The development of the Astrup micromethod for the determination of pH, carbon-dioxide tension, base excess, standard bicarbonate and actual bicarbonate in capillary blood has resulted in a new interest being taken in acid-base disturbances in the paediatric age-group, i.e. premature babies, newborns, infants and older children. The method has opened up a wide field for research but, more important, it enables the paediatrician to treat and manage acid-base disturbances on a rational basis. This can be regarded as perhaps one of the most important recent advances in paediatrics, as so many life-threatening illnesses affecting the paediatric age-group are associated with severe disturbances of acid-base metabolism.

Many clinicians still regard the interpretation and application of acid-base investigations as being beyond the scope of the 'ordinary mortal'. This negative attitude has been brought about mainly by the lack of uniformity in acid-base terminology; heated discussions of the latter in the pundit's in the medical literature; the difficulty in the past of collecting suitable blood (usually arterial) in order to carry out the necessary determinations for the assessment of the acid-base status of the patient; the difficulty of the interpretation of results; and the practical application of the results in the management of a given case.

These can be largely overcome by:

1. Accepting the terminology and classification for acid-base disturbances employed in this paper, until final agreement is reached in this matter.

2. Employing micromethods.

3. Making use of the various methods which give a graphic representation of the acid-base status of a patient.

4. Managing or correcting acid-base disturbances in a given disease:
   (i) by equations to determine the amount of acid or base required for the correction of a particular non-respiratory disturbance in a given patient; and
   (ii) by making use of the results obtained from acid-base studies to control any severe respiratory acidosis with augmented or intermittent positive-pressure respiration.

**TERMINOLOGY AND CLASSIFICATION**

The terminology and classification for acid-base disturbances employed in our laboratory are as follows:

1. A reduced pH and an increased pH are described as acidosis and alkalosis respectively.
2. Respiratory acidosis is defined as an excess of carbonic acid due to a rise in the PCO2.
3. Respiratory alkalosis is defined as a deficit of carbonic acid due to a lowered PCO2.
4. Metabolic acidosis is defined as an excess of non-volatile acid or a deficit of base.
5. Metabolic alkalosis is defined as an excess of base or deficit of non-volatile acid.
6. Base excess (BE) is defined as the titratable base on titration of blood (or plasma), with a PCO2 of 40 mm.Hg and a temperature of 38°C, to a normal pH of 7.40. It is regarded as a measure of metabolic alkalosis or metabolic acidosis. A positive value of BE signifies a metabolic alkalosis with a base excess or deficit of non-volatile acid, whereas a negative value of BE signifies a metabolic acidosis with a base deficit or excess of non-volatile acid.
7. The standard bicarbonate is the bicarbonate in plasma measured under the following standardized conditions: the haemoglobin must be fully oxygenated; the PCO2 must be 40 mm.Hg and the temperature 38°C. It is regarded as an expression of the metabolic aspect of acid-base metabolism.
8. The actual bicarbonate is the concentration of bicarbonate in plasma that is separated from the cells at the actual PCO2 and at a temperature of 38°C. It is regarded as the total available buffer, which includes bicarbonate, proteins, haemoglobin and phosphates.

**METHODS**

The determination of the acid-base equilibrium of a patient can be made either on arterial blood or arterialized capillary blood. Arterial blood is collected either from the umbilical, temporal, femoral or brachial artery, in a syringe in which the dead space is filled with concentrated heparin solution. Arterialized capillary blood can be collected from the warmed lobe of the ear in older children or from the warmed heel in infants, in heparinized capillary tubes. In the latter instance the foot must be thoroughly warmed for at least 10-15 minutes to promote the free flow of blood following a clean stab of the heel with a blade. Care must be taken:

(i) to avoid crying before and during the procedure as this would lower the PCO2;
(ii) not to squeeze the heel in order to avoid a possible admixture of tissue fluid or obstruction of the circulation;
(iii) to collect the blood anaerobically in the heparinized capillary tubes from quickly-forming unbroken drops of blood;
(iv) to mix the blood with the specially supplied stirring rods and magnet before clotting takes place;

(v) to exclude air and seal the ends of the capillary tubes with plasticine;
(vi) to store the capillary tubes in iced water unless estimations are carried out immediately;
(vii) to record the body temperature at the time the sample is taken in order to allow for corrections to be made in determining the actual pH of the patient.

This latter measurement is extremely important in the paediatric age-group, especially in premature and newborn infants in whom hypothermia occurs both easily and rapidly.

