■ CORRESPONDENCE

Severe ADU Desflurane Vaporizing Unit Malfunction

To the Editor:—A 77-yr-old man undergoing insertion of a J-splint for renal obstruction received general anesthesia delivered with an ADU anesthesia machine (Anesthesia Delivery Unit; Datex-Ohmeda, Stockholm, Sweden). A 5% desflurane vaporizer concentration setting with an O_2/N_2O mixture (2 and 3 l/min, respectively) resulted in stable inspired and expired desflurane concentrations (fig. 1). Immediately after lowering the fresh gas flow (FGF) to 0.35 l/min O_2 and 0.35 l/min N_2O, and while maintaining the same vaporizer concentration setting, a dramatic increase in inspired and expired desflurane concentrations to about 14% (15:45) was noticed, as shown in figure 1. The duration of this high concentration was short-lived (< 2 min) and did not trigger an alarm; the vaporizer concentration setting was decreased to 4.5% and was left unchanged throughout the remainder of the procedure (until 16:13). After a rapid decrease of the inspiratory and end-expiratory desflurane concentrations to about 7–8.5%, the concentrations started to increase again, leading to a gradual decrease in blood pressure. Because vaporizer malfunction was suspected, the FGF was increased to 5 l/min O_2/N_2O and was decreased again (to the previous settings) within a period of 1 min (15:56). Inspired and expired concentrations were noticed to decrease and increase again. This maneuver was repeated at 16:03, confirming that indeed something was wrong with the vaporizer output with the use of lower FGF (0.7 l/min). At 16:05, the FGF was therefore increased to 5 l/min. Vaporizer output itself was then checked at low FGF (0.7 l/min) by interrupting ventilation and having the sampling line of the multigas analyzer (Compact Airway Module M-CAiOV, Datex-Ohmeda, Helsinki, Finland) sample gases leaving the common gas outlet (16:10). Desflurane output read 14.5% (at 16:10) during the use of low flows, but matched the dialed 4.5% (16:12) when the FGF was increased again to its previous settings (O_2/N_2O mixture, 2 and 3 l/min, respectively). An alarm message appeared ("Service fresh gas unit."). Anesthesia was continued for a few more minutes for the remainder of the surgical procedure with desflurane and high FGF (5 l/min), and the patient was allowed to awaken without further incident.

On the same day of our observation, a similar case was reported by the Anesthesiology Discussion Group on GASNet.† With FGF of 0.6 l/min O_2 and 0.6 l/min air and a desflurane dial setting at 8%, the desflurane concentration on the agent analyzer display slowly approached 4.5–5.5%. Then, without warning, the desflurane concentration suddenly increased to 15%. It is unclear whether the events were the same as in our case.

The ADU vaporizing unit is an electronically controlled, flow-over, variable bypass, and measured flow vaporizer, and its mechanism of action and performance have been described recently.[1] Vaporizer output increased with lower FGF, with the largest error with FGF of 0.2 l/min (4.3 and 7.3% absolute output measured with 3% and 6% dialed, respectively, in a single instance). In the current case, however, substantially higher total FGFs (0.7 l/min) were used. Very preliminary testing by Datex-Ohmeda indicates that the one-way valve that prevents backflow of saturated vapor from the cassette *via* inspiratory channel toward the bypass channel may have failed to close after lowering the FGF (fig. 2).[2] This problem may be more significant when desflurane is used because the pressure in the desflurane Aladin cassette (Datex-Ohmeda, Stockholm, Sweden) may exceed 1 atm because of its high vapor pressure when the temperature is greater than 22.8°C (boiling point of desflurane at 1 atm pressure). A similar problem in
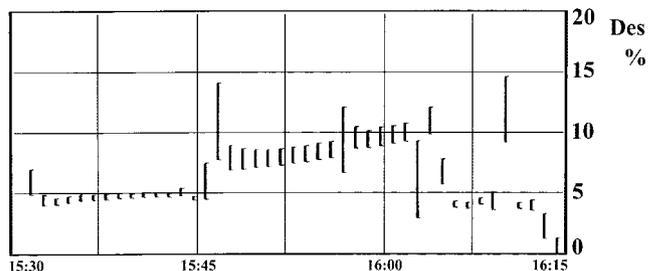

