Correspondence

Echocardiography in the critically ill

We wish to acknowledge the thorough and extensive review of echocardiographic principles and indications by Donovan and Colreavy,[1,2] emphasizing its potential in the Critical Care setting. They are strong protagonists of transthoracic and transoesophageal echocardiography in the critically ill patient.

However, we believe that they understate the limitations of the techniques, in particular that of transthoracic echocardiography. They state in the second abstract; "Both transthoracic echocardiography (TTE) and transoesophageal echocardiography (TOE) are extremely useful in managing critically ill patients".[2] Although there is considerable literature supporting this statement for transoesophageal echocardiography, many reviews on echocardiography in the Critical Care setting clearly state the limitations of the transthoracic approach, as only briefly outlined by Donovan and Colreavy. Furthermore, our clinical experience of transthoracic echocardiography in the Critical Care setting is that although useful for confirming or refuting certain diagnoses (i.e. cardiac tamponade), that frequently image quality limits the conclusions that can be made about valvular function and anatomy, and segmental left ventricular function due to limited echocardiographic windows.

Echocardiography conceptually is a very useful imaging modality as it is (relatively) noninvasive, portable and provides real-time data. We are advocates of its use in the Critical Care setting in selected cases. However, it is not the panacea of diagnostic imaging and by appreciating its limitations we are better able to use it to advantage in managing critically ill patients.

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In reply

We agree that echocardiography should be reserved for selected patients with specific cardiovascular problems. However we do not advocate that echocardiography should be used in all or even the majority of critically ill patients. The ACC/AHA guidelines[1] for the clinical application of echocardiography in the critically ill or injured patient states that echocardiography is "extremely useful for making the differential diagnosis in haemodynamically unstable patients." Other ACC/AHA Grade 1 (i.e. useful and effective) indications include evaluation of valvular heart disease and suspected aortic dissection or rupture and detection of source of emboli.

Ultrasound technology has advanced rapidly with harmonic fusion, automatic border detection, etc, being available in most modern machines. The vast improvements of imaging have impacted more on transthoracic echocardiography (TTE) than transoesophageal echocardiography (TOE). TOE provides superior image quality but is invasive and associated with a low but measurable morbidity and mortality. The safety of TOE in critically ill patients is insufficiently documented. We believe TTE to be underutilised in intensive care, perhaps because of the lack of 'on site' skilled sonographers. TTE should be *considered* first in critically ill patients requiring echocardiography. In many clinical settings TTE will suffice but when the superior image quality of TOE is essential (e.g. aortic dissection) or TTE images are suboptimal then TOE should be performed. Interestingly, a recent study[2] of the use of echocardiography in intensive care patients during cardiopulmonary resuscitation reported satisfactory transthoracic imaging in the majority of patients, despite this being the most difficult scenario for echocardiographic examination.

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Acid-base and strong ion difference

We wish to make further comment on the acid-base physiology debate recently highlighted in this journal.[1-3] Dr Worthley[2] suggests that the Bronsted-Lowry approach resolved the question: what is an acid? We beg to differ. In a very recent text on acid-base, Abelow[4] uses two definitions of an acid in the physiological setting. The first is the Bronsted-Lowry definition of a proton donor. The second definition - introduced much earlier by Arrhenius - is that an acid is a substance, which when added to a solution, increases the hydrogen ion concentration. The second definition allows molecular carbon dioxide to be defined as an acid. Rather than being fixed chemical laws, these differing definitions are tools to be selected for differing applications. The late Peter Stewart[5] used the second definition of an acid that allows the concept that an increase in plasma chloride concentrations is acidifying. Dr Worthley[2] suggests that describing the chloride anion as an acid is confusing. The same could be said for carbon dioxide using the Bronsted-Lowry approach. Confusion is in the mind of the observer.

Dr Worthley correctly states that, at all times, in distilled water, the hydrogen ion concentration is equal to the hydroxyl ion concentration due to the laws of conservation of mass and electroneutrality. Many clinicians incorrectly believe that the pH of plasma can only change because the ratio of hydrogen ions to hydroxyl ions changes. The pH is the logarithm of the reciprocal of the hydrogen ion concentration and does not reflect changes in the corresponding anions. As Dr Worthley reminds us, changing water temperature from 25 °C to 37 °C changes the pH from 7 (hydrogen ion concentration = 100 ηmol/L) to 6.8 (hydrogen ion concentration = 158 ηmol/L). This is a large change in clinical terms and yet the 50% increase in hydrogen ion concentration - or activity - cannot be due to the addition of acid. How could such a dramatic change in 'acidity' occur? There is only one answer. The hydrogen ions come from increased water dissociation. Stewart made this point repeatedly.

