefore, I stand by my conclusion that even with their new data, the authors have yet to demonstrate an increased margin of safety for M6G as compared with morphine.

Romberg *et al.* raised concerns regarding the placement of the "most important statement" of my Editorial View in a footnote. This was not

done to minimize the importance of model selection, but rather to avoid interfering with the flow of the associated text.

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Interdental Distance and Direct Laryngoscopy

To the Editor:—I read with interest the article by Calder *et al.*¹ showing that limitation of craniocervical extension to less than 36° limits interdental distance. They propose this as another possible mechanism for difficult airway management. However, the relation between interdental distance and difficult direct laryngoscopy is uncertain. Charters² has shown in a mathematical model that extreme mouth opening actually inhibits direct laryngoscopy. Clinical experience suggests that extreme mouth opening is not practiced as part of laryngoscopy technique. Previous studies showing a correlation between interdental distance and difficult laryngoscopy have included edentulous patients in the measurements,^{3,4} or they have not specified whether edentulous patients were included.^{5,6,7} Not having maxillary teeth (which undoubtedly improves the line of vision to the glottis) is an entity different from interdental distance *per se*.

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Mouth Opening, Craniocervical Extension, and Laryngoscopic Positioning

To the Editor:—I read with great interest the study of mouth opening and craniocervical extension by Calder *et al.*¹ Although the authors were circumspect in their interpretation, the relation between extension and mouth opening may provide an important basis for laryngoscopic positioning. Adnet *et al.*² demonstrated that for routine intubation, the "sniffing position" does not confer any advantage over simple head extension. Put another way (again, for routine intubation), the essential advantage of the sniffing position over the neutral position results from head extension. Other studies of Adnet *et al.*^{3,4} have shown that neither the sniffing position nor simple extension result in the alignment of the oral, laryngeal, and pharyngeal axes. Collectively, these data resulted in a fundamental question: If extension is the essential advantage of the sniffing position over neutrality and extension does not align the oral, laryngeal, and pharyngeal axes, wherein lies its value? The study of Calder *et al.* makes an important step toward

an answer: The value of extension may be that it facilitates mouth opening rather than axis alignment.

It has clearly been shown that limitations of mouth opening are associated with difficult intubation.^{5,6} The study by Karkouti *et al.*⁶ demonstrates that poor mouth opening and craniocervical extension are predictive of difficult intubation. These two "independent" variables have now been shown to be linked, suggesting that limitation of interdental distance is the ultimate disadvantage imposed by impaired extension. It is of interest to consider whether other predictors of difficult intubation, such as micrognathia or decreased thyromental distance, are also related to mouth opening or impaired extension. It is of further interest to consider the Mallampati classification as another measure of mouth opening. Mouth opening is expressed as interdental distance in the study of Calder *et al.*, whereas with the Mallampati classification, it can be expressed as palatoglossal distance. Stated

another way, interdental distance reflects true mouth opening, while palatoglossal distance reflects effective mouth opening, *i.e.*, the actual volume of free space achievable in the oral cavity independent of the interdental distance. When either interdental or palatoglossal distance are limited by conditions such as a decreased cervical mobility, micrognathia, or hyperglossia, intubation is rendered more difficult.

The study of Calder *et al.*, in conjunction with the studies cited above, suggests that craniocervical extension is a crucial aspect of laryngoscopic positioning and that its value lies in optimizing the degree of interdental distance. As the authors suggest, the next step is to investigate this in the setting of true laryngoscopic positioning, evaluating the maximal gape-facilitating angle of extension in anesthetized patients whose oral airways are actively being manipulated. I would further suggest that mouth opening—delineated as true (interdental distance) and effective (palatoglossal distance)—may emerge as the most important measure of difficult intubation and that its maximization may form the basis of rational laryngoscopic positioning.

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In Reply:—We thank Drs. Lee and Mashour for their interest in our article. We established that there is a link between mouth opening and craniocervical position in conscious volunteers. When the subjects were prevented from extending from the neutral position, we found that the *upper* 95% confidence limit of interdental distance was 37 mm, which is the same as the *lower* limit of normal interdental distance in young people.¹

The motions of the temporomandibular joint include depression, elevation, protrusion, retraction, and side-to-side grinding movements.² From the airway management point of view, although almost infinite protrusion should be advantageous, we agree with Dr. Lee that excessive depression can be inimical to our interests.

Dr. Mashour mentions the Mallampati examination. It was the unexpected success of the Mallampati examination in patients with cervical spine disease³ that led us to perform our investigation. As far as we know, the positive predictive value of the Mallampati examination has only exceeded 50% (the same as tossing a coin) in our study³ and Dr. Mallampati's original series.⁴ Unfortunately, the Mallampati examination has not proved to be a useful predictor of difficult direct laryngoscopy in any other patient population.⁵

Our findings may not be reproduced in anesthetized patients. Nevertheless, we suspect that one of the reasons that the sniffing position is popular for direct laryngoscopy⁶ is that it produces craniocervical extension and facilitates mouth opening.

Mouth opening is a complex phenomenon, and we have compli-

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cated its analysis because the craniocervical junction must now be included in the list of factors involved. We hope that some of those involved in airway management or oropharyngeal surgery will find it useful to know that active mouth opening can be facilitated by craniocervical extension and impeded by flexion.

