CLINICAL PROGRESS

Acid-Base Management for Open-Heart Surgery

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The occurrence of serious acid-base disturbance is not only more likely during open-heart procedures than with other forms of surgery, but its correction and the continued maintenance of good acid-base status is often critical. The operation, including surgery on the heart itself, offers considerable cardiovascular stress to the debilitated patient who has a limited cardiac reserve. The added insult of acid-base imbalance may be more than the heart can tolerate, and careful management, including correction of any imbalance, may prevent an adverse outcome to an otherwise successful operation.

Definition of Acid-Base Status

It is generally accepted that at 37°C, the pH of arterial blood has a normal value of 7.40. Acid-base imbalance may occur as either acidosis (pH below normal) or alkalosis (pH above normal). Acidosis is further defined as respiratory, which is due to retention of carbon dioxide producing an arterial pCO₂ above the normal value of 40 mm. Hg, or metabolic, which is due either to excess of acid metabolites or to decrease in available base. Similarly, alkalosis may be respiratory (pCO₂ below 40 mm. Hg) or metabolic (due to excess base or loss of acid). Respiratory and metabolic acidosis or alkalosis may be combined, thus creating the need for more definitive terms to describe fully the acid-base status. Hence acidosis or alkalosis may be mixed, uncompensated, partially compensated, or fully compensated. If it is the last, the choice as to whether it be described as fully compensated acidosis or fully compensated alkalosis depends on the apparent initial cause of the imbalance. Because of the confusion sometimes created by these terms, acid-base management is often considered to be a complicated matter. Primary consideration of the respiratory and metabolic components as separate entities, however, helps to clarify understanding of the situation, and the management of an imbalance becomes more straightforward. These two components may have an additive effect or a compensatory effect on one another but the treatment of each component must be considered in every instance. In some situations, treatment of one component may result in spontaneous correction of the other, whereas, in other cases, active treatment will be necessary for both.

The clinical picture resulting from acid-base imbalance is unreliable, as it varies with the types of acidosis or alkalosis and the degree of compensation. It may also be confused by the presence of other factors; for example, from their clinical appearance it is difficult to differentiate between the effects of metabolic acidosis and residual curarization.1 The aim of good management is primarily to prevent acid-base disturbance, and secondly to correct any imbalance and to maintain good acid-base balance with continued treatment if necessary. To attain this goal, accurate meas-
measurements must be made of the acid-base status as frequently as necessary to prevent major deviation from the normal.

Measurement of Acid-Base Status

There are many methods of determining acid-base status but certain requirements must be fulfilled if the method is to provide good control and allow good management. The acid-base status may change quickly leading to rapid deterioration in the patient’s condition or, equally rapidly, as a result of vigorous treatment, and information regarding such changes must be readily obtained. Information of the acid-base status from a blood sample taken an hour previously is not only presently inapplicable but, if acted upon, results in little better than blind treatment. Worse, during the time of awaiting the results upon which treatment is to be decided, the patient may have succumbed from gross acid-base imbalance. Therefore, any method used must be not only accurate and comprehensive but also rapid and capable of producing almost immediate results.

These requirements of a good, rapid, accurate method of ascertaining the acid-base status in our opinion appears to be best fulfilled by the thermostatically controlled triple electrode system, with use of a pH electrode, Severinghaus pCO₂ electrode, and modified Clark pO₂ electrode. Once this equipment is calibrated, it is possible to obtain values for pH, pCO₂, pO₂, and oxygen saturation (by calculation from pH and pO₂). Base excess or deficit is quantitated by a special application of the Siggaard-Andersen nomogram, as previously described. This value of base excess or deficit, in milliequivalents per liter as bicarbonate, is valid irrespective of temperature. Should the patient’s temperature be other than normothermic, appropriate corrections for pH, pCO₂, and pO₂ are applied. Thus a comprehensive quantitative assessment of the acid-base status is obtained within a few minutes of obtaining a 2- to 3-ml. sample of arterial blood.

The Astrup method of measuring pH, pCO₂, and base deficit or excess by equilibrating samples of blood with known tensions of carbon dioxide is also rapid and requires even less blood but this alone does not give a measure of the state of oxygenation and the values are only applicable under conditions of normothermia.