Dr Worthley predicts that the infusion of 6.5 liters of saline would change bicarbonate from 24 mmol/L to 22 mmol/L. In a study published this year, Scheingraber[6] and colleagues demonstrate a change in bicarbonate from 23.5 mmol/L to 18.4 mmol/L with less than 5 litres of saline in a group of patients with a mean body weight of 68 kg. One explanation for the divergence of this new human study from the older animal work quoted by Dr Worthley is interspecies differences.[7] Another is that conventional approaches are flawed.

Dr Worthley challenges the concept that hypoalbuminaemia induces an alkalosis, quoting the lack of respiratory compensation as evidence.[8] This study was performed in critically ill patients with multiple physiological derangements; a population that does not allow the hypothesis to be properly tested. Further, Rossing and co-workers[9] produced clear in vitro evidence that removing albumin alkalinises blood and that adding albumin acidifies blood.

Dr Morgan's editorial[1] examines the issue of strong ion difference and base excess as markers of severity of acid-base disturbance. Stewart[5] stated that base excess is a useful clinical indicator of the severity of acid-base disturbances, a point missed by Siggard-Anderson.[10] Stewart's aim was to explain the mechanisms of acid-base physiology rather than provide straightforward clinical indicators of disturbance.

Dr Morgan[1] is unsure of the value of the term post infusion acidosis. For the last 25 years the review by Garella, Chang, and Khan[11] has been influential in promoting the concept that bicarbonate is diluted when the term 'dilutional' acidosis is used to describe the effect of intravenous fluid administration on acid-base status. But, if Stewart were correct, dilution of bicarbonate has nothing to do with the acidosis following infusion of fluids. The recent study by Kellum et al,[12] provides further experimental evidence that Stewart was correct. Dr Morgan is incorrect in suggesting that Plasmalyte is unlikely to create a post infusion acidosis. Liskaser and colleagues[13] recently conducted a study examining acid-base changes following initiation of cardiopulmonary bypass. Following initiation of bypass, patients with a pump prime of Plasmalyte had a fall in standard base excess of 4.4 mmol/L. A group with a pump prime of Haemaccel and Ringer's Injection had a fall in base excess of 5.5 mmol/L. The difference between the groups was not statistically significant.

One difficulty in determining whether the Henderson-Hasselbalch approach or the Stewart approach better describes the mechanisms of acid-base homeostasis is that bicarbonate accounts for over half of the strong ion difference. Like the chicken and the egg, the question remains: which comes first, changes in bicarbonate or changes in the strong ion difference?

Dr Worthley argues that research throughout this century has supported the original Henderson proposal that bicarbonate is the key to 'metabolic' acid control. We ask, if one were to undertake these studies again starting from the assumption that the strong ion difference is an independent controlling factor, would these studies also support the Stewart approach? We believe so. Furthermore, while Stewart sought to understand the mechanisms of acid-base control, Hasselbalch's adaptation of the Henderson equation was merely intended to describe the state of acid-base balance. In our view, as we move into the next century,

the acid-base story is far from told. We hope that a healthy debate will continue as we seek to address what to us is now a major challenge: to design and execute studies that will define the mechanisms of acid-base regulation in health and disease. Both the Henderson-Hasselbalch and Stewart approaches will continue to have a role. Neither should be dismissed or clung to merely for reasons of history or comfort.

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In reply

Dr Story *et al*, have raised many important issues concerning the concept of acid-base that need to be clarified. Arrhenius defined acids as substances that *ionise* in water to produce hydrogen ions. However, as knowledge about reactions in non-aqueous solutions increased, the definition was found to be too restrictive. The Bronsted-Lowry definition of an acid and a base is independent of solvent, it not only includes all acids that were included by the Arrhenius definition, it includes many bases that were excluded.[1,2]

Carbon dioxide is the acid anhydride of carbonic acid. The latter is considered an acid by both the Arrhenius and Bronsted-Lowry theories. As carbon dioxide does not have a proton, it is not an acid; and Abelow in a later part of the text referred to by Story *et al*, acknowledges this by stating that "to designate carbon dioxide as [an] acid, is not strictly correct, since CO_2 does not itself donate protons".[3] In relation to the chloride ion, neither the Arrhenius nor the Bronsted-Lowry views consider it an acid: as a proton acceptor it is a base.

The question of neutrality of water and the temperature dependency of pH is an interesting one as the pH of pure water over the range that a human can experience (e.g. 25°C - 40°C) varies from 7.0 - 6.76.[4] The answer to the question of "how could such a dramatic change in 'acidity' occur?" being an "increased water dissociation", is not in dispute. However, the implication from the Stewart approach is - that with so much water about, and with its capacity to dissociate, body fluids should follow this range with the variations in body temperatures (e.g. hyperthermic acidosis?). Yet they don't. The reason is that pure water has no buffer capacity, whereas body fluids have an enormous buffer capacity, allowing the body to handle large acid loads with only minimal alteration in pH.