Ian Calder, F.R.C.A.,* John Picard, F.R.C.A., Martin Chapman, F.R.C.A., Caoimhe O'Sullivan, M.A., H. Alan Crockard, D.Sc., F.R.C.S.* The National Hospital for Neurology and Neurosurgery, London, United Kingdom. icalder@aol.com

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Patient Positioning and Ultrasound Guidance Are Important in Bilateral Cannulation of Internal Jugular Veins

To the Editor:—We congratulate Stocchetti *et al.*¹ on their interesting case report and the favorable outcome of the patient. We agree with the authors that bilateral cannulation of internal jugular veins may worsen intracranial hypertension. Nevertheless, we wish to make some comments:

For internal jugular vein cannulation, their patient's head and torso were placed in a flat position, and the head was even slightly rotated. All these procedures are known to increase intracranial pressure in

patients with reduced intracranial compliance.² In the context of increased intracranial pressure, we wonder why the internal jugular vein route was preferred for the insertion of the 8-French introducer and Swan-Ganz catheter to the subclavian or external jugular vein route.

In our opinion, Doppler or B-mode ultrasound guidance should always be used in these patients. Recently, we were able to demonstrate the safe cannulation even of the internal jugular veins in patients

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with reduced intracranial compliance under Doppler guidance without the need for change in body position.³

Following the arguments of Stocchetti *et al.* that the introducer itself was responsible for the obstructed cerebral venous drainage, a B-mode ultrasound image of the left internal jugular vein should have shown a small-sized vessel. With regard to the high thrombogenicity of large-bore catheters in small-sized veins, another access route would have been preferred.⁴ In this context, we refer to a fatal incident of intracranial hypertension we encountered after tracheostomy.⁵ During neck extension, the patient experienced brain herniation due to obstruction of the accessory brain drainage pathway (vertebral venous plexus) in the context of unrecognized bilateral internal jugular vein obstruction (left: thrombosis after central venous cannulation; right: hypoplastic vein).

Blood drains from the brain by two major routes: the internal jugular veins and the vertebral venous plexus.^{6,7} Valdueza *et al.*⁸ have shown that predominance of the jugular veins in cerebrovenous drainage is limited to the supine position. When outflow through the internal jugular veins is compromised, the vertebral system becomes the major channel for blood leaving the cranium.⁶

Various clinical implications, such as bilateral neck dissection, or metastatic spread of tumors, including the controversies about head positioning in increased intracranial pressure, underline the importance of a postural influence on cerebrovenous drainage.⁸

In patients at risk, such as patients with reduced intracranial compliance, central venous access procedures should be performed under ultrasound guidance.³

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A 4.42% Reduction in the Cross-sectional Area of a Jugular Vein Cannot Result in Doubling of the Intracranial Pressure

To the Editor:—We read with interest the case report by Stocchetti *et al.*¹ entitled “Bilateral Cannulation of Internal Jugular Veins May Worsen Intracranial Hypertension.” We agree with the authors’ hypothesis that bilateral cannulation of the internal jugular veins with large-bore catheters in patients with increased intracranial pressure (ICP) and limited buffering capacity of the intracranial system may cause an additional increase in the intracranial volume and hence ICP. We disagree, however, that their case report supports this hypothesis.

The authors describe a head-injured trauma patient with increased ICP to 30 mmHg. A 16-gauge catheter had been placed retrograde in the bulb of the right internal jugular vein. After placement of an 8-French introducer into the left internal jugular vein, a pulmonary artery (PA) catheter of unspecified size was inserted. With the patient supine, the ICP increased to more than 50 mmHg. After removal of the introducer, leaving the PA catheter in place, the ICP suddenly decreased to previous levels.

Although an increase in resistance of the jugular vein may theoretically result in an increase in ICP, we disagree with the authors that their case report illustrates this concept. To unequivocally prove this, the authors should have measured jugular venous pressure proximal to the insertion site both before and after the insertion of the catheters and shown a significant increase in jugular venous pressure after their insertion. Only then could they relate the increase in ICP to the obstruction of blood flow secondary to an increase in jugular venous resistance due to the presence of the catheters.

The authors believe the cause of the increase in ICP after insertion of the 8-French introducer (and a PA catheter of unspecified size) and the subsequent reduction of ICP after removal of the introducer (leaving the PA catheter in place) was solely related to the large size of the introducer. An increase in resistance in a blood vessel after the insertion of a catheter is directly related to the degree of reduction of the axial cross-sectional area of the blood vessel (due to the presence of

the catheter) and the length of catheter in the blood vessel. Seven French is the smallest size of PA catheter available in adults, which is not significantly smaller than an 8-French introducer. The axial cross-sectional area of an 8-French introducer is approximately 5.09 mm² versus 3.90 mm² for a 7-French PA catheter, a difference of 1.19 mm². Furthermore, knowing that increasing the length of a catheter in a blood vessel results in increasing resistance to blood flow and considering the greater length of the PA catheter inside the vessel as compared with the introducer, the difference on the effect of increased resistance between the introducer and PA catheter becomes even more insignificant. Therefore, the authors should not have observed such a significant reduction in ICP after removal of the introducer while leaving the PA catheter in place.