It is best to use arterial blood samples to determine the acid-base status, since venous or capillary blood values vary greatly depending on the degree of blood pooling and stasis that may occur in shock conditions. Arterial blood gas analysis may not give an accurate indication of the intracellular status; nevertheless, it would appear to give the best measure of the efficacy of the cardiovascular and respiratory systems. Samples are withdrawn, either from an arterial catheter or by arterial puncture, into a syringe previously wetted with heparin (1,000 units per ml. concentration).

Phase before Cardiopulmonary Bypass

From the time of administration of pre-anesthetic drugs until the commencement of cardiopulmonary bypass, acid-base imbalance is a potential hazard although in most cases it does not occur to any great extent.

Respiratory Acidosis

Respiratory acidosis may occur as a result of inadequate elimination of carbon dioxide from the lungs due to inadequate ventilation. It may also be the result of right-sided heart failure, since insufficient blood is being brought into contact with the alveolar membrane where diffusion of carbon dioxide into the alveoli occurs. Atelectasis results in perfusion of alveoli in which no gaseous exchange takes place and this, too, produces respiratory acidosis.

Respiratory acidosis consequent upon anesthesia and the open-chest procedure can usually be avoided during the phase of the operation before bypass if the anesthesiologist augments ventilation to insure adequate elimination of carbon dioxide from the lungs. Moderate hyperventilation, as provided by many anesthesiologists, is usually satisfactory at this time but gross hyperventilation, producing respiratory alkalosis, is to be avoided. If respiratory alkalosis occurs as the result of
overventilation during anesthesia, the body, in an effort to maintain a normal pH, may produce a compensatory base deficit. This base deficit is revealed as a metabolic acidosis when the arterial pCO₂ is allowed to return to a normal value.

**Metabolic Acidosis**

Metabolic acidosis is not usually a problem prior to bypass but may occur and on occasion becomes quite severe by the time bypass is commenced. The causes may be similar to those to be described in phases after bypass and after operation. Occasionally a progressive metabolic acidosis is encountered prior to bypass, even in the presence of adequate arterial pressure and pO₂, presumably due to a low cardiac output incident to anesthesia and thoracotomy.

Prevention and treatment of metabolic acidosis are important prior to bypass, just as in the other phases of the operation. Maintenance of good oxygenation and a reasonable arterial pressure should be ensured and in some cases vasopressor agents may be necessary. If, before the beginning of bypass, there is a base deficit of more than a few milliequivalents per liter as bicarbonate in the patient’s arterial blood, it should be corrected with sodium bicarbonate solution intravenously in sufficient amount to reduce the base deficit to zero. It is easier to maintain good acid-base status throughout the bypass period if the patient is not in base deficit when bypass is started.

**Bypass Phase**

Gross acid-base changes may occur during bypass. If considerable imbalance does develop and remain uncorrected at the end of the bypass period, this may add markedly to the stress of the phase after bypass and result in increased postoperative morbidity. Disturbances occurring during bypass have been greatly lessened since the introduction of high-flow perfusion technics. Nevertheless, good acid-base maintenance still requires constant attention. If gross respiratory alkalosis is permitted during bypass, it may result in a progressive base deficit. There is evidence that respiratory alkalosis produces a shift in the hemoglobin oxygen dissociation curve to the left, leading to tissue anoxia, since under these conditions oxygen is not liberated by the hemoglobin so readily. It may be also that under conditions of respiratory alkalosis there is decreased peripheral tissue perfusion.

When the temperature is kept relatively unchanged at 37°C, it is only necessary to maintain a pH of 7.40 and a pCO₂ of about 40 mm. Hg by adding a low percentage of carbon dioxide to the pump oxygenator gas mixture. Then, if the perfusion is satisfactory, acid-base imbalance is not usually a problem. Should the tissue perfusion be inadequate, it may be necessary to correct any base deficit with sodium bicarbonate, which may be added to the blood on the venous side of the oxygenator prior to discontinuation of bypass.

If hypothermia is to be employed, then acid-base management becomes more important and more involved, since gross imbalance may result under these conditions. Throughout the operation optimal pH should be maintained but it is important to realize that a pH of 7.40 is not the optimal pH at a reduced temperature. Edmark suggested that the pH should be reduced during hypothermia; to achieve this he advocated the use of 0.3 N hydrochloric acid. When carbon dioxide is used instead of a hydrochloric acid drip to control the pH of the blood in the pump oxygenator and with a pH-temperature scale similar to that suggested by Edmark (fig. 1), it has been possible to avoid the occurrence of base deficit or metabolic acidosis during hypothermic bypass. The pH is reduced by 0.0147 of a pH unit for each degree Centigrade below 37. Using this technic, we have been able to prevent other phenomena usually associated with hypothermia. For example, ventricular fibrillation and cessation of spontaneous respiratory effort have been commonly observed when the temperature is lowered about 30°C.; with pH control, however, spontaneous respiratory effort continues at temperatures below 25°C., and ventricular fibrillation usually does not occur unless there is interference with coronary blood flow or severe cardiac manipulation by the surgeon. If ventricular fibrillation is re-
quired for the surgery it must be induced electrically, sometimes repeatedly or continuously.