In the present studies arterial blood is usually collected from the umbilical artery in premature and full-term newborns during the first 48 - 72 hours of life; from the temporal artery in premature or full-term infants and from the femoral artery in older infants and children. Arterialized capillary blood is collected in premature babies, infants and older children with good peripheral circulations. Blood is analysed either (i) within minutes after collection, or (ii) after storage for a period of less than 1 hour in ice water. Temperature corrections are made for the actual pH measurements by adding a factor of 0.015 for every 1°C that the patient is below 38°C to the pH value measured at 38°C. When the patient’s temperature is over 38°C this factor is subtracted.\(^{21}\)

NORMAL VALUES

Normal values for the acid-base status of premature and full-term infants during the first 72 hours of life\(^{3}\) (Table 1) and premature infants during the first 5 weeks of life\(^{8}\) (Table II) have been established in our laboratory. Values of arterial blood, arterialized capillary blood and venous blood have not been clearly established in the literature for infants and children belonging to older age-groups. It is stated that the acid-base status alters with age, so that a wholly or partly compensated metabolic acidosis is found in children, which gradually decreases with growth, disappearing before the age of 17.\(^{21}\) At 2 - 3 years of age the BE in the blood is approximately 2 mEq./l. lower and the PCO\(_2\) is approximately 4 mm. lower than in adults.\(^{19}\) Children over 6 years of age are regarded in our laboratory as having normal acid-base values within the range reported by Sigggaard-Andersen for adults.\(^{22}\)

TABLE II. ACID-BASE VALUES IN 52 PREMATURE INFANTS DURING FIRST 5 WEEKS OF LIFE

<table>
<thead>
<tr>
<th>Number of determinations</th>
<th>2-4 days</th>
<th>5-8 days</th>
<th>9-12 days</th>
<th>3rd week</th>
<th>4-6 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.30</td>
<td>7.32</td>
<td>7.30</td>
<td>7.30</td>
<td>7.31</td>
</tr>
<tr>
<td>S.D.</td>
<td>±0.13</td>
<td>±0.03</td>
<td>±0.04</td>
<td>±0.02</td>
<td>±0.03</td>
</tr>
<tr>
<td>PCO(_2) (mm.Hg)</td>
<td>34.6</td>
<td>39.4</td>
<td>40.4</td>
<td>39.9</td>
<td>42.1</td>
</tr>
<tr>
<td>S.D.</td>
<td>±12.3</td>
<td>±10.2</td>
<td>±6.3</td>
<td>±9.7</td>
<td>±5.3</td>
</tr>
<tr>
<td>BE (mEq./l.)</td>
<td>-4.8</td>
<td>-6.8</td>
<td>-6.4</td>
<td>-6.6</td>
<td>-5.7</td>
</tr>
<tr>
<td>S.D.</td>
<td>±2.2</td>
<td>±2.4</td>
<td>±2.9</td>
<td>±2.8</td>
<td>±5.3</td>
</tr>
<tr>
<td>Actual HCO(_3)</td>
<td>19.9</td>
<td>19.5</td>
<td>19.3</td>
<td>18.4</td>
<td>20.4</td>
</tr>
<tr>
<td>S.D.</td>
<td>±3.0</td>
<td>±4.8</td>
<td>±3.4</td>
<td>±4.4</td>
<td>±2.3</td>
</tr>
</tbody>
</table>

For practical purposes, however, we regard this difference between the age-groups to be only of importance in the immediate newborn period and in premature infants during the first 6 weeks of life. The assessment of acid-base disturbances in the former is of importance in cases of the respiratory distress syndrome, and in the latter in the management of cases of late metabolic acidosis of prematurity.

Graphic Presentation of the Acid-Base Status

The assessment of the acid-base status cannot be determined from one parameter alone and it is therefore necessary to know:

(i) the pH to decide whether an acidosis or alkalosis is present;

(ii) the PCO\(_2\) to evaluate the respiratory component of acid-base metabolism; and

