Is there a role for Sodium Bicarbonate in NICU?

Stephen Wardle
Consultant Neonatologist
Nottingham University Hospitals
Aim / Objectives

To persuade you:-

• there is no evidence in favour of using bicarbonate and some significant adverse effects

• to think about acid base and fluid management in a different way to avoid the need for bicarbonate


SODIUM BICARBONATE
also known as Baking Soda

* Alkalizes the body
* Absorbs heavy metals
* Absorbs radiation
* Purifies air
* Extinguishes fires
* Deodorizes
* Soothes bug bite itching
* Natural toothpaste
* Non-toxic deodorant

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Possible Uses of Bicarbonate on NICU

1. During resuscitation

2. To correct acidosis in babies with pulmonary hypertension (PPHN)

3. To correct ‘metabolic’ acidosis in preterm infants
Examples

28 week baby  
6 hours old

pH  7.19  
CO2  5.5 kPa  
BE  -12  
Lactate 8.2 mmol/l

25 week baby  
6 days old

pH  7.19  
CO2  5.5 kPa  
BE  -12  
Lactate 1.2 mmol/l

Term infant

FiO₂  100%  
SpO₂  89%  
pH  7.26  
CO₂  4.5  
BE  -10  
Lactate 6.5
Resuscitation

Recommended for use by NLS / AAP

‘May reverse intra-cardiac acidosis…

But ..lack of evidence …

Strong opinions in both directions
A study from 1966 in five fetal monkeys which infused TRIS and glucose together

Daniel, Dawes et al BMJ 1966
Arguments against:

- Lack of effectiveness in animal and human studies
- Increases CO2 (and increases acidosis)
- Even if arterial CO2 is normal, increased venous CO2 causes decreased intra-cellular pH
- Reduces pH of CSF

Roberton text book
PPHN

Meconium Aspiration

Congenital diaphragmatic hernia

Pulmonary hypoplasia etc
PPHN

Strategy to maintain high pH using bicarb is possible

But

No evidence of effectiveness

Very high pH is needed to improve PaO$_2$

Other strategies (e.g. NO) have more evidence / more effective

Maintain normal pH and avoid acidosis – can be achieved without bicarb

Many adverse effects:

- Hypernatraemia
- Effect on intracellular pH
- Effect on CBF / oxygen availability
Preterm Babies - Historical Perspective

1950s
Hypoglycaemia, azotaemia, hyperkalaemia and metabolic acidosis common before death in premature infants

1963 Usher
IV glucose and bicarbonate proposed
reduced mortality compared to historical controls

1967 Usher
bicarb to correct acidosis in RDS
mortality increased, IVH rate doubled

1977 Corbett
RCT of bicarb
No effect on mortality / IVH rate
pH improved without bicarb
Rennie Roberton 3rd Edition 1999
Bicarbonate 14 page references

Rennie Roberton 4th Edition 2005
Bicarbonate 2 page references
Why not just use bicarbonate?

Giving bicarb may cause:-

- Increased risk of IVH (Usher 1968)
- Effects on CBF
- Possible detrimental effect on cardiac function
- Increased CO$_2$
- Hypernatraemia
- Skin damage
Preterm Babies

What is the cause of the acidosis?

Most early acidosis is lactic acidosis
Prevalence and Severity

- 23 infants <1000g were studied
- 56.5% became acidotic in the 1st 7 days
- 30% became acidotic on the 1st day, always with an increased lactate concentration
- 100% showed a low base excess in the 1st 7 days

Acidotic babies more likely to die (p = 0.012)
Lactic Acidosis

Requires treatment of the cause:

- Peri-partum hypoxia-ischaemia
- Low systemic output
- NEC
- Significant IVH
- Significant acute blood loss etc
If lactate is normal what is the cause of the acidosis?
Conventional Acid-Base balance

- Henderson - Hasselbach Equation:

\[
pH = 6.1 + \frac{\text{‘metabolic’ } [\text{HCO}_3^-]}{\text{‘respiratory’ } 0.03 \text{ pCO}_2}
\]
Metabolic Acidosis – conventional approach

• Accumulation of acid other than CO$_2$ and associated with a decrease in [HCO$_3^-$]

• The acid can be
  • Lactic
  • Something else – bicarb loss??
The Anion Gap

Difference between measured plasma cations and anions

\[ ([Na^+] + [K^+]) - ([HCO_3^-] + [Cl^-]) \]

Normal pH

Metabolic Acidosis

Lactic Acidosis

Raised

Durward 2002
Giving sodium chloride generates an acidosis

This cannot be easily explained by Henderson Hasselbach

This is the commonest cause of non-lactic acidosis in preterm babies

not bicarb loss!
Stewart’s Strong Ion Theory

Can. J. Physiol. Pharm. 1983

pH is affected by 3 independent variables

- $\text{PCO}_2$ (↑ in acidosis)
- Weak acids (albumin and phosphate) (↑ in acidosis)
- Strong ion difference (SID) (↓ in acidosis)
All of which affect bicarbonate concentration
Strong Ion Difference (SID)

• Strong ions are fully dissociated at physiological pH
• $\text{SID} = [\text{Strong cations}] - [\text{Strong anions}]$

Cl to Na ratio is a good approximation

Durward 2002
Strong Ion Gap (SIG)

\[
\text{SID} = (\text{Strong Cations} - \text{Strong anions}) = \text{Weak anions}
\]

\[
\text{SIG} \neq (\text{Strong Cations} - \text{Strong anions}) \neq \text{Weak anions}
\]
<table>
<thead>
<tr>
<th>Fluid</th>
<th>Sodium mmol/l</th>
<th>Chloride mmol/l</th>
<th>SID</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood (plasma)</td>
<td>140</td>
<td>100</td>
<td>40</td>
</tr>
<tr>
<td>0.9% saline</td>
<td>150</td>
<td>150</td>
<td>0</td>
</tr>
<tr>
<td>4.5% HAS</td>
<td>150</td>
<td>150</td>
<td>0</td>
</tr>
<tr>
<td>Ringers Lactate</td>
<td>130</td>
<td>109</td>
<td>21</td>
</tr>
<tr>
<td>Hartmanns</td>
<td>129</td>
<td>109</td>
<td>27</td>
</tr>
<tr>
<td>PN (typical)</td>
<td>33</td>
<td>0</td>
<td>33</td>
</tr>
</tbody>
</table>
Acidosis is therefore usually due to infusions of fluid with low Strong Ion Difference

E.g. Sodium chloride 0.9% at 0.5 ml/hr in 500g baby

\[
= 24 \text{ mls} / \text{kg} / \text{day}
\]

Plus flushes, other infusions etc

\[
= \text{significant amount of low SID fluid}
\]

\[\text{→ significant acidosis}\]
How can this be avoided?

Avoid low SID fluids where possible

Avoid saline / albumin

Acetate in TPN instead of chloride  \textit{(Peters ADC 1997)}

Do not treat base deficit – determine the cause and manage appropriately
Conclusions

No evidence for sodium bicarb in resuscitation

Better approaches to managing PPHN
Conclusions

Avoid acidosis in preterm babies with:

• Better understanding of Stewart strong ion theory

• Greater use of infusions with higher strong ion difference (acetate in PN)

• Avoid infusions of low strong ion difference fluids (saline)