One of the reasons for differentiating pH from H^+ concentration is to separate 'intensive' and 'extensive' variables, the significance of which has long been recognised in thermodynamics.[5] Confusing acidic intensity (as measured by pH, i.e. extremely small quantities of free H^+ or H_3O^+) with acidic capacity (as measured by titratable acidity, i.e. extremely large quantities of H^+) has lead to the misunderstanding of the pH and acid load effects of intravenous solutions,[6] and an overestimation of the effects of 20 mmol of HCl in a

70 kg man,[7] when up to 360 mmol of HCl has been administered without the proposed theoretical effects being observed.[8]

That intravenous 0.9% saline can cause an acidaemia is also not in doubt (caused by a reduction in bicarbonate concentration not an elevation in chloride concentration),[9] and the study by Scheingraber *et al*,[10] referred to by Story *et al*, confirmed that the bicarbonate concentration calculated using either the Henderson-Hasselbalch equation or the Stewart approach produced equivalent results (which should come as no surprise as $HCO_3^- = SID - A^-$).[9] What I found interesting was the fact that the difference between the bicarbonate changes found in humans in this study, compared with that found in animals in other studies, was not explained by the Stewart approach, and that no other reason could be proposed other than the obvious species (and therefore buffering) difference.[11]

Finally, the fact that removing albumin from blood in an *in vitro* experiment alters the pH of blood is also not in dispute; albumin is one of the body's buffers.[12] What is in dispute is the proposal of hypoproteinaemic alkalosis and hyperproteinaemic acidosis as clinical entities.[13] In this regard, the study by Wilkes provided valuable *in vivo* information in a clinical situation where a perceived pH abnormality by the human organism should have been detected, but was not.[14]

While there is a clear relationship between HCO_3^- and the SID,[9] it is the regulation and maintenance of acid base that is important. In man, acid-base balance is regulated by the renal and respiratory system regulation of the bicarbonate pair (a point also made clear by Abelow).[15] Currently, there are no studies that have demonstrated a regulation of body acid-base status by regulation of SID and A_{TOT}.

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In reply

Dr Story and colleagues take issue with a couple of points in the editorial concerning the relative importance of Stewart's physical chemical approach.[1] These objections notwithstanding, I am pleased to note that they do not dispute the main thrust of the argument, and in particular two important points. The first is that Stewart's analysis is one of at least three valid approaches. Devotees from each of the three schools should be capable of peaceful co-existence, as has been argued with such clarity by Schlichtig, Grogono and Severinghaus.[2] Secondly, Dr Story *et al*, concede that while Stewart's analysis can be a useful conceptual tool, it does not have much clinical utility at the bedside.

I am happy to respond to the objections raised. Firstly, they may have misunderstood my position on the proposed term, 'post infusion acidosis'. I merely point out that it is equally possible to cause a 'post infusion alkalosis', for example after infusion of 8.4% sodium bicarbonate (leaving aside all questions of transient paradoxical intracellular acidosis). Similarly metabolic alkalosis can occur after massive infusion of blood anticoagulated with citrate. In other words, although infusion of fluids with an intrinsic strong ion difference (SID) of zero will tend to alter the PCO_2/pH relationship in the direction of metabolic acidosis, there are other

fluids which on infusion have entirely different effects on metabolic acid-base status. The term 'post infusion acidosis' seems merely confusing.

Dr Story *et al*, also object to my contention that Plasmalyte, which has an intrinsic SID of around 50 mEq/L after metabolism of the contained acetate and gluconate, is unlikely to cause a metabolic acidosis unless these organic anions are incompletely metabolised. They refer to a study involving patients subjected to cardiopulmonary bypass. This study is unpublished at the time of writing and thus it is hard to comment without knowledge of the detail. Nevertheless, we were originally informed that in patients for whom the pump had been primed with Plasmalyte, the median arterial base excess post-bypass increased by 1.1 mEq/L compared with pre-bypass values. Dr Story *et al*, now tell us that in these same patients, standard base excess initially fell by 4.4 mEq/L on commencement of bypass. If the original information is correct, they can not claim that priming with Plasmalyte produced a metabolic acidosis post-bypass. In fact the trend was towards a metabolic alkalosis. I can only assume that the authors were able to show that the transient reduction in base excess while on cardiopulmonary bypass was not due to my suggested mechanism – namely incomplete metabolism of acetate and gluconate. If that was not shown, I do not understand the objection. I await the paper with interest.