Fig. 1. Desflurane (Des) concentrations over time. The *bars* represent maximum and minimum concentrations of desflurane over the time period (1 min).
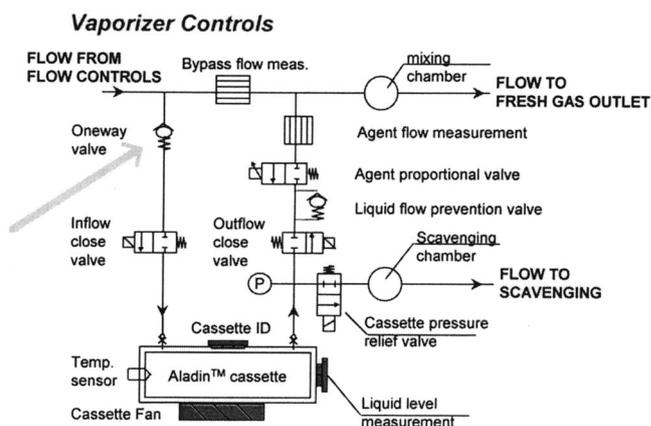

Fig. 2. Diagram of the ADU vaporizing unit. The *arrow* indicates the one-way valve preventing backflow of saturated vapor into the bypass channel. Adapted from reference 2 with permission.

1999 prompted a redesign of this one-way valve and an upgrading of all ADU anesthesia machines in service worldwide (Mr. Ola Lassborn, Quality Manager, Datex-Ohmeda, Stockholm, Sweden, verbal personal communication, March 2003). Despite the new design, this report suggests a continued problem with this valve with the possible delivery of unintended high concentrations of inhaled anesthetics. It is unclear whether the valve still has a design problem or whether only a few defective valves exist (a manufacturing issue). This safety issue is being addressed by Datex-Ohmeda. For now, it is advisable to monitor carefully for excessive agent concentrations when using the ADU Datex-Ohmeda anesthesia machines, especially if desflurane is administered.

Jan F. A. Hendrickx, M.D., Rik M. Carette, M.D., Thierry Deloof, M.D., Andre M. De Wolf, M.D.* *Department of Anesthesiology, OLV Hospital, Aalst, Belgium. a-dewolf@northwestern.edu

References

1. Hendrickx JFA, De Cooman S, Deloof T, Vandeput D, Coddens J, De Wolf AM: The ADU vaporizing unit: A new vaporizer. Anesth Analg 2001; 93:391–5
2. Datex-Ohmeda AS/3 TM Anesthesia Delivery Unit, Technical Reference Manual. Document 893811–3. Bromma, Sweden, Datex-Ohmeda, December 1999, part II, section B, p B-8

---

† Tallmeister A: Runaway vaporizer. Anesthesiology Discussion Group, GASNet, March 14, 2003. Available at: http://www.gasnet.org (Internet, Anesthesiology Discussion Group, Archive Pages, February 2003).

1459

In Reply:—This article is related to a particular customer complaint reported to us on March 17, 2003. We investigated the Fresh Gas Control Unit related to the report and identified the root cause as being a malfunction in the one-way valve. The corrective action for this customer was to install a replacement Fresh Gas Control Unit, including a new one-way valve. As a preventive action, we have initiated an investigation aiming to evaluate if this was an individual component failure or a potential systematic issue.

Ola Lassborn, M.Sc. Datex-Ohmeda Division, Instrumentarium AB, Stockholm, Sweden. ola.lassborn@se.datex-ohmeda.com

---

Noninvasive Cardiac Output Performance Improved after Sufficient Stabilization Time following Decrease of Ventilation

To the Editor:—Tachibana *et al.*[1] carefully designed their study to explore two issues that have not received much attention in the literature: performance of the $NICO_2$ monitor (Respironics-Novametrix, Wallingford, CT) during low tidal volume controlled ventilation and during spontaneous/mixed ventilation. For low minute volume controlled ventilation, the authors report that the $NICO_2$ monitor underestimates thermodilution cardiac output and shows decreased precision and accuracy. It is our opinion that the problem was not with the low minute volume. Instead, there are indications that the time allowed for the patients to stabilize following the 50% decrease in minute volume may not have been sufficient.

Taskar *et al.*[2] showed that the time constant for carbon dioxide output (and end-tidal carbon dioxide) is much longer following a decrease in ventilation than an increase in ventilation. The time constant for a decrease in ventilation was 35 ± 10.7 min after a 10% decrease in ventilation. In other reports, only half of the final change in end-tidal carbon dioxide was realized 16 min after a decrease in ventilation.[3] Changes in end-tidal carbon dioxide and carbon dioxide output are slow because of the time needed for the venous carbon dioxide partial pressure to adjust to the new level needed to allow carbon dioxide excretion to match metabolic carbon dioxide production.

