ACIDOSIS DURING CLINICAL ANESTHESIA *

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For many years anesthetists remained either perplexed or indifferent to the condition termed acidosis, because the significance of experimental results had not become evident to the anesthetist. If acidosis did occur during anesthesia it was not always apparent. In other words, the anesthetist had not yet learned how to detect it, nor had he become aware of its insidious harm to the patient. Today, however, it is very different, for the anesthetist is fully alive to the importance of this condition in every anesthetic which he administers. For this changed attitude of mind we must give credit both to the laboratory work of Van Slyke, Stehle, Seevers and their associates, and to the clinical observations of Bourne and Waters. This paper summarizes the ideas of these investigators and, at the same time, answers a few of the simple questions concerning acidosis in anesthesia.

Acidosis is the state in which there is a rise of H-ions of the blood above the normal limits. This excess of acid ions may be due either to the volatile carbonic acid of the blood, in which case it is termed gaseous acidosis, or to the non-volatile acids of the blood, such as lactic, phosphoric, sulphuric or hydrochloric, in which instance it is called non-gaseous acidosis. Because of the clinical importance of non-gaseous acidosis in diabetes and nephritis, the accumulation of these non-volatile acids is commonly measured by the decrease which they cause in the carbon dioxide combining power of the blood or plasma. A decrease in this value is often spoken of rather loosely as acidosis, even if it has not progressed so far as to increase the H-ion concentration above normal value. It must not be construed that acidosis is either strictly gaseous or non-gaseous. Under certain circumstances, it is possible to have a mixture of both gaseous and non-gaseous acidosis.

These two types of acidosis occur frequently in anesthesia. In fact the anesthetist can deduce that under any one of the following circumstances, or combination of conditions, there is a degree of gaseous acidosis because of failure to eliminate the carbon dioxide: depressed respiration and circulation commonly seen in barbiturate, tribromethanol or spinal anesthesia; depressed respiration as seen in cyclopropane anesthesia and opiate depression; obstructed air passages; increased carbon dioxide of the inspired air as when open drop masks are too heavily banked with towels, when there is an insufficient flow of

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gases during the partial rebreathing technic, or when faulty valves or spent soda lime occurs in the absorption technic.

In addition to the foregoing obvious instances of gaseous acidosis non-gaseous acidosis may be equally evident. In ethyl ether, vinyl ether, and chloroform anesthesia and to a less extent with cyclopropane, there is a non-gaseous acidosis. Ronzoni and her co-workers have detected a rise in the lactic acid of the blood during ether anesthesia. Stehle and Bourne noted a fall in muscle phosphate and a rise in liver phosphate during ether and chloroform anesthesia, and concluded that the phosphoric acid of the blood must increase during anesthesia.

During clinical anesthesia non-gaseous acidosis is frequently associated with gaseous acidosis. When a patient is deeply anesthetized with ether the respirations become depressed, and carbon dioxide is not eliminated, thereby adding a gaseous acidosis to a non-gaseous one. Similarly, faulty valves or spent soda lime can easily increase the carbon dioxide of the inspired air, and so superimpose a gaseous acidosis on a patient who may already have a non-gaseous one from diabetes, uremia, or ether anesthesia. Perhaps the most common example is the anesthetization of the patient with acute appendicitis or cholecystitis. From starvation, the patient will have a non-gaseous acidosis; from the anesthetic agent—let us say ether—he has additional non-volatile acidosis. Then something goes wrong with the anesthetic machine, or if there are too many towels around the mask, interfering with the carbon dioxide elimination, a gaseous acidosis is added to a very marked non-gaseous one.

The mechanism producing acidosis involves several factors. Acidosis occurs because the body fails either to eliminate acid ions rapidly enough or to neutralize them with adequate buffer alkalis. Gaseous acidosis is due to the failure of the body to get rid of carbon dioxide through the lungs, with a steady rise in the carbonic acid of the blood. It is obvious from this that when the respiratory system is under the influence of certain drugs, such as morphine, tribromethanol, barbiturates and cyclopropane, it can no longer react by hyperventilation to small increases in carbon dioxide. Gaseous acidosis is, therefore, not a matter of neutralization or of overproduction of acid ions. It is a consequence of insufficient elimination of the carbon dioxide. Non-gaseous acidosis, on the other hand, is due to a failure to neutralize the excessive fixed or non-volatile acids in the blood, because there is inadequate alkali reserve or buffer alkali in the body to handle these acids. This preponderance of non-volatile acids, such as occurs in ether anesthesia, is probably caused both by an overproduction of these acids and also by a retarded elimination, as evidenced by the suppression of urine during anesthesia. The large reserve of alkali is then not enough to buffer this huge increase in acid H-ion concentration, and, consequently, non-gaseous acidosis results.