TABLE I. SUMMARY OF BLOOD VALUES AT VARIOUS AGES\(^{8}\) IN PREMATURE AND FULL-TERM INFANTS

<table>
<thead>
<tr>
<th>Hours</th>
<th>Premature</th>
<th>Standard deviation</th>
<th>Range</th>
<th>Full-term</th>
<th>Standard deviation</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>4</td>
<td>7.35</td>
<td>0.04</td>
<td>7.28-7.42</td>
<td>7.37</td>
<td>0.04</td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>7.39</td>
<td>0.04</td>
<td>7.33-7.48</td>
<td>7.42</td>
<td>0.05</td>
</tr>
<tr>
<td></td>
<td>48</td>
<td>7.37</td>
<td>0.05</td>
<td>7.29-7.46</td>
<td>7.42</td>
<td>0.04</td>
</tr>
<tr>
<td>PCO(_2) (mm.Hg)</td>
<td>4</td>
<td>43.4</td>
<td>8.2</td>
<td>31.7-57.8</td>
<td>36.7</td>
<td>5.3</td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>35.8</td>
<td>4.0</td>
<td>27.6-43.0</td>
<td>34.9</td>
<td>4.0</td>
</tr>
<tr>
<td></td>
<td>48</td>
<td>39.5</td>
<td>6.1</td>
<td>33.0-57.0</td>
<td>34.0</td>
<td>4.5</td>
</tr>
<tr>
<td></td>
<td>72</td>
<td>36.8</td>
<td>5.2</td>
<td>29.0-45.0</td>
<td>35.5</td>
<td>5.4</td>
</tr>
<tr>
<td>Base excess (mEq./l.)</td>
<td>4</td>
<td>-2.3</td>
<td>2.6</td>
<td>-6.6-2.9</td>
<td>-2.5</td>
<td>1.6</td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>-2.6</td>
<td>1.8</td>
<td>-7.4-0</td>
<td>-1.4</td>
<td>1.8</td>
</tr>
<tr>
<td></td>
<td>48</td>
<td>-3.3</td>
<td>2.9</td>
<td>-9.6-0.5</td>
<td>-0.8</td>
<td>2.4</td>
</tr>
<tr>
<td>Buffer base (mEq./l.)</td>
<td>4</td>
<td>47.8</td>
<td>3.0</td>
<td>44.0-53.2</td>
<td>47.5</td>
<td>1.8</td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>46.6</td>
<td>2.7</td>
<td>40.8-50.3</td>
<td>47.6</td>
<td>2.7</td>
</tr>
<tr>
<td></td>
<td>48</td>
<td>46.2</td>
<td>2.8</td>
<td>41.8-51.0</td>
<td>47.8</td>
<td>3.2</td>
</tr>
<tr>
<td></td>
<td>72</td>
<td>46.5</td>
<td>3.3</td>
<td>40.6-50.2</td>
<td>49.0</td>
<td>3.3</td>
</tr>
<tr>
<td>Standard HCO(_3) (mEq./l.)</td>
<td>4</td>
<td>22.2</td>
<td>2.2</td>
<td>19.2-26.2</td>
<td>22.0</td>
<td>1.3</td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>21.9</td>
<td>1.6</td>
<td>18.4-24.0</td>
<td>22.8</td>
<td>1.6</td>
</tr>
<tr>
<td></td>
<td>48</td>
<td>21.9</td>
<td>1.7</td>
<td>18.4-24.4</td>
<td>23.0</td>
<td>1.6</td>
</tr>
<tr>
<td></td>
<td>72</td>
<td>21.4</td>
<td>2.0</td>
<td>17.1-23.5</td>
<td>23.3</td>
<td>2.1</td>
</tr>
<tr>
<td>Actual HCO(_3) (mEq./l.)</td>
<td>4</td>
<td>22.9</td>
<td>3.7</td>
<td>17.0-30.5</td>
<td>21.4</td>
<td>1.4</td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>20.8</td>
<td>1.9</td>
<td>16.2-24.6</td>
<td>21.7</td>
<td>1.9</td>
</tr>
<tr>
<td></td>
<td>48</td>
<td>21.6</td>
<td>1.6</td>
<td>17.6-24.6</td>
<td>21.3</td>
<td>2.0</td>
</tr>
<tr>
<td></td>
<td>72</td>
<td>20.6</td>
<td>2.7</td>
<td>14.4-23.6</td>
<td>22.2</td>
<td>2.1</td>
</tr>
</tbody>
</table>
(iii) the base excess or standard bicarbonate or actual bicarbonate to assess the metabolic component of acid-base metabolism.

Thus, for instance, it may be completely misleading to evaluate the acid-base status of a patient from the BE or standard bicarbonate or actual bicarbonate values alone. An elevated value for any of these measurements can be the result of a metabolic alkalosis, but can also be the result of a compensatory rise in the presence of a respiratory acidosis. A correct assessment can only be made if the PCO₂ value is known.