The essence of management is still maintenance of optimal pH but, since optimal pH is changing as the temperature is altered, it is necessary to vary the percentage of carbon dioxide in the pump oxygenator gas mixture to maintain the desired pH. For this reason, pH measurements are obtained from samples of blood taken frequently from the arterial side of the oxygenator. Since it is difficult to maintain the electrode bath at the changing temperature of the blood in the oxygenator, it is more convenient to maintain the electrodes at 37C. and to apply correction factors for the temperature difference between 37C. and that of the blood.\textsuperscript{6-8} One of us (S.A.A.C.) has designed a circular slide rule for application of the Rosenthal pH-temperature correction factor, which eliminates the necessity for calculation and thus speeds the results upon which the carbon dioxide percentage of the pump respiratory mixture is altered.

Throughout the bypass procedure it is important to maintain optimal pH at all times including the rewarming period when the carbon dioxide percentage may have to be greatly reduced. Since the percentage must vary according to the temperature and especially with the rate of change of temperature, it is not possible to maintain good acid-base balance by administering a predetermined fixed percentage of carbon dioxide into the pump oxygenator throughout bypass.

If the priming fluid of the pump oxygenator system is not entirely whole blood, the buffer capacity of this mixture must be considered. In order to ensure adequate buffer capacity, sodium bicarbonate must be added to the extent of 45 mEq. per liter of diluent fluids. This provides an over-all buffer capacity approximating that of whole blood. Freshly drawn heparinized blood is preferable for priming purposes as older blood or acid-citrate-dextrose blood may be quite acidotic. Nevertheless, after the pump is primed and following recirculation with an oxygen-low CO\textsubscript{2} mixture in the oxygenator, it is advisable to measure the degree of base deficit and correct this by adding more sodium bicarbonate. [Base deficit (mEq./L.) \times priming volume (liters) = amount of sodium bicarbonate (mEq.) necessary to correct the base deficit.]

Metabolic acidosis may occur during bypass in spite of careful management. Usually this must be assumed to have been the result of inadequate tissue perfusion, provided good oxygenation was maintained in the oxygenator. If a base deficit of more than a few milliequivalents per liter as bicarbonate exists toward the end of the bypass period, it should be corrected before discontinuing the perfusion. Addition of sufficient sodium bicarbonate to reduce the base deficit to zero.

\textit{Figure 1}

Temperature-optimal pH relationship similar to that originally suggested by Edmark (upper line); optimal pH reading when uncorrected for temperature with the electrode maintained at 37C. (lower line). For example, at a temperature of 20C. the optimal pH would be 7.15 but with the electrode maintained at 37C. the instrument reading would be 6.90.
is advisable, for it ensures that the heart will not be depressed by metabolic acidosis upon resumption of the normal circulation.

**Phases after Bypass and Operation**

Even if the acid-base status is good at the end of bypass, a serious acid-base imbalance may subsequently develop after bypass or operation. Alkalosis is to be avoided at these times just as earlier. It seldom occurs spontaneously but rather as the result of overly vigorous treatment. The usual problem is almost invariably one of either respiratory or metabolic acidosis.

**Respiratory Acidosis**

During the post-bypass period the anesthesiologist can usually avoid respiratory acidosis just as in the period before bypass by adequately eliminating carbon dioxide from the lungs. Hyperventilation producing respiratory alkalosis should be similarly avoided.

In the immediate postoperative phase, respiratory acidosis from inadequate ventilation is common. Pain from the large incision may cause gross limitation of chest movement so that the ventilatory effort is mostly diaphragmatic. Diaphragmatic breathing exercises before operation may greatly contribute to the adequacy of the respiration in the recovery ward but relief of the chest pain often corrects the situation most dramatically. A long-acting intercostal nerve block, performed at the conclusion of the operation, may be all that is required. Sometimes moderate doses of analgesics given intermittently are also necessary, but respiratory depression due to overdosage of these drugs in combination with residual effects of anesthesia is to be anticipated and avoided if possible. In addition, among other effects, severe metabolic acidosis causes depression of respiration, and correction of metabolic acidosis may result in a marked increase in ventilation.