The authors observed an increase in ICP to greater than 50 mmHg, which is approximately twice the value noted before the insertion of the introducer (fig. 1 in their published article).

Because cerebral blood flow remained constant according to the authors, the only remaining variable that could possibly explain the doubling of the ICP is a 50% reduction in the cross-sectional area of the internal jugular vein due to insertion of an 8-French introducer. Comparing the axial cross-sectional area of the left internal jugular vein (114.93 mm²)² and that of the introducer (5.09 mm²), one may expect an approximate 4.42% increase in resistance, and hence ICP, compared with baseline values when the introducer is placed into the vein. Considering bilateral drainage of cerebral blood, one would expect an even smaller increase in resistance, approximately half or 2.21%.

The authors state, “Leaving the Swan-Ganz catheter in place, we withdrew the introducer from the vein; the ICP suddenly decreased to previous values (fig. 1).” However, their figure 1 actually illustrates a reduction in the ICP after removal of the introducer to levels even lower than the baseline values of 20–30 mmHg into the range of less than 10 mmHg. There has been an inexplicable “overshooting” of the

decrease in ICP greater than that which should have occurred by merely removing the introducer. Therefore, removal of the introducer cannot be the explanation for their observations. We believe their explanation is erroneous in that it falls short of providing adequate justification for the observed phenomena.

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In Reply:—We are grateful to both correspondents for their stimulating comments. The experience of Drs. Schummer with ultrasound is highly informative and potentially useful for preventing both short-term and long-term complications of jugular cannulation. We agree with them that a previous estimation of the left internal jugular vein (IJV) size in our case could have been extremely useful and that alternative venous accesses should have been used.

The calculations given by Khorasani *et al.* are detailed and intriguing but based on questionable assumptions and partially incorrect. Briefly, they assumed a caliber of the left IJV, calculated a reduction in its caliber, extrapolated a small reduction in flow, and concluded that this reduction could not explain the observed massive intracranial pressure (ICP) increase.¹ The caliber of the left IJV in our patients is unknown. It has been reported that the caliber of the IJV is highly variable and that there is asymmetry between the two sides. In fact, the IJV diameter may range from 2.5 to 12 mm.^{2,3} The assumed left IJV cross-sectional area is based on an article⁴ in which the authors acknowledged as a limitation of the study the inaccuracy of the method used to estimate the jugular vein diameter. We would be reluctant to apply data from that article to this specific patient. Khorasani *et al.* calculated the reduction of the left IJV caliber due to cannulation as the difference between cross-sectional areas, which is appropriate for perfectly coaxial tubes, without considering that the catheter enters into the vessel with a slope. The vein wall is thin and extremely deformable; therefore, the geometry at the level of cannulation could have been profoundly distorted.

However, even accepting these assumptions, Khorasani *et al.* did not consider that the resistance to flow is inversely proportional to the forth power of the radius, if laminar flow is likely to occur. Therefore, in their simplified model, the reduction of the left IJV radius caused by the introducer would cause an 11% resistance increase, rather than a 4.4% increase.

Finally, Khorasani *et al.* state that a 50% reduction in the cross-sectional area is required for almost doubling ICP: This assumption is definitely wrong. Intracranial pressure is not linearly related to cerebral blood flow because it depends on intracranial volumes, including cerebral blood volume, and intracranial elastance. When intracranial elastance is critically reduced, an increase in cerebral blood volume as small as 2.8 ml may increase ICP from 30 to 50 mmHg (assuming a

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pressure volume index of 13 ml, which is the critical value reported by Marmarou *et al.*⁵). Therefore, a small change in venous outflow can cause a sharp ICP change if cerebral blood volume changes even slightly in the context of exhausted intracranial compensatory mechanisms.

The response of the intracranial vasculature to an abrupt ICP increase is complex and usually implies a marked vasodilatation to keep cerebral blood flow constant despite the reduced cerebral perfusion pressure. The opposite happens when the venous outflow improves, because an increase in cerebral perfusion pressure could induce vasoconstriction, which may explain the “overshooting” of ICP after removing the introducer.⁶ We cannot prove this assumption in our clinical case because we did not measure blood flow and intracranial elastance; therefore, we propose our interpretation of the facts as probable rather than proven, as we specified in our case report.

We believe that increased resistance to blood flow in the IJVs due to vascular cannulations remains a potential cause of sudden and deleterious ICP increase.

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Do We Need a Low Dose of Succinylcholine to Facilitate Intubation in Teaching Hospitals?

To the Editor:—We read with interest the article by Naguib *et al.*¹ in which the authors recommended using 0.5–0.6 mg/kg succinylcholine to facilitate tracheal intubation in a rapid-sequence induction. In their study, all of patients were intubated successfully; however, the authors did not provide data regarding whether all patients were successfully intubated on the first try or some patients required two or more tries.