In most instances of gaseous acidosis, intoxication from an excess of carbon dioxide seldom occurs. When depression of circulation or of

respiration, or obstruction to respiration occurs, the threat to oxygenation becomes a real danger long before carbon dioxide retention begins to endanger the patient's life. However, when there is an increased carbon dioxide of the inspired air, and pulmonary ventilation and the supply of oxygen is adequate, then the carbon dioxide can increase to very dangerous levels and may reach concentrations which are narcotic. This is especially true when the anesthetist is supplementing tribromethanol or barbiturate anesthesia, where a volatile acidosis is already present. This excess of carbon dioxide in the inspired air can be very harmful in cases such as acute appendicitis or cholecystitis, where there is already non-volatile acidosis from starvation. If these patients are given ether anesthesia, respirations may become very rapid, and, in some instances, convulsions may be seen, as has been repeatedly emphasized by Waters. An hour or so of extremely rapid respirations may cause the patient to die on the operating table from sheer exhaustion; if he survives he will have a stormy recovery with malaise, nausea, headache and dehydration. The probable explanation is this: First, the patient has a non-gaseous acidosis from starvation before operation. The ether intensifies this non-gaseous acidosis. Still further, a mask placed on the face decreases the carbon dioxide elimination, adding a gaseous acidosis to an already high non-gaseous acidosis. As Peabody has demonstrated, it required a very slight retention of carbon dioxide to cause a marked hyperpnea when non-gaseous acidosis is already present. Apart from these more specific deleterious effects of non-volatile and volatile acidosis, it has been demonstrated that with increasing H-ion concentration, liver function is impaired so that the liver fails to store glycogen, and the blood sugar rises. In addition, the increased H-ion concentration, however produced, will also impair the power of hemoglobin to transport oxygen, and this may be contributory, along with other causes, to produce relative oxygen starvation of the tissues. This in turn will lead to the formation of lactic acid, and increase the acidosis by a vicious cycle. From this, it is obvious that no anesthetist can pass over lightly the practical importance of acidosis.

These two types of acidosis can be combated. In gaseous acidosis, as where respiration and circulation are depressed by barbiturate, tribromethanol, or spinal anesthesia, and where respiration alone is affected by morphine, the acidosis is automatically reduced when the anesthetist administers analeptics, or does artificial respiration to relieve an acute oxygen want. Similarly, in instances of depressed respiration with cyclopropane, intermittent manual compression of the breathing bag will help to reduce the retention of carbon dioxide. Where respiration is obstructed, removal of the obstruction should be as rapid as possible. The relaxed tongue, glottic spasm and narrow bore endotracheal tubes are common respiratory obstructions which the anesthetist is often prone to neglect. Finally, great care must be exercised to maintain the carbon dioxide of the inspired air at a minimum. In inhalation anesthesia, increases in pulse rate, respiratory rate and volume, rises in

blood pressure, or chemical indicators, detect increased carbon dioxide of the inspired air. When using open drop ether, especially in the cases with an acute infection of the gastro-intestinal tract, the anesthetist should avoid hyperpnea or other signs of carbon dioxide retention by lifting the mask from the face at frequent intervals, or insufflating ether through the nipple of a pharyngeal airway. It might be advisable in some instances to administer tracheal insufflation of ether. In the absorption technic, artificial aid to respiration, or controlled respirations, may be advisable. Where there is marked non-gaseous acidosis, it is important to try to reduce this with any of the well known antiacidotic fluids, given intravenously or rectally. In the better equipped hospitals it is quite simple to follow the degrees of acidosis in the patient by using the method of Shock and Hastings, making determinations on 0.1 cc. of blood taken from a finger tip.

To summarize what has been said concerning acidosis in anesthesia: Acidosis is an increase in the H-ion concentration of the blood beyond the normal limits of variation.

Two types occur: gaseous acidosis due to a carbon dioxide retention, and non-gaseous due to an increase in the fixed acids of the blood.

Gaseous acidosis is due to the failure of carbon dioxide to be eliminated through the lungs. Non-gaseous acidosis is due to an overproduction of non-volatile acids and to the failure of elimination through the kidneys. There is an insufficient reserve of alkali to buffer this preponderance of fixed acids.

Acidosis may be responsible for exhaustion or convulsions during anesthesia, and may contribute to anoxemia, or to malaise, headache, nausea, and dehydration postoperatively.

The two types of acidosis can be combated. In the first place, every effort should be made to aid the body in eliminating carbon dioxide; secondly, a suspected non-volatile acidosis should be treated with antiacidotic measures before, during and after anesthesia.

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