Postoperative atelectasis is a common sequel to open-heart surgery although in most cases it may be localized. Diffuse and gross atelectasis occurring after bypass has been described. This condition resembles hyaline membrane disease of the neonate and has been attributed to absorption during bypass of the surface active material that normally lines the alveoli. Since carbon dioxide dif-

### Table 1

<table>
<thead>
<tr>
<th>Operation</th>
<th>Oxygenator blood temperature (°C)</th>
<th>CO₂ in pump respiratory mixture (%)</th>
<th>Elapsed time on bypass, hr, min.</th>
<th>Base deficit (mEq/liter as bicarbonate)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I.V.S.D. repair</td>
<td>31.0 Cooling</td>
<td>2</td>
<td>9</td>
<td>-6.5</td>
</tr>
<tr>
<td>28.0 Stable</td>
<td></td>
<td></td>
<td>45</td>
<td>-8.5</td>
</tr>
<tr>
<td>36.0 Rewarming</td>
<td></td>
<td></td>
<td>1,47</td>
<td>-8.0</td>
</tr>
<tr>
<td>I.V.S.D. repair and</td>
<td>25.0 Cooling</td>
<td>2</td>
<td>21</td>
<td>-8.7</td>
</tr>
<tr>
<td>modif. pul. stenosis</td>
<td>22.5 Stable</td>
<td></td>
<td>52</td>
<td>-10.0</td>
</tr>
<tr>
<td>32.0 Rewarming</td>
<td></td>
<td></td>
<td>1,18</td>
<td>-10.0</td>
</tr>
<tr>
<td>Atrial septal construction</td>
<td>31.5 Cooling</td>
<td>2</td>
<td>9</td>
<td>-5.0</td>
</tr>
<tr>
<td>27.5 Stable</td>
<td></td>
<td></td>
<td>45</td>
<td>-6.0</td>
</tr>
<tr>
<td>34.5 Rewarming</td>
<td></td>
<td></td>
<td>2,31</td>
<td>-10.0</td>
</tr>
<tr>
<td>Pul. valvulotomy</td>
<td>28.6 Stable</td>
<td>2</td>
<td>18</td>
<td>-2.0</td>
</tr>
<tr>
<td>28.6 Stable</td>
<td></td>
<td></td>
<td>48</td>
<td>-11.0</td>
</tr>
<tr>
<td>35.7 Rewarming</td>
<td></td>
<td></td>
<td>1,3</td>
<td>-10.0</td>
</tr>
<tr>
<td>Aortic valve</td>
<td>28.0 Stable</td>
<td>2</td>
<td>9</td>
<td>-5.0</td>
</tr>
<tr>
<td>commissurotomy</td>
<td>33.0 Rewarming</td>
<td></td>
<td>1,15</td>
<td>-9.5</td>
</tr>
<tr>
<td>35.0 Rewarming</td>
<td></td>
<td></td>
<td>1,50</td>
<td>-9.0</td>
</tr>
</tbody>
</table>

* This does not include the CO₂ reaching the oxygenator via the coronary suction when the surgical field is being flooded with 100 per cent CO₂ to prevent air embolism.
fuses much more readily across the alveolar membrane than does oxygen, this condition of diffuse atelectasis may be revealed by a low or falling arterial pO₂ in the presence of a nearly normal arterial pCO₂.

Gases may be absorbed from the alveoli by the bronchial circulation during bypass and this may predispose to alveolar collapse and atelectasis. For this reason, it may be useful during bypass to maintain expansion of the lungs with air rather than anesthetic gas mixtures of high oxygen content. The incidence of atelectasis after bypass might then be less, since the nitrogen of air is not so readily absorbed from the collapsed lung. At any rate, it is essential for the anesthesiologist to inflate the lungs fully at the end of bypass and during these efforts the lungs should be free in the chest and unrestrained by surgical retractors or packing.