Finally there is the question of 'hypoproteinaemic alkalosis'. Many advocates of the Stewart approach, including Dr Story and colleagues, insist that this is a clinical entity. However the evidence for its existence *in vivo* is at best slim. The *in vitro* study referred to merely confirms that alteration of A_{TOT} (which in plasma is primarily albumin) changes the normal range of SID (but not PCO_2). This is no surprise.[3] However it does not mean that hypoalbuminaemia causes metabolic alkalosis *in vivo*. For example, one of the most dramatic acute reductions in plasma albumin concentrations occurs in burns patients resuscitated with crystalloid 4mL/kg/% burn in the first day. At the end of that time total protein concentrations are often halved and plasma albumin almost disappears. The only metabolic acid-base disturbance seen under these circumstances is metabolic acidosis and hyperchloraemia, (usually without elevation of plasma lactate concentrations or renal dysfunction). If acute hypoproteinaemia is a genuine cause of metabolic alkalosis, why don't we see it in any of these extreme examples?

All of this aside, it should be pointed out that the appropriate reference compartment *in vivo* for acid-base status is not plasma - it is the extracellular space. The stability of standard base excess (an extracellular space parameter) in acute respiratory acid-base disturbances supports this contention.[4] In the extracellular space, haemoglobin is the predominant contributor to A_{TOT} . Surely Dr Story and colleagues are not arguing that acute anaemia causes metabolic alkalosis, or that polycythaemia causes metabolic acidosis? As has already been pointed out,[2] it is a trap to focus on plasma rather than the ECF when interpreting acid-base status, or to ignore Gibbs-Donnan equilibria, particularly across red cell membranes. Incorrect and confusing concepts such as hypoproteinaemic alkalosis then emerge.

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Difficulty with the Ciaglia Blue Rhino™ dilator

Percutaneous tracheostomy has become the method of choice for tracheostomy insertion in the intensive care patient and numerous techniques have been described.[1-5] In general, the percutaneous techniques that have been used to facilitate the tracheal stoma formation involve either a progressive dilatation (e.g. Ciaglia percutaneous tracheostomy introducer set, C-PTS-100),[3] or a single dilation technique using, forceps,[5] tracheostome,[4] or cutting trocar[1,2].

Recently, a Ciaglia Blue Rhino™ Percutaneous Tracheostomy Introducer Set (C-PTIS-100-HC, Cook Critical Care, Bloomington, USA), has been introduced, where the progressive dilatation stage (previously achieved using different dilators of increasing size) has been changed to a 'one stage' dilation using a single dilator (Ciaglia Blue Rhino™ dilator) that tapers to 38F gauge.

In a patient who had normal neck and tracheal anatomy and required a tracheostomy for long term airway access, the Ciaglia Blue Rhino™ Percutaneous Tracheostomy Introducer Set was used. The procedure

was performed using 300 μg fentanyl and 8 mg vecuronium intravenously. The endotracheal tube was withdrawn until the tip was at the glottis, the balloon was then inflated and the tip of the endotracheal tube was inserted into the glottic entrance. After a standard sterile preparation of the anterior skin area of the neck, the cricoid cartilage was identified and lignocaine 1% with adrenaline 1:200,000 was injected in the subcutaneous tissues immediately below the cricoid. A 2 cm midline transverse cutaneous incision was made at this level, and the anterior neck structures between the incision and the trachea were separated using blunt dissection. A 14 gauge needle and cannula was inserted in the midline of the incision, and directed posteriorly whilst withdrawing on the plunger of the attached syringe. The needle was directed to pass between the first and second tracheal rings and the trachea was identified. The outer plastic cannula was advanced into the lumen of the trachea and the inner needle was removed. The tracheal lumen was once again identified by air being withdrawn from the cannula. A J-tipped Seldinger wire was introduced into the trachea. The short 11 French introducing dilator was then inserted into the trachea over the Seldinger wire to enlarge the hole in the anterior tracheal membrane.

The wet Ciaglia Blue Rhino™ dilator (with the white guiding catheter) was then placed over the Seldinger wire and an attempt to insert the unit into the trachea was made. However, while some difficulty was encountered, after using some force, it was inserted up to the skin mark. Upon removal, the unit was grossly misshapen (Figure 1).

The Seldinger wire and white guiding catheter were replaced and two previously reused 12 and 18 French dilation catheters were used to enlarge the tracheal access site. The Ciaglia Blue Rhino™ was then reused providing the tracheal stoma width required. A 36 French tracheostomy tube was then inserted successfully.

Figure 1. The misshapen Ciaglia Blue Rhino™ dilator with outer plastic cannula and J-tipped Seldinger wire.

The one stage dilation provided by the Ciaglia Blue Rhino™ dilator is a useful addition to the percutaneous tracheostomy technique, however the tip of the dilator is softer than the standard Ciaglia blue dilators, and particular care needs to be taken to ensure the initial dilation by the short 11 French introducing dilator provides a large enough hole to allow the tip of the Ciaglia Blue Rhino™ dilator into the trachea before applying force to the dilator to provide the necessary stomal size.

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