



Adult Acute Care

July / August / September 2003

Bulletin

America's ARDS Protocol? No, But It Has a Role in Clinical Practice

by Richard Kallet, RRT, San Francisco General Hospital, San Francisco, CA

As a clinical coordinator for the ARDS Network and an ardent supporter of our work, I certainly have an opinion on the issues raised by the Alliance for Human Research Protection and Dr. Peter Eichacker and colleagues. However, since this is an ongoing issue I will refrain from direct comments on the supposed "controversy" and instead direct the interested reader to publications that speak much more eloquently than I can.

In particular, I would suggest reading the recent paper by Robert Steinbrook in the *New England Journal of Medicine*, 348 (14):1393-1401 to learn the complete history of the "controversy." Then turn to the accompanying editorial by Jeffrey Drazen (1377-1380). Additional information can be found in the correspondence section of the *American Journal of Respiratory and Critical Care Medicine*, 167:933-936. The four letters found there (including one from Tom Petty, MD, FAARC) point out the numerous flaws in the work of Eichacker et al. Last, the ARDS Network's response, also published in the *American Journal of Respiratory and Critical Care Medicine*, 166:1515-1517 provides a succinct (as much as possible in these situations) refutation of Eichacker et al.

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ARDSNet: The Saga Continues

EDITOR'S NOTE: Earlier this year, we ran a short article in the Bulletin outlining the controversy surrounding the ARDSNet protocol, which calls for some ARDS patients to receive low tidal volume mechanical ventilation. Specifically, the story outlined opposition from Dr. Peter Q. Eichacker, of the National Institutes of Health, to the use of the protocol in the Fluid and Catheter Treatment Trial, or FACCT. Dr. Eichacker, with support from the Alliance for Human Research Protection, argued the study on the original protocol was flawed. The FACCT study was subsequently put on hold by the medical centers involved. Other researchers, however, refuted that opposition, calling the ARDS protocol study "well designed, safe, and important," and safety panels at the universities involved in the FACCT study reached the same conclusion. The Bulletin story sparked interest among section members, and I posted a query to the section e-mail list for input on specific Vts being used around the country pre- and post-ARDSNet. Here are the pertinent excerpts from that original post, followed by "sound bites" from the responses sent in by members. See what your fellow RTs have to say, then read "America's ARDS Protocol? No, But it Has a Role in Clinical Practice." by Richard Kallet, RRT.

Part of the ARDSNet controversy centers around whether the ARDSNet study compared "current practice" to the 6 mL/kg wing. At the community hospital where I worked (both before and after the ARDSNet study), prior to the study, the Vt would usually run 8-12 mL/kg. Since ARDSNet, Vts are running 6-10 mL/kg FOR ALL PATIENTS. The cardiac surgeons still don't like to use PEEP over 5 on their patients, even if FIO₂ is 1.0, opting instead to just increase Vt. However, the anesthesiologists who order initial vent settings post-op now order about 8-10 mL/kg. The pulmonologists are much quicker to order the smaller range for all comers, and especially the ARDS patients. The cardiac team still uses measured weight/kg, and the pulmonology group strives for the predicted weight. However, the ARDSNet protocol is not used as much for two reasons: (1) The culture (heavy from cardiac surgeons) is that PEEP always drops the cardiac output. PEEP is increased from 5 to 7.5, then maybe to 10. This concept is heavily indoctrinated into the nursing staff. (2) One of our pulmonologists just recently started quoting Dr. Eichacker's criticisms.

My question for the section is this: What Vts were being used at your facility and how were they determined, both pre- and post-ARDSNet? If possible, differentiate between ALI/ARDS patients and other patients on mechanical ventilation. — **Jeff Whitnack, RRT, RPFT.**

Regarding cardiac surgery patients, we have adopted a protocol (loosely based on the ARDSNet) where all our heart patients are initially placed on SIMV/PCV rate 10-12, 8-10 mL/kg IBW, 60% FIO₂, and 8-10 of PEEP for initial vent settings. I personally use closer to 8 mL/kg, rate 12, and 10 PEEP. We all know how a little PEEP can adversely affect the hemodynamics of a dry patient post-op, so we watch for it, and adjust accordingly. The lower peak pressures coupled with the higher mean airway pressures usually provide for improved oxygenation and resolution of atelectasis without much affect on Cardiac Output/Index. — **Jeffrey Davis, RRT, supervisor, respiratory therapists, The Cleveland Clinic Foundation, Cleveland, OH.**

Pre-ARDSNet we routinely used 10 mL/kg in all our vent environments. Post-ARDSNet we seem to still use the same. (Must be that easy calculation!) Post-open heart patients often went as high as 12mL/kg, and that has stayed pretty consistent, at 10-12 mL/kg both pre- and post-ARDSNet protocol in the absence of ALI/ARDS. If ALI/ARDS develops, the pulmonologists are always consulted, and most of them then order the ARDSNet protocol for the RT to manage accordingly to patient response.