### Treatment of Respiratory Acidosis

The treatment of respiratory acidosis is to ensure adequate elimination of carbon dioxide through the lungs. If adequate ventilation cannot be promoted by pain relief and counteraction of depressive effects, then respiration must be augmented mechanically. The use of a mechanical ventilator must not be undertaken lightly as this of itself can cause complications. Ventilation should be sufficient to reduce the pCO₂ to a normal value but gross

### Table 2

**Cardiopulmonary Bypass with Hypothermia. Examples of Cases during Which Sufficient CO₂ Was Administered in Pump Oxygenator to Maintain pH Optimal with Temperature According to Edmark's Scale**

<table>
<thead>
<tr>
<th>Operation</th>
<th>Oxygenator blood temperature (°C)</th>
<th>CO₂ in pump respiratory mixture (%)</th>
<th>Elapsed time on bypass, hr.,min.</th>
<th>Base deficit (mEq./liter as bicarbonate)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Part. repl.</td>
<td>34.5 Cooling</td>
<td>8</td>
<td>6</td>
<td>-2.5</td>
</tr>
<tr>
<td>mitral valve</td>
<td>27.8 Stable</td>
<td>5</td>
<td>1, 4</td>
<td>-5.0</td>
</tr>
<tr>
<td></td>
<td>33.5 Rewarming</td>
<td>1</td>
<td>2,17</td>
<td>-5.7</td>
</tr>
<tr>
<td>Total repl.</td>
<td>28.5 Cooling</td>
<td>7</td>
<td>19</td>
<td>0</td>
</tr>
<tr>
<td>aortic valve</td>
<td>25.5 Stable</td>
<td>5</td>
<td>1, 6</td>
<td>-3.0</td>
</tr>
<tr>
<td></td>
<td>35.2 Rewarming</td>
<td>2</td>
<td>3,20</td>
<td>-2.5</td>
</tr>
<tr>
<td>Total repl.</td>
<td>31.0 Cooling</td>
<td>8</td>
<td>12</td>
<td>0</td>
</tr>
<tr>
<td>aortic valve</td>
<td>28.5 Stable</td>
<td>4</td>
<td>2,26</td>
<td>-3.0</td>
</tr>
<tr>
<td></td>
<td>33.0 Rewarming</td>
<td>2</td>
<td>3,54</td>
<td>-5.3</td>
</tr>
<tr>
<td>Mitral commissurotomy</td>
<td>34.5 Cooling</td>
<td>4</td>
<td>7</td>
<td>-4.5</td>
</tr>
<tr>
<td></td>
<td>27.0 Stable</td>
<td>4</td>
<td>51</td>
<td>-2.5</td>
</tr>
<tr>
<td></td>
<td>35.2 Rewarming</td>
<td>2.5</td>
<td>1,51</td>
<td>-2.5</td>
</tr>
<tr>
<td>Mitral commissurotomy†</td>
<td>30.0 Cooling</td>
<td>10</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>27.5 Stable</td>
<td>6</td>
<td>45</td>
<td>-2.0</td>
</tr>
<tr>
<td></td>
<td>34.8 Rewarming</td>
<td>4</td>
<td>1,36</td>
<td>-1.8</td>
</tr>
<tr>
<td>Mitral annuloplasty and</td>
<td>29.0 Cooling</td>
<td>10</td>
<td>11</td>
<td>0</td>
</tr>
<tr>
<td>commissurotomy†</td>
<td>28.7 Stable</td>
<td>10</td>
<td>24</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>34.6 Rewarming</td>
<td>2</td>
<td>50</td>
<td>-3.0</td>
</tr>
<tr>
<td>Repair of torn mitral chordae and mitral annuloplasty†</td>
<td>28.8 Cooling</td>
<td>12</td>
<td>13</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>27.0 Stable</td>
<td>8</td>
<td>35</td>
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</tr>
<tr>
<td></td>
<td>33.0 Rewarming</td>
<td>2</td>
<td>1,16</td>
<td>-1.2</td>
</tr>
</tbody>
</table>

* This does not include the CO₂ reaching the oxygenator via the coronary suction when the surgical field is being flooded with 100 per cent CO₂ to prevent air embolism.  
† In these cases sufficient sodium bicarbonate was added to the pump priming mixture and intravenously to the patient to correct any base deficit prior to bypass. In the previous cases some sodium bicarbonate was added to the pump priming mixture but no attempt was made to correct accurately for base deficit in either the pump or the patient prior to bypass.

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hyperventilation should be avoided in the recovery room just as during the other phases and for the same reasons. Some means of humidification must be provided or plugs of accumulated mucus may inspissate and lead to further atelectasis.