To simplify the interpretation by the clinician of acid-base disturbances, methods for the graphic representation of the latter have been developed. These methods are also of great help in the management of such cases.

The pH, HCO₃⁻ coordinate system proposed by Van Slyke³ has been popularized in recent years by Davenport⁴ and Siggaard-Andersen.⁵ Siggaard-Andersen’s system is shown in Fig. 1. The addition of a table next to the graph introduces a time factor and permits the recording of serial acid-base and other data, treatment given to the patient, etc. By plotting a point on the graph, the acid-base status of the patient can be determined immediately by noting the area in the diagram in which the point falls. Each area represents a specific state of acid-base disturbances. The following indicate zones on the graph:

- 0. Normal
- 1. Uncompensated respiratory acidosis
- 2. Partly compensated respiratory acidosis
- 3. Fully compensated respiratory acidosis
- 4. Partly compensated metabolic alkalosis
- 5. Uncompensated metabolic alkalosis
- 6. Combined metabolic alkalosis and respiratory alkalosis

Fig. 1. Example of graphic representation of acid-base status.

AETIOLOGY OF ACID-BASE DISTURBANCES IN THE PAEDIATRIC AGE-GROUP

Over the last 2 years approximately 7,000 acid-base determinations were carried out in premature infants, full-term newborns, neonates, infants and child:en suffering from a wide spectrum of disease processes.

The following conditions were found to be associated with significant acid-base disturbances in many cases:

- Newborns
  - *Metabolic acidosis*—infectious and septicaemic conditions; congenital heart disease; diarrhoea; acute haemorrhagic shock.
  - *Respiratory acidosis*—Asphyxia neonatorum; neonatal pneumonia; pneumothorax; meconium aspiration; clinical hyaline membrane disease; neonatal disseminated atelectasis.
  - *Combined acidosis*—Clinical hyaline membrane disease; neonatal disseminated atelectasis; post-exchange transfusion; cerebral birth trauma; meconium aspiration; neonatal tetanus; neonatal surgical conditions.

- Prematures
  - As above; and late metabolic acidosis of prematurity.

- Infants
  - *Metabolic acidosis*—Gastroenteritis; overwhelming infections; renal disease; surgical conditions; salicylate intoxication.
  - *Respiratory acidosis*—Pneumonia; bronchiolitis; laryngo-tracheo-bronchitis; foreign body; asthma; CNS depression secondary to poisons.
  - *Metabolic alkalosis*—Pyloric stenosis; iatrogenic, e.g. overtreatment with sodium bicarbonate.
  - *Respiratory alkalosis*—Encephalitis.

- Older Children
  - *Metabolic acidosis*—Renal disease; late salicylate poisoning; diabetic pre-coma.
  - *Respiratory acidosis*—Ascending myelitis; Guillain-Barré syndrome; poliomyelitis; tetanus; asthma.
  - *Metabolic alkalosis*—Excessive vomiting; associated with intestinal obstruction.
  - *Respiratory alkalosis*—Hyperventilation:
    - (i) Iatrogenic—IPPR
    - (ii) Encephalitis
    - (iii) Asthma
    - (iv) Early salicylate poisoning

Acidosis, either metabolic, respiratory, or a combination of both, was the commonest significant disturbance encountered. Severe metabolic acidosis was especially common during periods of stress during the first year of life. Significant metabolic alkalosis was rare and mainly seen in conditions associated with severe and prolonged vomiting such as pyloric stenosis.

SUMMARY

The development of micromethods for acid-base determinations is of particular interest to the paediatrician, as many life-threatening illnesses affecting the paediatric age-group are associated with severe disturbances of acid-base metabolism.

The interpretation and application of acid-base investigations can be simplified by understanding the terminology used, knowing the normal values for the different acid-base parameters at various age-groups, and by making use of methods which give a graphic representation of the acid-base status of the patient.

In a survey of acid-base disturbances in premature infants, full-term newborns, neonates, infants and older children...
suffering from a wide spectrum of disease processes, acidosis which was either metabolic, respiratory or a combination of both was the commonest significant disturbance encountered. Severe metabolic acidosis was especially common during periods of stress in premature infants, full-term newborns and infants up to the age of 1 year.

The management of metabolic acidosis and respiratory acidosis will be discussed in further articles.