On the basis of these data, it is likely that the venous carbon dioxide levels were still increasing at 15 min after the large decrease in minute volume described by Tachibana *et al.*[1] This is supported by the authors' report that average carbon dioxide output during the low minute volume mode was still 28% less than in the normal minute volume tests. If we can assume that the patients' metabolic rates did not change with the ventilator adjustment, then the venous carbon dioxide level was not yet stable at the time of measurement. Tachibana *et al.* noted that the $NICO_2$ monitor values appeared to be stable during the hypoventilation stage. This does not necessarily imply that the venous carbon dioxide levels were stable, only that they were increasing more slowly. Increasing venous carbon dioxide levels would be expected to cause partial rebreathing cardiac output to read low. This effect would be exacerbated at high cardiac outputs, as the differential end-tidal carbon dioxide signal is smaller.

Also, precision reported for the low minute ventilation test improves considerably (1.27–1.07 l/min), when the single outlier shown in the scatter plot (fig. 1C of the article) is rejected as an artifact (reducing n from 25 to 24). Moreover, not forcing the regression to pass through the origin improves the correlation significantly, from $r = 0.34$ to 0.72.

It is our opinion that had the patient remained in hypoventilation until the carbon dioxide output reached its preadjustment level, then the $NICO_2$ monitor readings would have been as accurate as those observed under normal ventilation. The $NICO_2$ monitor has been designed to calculate cardiac output after changes in ventilation that are typical in clinical practice. The severe decrease in ventilation tested by Tachibana *et al.*[1] was outside the typical expected range of change.

Kai Kück, Dipl.-Ing. Joseph A. Orr, Ph.D.* Lara M. Brewer, M.S.
*University of Utah Health Sciences Center, Salt Lake City, Utah.
jorr@abl.med.utah.edu

References

1. Tachibana K, Imanaka H, Takeuchi M, Takauchi Y, Miyano H, Nishimura M: Noninvasive cardiac output measurement using partial carbon dioxide rebreathing is less accurate at settings of reduced minute ventilation and when spontaneous breathing is present. ANESTHESIOLOGY 2003; 98:830–7
2. Taskar V, John J, Larsson A, Wetterberg T, Jonson B: Dynamics of carbon dioxide elimination following ventilator resetting. Chest 1995; 108:196–202
3. Lumb AB: Nunn's Applied Respiratory Physiology, 5th edition. Boston, Butterworth Heinemann, 2000, p 239

In Reply:—We thank Kück *et al.* for their response to our study of the $NICO_2$ monitor,[1] because dialogue between the designers of equipment and those who put it to practical use is valuable. In this case, the clinical implications of our findings do not seem to be apparent to the authors, possibly because of different interpretations of the role of continuous monitoring devices. For the $NICO_2$ to be of clinical use as a continuous cardiac output (CO) monitor, it should display changes in CO values accurately immediately after minute ventilation ($\dot{V}_E$) is decreased. In fact, we found that after 15 min it still underestimates CO.[1,2] Claiming that the decrease in $\dot{V}_E$ was outside the typical range of change, Kück *et al.* then suggest that we should wait even longer.

Their case would be strengthened if they could specify appropriate delay periods for different levels of change: for example, how long must we wait for correct readings when $\dot{V}_E$ reduction is less than 20%? Clinicians need to acquire reliable data in real time or, at least, know how to interpret values. If we must wait before getting reliable CO data from the $NICO_2$ and can trust the values only when $\dot{V}_E$ is stable, its clinical value as a continuous monitoring device in all situations is open to question.

Having already reported the implications of $\dot{V}_E$ as a factor in decreased precision and accuracy of $NICO_2$ monitoring,[1] we were already aware that 15 min was insufficient time for venous carbon

dioxide ($P\bar{v}_{CO_2}$) to stabilize. We do not, however, fully agree when Kück *et al.* claim that a baseline drift in $P\bar{v}_{CO_2}$ ($\Delta P\bar{v}_{CO_2}$) leads to the underestimation by NICO$_2$. We assume that $P\bar{v}_{CO_2}$ changes exponentially with a time constant of 35 min[3] and that $P\bar{v}_{CO_2}$ and Pa$_{CO_2}$ change in parallel to reach eventual values. Thus, because Pa$_{CO_2}$ increased from 35.5 mmHg to 51.9 mmHg,[1] $\Delta P\bar{v}_{CO_2}$ during the 50-s rebreathing period is calculated as 0.25 mmHg.