If atelectasis is not widespread, the periodic use of a respirator for a few minutes of intermittent positive-pressure breathing may be sufficient. In some cases, it may be necessary to use the respirator continuously to avoid respiratory acidosis. Tracheotomy is often necessary in these patients. If such trouble is especially anticipated, it may be advisable to perform the tracheotomy several days prior to the major surgery, to avoid problems of infection or bleeding or the necessity of this procedure as an emergency under adverse conditions.

Even in the absence of respiratory acidosis, mechanical ventilation may be indicated in some cases. It is sometimes possible for a patient spontaneously to maintain a nearly normal pCO₂ but at great cost in oxygen consumption, due to the work of respiration. In the presence of low cardiac output and arterial oxygen desaturation, removing the work of respiration may be beneficial in lessening the demand on an overworked heart.¹³

Augmentation of spontaneous respiration, rather than controlled artificial respiration is the technic of choice, although, in a minority of cases, controlled ventilation may be necessary to maintain a satisfactory pCO₂. A ventilator triggered by the patient's own effort usually ensures that gross respiratory alkalosis will not occur. Gaining control of respiration, however, or initial synchronization of the spontaneous respiratory effort with the ventilator may be difficult and require a period of gross hyperventilation. To avoid this, administration of a relaxant may occasionally be necessary at first.

Metabolic Acidosis

In the presence of normal urinary excretion of acid metabolites, metabolic acidosis occurs primarily under conditions of anerobic tissue metabolism, which releases excess acid metabolites into the blood. Hence anything that interferes with oxygenation of the tissue cells may cause metabolic acidosis.

Poor oxygenation of the blood in the lungs will result in arterial oxygen desaturation and consequently there will be less oxygen available to the tissues. The cellular hypoxia so produced results in some degree of anerobic metabolism, which causes metabolic acidosis. This poor oxygenation may arise from several causes. There may be a low oxygen concentration in the respired gases or ventilation may be insufficient. Atelectasis hinders oxygenation of the blood, since blood perfusing unventilated areas of lung cannot be oxygenated.

Shift in the hemoglobin oxygen dissociation curve to the left, known to occur with respiratory alkalosis,⁹ may lead to tissue anoxia, since under these conditions oxygen is not liberated as readily by the hemoglobin. This is another reason for avoidance of gross hyperventilation following bypass.

If the blood is being adequately oxygenated in the lungs and the oxygen is readily supplied by the blood to the tissue cells, the occurrence of metabolic acidosis must then be due to cellular hypoxia from poor perfusion of tissue, resulting from peripheral vasoconstriction or low cardiac output. If the cardiac output is inadequate, this, in itself, will result in peripheral vasoconstriction and usually the two factors go hand in hand.

Prevention of Metabolic Acidosis

Good oxygenation of the blood must be achieved by adequate ventilation. The patient is usually placed in an oxygen-enriched atmosphere postoperatively, by use of an oxygen tent, and it may be necessary to administer oxygen instead of air when a ventilator is employed, especially if atelectasis is present. In the presence of severe atelectasis hyperventilation with oxygen may be necessary to provide adequate oxygenation of the blood. Under these circumstances some form of isocapnic hyperventilation technic may be necessary in an attempt to achieve adequate arterial oxygen saturation without incurring gross respiratory alkalosis. Cold may cause marked vasoconstriction; for this reason the use of thermal blankets to maintain a normal
temperature is advocated postoperatively. Shivering should be avoided, as this can account for considerable oxygen consumption. Retention of acid metabolites in the blood must be avoided and thus good urinary output should be maintained. Osmotic diuretics such as mannitol are often given prophylactically for this purpose.

Following a bypass procedure, there may be several reasons for a low cardiac output. Often the heart is grossly debilitated, even before surgery, and does not have much reserve. The corrective surgery in the heart itself, in some cases including elimination of a shunt, may present considerable increase in work to the myocardium, which may be unable to cope with this load for a time after resumption of the normal circulation. Inadequate blood volume may be the cause of both low cardiac output and peripheral vasoconstriction and thus blood replacement should be made on the basis of blood volume studies or central venous pressures. If the patient was previously maintained on digitalis, this should be continued throughout surgery and postoperatively. Following multiple blood transfusions, accumulation of citrate may result in low cardiac output which may improve after intravenous administration of calcium gluconate or calcium chloride.