We work in a teaching hospital, and half of the employees in our department are residents and trainees. Routinely, these young anesthesiologists have at most two tries; if they fail, the attending anesthesiologists perform the intubation, when at least 2–3 min has passed since succinylcholine was injected. Although recovery of adductor pollicis from a dose of 0.5–0.6 mg/kg succinylcholine (T₁ to 10%) occurs at

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approximately 5 min after drug is given,² the duration could be much shorter at the laryngeal muscles. During rapacuronium-induced (1.5 mg/kg) neuromuscular blockade, the time of recovery of T₁ to 25% was 3.7 min at the larynx and 10.2 min at the adductor pollicis.³ Therefore, the attending anesthesiologist may face a worse intubation condition than the resident or fellow met. Sometimes an additional dose of succinylcholine is required.

We want to emphasize the advantages of using 1.0 or 1.5 mg/kg succinylcholine for intubation in this condition. First, the quality of the intubation condition is expected to increase with dose. In the study of Naguib *et al.*,¹ the incidence of acceptable intubation was not significantly different whether patients received 0.6 or 1.0 mg/kg succinylcholine, but the incidence of excellent intubation condition was higher if 1.0 mg/kg was administered (80% *vs.* < 60%, 1.0 mg/kg *vs.* 0.6 mg/kg, respectively). This is important for an inexperienced anesthesiologist to perform a difficult intubation under better conditions. Second, the senior anesthesiologist will still have chance for intubation under good conditions without giving additional succinylcholine when a junior doctor fails after one or two tries. The duration of apnea after

1.5 mg/kg succinylcholine is approximately 2 min longer than that after 0.5 mg/kg succinylcholine.⁴ This 2 min is enough for one attempt at performing intubation but is not a difficult problem for anesthesiologists to maintain the artery oxygen saturation of patients by manual ventilation *via* facemask or laryngeal mask airway.

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Should the Intubating Dose of Succinylcholine Be Revisited?

To the Editor:—We read with interest the article of Naguib *et al.*¹ and the Editorial View of Donati² regarding optimal succinylcholine dose used for intubation. In their article, Naguib *et al.*¹ conclude that the recommended dose of 1 mg/kg succinylcholine for intubation at 60 s may be excessive and that reducing the dose to 0.56 mg/kg would provide same intubating conditions in 95% of patients with normal airway anatomy and allow a more rapid return of spontaneous respiration.

In our opinion, patients with normal airway anatomy, as those included in the study of Naguib *et al.*,¹ are not the challenging cases to anesthesiologists. They can easily be intubated even with a reduced dose of succinylcholine, and it is indifferent whether return of spontaneous respiration in these patients is rapid or slow. However, patients who are muscular, are obese, or have abnormal airway anatomy and parturients undergoing cesarean delivery present a challenge to anesthesiologists during intubation. Profound and intense muscular relaxation is necessary to obtain an optimal/best attempt at intubation in these patients, and we question whether this can be achieved using

a reduced dose of 0.56 mg/kg succinylcholine. Finally, if a difficult intubation is anticipated before initiation of general anesthesia, we would be prone to follow the American Society of Anesthesiologists algorithm for difficult intubation rather than using a reduced dose of succinylcholine with a presumption that it would allow a more rapid return of spontaneous respiration in case of failure to intubate.

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What Is the Correct Dose of Succinylcholine?

To the Editor:—Although we find the two studies by Naguib *et al.*¹ and Kopman *et al.*² to be theoretically interesting, we believe that it is dangerous to link them with our everyday anesthetic practice.

The data sheet for Scoline (Evans Medical, Surrey, United Kingdom),³ which used to be one of our available preparations of succinylcholine in the United Kingdom, states in its precautions that "Scoline must never be given when there is doubt about the ability to ventilate the lungs." In keeping with this, it has never been our practice to rely on the short duration of action of succinylcholine as a "get-out clause" in the event of a failed rapid sequence intubation.

The wide variability in a patient's breakdown of succinylcholine is well known, and surely both of these studies show that using a lower dose of succinylcholine makes an intubation attempt more likely to fail. There may then still be the problem of prolonged paralysis.

Because a successful intubation is of paramount importance in this group of patients, we want to have the best intubation conditions. Therefore, on the basis of these studies, we believe it would be more

clinically relevant to have studied the effects on the intubating conditions of using a higher dose of succinylcholine than the standard dose of 1.0 mg/kg rather than studying lower doses.

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Optimal Intubating Dose of Succinylcholine

To the Editor:—Regarding the optimal intubating dose of succinylcholine,^{1,2} I vividly recall the late Francis Foldes, M.D., then Chairman of the Department of Anesthesiology at Montefiore Medical Center, Bronx, New York, exhorting his residents to not dare use any more than 0.6 mg/kg for tracheal intubation. Furthermore, he taught us to administer the drug over 30–60 s, thus virtually eliminating fasciculations and myalgia, a benefit that neither Naguib *et al.* or Kopman *et al.* mentioned.

In their seminal reports 50 yr ago, Foldes³ and Hampton⁴ point out that the main advantage of succinylcholine is that it provides brief intense relaxation for intubation thus permitting the anesthesiologist to use smaller amounts of nondepolarizing muscle relaxants as dictated by the nature of the case. In the past decade, with the proliferation of newer short-acting nondepolarizing muscle relaxants and inexpensive train-of-four stimulators, this advantage seems to have been deemed insignificant or entirely forgotten. For example, to avoid large doses of nondepolarizing muscle relaxants, Caldwell⁵ suggested intubating with

high doses of propofol or opioids and never mentioned succinylcholine even to condemn its use.