The NICO$_2$ system uses the following equation:

$$(-\Delta\dot{V}_{CO_2}) = CO \times (\Delta Ca_{CO_2} - \Delta C\bar{v}_{CO_2}) \qquad (1)$$

Here, $\Delta\dot{V}_{CO_2}$ is the change in carbon dioxide production between normal breathing and carbon dioxide rebreathing; $\Delta C\bar{v}_{CO_2}$ is the change in venous carbon dioxide content; and ΔCa_{CO_2} is the change in arterial carbon dioxide content. If the carbon dioxide dissociation curve is linear, the error caused by baseline drift in $C\bar{v}_{CO_2}$ should mirror the ratio of $\Delta C\bar{v}_{CO_2}/\Delta Ca_{CO_2}$, which is equal to $\Delta P\bar{v}_{CO_2}/\Delta Pa_{CO_2}$. Because ΔPa_{CO_2} during the rebreathing is approximately 2 to 6 mmHg,[4] baseline drift of $P\bar{v}_{CO_2}$ at 0.25 mmHg may be responsible for 4–13% underestimation, much smaller than the value we observed (30% underestimation).[1] The designers have proposed a new algorithm in which it is unnecessary for $P\bar{v}_{CO_2}$ to reach a constant during carbon dioxide rebreathing CO measurement.[5] We do not know if the system that we evaluated incorporated this revision, but even if it did, something else may be causing the discrepant underestimation.[6]

In clinical situations, whether intentional or unintentional, reduction of $\dot{V}_E$ to half is common. Mechanical ventilation at low tidal volume is a standard technique in cases of acute lung injury or adult respiratory distress syndrome. For example, in treating a patient with adult respiratory distress syndrome due to sepsis, during ongoing CO monitoring with NICO$_2$, tidal volume would be decreased. In an actual example, Amato *et al.* reported decreasing tidal volume from 661 ml to 362 ml and observed Pa$_{CO_2}$ to increase from 38.1 mmHg to 58.2 mmHg.[7] In instances of pneumothorax, asthma, pulmonary bleeding, endotracheal tube misplacement, and numerous other clinical situations, $\dot{V}_E$ is likely to suddenly decrease.

Although it seems reasonable to reject a single outlier in scatter plots (figs. 1C and 2C of the article)[1] for linear correlation and bias analysis, the statistical effects of disregarding this point are minor: correlation coefficient, 0.34 to 0.40; slope of linear regression, 0.70 to 0.68; bias, −1.73 to −1.88; and precision, 1.27 to 1.07. Ultimately, reducing n from 25 to 24 is useful because it enables us to observe a more consistent underestimation of CO after decreased minute ventilation.

The purpose of our study was to probe the limits of clinical usefulness of the NICO$_2$ system. We appreciate that because it is noninvasive, easy to use, and works well when $\dot{V}_E$ does not dramatically change, the system generally provides convenient and effective clinical monitoring. Without first evaluating the parameters within which a device provides useful data, however, it would not be prudent, in critical situations, to rely on information from any monitor. Unless clinicians are aware of the limits of the NICO$_2$ system, blind trust may result in unnecessary use of catecholamines and other less-than-optimal judgment. We thank the designers for providing a piece of equipment that makes it easier for physicians to provide attentive care to patients, and we welcome this opportunity to reiterate that it is crucially relevant for clinicians to be aware that the real-time monitoring accuracy of the NICO$_2$ system is affected by changes in $\dot{V}_E$.