We should like to thank Dr. J. G. Burger, Medical Superintendent of Groote Schuur Hospital, for permission to publish; Prof. F. J. Ford, for editing; paediatric and surgical colleagues for referring cases to us; Sister N. N. Duk for her cooperation in this study; and Mrs. O. M. Cartwright for her help in the preparation of the paper. We acknowledge with gratitude the financial assistance given to us by the Council for Scientific and Industrial Research of South Africa, the Wellcome Trust and the Teaching Hospital Board Staff Research Fund.

REFERENCES


REFLECTIONS ON CLINICAL TEACHING

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In recent years medical education appears to have stimulated considerable interest from all quarters of the profession. The emphasis, however, has been mainly on the criteria of student selection, and revision and reorientation of the medical curriculum. There has not been sufficient thought on actual teaching, and much of the present deficiency in our training methods may well be a reflection of this. Evidently a particular kind of teaching with special relevance to general principles is urgently required not only to fill the long-felt need, but also to meet the ever-increasing demands on students. Consequently it is thought desirable to re-examine some of the methods used and to re-emphasize those that may mean much to the progress of medical education and student knowledge.

Shortcomings

Any shortfall in our present system may be the result of our taking for granted the actual process of imparting knowledge. A blueprint curriculum is presented to the teachers and students without adequate liaison between the two. There is a constant attempt to revise the curriculum and to make it as comprehensive as possible with due regard to the changing pattern and advance in medical science and practice.

Similarly, methods of student selection are constantly improved. A similar parallel is not evident with regard to the selection of teachers. The position therefore is that many schools are quite content to have a good curriculum presented to a highly selected student group with teachers to guide them in a situation where learning is expected to take care of itself. It is therefore questionable whether the present criteria for appointments are the best, wherein the ability and desire to teach do not appear to be primary determinants. Furthermore, there is no organized training or apprenticeship for those contemplating a teaching career, as this is thought to be unnecessary for teaching at a university.

The Clinical Teacher

A clinical teacher should be a good clinician and a keen teacher with an interest in research. Furthermore, it is desirable for him to have had a period of training in the art of teaching. These qualities are complementary and have an important bearing on the end-product of undergraduate education. Dedication in teaching is as important as dedication in medicine. Enthusiasm and absorbing interest in teaching soon reflect themselves in the interest displayed by students in their learning.

The teacher should have an especially competent command of the language medium used and should be able to express himself fluently and lucidly. He should also possess a dynamic personality and should gain the respect and admiration of his students with regard to his competence, knowledge, personality and honesty. His bedside manner should be impeccable. That a clinical teacher should be a good clinician is self-explanatory, but there may be a division of opinion regarding his involvement in research. As students tend to emulate a good teacher, it is relevant that he should in addition to his teaching duties also make original contributions, provided that this does not interfere with his teaching programme.

The training of teachers is a recent concept that is receiving more and more prominence. It is important to appreciate why such training is necessary. Basic psychology in teaching and general principles are best appreciated in an organized training programme. What to teach, how to teach, and the purpose of teaching, are outlined in the training. When institutions for the training of teachers are established and teaching is made more attractive as a career, the men most suited for this vocation are bound to come forward more readily. As the specialty of teaching becomes firmly established, conferences on training teachers and methods of teaching are likely to be held. Such exchange of experience will prove valuable, and progress in medicine will then be matched adequately with improvement in the quality of teachers and teaching.

PRINCIPLES IN CLINICAL TEACHING

There are certain general principles in clinical teaching that are worth outlining. For example, it is important to emphasize that each patient is a unique biological phenomenon of a kind which makes it permissible to regard him as an experimental subject not analogous to a guinea-pig, but as relevant to his emotional behaviour and therapeutic programme. Implicit in this consideration is the fact that his response to treatment is not easily predictable and may have to be modified or altered from time to time depending on his reaction as an organism and as a human being.

As it is not possible nor desirable for students to know everything in their syllabus, it is incumbent on the teacher to refrain from spending an inordinate amount of time on teaching facts and figures. He should instead teach them how to refrain from spending an inordinate amount of time on teaching facts and figures. He should instead teach them how to learn. In their early clinical training they are thus placed in a situation where their main aim is to learn how to learn. This principle cannot be emphasized too strongly as medicine is a lifelong study and learning is an art that can be adequately acquired only through proper guidance. Since memory is so important in medicine, it is condescending to the techniques of memory training may prove valuable. Clinical memory training is dependent on keen observation as distinct from looking. It is the duty of the clinical teacher to point out actively the nature and importance of clinical observation, particularly when suitable examples present themselves.