It is nice to see low-dose succinylcholine revisited.

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In Reply:—In their letter, Drs. Lu and Yu posed two questions. The first one concerns the number of attempts at tracheal intubation in our patients.¹ The second question relates to the need for high doses of succinylcholine when teaching residents. All patients in our study¹ were successfully intubated on the first attempt. This study demonstrated that, for routine tracheal intubation, there is no need to administer more than 0.6 mg/kg succinylcholine. I believe that, in certain circumstances, as addressed in our article, there might be a need to administer greater than 0.6 mg/kg succinylcholine. Provided that the patient's airway anatomy is normal and it is possible to ventilate the lungs easily (and to maintain anesthesia), there should be no problem with higher doses of succinylcholine in the scenario described by Drs. Lu and Yu. However, when unanticipated difficult tracheal intubation is encountered and accompanied with an inability to ventilate the patient's lungs, the duration of apnea associated with high doses of succinylcholine could be disastrous. Inadequate ventilation and unanticipated difficult tracheal intubation represented, respectively, 38% and 17% of the 1,541 claims in the American Society of Anesthesiologists Closed Claims database.²

I disagree with Dr. Tabboush's contention that patients with abnormal airway anatomy should be given higher doses of succinylcholine. Similarly, morbid obesity is frequently associated with factors that could impose difficulties for mask ventilation, rigid laryngoscopy, and/or intubation (for review, see Adams and Murphy³). It was never suggested or implied in our article that succinylcholine (even in small doses) should be used in patients with anticipated difficult airways. If a difficult airway is anticipated, I believe that the most appropriate and safe course of action is to proceed with an awake fiberoptic intubation. Therefore, it seems that Drs. Messent and Lim must have also misinterpreted our study. I also cannot agree with their comments about the

study being either "theoretical" or "dangerous." Similarly, it has never been suggested that parturients undergoing cesarean delivery should be given less than 1.0 mg/kg succinylcholine. It is clearly stated in our article, "The dose of succinylcholine must be individualized depending on the clinical situation," and "in a patient with increased intracranial pressure or in a patient with a full stomach, decreasing the dose of succinylcholine to less than 1.0 mg/kg might increase morbidity." Parturients undergoing cesarean delivery are included in the latter category.

Increasing the dose of succinylcholine to greater than 1.0 mg/kg, as suggested by Drs. Messent and Lim, will never guarantee "the best intubation conditions" in all of their patients. The effect of increasing succinylcholine doses and the factors contributing to intubating conditions are discussed in detail in our article.

I thank Dr. Kron for his kind comments. However, I believe that slow administration of succinylcholine has never been shown to be effective in preventing fasciculations or myalgia.

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In Reply:—We certainly have no argument with the position of Drs. Messent and Lim that when serious doubt exists about one's ability to secure a patent airway, the administration of neuromuscular blocking agents is contraindicated. As our data¹ make clear, even after doses of succinylcholine as low as 0.40 mg/kg, significant recovery (at the adductor pollicis) may on occasion not begin until 6–7 min have elapsed. Nonetheless, we feel compelled to point out that even experienced anesthetists may misjudge the ease of tracheal intubation. In these circumstances, the shorter the period of “cannot intubate, cannot ventilate” is, the better.

Forty years ago, when the senior author was a resident, a common intubation sequence consisted of little more than 4 mg/kg thiopental and 0.60 mg/kg succinylcholine. In that era, this protocol produced some fairly “ugly” intubations. Four decades later, after an induction consisting of propofol and a short-acting opioid, the same dose of succinylcholine produces highly satisfactory conditions for tracheal intubation in the majority of patients. For routine nonemergent intubations, we rarely exceed this dose. Nevertheless, as we pointed out in our conclusion, when complete neuromuscular block is critical, doses of succinylcholine as high as 1.0–1.5 mg/kg may be still be appropriate.

We would also like to thank Dr. Kron for his kind remarks. However, we think that the efficacy of the slow administration of succinylcholine

as a means of reducing the magnitude of fasciculations and the incidence of postoperative myalgia is controversial. Certainly, this stragem has been offered by several authors.^{2–3} On the other hand, there is equally convincing data to suggest that this technique is not efficacious.^{4,5} We would suggest that the hypothesis that small initial doses of succinylcholine produce a “self-taming” effect remains unproven.

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Outbreak of Severe Acute Respiratory Syndrome in Singapore and Modifications in the Anesthesia Service

To the Editor:—The World Health Organization issued a global alert on atypical pneumonia and severe acute respiratory syndrome (SARS) and the risk to healthcare workers on the March 12, 2003, after reports from Hong Kong, China, and Vietnam.* This letter reports the changes in anesthetic practice in our hospital during the 2-month outbreak in Singapore.

All patients who had fever and dyspnea were treated as suspect SARS patients, even if they had no SARS contact history. All elective surgery was postponed during the outbreak; only emergency and cancer operations were performed. The operating complex reception staff completed a checklist (table 1) and checked the temperature of all patients. Patients with unexplained temperatures 37.5°C or higher had surgery delayed until a cause was established. Patients were categorized as low-, medium-, and high-risk patients (table 2).