Kazuya Tachibana, M.D.,* **Hideaki Imanaka, M.D. Muneyuki Takeuchi, M.D. Masaji Nishimura, M.D.** *National Cardiovascular Center, Osaka University Hospital, Osaka, Japan. ktachiba@hsp.ncvc.go.jp

References

1. Tachibana K, Imanaka H, Takeuchi M, Takauchi Y, Miyano H, Nishimura M: Noninvasive cardiac output measurement using partial carbon dioxide rebreathing is less accurate at settings of reduced minute ventilation and when spontaneous breathing is present. ANESTHESIOLOGY 2003; 98:830–7

2. Tachibana K, Imanaka H, Miyano H, Takeuchi M, Kumon K, Nishimura M: Effect of ventilatory settings on accuracy of cardiac output measurement using partial CO$_2$ rebreathing. ANESTHESIOLOGY 2002; 96:96–102

3. Taskar V, John J, Larsson A, Wetterberg T, Jonson B: Dynamics of carbon dioxide elimination following ventilator resetting. Chest 1995; 108:196–202

4. Haryadi DG, Orr JA, Kuck K, McJames S, Westenskow DR: Partial CO$_2$ rebreathing indirect Fick technique for non-invasive measurement of cardiac output. J Clin Monit 2000; 16:361–74

5. Kuck K, Haryadi DG, Brewer LM, Orr JA: Mixed venous CO$_2$ does not need to remain constant during CO$_2$ rebreathing cardiac output measurements (abstract). ANESTHESIOLOGY 2000; 93:A331

6. Yem JS, Tang Y, Turner MJ, Baker AB: Sources of error in noninvasive pulmonary blood flow measurements by partial rebreathing: A computer model study ANESTHESIOLOGY 2003; 98:881–7

7. Amato MBP, Barbas CSV, Medeiros DM, Magaldi RB, Schettino GPP, Lorenzi-Filho G, Kairalla RA, Deheinzelin D, Munoz C, Oliveira R, Takagaki TY, Carvalho CRR: Effect of a protective-ventilation strategy on mortality in the acute respiratory distress syndrome. N Engl J Med 1998; 338:347–54

(Accepted for publication August 2, 2003.)

Noninvasive Cardiac Output Monitor Algorithms Are More Sophisticated and Perform Better than Indicated in Modeling Paper

*To the Editor:—*Yem *et al.* used a computer model to describe potential sources of systematic error in the partial rebreathing method of measuring cardiac output.[1] They use a complex model to test the basic form of the partial Fick equation first described by Gedeon *et al.*[2] Although the mathematical model of the cardiovascular system described by the authors seems to be elegant and realistic, the partial rebreathing technique that was analyzed using the model bares little resemblance to what is implemented in the actual commercially available system.

It is incorrect to assume that the algorithm used to calculate pulmonary blood flow in a commercially available system is as simple as what

is described in the sales literature. Most modern monitoring devices are based on well-known, yet idealized, equations and derivations of these equations. These equations are only used in actual clinical devices after a series of compensations and corrections have been applied to the raw data. Examples of such devices include thermal dilution cardiac output computers and pulse oximeters. Similarly, currently available partial rebreathing cardiac output computers apply various corrections that compensate for the systematic errors described by Yem *et al.*

Specifically, Yem *et al.* describe excessive rebreathing time as a source for underestimation at high cardiac outputs and insufficient rebreathing times as a source of error when cardiac output is low. Yem *et al.* assume that because the patient rebreathes for 50 s, the partial

Fick equation must be applied using the data from the last breath observed during rebreathing. Because data are collected throughout the rebreathing period, the algorithm inside the monitor may select any of the breaths that occur during rebreathing, thereby eliminating the need for variable length rebreathing periods.

The other systematic error source cited by Yem *et al.* occurs when the rebreathing time is insufficient for equilibrium of the end-tidal carbon dioxide signal to occur. This problem is well documented in the literature related to cardiac output measurement using the total rebreathing method. Various correction techniques dealing with this problem have been described and compared in the literature,[3] and one of these techniques is applied in the $NICO_2$ system.

These and other corrections based on a mathematical model of the lung similar to what Yem *et al.* have described operate within the $NICO_2$ partial rebreathing cardiac output system (Novametrix-Respironics, Wallingford, CT). Jaffe[4] and Haryadi *et al.*[5] describe many of these corrections in two articles; the former is referenced by Yem *et al.* Adequate compensation for the error sources described by Yem *et al.* is evidenced in the results published by Odenstedt *et al.*[6] Although Yem *et al.* should be complimented on what appears to be an elegant mathematical model, it would be more interesting if this model were used to analyze a realistic partial rebreathing algorithm as implemented in an actual, commercially available device rather than a simple idealized equation.