**Correction of Metabolic Acidosis**

It has been well established that metabolic acidosis will cause depression of cardiac function. Since a low cardiac output results in further metabolic acidosis, a vicious circle may be established that must be broken if the patient is to survive. "Flogging" the already failing heart with cardiac stimulants and administering large amounts of vasopressors are ill advised and may actually cause the demise of the patient because of further vasoconstriction and thus more metabolic acidosis. Furthermore, vasopressors are less effective in the presence of acidosis, and ultimately may fail to maintain adequate arterial pressure.

It is more rational to correct the metabolic acidosis and continue to maintain good acid-base status by the administration of drugs that will buffer the acid metabolites in the blood and eventually correct the environment of the tissues. In addition to the use of buffers it may still be necessary to support blood pressure with vasopressors but, if so, much smaller amounts of these drugs will be required. Correction of metabolic acidosis itself should not be considered as sufficient treatment but rather an expedient method of "buying time" during which the cause of the circulatory inadequacy may be treated, e.g., by blood transfusion, improved ventilation and oxygenation, and intravenous administration of cardiac glycosides.

Sodium bicarbonate and THAM (trihydroxymethyl aminomethane)* are the two buffers most commonly used. In cases of moderate acidosis sodium bicarbonate would appear to be satisfactory. The use of sodium bicarbonate alone in combating severe metabolic acidosis may result in a large amount of sodium being given. Furthermore, bicarbonate becomes totally ionized when introduced into the blood stream and does not enter the cells where the metabolic acidosis originates, whereas THAM has been shown to remain about 30 per cent un-ionized, and it is thought that this portion enters the cells where it may act as an intracellular buffer. THAM also has an osmotic diuretic action that helps to maintain good urinary output.

When correcting metabolic acidosis, we give sodium bicarbonate at first up to a total dosage of about 2 mEq./Kg. If this is not sufficient to establish and maintain good acid-base status, buffer administration is continued with 0.3-molar THAM. If possible, the THAM is given through a catheter placed into a central venous stream, as this substance has been shown to cause local tissue necrosis if it extravasates into the subcutaneous tissue.

The correction of metabolic acidosis with intravenous buffers should not be undertaken in haphazard fashion. Administration of a fixed amount of sodium bicarbonate in a dosage according to the weight of the patient may produce some temporary improvement but is usually inadequate if the cause of the

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* Talatrol—Abbott Laboratories.
metabolic acidosis is still active. On the other hand, when acid-base status is rapidly and repeatedly assessed through blood-gas analyses, it is then possible to titrate the buffers intravenously according to their effect and by this means not only to correct the metabolic acidosis but also to maintain good acid-base status as long as is necessary with buffers; i.e., until the cardiac output is sufficient to maintain good circulation. In this way, the heart no longer has to cope with the depressant effect of the metabolic acidosis and has a better chance to recover. The blood gas analyses are made as often as necessary to maintain good control. The buffer is infused by intravenous drip in sufficient amount to reduce the base deficit to zero, and administration is then continued at a rate that will hold the arterial blood at about zero base deficit, the drip being slowed if there is a tendency toward base excess and increased if base deficit should recur. It may be necessary to continue administration in this way for several hours and the total dosage of buffer may be quite large. So far no ill effects have been seen by us with this titration method of administration. Complications previously attributed to administration of buffers may have been, in some cases, due to overdosage producing alkalosis. However, it seems more likely that in most instances inadequate amounts of buffer have been given and the effects of the continuing metabolic acidosis have been mistaken for side effects of buffer.

**Mixed Acidosis**

The occurrence of a mixed acidosis may be due to a combination of separate causes for the respiratory and metabolic components. Accordingly, the mixed acidosis may be managed usually by improving ventilation, correcting the metabolic acidosis, and treating the original causes of the imbalance.

In the period after bypass a mixed acidosis may occur, the cause of which is unresponsive to medical treatment. This may happen if prior to surgery there was a high pulmonary vascular resistance combined with a shunt that allowed some of the output of blood from the right heart to bypass the lungs. Such cases are really considered inoperable but occasionally in a borderline situation surgery is undertaken. After the corrective surgery with elimination of the shunt, the load of forcing its total output through the lungs in the face of the high pulmonary vascular resistance may prove to be too much for the right ventricle. This results in right heart failure and respiratory acidosis. Consequent low output from the left side of the heart results in poor tissue perfusion and metabolic acidosis. If the pulmonary vascular resistance is not too high, then “buying of time” through correction of the acidosis with buffers plus medical treatment of the heart failure, e.g., by rapid digitalization, may be sufficient. If this is to no avail then surgical re-establishment of the shunt may be necessary to ease the load on the right heart. Even though this is a retrograde step in the operation, it may be the only way to prevent early death of the patient from acute right heart failure. Here, once more, repeated blood gas analysis provides rapid assessment of developing changes and allows interpretations and decisions that facilitate management.