All staff were provided with thermometers to check and report their temperature thrice daily. Anyone with a temperature 37.5°C or higher had to stop work and have a checkup at the staff clinic. All staff kept a log of all patients they had contact with, to facilitate contact tracing, and the information was collated daily centrally. The anesthesiology and operating room (OR) staff were grouped into two teams working alternate stretches, so that if one team became infected, the other team could take over and maintain the service. Contact between the two teams was strongly discouraged, and all department meetings were postponed.

All staff had to pass an assessment on the correct use, removal, and disposal of personal protection equipment. At mask-fitting and leak-testing exercises, different makes of masks were tried, and those who were unable to achieve a good mask fit were posted out of the OR to areas with low risk of SARS exposure. For low-risk patients undergoing

surgery in the general ORs, all OR staff donned N95 masks, goggles, splash-proof gowns, and gloves. The anesthesiologists practiced double gloving and removed the outer gloves immediately after any invasive or airway procedure before handling clean equipment. Eight medium- and high-risk patients were considered SARS exposed or suspect SARS patients, and they underwent surgery in the isolation OR. These patients were transferred into the operating complex wearing an N95 mask if they had no dyspnea, and the anesthesiologists used additional protection of powered air-purifying respirator systems (3M Jupiter Air filter unit and HT-101 hood; 3M UK PLC, Bracknell, United Kingdom; or T4 Personal Protection System; Stryker Instruments, Kalamazoo, MI) and surgical gowns. Probable SARS patients were not operated on in this hospital but were transferred to a designated SARS hospital.

The OR at the end of the last bank of ORs, closest to an entry point into the operating complex, was designated the isolation OR. Access to this OR was reduced to two points with double doors, and the other doors were sealed. All equipment not used during a particular opera-

Table 1. Checklist for Suspected SARS before Admission of Patient into the OR

Cough, shortness of breath, breathing difficulty, and body aches accompanied by increase of body temperature 37.5°C or higher
Close contact with a person who has been diagnosed with SARS or any SARS hospital staff/patient/visitor in the past 3 weeks (<i>close contact</i> means having cared for, having lived with, or having had direct contact with)
Having been discharged from, worked in, or visited the SARS hospital in the past 3 weeks
History of travel to areas reporting cases of SARS
Having been issued with a quarantine order

OR = operating room; SARS = severe acute respiratory syndrome.

* World Health Organization: Severe acute respiratory syndrome (SARS). March 12, 2003. Available at: http://www.who.int/csr/don/2003_03_12/en/. Accessed September 8, 2003.

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Table 2. Classification of Patients Based on Checklist

Patient Response	Category of Risk
No to all five questions in checklist and temperature < 37.5°C	Low
Yes to any question and temperature < 37.5°C	Medium
No to all questions and temperature ≥ 37.5°C	Medium
Yes to any question and temperature ≥ 37.5°C	High

tion was removed before patient entry. To minimize turbulence, all equipment needed for the procedure was kept within the OR rather than in the preparation room.

Our ORs are usually at a higher pressure (0.038–0.075 mmHg) with respect to the scrub room, preparation room, and corridors so that air flows from a clean to a less clean area. Although this system may help to prevent surgical infection, it may hinder containment of a virulent infection within the isolation OR. To reduce the spread of potentially contaminated air out of the isolation OR, its ventilation system inflow was reduced and the exhaust was increased, making its pressure 0.038 lower than its scrub and preparation rooms, 0.075 lower than the other ORs, and neutral with the corridor. With this rebalancing, the air changes were reduced from 334 changes/h over the operating zone and 56 changes/h for the whole OR to 192 and 36 changes/h, respectively. Although the shielding over the surgical zone was no longer as efficient, the laminar airflow still ensured that there was minimal turbulence over the surgical zone. Pressure measurements and smoke tests performed in accordance with Center for Disease Control guidelines showed that there was no escape of air out of the isolation OR.¹

To reduce aerosol production and transmission, and equipment and environmental contamination, we used regional anesthesia whenever

possible, and when general anesthesia was required, we used low fresh gas flows, including during bag-mask ventilation. We stopped using nebulizers in the operating complex. We used and changed a heat-moisture exchanger filter, a second microbial filter at the end of the expiratory tubing, circle system tubing, and carbon dioxide absorbent after every patient. To reduce wastage, only one absorbent canister was filled to a quarter. It was not practical to change the fixed tubings and ventilator bellows of the anesthetic machine.

When it was safe to do so, tracheal intubation was performed under full paralysis, and patients were extubated “deep” to minimize coughing and gagging. When disconnection of the patient and the circuit was required to facilitate positioning, the disconnection was made, leaving the heat-moisture exchanger filter attached to the endotracheal tube or laryngeal mask. Low-risk patients recovered in the PACU, whereas medium- and high-risk patients undergoing surgery in the isolation OR recovered fully there and were then transferred to the ward, avoiding the PACU.

Because SARS is a new disease, these changes were not evidence based. Although no patients or staff were infected in the OR, we are unable to determine which interventions were necessary, because everything was done at once. In particular, grouping of staff into cohorts may not have been useful, and these changes have greatly affected operating efficiency and cost.

