

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 This study is unpublished at the time of writing and thus it is hard to comment without knowledge of the detail.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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REFERENCES

1. Morgan T. Finding common ground in acid-base. *Critical Care and Resuscitation* 1999;1:123-126.
2. Schlichtig R, Grogono AW, Severinghaus JW. Current status of acid-base quantitation in physiology and medicine. *Anesthesiology Clinics of North America* 1998;16:211-233.
3. Rossing TH, Maffeo N, Fencl V. Acid-base effects of altering plasma protein concentration in human blood *in vitro*. *J Appl Physiol* 1998;84:1740-1748.
4. Schlichtig R, Grogono AW, Severinghaus JW. Human $PaCO_2$ and standard base excess compensation for acid-base imbalance. *Crit Care Med* 1998;26:1173-1179.

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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REFERENCES

1. Sheldon CH, Pudenz RH, Tichy FY. Percutaneous tracheostomy. *JAMA* 1957;165:2068-2070.
2. Toye FJ, Weinstein JD. A percutaneous tracheostomy device. *Surgery* 1969;65:384-389.
3. Ciaglia P, Firsching R, Syniec C. Elective percutaneous dilational tracheostomy *Chest* 1985;87:715-719.
4. Schachner A, Ovil Y, Sidi J, Rogev M, Heilbronn Y, Levy MJ. Percutaneous tracheostomy-A new method. *Crit Care Med* 1989;17:1052-1056.
5. Griggs WM, Worthley LIG, Gilligan JE, Thomas PD, Myberg JA. A simple percutaneous tracheostomy technique. *Surg Gynecol Obst* 1990;170:543-545.