Joseph A. Orr, Ph.D.,* **Kai Kück, Ph.D., Lara M. Brewer, M.S.**
*University of Utah Health Sciences Center, Salt Lake City, Utah.
jorr@abl.med.utah.edu

References

1. Yem JS, Tang Y, Turner MJ, Baker AB: Sources of error in noninvasive pulmonary blood flow measurements by partial rebreathing. ANESTHESIOLOGY 2003; 98:881–7
2. Gedeon A, Forslund L, Hedenstierna G, Romano E: A new methods for noninvasive bedside determination of pulmonary blood flow. Med Biol Eng Comput 1980; 18:411–8
3. Heigenhauser GJF, Jones NL: Measurement of cardiac output by carbon dioxide rebreathing methods. Clin Chest Med 1989; 10:255–64
4. Jaffe MB: Partial CO_2 rebreathing cardiac output: Operating principles of the NICO™ system. J Clin Monit Comput 1999; 15:387–401
5. Haryadi DG, Orr JA, Kuck K, McJames S, Westenskow DR: Partial CO_2 rebreathing indirect Fick technique for non-invasive measurement of cardiac output. J Clin Monit Comput 2000; 16:361–74
6. Odenstedt H, Stenqvist O, Lundin S: Clinical evaluation of a partial CO_2 rebreathing technique for cardiac output monitoring in critically ill patients. Acta Anaesthesiol Scand 2002; 46:152–9

(Accepted for publication August 2, 2003.)

In Reply:—We thank Orr *et al.* for their interest in our article and are pleased to take this opportunity to reply to their comments.

The objective of our article was the analysis of errors that are intrinsic to the standard partial rebreathing technique as reported by Gedeon *et al.*[1] and others. We did not set out to analyze the performance of any specific instrument. We mentioned the $NICO_2$ in the introduction of our article[2] because it is one of the more successful instruments commercially available for monitoring pulmonary blood flow using the partial rebreathing method, and because recent studies have questioned its accuracy at both low and high cardiac outputs.[3-5]

We accept that the $NICO_2$ instrument may use algorithms that are more sophisticated than the standard partial rebreathing equations and that those algorithms do address some of the sources of error we described.[2] Some of the corrections described by Haryadi *et al.*[6] and Jaffe,[7] however, are intended to compensate for right-to-left shunt and parallel dead-space. In our analysis,[2] our computer model was set up to simulate a healthy lung with negligible shunt and parallel dead-space ventilation, so these corrections were not relevant to our study. We used the same blood gas dissociation curves for calculating carbon dioxide content in our model and partial rebreathing equations, so this potential source of error was also removed.

In their letter, Orr *et al.* refer to an algorithm that is used to avoid excessive rebreathing times, but there is not enough information in their letter or in the references cited to enable an independent evaluation of the algorithm. They refer to Heigenhauser and Jones,[8] who describe a number of approaches for correcting estimates of quasi-equilibrium end-tidal pressure of carbon dioxide when rebreathing times are inadequate, but do not indicate which method is used in the $NICO_2$. Haraydi *et al.*[6] report an algorithm for correcting carbon dioxide output estimates for differences between airway and pulmonary capillary carbon dioxide flux during rebreathing. This algorithm requires knowledge of alveolar volume, which appears to be calculated as a function of the Fowler dead-space,[6] but the relevant equations are not reported or referenced.

We understand that commercial interests might motivate against the publication of some of the intellectual property developed for commercial instruments. However, it is unfortunate that some of the more critical algorithms used in the $NICO_2$ to improve the partial rebreathing method have not been published in enough detail to allow the independent evaluation suggested by Orr *et al.*

Johnny S. Yem, B.E.(Hon), B.Sc. Yongquan Tang, M.B.B.S. Martin J. Turner, Ph.D., M.Sc.(Eng.),* **A. Barry Baker, M.B.B.S, D.Phil., F.A.N.Z.C.A., F.R.C.A., F.J.F.I.C.M.** *University of Sydney, Royal Prince Alfred Hospital, Camperdown, Australia.
mjturner@mail.usyd.edu.au