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Modified Protective Suits for Anesthesiologists Performing Tracheal Intubation for Severe Acute Respiratory Syndrome Patients in Taiwan

To the Editor:—Severe acute respiratory syndrome (SARS) is a disease associated with coronavirus that has been recently described in patients in Asia, North America, and Europe.^{1–3} The outbreak of SARS occurred in northern Taiwan since March 2003 and spread all over the island in the following 2 months. Here we report our experience of using modified personal protective suits during tracheal intubation procedures in 40 patients who contracted SARS.

Since April 2003, there were 70 probable and suspected SARS patients in our medical center, which is a 2,400-bed tertiary hospital. The SARS patients were kept in negatively pressurized isolation units for intensive care. Among them, 14 patients eventually died because of intractable respiratory failure. All criteria for the diagnosis of probable and suspect SARS cases were based on the guidelines made by the World Health Organization. During the outbreak, 40 probable SARS patients were intubated by 8 anesthesiologists and 23 nurse anesthetists. Our practice guideline for tracheal intubation in SARS patients was similar to those mentioned by others.⁴ Because the personal protective equipment recommended by the Centers for Disease Control and Prevention, such as Stryker T4 (Stryker Instruments, Kalamazoo, MI) and powered air-purifying respirators (e.g., 3M AirMate; 3M, St. Paul, MN) were not available during the outbreak, we had to modify our own protective suits and equipment against SARS. The safety and efficiency of our modified suits, however, had not yet been proven. Figure 1 displays mode 1 (an N-95 mask [3M], goggles, protective suits

[Tyvek; DuPont, Wilmington, DE], and gloves), mode 2 (a half face piece with P-100 filter; 3M), and mode 3 (a half face piece with P3-level filter; SR-100, Sundstrom; The SEA Group, Warriewood, NSW, Australia). On top of one of three modes, a protective hood (SR-64; The SEA Group) and a full face shield, which was made of a piece of plastic cloth and a transparency film, were used to cover the head, neck, and front chest (fig. 2, left and middle panels). We also adapted mode 3 by connecting a nonrebreathing oxygen mask with a reservoir bag to the P3 filter (fig. 2, right panel). With such a device, it provided pure oxygen from a portable oxygen cylinder with a flow rate greater than 10 l/min. When air might be drawn from outside the mask when minute ventilation exceeded airflow rate, the virus load of inhaled air should be diluted. With the modified protective suits, 8 anesthesiologists and 23 nurse anesthetists participated in intubating 40 patients during the SARS outbreak. None of our anesthesia staff members contracted SARS.

Case reports of SARS to the World Health Organization were updated on July 2, 2003, including 8,442 cumulative cases and 812 deaths. Surveillance indicates 76% of infections were acquired in a healthcare facility. In 10–20% of cases, the respiratory illness was severe enough to require intubation and mechanical ventilation. The fatality rate among persons with illness meeting the current World Health Organization case definition for probable and suspected cases of SARS was around 3.4%. In Taiwan, there were 674 probable cases and 84 deaths (including 2 internists and 5 nurses) up until July 2. In our medical center, 14 of 70 SARS patients died (including 1 internist). Sixteen healthcare workers were infected, and 13 of them had partic-

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Fig. 1. Modified personal protective suit with mask. (Left) N-95 mask; **(middle)** half face piece with P-100 filter; **(right)** half face piece with P3-level filter. Protective suit (Tyvek) with goggle and surgical gloves are the basic requirements.



Fig. 2. Hood, face shield, and positive flow of oxygen. (Left) SR-64 protective hood; **(middle)** SR-64 hood with face shield made of plastic cloth and transparency film; **(right)** oxygen (greater than 10 l/min) was delivered to mask with P3-level filter from a portable oxygen cylinder. A nonrebreathing mask with a reservoir bag was attached to the P3 filter. The oxygen flow rate should be adjusted according to different minute volumes.



ipated in tracheal intubation procedures on three SARS patients. None of them had worn any protective suits during the intubation procedure and caused a high infectivity rate of SARS (100%, 13 of 13). Although standard precaution against droplets and contact has been shown effective for routine procedure,⁴⁻⁵ a higher level of personal protective equipment is recommended by the Centers for Disease Control and Prevention for high-risk procedures such as tracheal intubation. Because of the limited medical resources during the outbreak, powered air-purifying respirators and Stryker T4 protection systems were beyond our expectation. Using the modified protective suits during tracheal intubation procedures in 40 SARS patients (figs. 1 and 2), none of our anesthesia staff members contracted SARS (0%, 0 of 31). It should be noted, however, that not all of the commercially available protective equipment have been proven to be totally effective against coronavirus infection. Therefore, our limited experience on this suit modification should not be regarded as an accepted standard but only an alternative option when powered air-purifying respirators and Stryker systems are not available. Based on the Centers for Disease Control and Prevention recommendation and a recent editorial viewpoint,⁴ powered air-purifying respirators or Stryker T4 systems should be the minimum requirement when intubating SARS patients cannot be avoided.