**Discussion**

Among the commonest problems in open-heart surgery are those of cerebral complications resulting sometimes in irreversible brain damage. Prevention of respiratory alkalosis may help to maintain better cerebral circulation throughout an open-heart operation so that the brain is well nourished at all times.

Clemmesen, Juul-Christensen, and Wandall have demonstrated in dogs that if the pulmonary circulation is perfused on bypass, pulmonary edema develops when severe alkalosis due to carbon dioxide deficiency is present. Although the pulmonary circulation is not perfused during open-heart operations, the bronchial circulation is intact and consequently perfused; if perfusion is with alkaliotic blood, this may be responsible for some lung damage.

Bindslev, Juul-Christensen, and Wandall have demonstrated an increased fragility of red cells with alkalosis and report that in
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hyperventilation studies on dogs, intravascular hemolysis was produced.

During the past year, using the technic here- in described, we have noticed a decided improve- ment in morbidity following open-heart surgery with very few unexplained cerebral, pulmonary, and hemolytic problems. With careful acid-base management throughout the operation we have found that (1) the pa- tient regains consciousness rapidly at the conclusion of the anesthesia, (2) there is seldom any respiratory depression or subsequent abnormal respiratory pattern, (3) there is seldom any obvious vasoconstriction or cya- nosis of extremities, (4) the blood pressure is usually good and is easily obtained by the cuff method, and (5) intensive supportive measures, such as the use of vasopressors and continuous artificial augmentation of ventila- tion, are rarely necessary.

The occurrence of metabolic acidosis after bypass may be prevented in large part by avoidance of respiratory alkalosis during bypass. One of the most important realizations is that during hypothermia an arterial blood pH of 7.40 and pCO₂ of 40 mm. Hg is not optimal but may in fact represent a relative respira- tory alkalosis. Because of all these considera- tions, we believe that severe respiratory alkalosis should not be permitted at any time during the operation. If acidosis occurs for any reason, it should be corrected forthwith. More- over, the longer the bypass period, the more important this becomes. With only a short time on bypass at normothermia, such as in repair of an atrial septal defect, it is relatively unimportant; with prolonged bypass proce- dures and especially under hypothermic con- ditions it becomes very important indeed. Eradication of the severe insult which acidosis exerts on the debilitated cardiovascular system may produce great improvement in the pa- tient’s condition.

Much has yet to be learned regarding acid- base metabolism and heart surgery. It is hoped that further data related to these changing concepts will be forthcoming in the near future.

Summary

An important part of managing patients during open-heart surgery is continuous moni- toring of the acid-base status prior to, during, and following cardiopulmonary bypass. The use of a thermostatically controlled triple elec- trode system whereby arterial pH, pCO₂, and pO₂ can be measured and base excess or base deficit quantitated by special application of the Astrup-Andersen nomogram is enthusiastically endorsed.

In the opinion of the authors, respiratory alkalosis is to be avoided throughout the en- tire operation and postoperatively, as this ap- pears to result in a base deficit, which is sub- sequently revealed as a metabolic acidosis. Particular consideration is given to the bypass phase when hypothermia is employed, since “optimal” pH appears to vary with tempera- ture.

Continued maintenance of good acid-base status with buffers is advocated. It is con- sidered that buffer administration should be on a “titration” principle with frequent arterial blood gas analyses.

Possible reasons are presented for the im- proved postoperative morbidity when optimal acid-base status is maintained throughout the operation and postoperatively.

References


Articles should be so written that not only intelligent people but specialists in fields other than those of the writer may read them with pleasure and profit. No article, even the most learned, needs be so written as to rebuff all readers except those strengthened to their task by animus or malice. An author too great or too lazy to consider and labor for the comfort of his readers, is either too grand for the condescension, or too low for the privilege, of print.—William Mills Ivins, Jr., 1881-1961. Late Curator of Prints, Metropolitan Museum of Art, New York City. Bulletin of the Metropolitan Museum of Art, February 1963. Submitted by Miss Dorothy Vickery, American Heart Association, New York.
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