References

1. Gedeon A, Forslund L, Hedenstierna G, Romano E: A new method for noninvasive bedside determination of pulmonary blood flow. Med Biol Eng Comput 1980; 18:411–8
2. Yem JS, Tang Y, Turner MJ, Baker AB: Sources of error invasive pulmonary blood flow measurements by partial rebreathing, ANESTHESIOLOGY 2003; 98:881–7
3. Tachibana K, Imanaka H, Miyano H, Takeuchi M, Kumon K, Nishimura M: Effect of ventilatory settings on accuracy of cardiac output measurement using partial CO_2 rebreathing. ANESTHESIOLOGY 2002; 96:96–102
4. Nilsson LB, Eldrup N, Berthelsen PG: Lack of agreement between thermodilution and carbon dioxide-rebreathing cardiac output. Acta Anaes Scand 2001; 45:680–5
5. van Heerden PV, Baker S, Lim SI, Weidman C, Bulsara M: Clinical evaluation of the non-invasive cardiac output ($NICO_2$) monitor in the intensive care unit. Anaesth Intensive Care 2000; 28:427–30
6. Haryadi DG, Orr JA, Kuck K, McJames S, Westenskow DR; Partial CO_2 rebreathing indirect Fick technique for non-invasive measurement of cardiac output. J Clin Monit Comput 2000; 16:361–74
7. Jaffe MB: Partial CO_2 rebreathing cardiac output: Operating principles of the $NICO_2$ system. J Clin Monit Comput 1999; 15:387–401
8. Heigenhauser GJ, Jones NL: Measurement of cardiac output by carbon dioxide rebreathing methods. Clin Chest Med 1989; 10:255–64

(Accepted for publication August 2, 2003.)

Ralph Milton Waters, M.D.

To the Editor:—I read with great interest the erudite review[1] of *Holding Court with the Ghost of Gilman Terrace: Selected Writings of Ralph Milton Waters, M.D.*, and I have a few comments. First, Waters developed the first academic department not only in the United States but also in the *world*. I base this statement on the knowledge that Sir Robert Macintosh, the first professor of anesthesia in Europe, who developed the department of anesthesia in Oxford, England, made his "first pilgrimage to Madison, The Mecca of anesthetists in the early 1930s" (quotation from Sir Robert) when he was in private practice and there was no interest in Britain in teaching anesthesia at the postgraduate level.[2]

Second, Waters' department, even in its early days, incorporated all the ingredients of current successful academic departments. Teaching and research existed hand in hand with clinical service. The research included work in the laboratory and the operating room. The teaching included frequent departmental meetings.[2]

Third, I have been puzzled for years about the reason(s) for the lack of adequate recognition of this pioneer of our specialty. I know that since 1966 a Ralph Waters award and its companion lecture have been presented in a regional anesthesia meeting (The Midwest Anesthesia Conference), and a recent conference was held in Madison, Wisconsin, but are these adequate tributes for *the* outstanding man of our specialty over the past century? When I started learning anesthesia, I became aware of the name Waters as associated with a "mysterious" (to me) city called Madison, Wisconsin. When I came to Iowa City, Iowa, I made the "pilgrimage" to the city of my hero, which I discovered to be within a few hours' drive. Although a charming university town, I was disappointed by the lack of any tributes I could find to this giant of academic anesthesia and anesthesia in general. I have been told that his reclusiveness after he retired may have contributed to this state of partial neglect. Maybe someone in the future will research his life and his reasons for keeping his distance from the field of anesthesia after his retirement. Perhaps, it was axiomatic that Ralph Waters introduced John Snow, the father of scientific anesthesia, to our specialty in the 1930s.[3] Now is the time to properly introduce Waters, the father of academic anesthesia.

Finally, the review stated elegantly that Waters' thoughts, observations, and recommendations are as relevant to our specialty currently as they were in his time. I venture to add that they are even more poignant in the current crisis facing academic anesthesia.

Mohamed M. Ghoneim, M.D. University of Iowa, Iowa City, Iowa. mohamed-ghoneim@uiowa. edu

References

1. Todd MM: Holding Court with the Ghost of Gilman Terrace: Selected Writings of Ralph Milton Waters, M.D. (book review). ANESTHESIOLOGY 2003; 98:1525–6
2. Macintosh RR: Ralph M. Waters Memorial Lecture. Anaesthesia 1970; 25: 4–13
3. Waters RM: Carbon dioxide absorption from anaesthetic atmospheres. Proc R Soc Med 1936; 30:11–22

(Accepted for publication August 2, 2003.)

Who Invented the "Jaw Thrust"?