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Unanticipated Difficult Intubation due to Significant Change in Mobility at the Neck and Jaw on Assuming Supine Position

To the Editor:—Airway management is a major responsibility for the anesthesia personnel. Patients with obvious signs suggestive of difficult direct laryngoscopy and intubation (*i.e.*, orofacial abnormalities, short thick neck, protruding teeth, high arches palate, and others) are usually treated with extreme caution. However, some patients who appear normal on conventional examination may still present an unanticipated difficult airway. It is all the more alarming in morbidly obese patients who have a higher-than-usual incidence of difficult mask ventilation and precipitous oxygen desaturation.¹ Here I present a case of unanticipated difficult intubation in a morbidly obese patient in whom the difficulty in performing direct laryngoscopy and intubation was a direct result of altered neck extension and mouth opening resulting from a change in the position of the patient from the sitting to the supine position.

A 35-yr-old African-American man (weight, 190 kg; height, 178 cm) presented for gastric bypass surgery under general anesthesia. He had no significant medical history. His preoperative airway examination revealed relatively large face, a thick neck, adequate mouth opening (> 4 cm), and intact upper and lower dentition with no evidence of overbite. He was assigned an airway Mallampati score of II. He had a full range of active neck flexion and extension. All examination was performed while the patient was in the sitting position. A doughnut-shaped pillow (10 cm thick) was placed under his head. Anesthesia was induced with thiopental sodium, and muscle relaxation for intubation was achieved with succinylcholine. Soon after fasciculation had passed, we faced significant difficulty extending the neck and opening his mouth. A slight extension at the neck allowed us to open the mouth enough to introduce the Macintosh No. 4 blade. Further attempts at neck extension were unsuccessful. The headboard of the operating table was lowered, with no improvement in neck extension. A roll of blanket was placed under the shoulder, which seemed to worsen the overall extension at the neck. At that point, the head was supported, and the intubation was accomplished successfully on the second attempt with only the posterior tip of the arytenoids visualized.

At the end of the surgical procedure, the patient was extubated while awake. On the third postoperative day, I reexamined the airway with the patient in the sitting position, with no change in the findings. However, when he was asked to lie on the bed and open his mouth, he could open his mouth only while actively extending his head. On closer examination of his neck, it was clear that his skin and tissues at the back of his neck and, especially, the upper shoulder were thick and excessive. Interestingly, the full range of motion at the neck and adequacy of mouth opening was well persevered on asking the patient to perform these maneuvers while in the lateral position.

Various investigators have found that a Mallampati score of III or IV was a risk factor for difficult intubation.¹ However, the sensitivity, specificity, and negative predictive value of the Mallampati score are poor. In addition, investigators have found no correlation between body mass index and difficult intubation in obese patients.¹

Adequate mouth opening and neck mobility are the two most important variables defining ease of direct laryngoscopy and intubation.² It is generally understood that these variables are well persevered in the supine position. Tham *et al.*,³ in their study evaluating the effect of posture on the Mallampati class, concluded that posture has no effect on the class assigned in the Mallampati test performed in the sitting position. It is well known that both Mallampati and the later suggested

Samsoon and Young modification indicate that these assessments should be performed with the patient in the sitting position.⁴

In the current case, a closer look revealed that as the patient was asked to lie down on the operating table, the tissues on the posterior upper aspect of the shoulder were squeezed in the direction of the back of the neck, thereby crowding the area behind the neck. This tissue at the back of the neck in the supine position seemed to restrict the extension at the neck whenever the patient was placed in the supine position with his shoulder positioned over the relatively hard operating room mattress. Interestingly, the patient could extend his neck to a greater extent when he was allowed to support his upper body on the elbows while in the supine position. Placing a roll of towels under the neck seemed to further limit the extension at the neck by facilitating the redistribution of the tissues to behind the neck.

Yet another interesting finding in this patient was our inability to open the mouth adequately when we placed him supine on the operating room table with his head resting on the doughnut-shaped pillow. The cause of this is unclear, although the patient could demonstrate adequate mouth opening in supine position for as long as he could extend his neck simultaneously. Theoretically, it is possible for the tissues in the neck, especially those in the posterior region of the neck (behind the angle of the mandible), to become crowded enough. Perhaps it is this change in the range of passive mobility at the atlantooccipital joint coupled with restricted jaw mobility in the anesthetized paralyzed patient placed in supine position, which may be one of the causes of unanticipated difficulty in intubation. Because during passive jaw opening the angle of the mandible moves backward, the ease of doing that depends in part on the softness of the tissues that it displaces. In my personal experience, I have encountered this problem most often in patients with thick muscular necks with prominent sternocleidomastoids.

The difficult tracheal intubation is more common among obese than nonobese patients. Unfortunately, among the classic risk factors for difficult intubation, only a Mallampati score of III or IV has been identified as a risk factor in obese patients. It includes assessment of range of extension of the cervical spine, mobility at the temporomandibular joint, maximum mouth opening, and tongue size. Here I have identified two such factors, which may contribute to the increased incident of difficult laryngoscopy and intubation in a subset of obese patients. Consequently, I recommend that all obese patients and patients with thick, short necks be reevaluated while in the supine position for adequacy of range of motion and mouth opening to avoid unanticipated difficult airway on induction.

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