This past January we implemented the PB 840 vent. The RTs are staying pretty consistent with the utilization of its built-in low-volume strategy at 7.25 mL/kg, with most all patients on this strategy regardless of ALI/ARDS status. We also changed our ventilator set-up policy/guidelines (in the absence of physician order) to be 6-10 mL/kg from a previous 8-12 mL/kg.

Both our intensivists and pulmonologists usually let the RT choose the initial VT setting

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Adult Acute Care Bulletin

published by the
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Case Study: Why Did This Patient Recover, Part Two?

by Jeff Whitnack, RRT

In the last Bulletin, William A. French presented a case titled, "Why Did This Patient Recover?" Reading his account nudged my memory of a case I witnessed many years ago. The reasons for the seemingly spontaneous recovery of the patient presented by Mr. French are a bit of a mystery. My guess is that the Ativan – and just leaving him alone – allowed him to rest and recover. (Or it could have been a transient P.E.?) The patient I present below holds no such mystery. Yet the irony of it all, along with other lessons, continues to make for a story to be told.

Before I dive into the case I need to describe the setting. I was working in a community hospital. The MDs would see their patients on the wards, write the orders, and then go home. Coverage would be provided, but not necessarily by an MD familiar with the patient. The ER had an MD, yet he could only see the patient if requested by the covering MD, or if an outright code blue was called. The problems would begin, obviously, when patients started to "go south." As every decompensation clinically unveiled itself in a respiratory manner (breathing too fast, too slow, just too downright funky, too low an SpO₂) I would be called. Sometimes it would be because the RN had called the MD and a "Prevent All" treatment was ordered in what seemed to be a reflex order. At other times, a concerned RN would want to discuss the patient with me first before calling the MD. So when a patient who had "been fine" just hours earlier was suddenly tachypneic, tachycardic, diaphoretic, and having a barely tenable SpO₂ only with higher O₂ levels administered, the RN and I would be there discussing the various possibilities (MI, sepsis, P.E.) and scrambling to stabilize and do the "diagnostic tango". If this were a movie script the background song would be from Bob Dylan's *Ballad of a Thin Man*: "you know something's happening, but you don't know what it is, do you Mr. Jones?"

Often a routinely predictable scenario would arise. As we partially stabilized and evaluated the patient, with a call put out to the MD (who we hoped would be one both covering and familiar with the patient), the focus of the communication would assume a certain path between myself and the RN. The floor RN would try to get me to agree (or, better yet, strongly suggest to the MD) that "the patient needs to be transferred to the ICU." While such a transfer might indeed be in the cards, I would often point out that effective stabilization and diagnosis shouldn't wait for a transfer – that we should "bring the mountain to Mohammed vs. Mohammed going to the mountain." And, of course, if the ICU RNs heard of the possibility of an admission I would always be asked, "is he really that bad that he needs to come to the ICU?" Often my reply would be that the patient's care needs are indeed both intensive and critical, but whether care takes place on the floor unit, the roof, or in the ICU isn't the primary issue. My point was that if one of the ICU nurses, staffing permitting, could come out and help when such a floor patient was "going south," perhaps we could avert the transfer entirely.

So, with that hospital background in mind, let me tell you the tale.

Mrs. B was in her late 80s. I don't recall the reason for her hospital admission. She was on a floor unit, unmonitored with last SpO₂ 96% on room air. Several hours earlier all her vital signs had been stable. Suddenly, I was called stat at about 11 p.m. She was on 6 L/min nasal cannula with an SpO₂ of 87%, RR was 40+, breathing labored, she was diaphoretic, and her HR was 130. There was no wheezing. By the time I arrived, a quick call to the MD (one not familiar with her case) had given rise to an order for, believe it or not, an Albuterol treatment. The MD was to be called back after that. With not even a remote hope that an Albuterol treatment would have any impact, yet wanting to establish its uselessness as quickly as possible, I gave the Albuterol treatment ASAP using a face mask with O₂, with a NRB mask flush piggybacked on top. SpO₂ came up to barely 92%. There was no improvement after the Albuterol treatment. Meanwhile EKGs and ABGs were being done. Mrs. B's EKG showed no smoking gun. The ABGs by themselves looked normal, but being on a flush NRB mask and having a minimal PaO₂, along with a normal pH/PaCO₂ with an RR hovering around 40 is also hardly normal. So we called the MD back to report the ABG results, the EKGs, and the indiscernible response to the "Prevent All" treatment. The MD asked me, "What do you think is going on?" while

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CASE STUDY: WHY DID THIS PATIENT RECOVER, PART TWO?

informing me that he is totally unfamiliar with Mrs. B. I respond that I don't really know but that it's probably something pretty serious. I predict that unless we can quickly figure out and treat the cause, she will probably end up intubated in the ICU. I mention the patient's advanced age and suggest that he speak with the patient and/or family about whether they want to go that route, as opposed to other options, such as comfort care. The MD says he'll call the family and asks to speak to the RN caring for the patient.