To the Editor:—Marshall Hall in 1856 was the first to describe pharyngeal obstruction and suffocation by the tongue falling back during deep chloroform anesthesia,[1] an explanation that Snow had previously rejected.[2] Hall advised turning the patient to the prone position to relieve the obstruction. Shortly thereafter, pulling the tongue outward became common practice to treat such obstruction, and various forceps were designed to grasp the tongue without damaging it.[3,4]

The "jaw thrust," *i.e.*, pulling the mandible upward and forward with the head slightly extended to retract the tongue from the posterior pharyngeal wall, was described by the noted German surgeon, F. von Esmarch, in his 1877 textbook of military surgery.[5] On the Continent, Esmarch is generally regarded as the maneuver's inventor, and the jaw thrust is often called "Esmarch's maneuver" (Esmarchs Handgriffe).[3,4]

However, in 1992, D. J. Wilkinson reported[6] that Jacob M. Heiberg, a professor of surgery in Christiansen (now Oslo), Norway, had already described the jaw thrust in a British medical journal in 1874,[7] thus preceding Esmarch by 3 years. A British textbook of anesthesia now cites Heiberg as the technique's inventor.[8]

The complete history of the discovery of the jaw thrust is even more intricate and complex. In the same year (1874) in which Heiberg reported his maneuver in Great Britain, he published an identical article in the *Berliner Klinische Wochenschrift*, a widely read German medical weekly.[9] His paper prompted C. Langebuch, a German surgeon, to write to the journal's editor.[10] Langebuch disputed Heiberg's priority and claimed to have learned the technique from Esmarch in 1866 when he was a student in Kiel. Heiberg answered Langebuch's letter in a later issue of the *Berliner Klinische Wochenschrift*.[11] He had, he said, written to Esmarch to apologize for his hasty and mistaken claim. Esmarch had answered that he had indeed used the jaw thrust since 1866 but that he was not its inventor. He had learned it from J. S. Little, a British surgeon who had visited him in Kiel in 1866, before leaving for India in 1868. Esmarch's letter may have prompted O. Kappeler to mention "a Dr. Little (?)" as the possible inventor of the technique in his textbook *Anaestetica*, published in 1880.[3]

An extensive search of the British medical directories and rosters of the surgical colleges has failed so far to identify a J. S. Little answering to Esmarch's description.

In fact, a few years before Esmarch or Heiberg, Joseph T. Clover had described the jaw thrust in minute detail in an 1868 lecture to London dentists.[12] He emphasized its importance to anesthetists in later articles in 1871 and 1874.[13,14]

Who was first? So far, one must view J. T. Clover or J. S. Little as the discoverer of the jaw thrust. Little, of course, may well have been a student of Clover. Thus, who was first remains unclear, but it certainly was not Esmarch or Heiberg.

Ray J. Defalque, M.D., Amos J. Wright, M.L.S. School of Medicine, University of Alabama at Birmingham, Birmingham, Alabama. ajwright@uab.edu

References

1. Hall M: Asphyxia, its rationale and its remedy. Lancet 1856; 1:393–4

2. Snow J: On Chloroform and Other Anaesthetics: Their Action and Administration. London, J. Churchill, 1848, p 245

3. Kappeler O: Anaesthetica. Stuttgart, F. Enke, 1880, pp 125–6

4. Killian H: Narkose zum Operativen Zwecken. Berlin, Springer, 1935, p 240

5. Esmarch F: Handbuch der kriegschirurgischen Technik. Hannover, C. Rumpler, 1877, pp 115–7

6. Wilkinson DJ: Keeping the airway open: Esmarch's maneuver or Heiberg's heave? Third International Symposium of the History of Anesthesia. Edited by Fink BR, Morris, LE, Stephen, CR. Park Ridge, Illinois, Wood Museum Library, 1992, pp 443–6

7. Heiberg J: A new expedient in administering chloroform. Med Surg Gaz 1874; 1:36

8. Atkinson RS, Rushman GB, Davies NJH: Lee's Synopsis of Anaesthesia, 11th edition. Oxford, Butterworth-Heineman 1993, pp 261–2

9. Heiberg J: Zum neuen Handgriff bei der Chloroformirung. Berlin Klin Wochenschr 1874; 11:49

10. Langebuch C: Zum neuen Handgriff bei der Chloroform-Narkose. Berlin Klin Wochenschr 1874; 11:529

11. Heiberg J: Zum neuen Handgriff bei der Chloroformirung. Berlin Klin Wochenschr 1874; 11:657–8

12. Clover JT: On the administration of chloroform in dental operations. Br J Dental Sci 1868; 11:123–41

13. Clover JT: Chloroform accidents. BMJ 1871; 2:33–4

14. Clover JT: Remarks on the production of sleep during surgical operations. BMJ 1874; 1:200–3