After speaking with the MD, I call the ICU and tell them of the potential impending admission. A bit later the charge nurse comes over and begins to look over the chart. Mrs. B seems to be at least "treading water" in her unstable state. The MD calls back. After discussing the course with Mrs. B's directed decision maker, the decision is made to just make her as comfortable as possible. No ICU transfer, no intubation or CPR. Valium is ordered and given.

Meanwhile the ICU charge nurse is outside reading the patient's chart. Upon reading the last MD entry in the progress notes she sees a "probable DTs" predicted. ("DTs" refers to delirium tremens, or alcohol withdrawal). No medications are ordered to cover such an impending predicted occurrence; apparently nothing about impeding DTs is in the RN care plan either.

After receiving the Valium, Mrs. B makes a rather sudden and "miraculous" recovery. It seems that the catecholamine storm brought on by the alcohol withdrawal put too much stress on the cardiovascular system. The demands of a high HR, along with the anxiety and high RR, sent everything into a downward spiral. Since the treatment for DTs includes Valium, other sedatives, and medications, the intended palliative treatment course was in fact therapeutic in this ironic case.

A strong example of the need for multidisciplinary care. (And I was glad it was only an Albuterol treatment I had given. I wondered what might have transpired had this happened back in the days of Isuprel HHN treatments.) ♦

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AMERICA'S ARDS PROTOCOL? NO, BUT IT HAS A ROLE IN CLINICAL PRACTICE

That said, I will share our experience with the protocol at San Francisco General. We have been using the ARDS Network low Vt protocol as a virtual standard of care for ALI/ARDS patients since September 2000. I hope to be reporting on our experience with the first 170 patients in Monterey and Las Vegas later this year.

I would not go so far as to claim that not using the protocol constitutes malpractice. However, to continue to use high Vt ventilation (≥ 10 mL) with high plateau pressures in patients with ARDS could result in a malpractice case someday. The idea that we should use 6 mL/kg on everyone is just silly and a misreading of the pathophysiologic rationale for the therapy! In the compendium paper to the ARDSNet study, which was published in *RESPIRATORY CARE* (October 2001; 1024-1037), we state up front that there is absolutely no evidence that this protocol should be adapted as standard ventilator practice outside of ALI/ARDS. However, there are other situations where the possibility of ventilator-induced lung injury might be reasonable (for example, trying to cram a 12 mL/kg Vt in a patient with a markedly decreased surface area for ventilation, pneumonectomy, or severe unilateral pneumonia). Perhaps we should just limit people to a Vt of 8-9 mL/kg if it is anticipated that they may require more than 24 hours of support?

Finally, for the record: folks in the ARDS Network are proud of the study, and we think the adaptation of our protocol in some reasonable fashion would be helpful for clinical practice. The most important thing is to get the Vt and plateau pressure down in the neighborhood we played in during the study. But I know most of us would cringe at the hubris that our protocol should somehow morph into the respiratory care version of the Dallas Cowboys – in other words, "America's ARDS Protocol." (My apologies to my friends in Dallas!) ♦

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ARDSNET: THE SAGA CONTINUES

in stable patients. If ARDS/ALI is the diagnosis, they almost always order the ARDSNet protocol. — **Craig Gordon BS, RRT, LRCP, clinical coordinator, pulmonary services, Riverside Methodist Hospital, Columbus, OH**

Our protocol is 6-10 mL/kg IBW (with the option to adjust as needed for optimal blood gas values). After ARDSNet we went down a common path – typically ventilated all patients (excluding cardiac surgery) in the lower range. Now that we've had a few years to look at results, we are ventilating the average vent patient (nonARDS) in a more moderate range (8-10 mL/kg). I gave a short presentation on this topic at our most recent Critical Care Committee meeting, explaining how the current literature suggests that we're potentially causing derecruitment and atelectasis by ventilating patients with normal lung compliance at such low volumes. The cardiac surgeons were very interested and now wish to meet to discuss weaning strategies. — **Merrill Shippy, BS, RRT, director, respiratory care services/Sleep Disorders Center, Olathe Medical Center, Inc., Olathe, KS**

We were using 8-10 mL/kg pre-ARDSNet pretty much across the board. Some cardiac surgeries had larger values, and still do post-ARDSNet. The prevailing thought is that this population is not suffering ALI/ARDS. Post-ARDSNet, 6-8 mL/kg seems to be much more prevalent. When I voice concerns – which I do if I believe that is inadequate for a given patient – we generally compromise in the 8-9 mL range. Often we are shooting for the ARDSNet recommendation of MAP < 30 cmH₂O rather than fixating on tidal volume. — **William S. Demaray, BS, RRT, University Hospital, Albuquerque, NM** ♦

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