Three Aspects of Problems Related to the Geriatric Patient

Cincinnati, Ohio

PART I

As nurse anesthetists today we must have a thorough knowledge of anesthesia and the ability to apply this to the millions of aged ill patients in our population. Although science has given us immunity to many communicable diseases, we are on the frontier of the growing forces of chronic and disabling diseases that surgeons have learned to treat and improve. Comprehensive health care will lead to the prevention and health control of disability and dependency. (Table II)

The number of persons over 65 (Fig. 1) will jump from 17 million in 1960 to an estimated 25 million in 1980—an increase of 44 per cent. (Table I)

![Fig. 1: A 96 year old man tells his wife about his feelings six years after artificial valves were placed in his heart.](image)

- **STATISTICS**
  - **LIFE EXPECTANCY**
    - 1900 — AGE 48
    - 1950 — AGE 68
    - 1970 — AGE 70
  - **PERCENTAGE OF OPERATIONS**
    - 1935 — 10 %
    - 1950 — 25 %
    - 1960 — 47 %

<table>
<thead>
<tr>
<th>Table I</th>
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*From the School of Anesthesia, Cincinnati General Hospital, Cincinnati, Ohio. Presented at the annual meeting, Tri-State Assembly of Nurse Anesthetists, Chicago, May 1, 1967.*
pulmonary modifications are repeatedly encountered. Assessment of circulating blood, electrolytes and nutritional factors should be evaluated by laboratory procedures and deficits corrected before anesthesia is superimposed.

Many important advances have recently been made in laboratory determinations. These have resulted from a combination of many factors. Determinations for the hematocrit and hemoglobin are not always indicative of loss or excess of blood volume. Blood volume studies with modern techniques can give an accurate need of the patient with signs of deficiencies or overloading without depending on clinical manifestations.

Minimal requirements of laboratory work for hospital admission are prescribed by the Joint Commission on Accreditation of Hospitals. These are hemoglobin, hematocrit, white blood count and urinalysis. In acute emergencies, some assumptions often must be allowed. If a complete battery of electrolyte studies can be made as basic laboratory studies, they should be a part of the workup and appreciated by the anesthetist. The knowledge that electrolyte alterations and acid-base balance disturbance may play a role in cardiovascular collapse cannot be emphasized too strongly. Particular attention should be focused on sodium, potassium and calcium determinations and their relationship to chloride and carbon dioxide combining power.

Let us remember that the liver is the organ of metabolism and serves as a storage reservoir for glycogen, proteins and vitamins. Impaired liver function in the geriatric patient should be evaluated before surgery to determine the degree of liver damage. Diets high in protein should be given before elective surgery where nutritional deficits have been present. Although some replacement may be given intravenously, the oral route is still the most effective. Hepatic dysfunction may make it impossible to detoxify drugs or to reduce the tendency to shock.

Effective ventilation during anesthesia has already been obtained by maximal vigilance but preoperative chest x-rays have had a relatively slow acceptance. Yet we are all aware of the changes in the respiratory system with the process of aging.

DETERMINATION OF ANESTHETIC TOLERANCE

So far no drug can be classified as "ideal" in all respects yet we are constantly searching for the agent which affords the patient the greatest safety and at the same time provides the surgeon with the best operating conditions. A patient with no emotional disturbance accepts any kind of anesthesia easily and emerges from it the same way with little aftereffect. The use of the vein or the ability to operate a gas machine can never compensate for the compassion shown to allay apprehension.

Most anesthetists are so accustomed to certain techniques and agents that they have become specialized in their use. The reasons for the advantage of one anesthetic over another may vary from clinic to clinic and from anesthetist to anesthetist. We should evaluate each drug on the basis of the toll it takes. Our lives would be made easier if there were one specific drug that could fulfill all requisites.

An induction of a 0.2% solution of pentothal sodium will quiet the ner-
vous and fearful patient and allow us to approach the maintenance of anesthesia rapidly while still preserving the patient’s cardiac and respiratory reserve. Complete depression of the central nervous system in the already narcotized patient before adjusting him to the mask for inhalation equilibrium should be avoided at all costs. With a word of reassurance the patient, especially in the geriatric group, will offer little resistance to the external stimulus of a mask or the initiation of conduction anesthesia.

RESPIRATION

All measures should be maintained to guard the airway. Hypoventilation is a term which describes the movement of gases inadequate for the needs of the individual. Apnea is an extreme degree of hypoventilation. The record is not good concerning the detection of hypoventilation. The mere absence of hypoxia does not rule out hypercarbia because of the capacity of carbon dioxide to diffuse more readily. Blockade of air flow in the alveoli is significant in anesthesia because of the delay in gaseous exchange between the alveoli and arterial blood. This delay not only interferes with oxygen and carbon dioxide transport but also impedes the flow of anesthetic gases or vapors and retards induction and recovery.

CIRCULATION

There have been many important advances in the treatment of cardiac arrhythmias. Improved methods of diagnosis have resulted with the availability of portable electrocardiographic apparatus. Training of all doctors in diagnosis of specific dysfunctions has resulted in a more definitive therapy. Vagal stimulation-like drugs of which digitalis is the most popular are often administered. Digitalis slows conduction between the atrium and ventricle. Digitalis toxicity will produce almost any type of arrhythmia. (Table III) It is well to remember that the therapeutic dose is always 50 to 60% of the toxic dose. The most common signs of overdosages are:

1. extrasystole
2. slowing of heart rate
3. varying degrees of heart block
4. tachycardia, both atrial and ventricular
5. fibrillation, both atrial and ventricular

Alterations in acid-base balance are associated with shift in plasma electrolytes and may manifest a profound effect on the production of arrhythm-

<table>
<thead>
<tr>
<th>DIGITALIS</th>
<th>Therapeutic dose</th>
<th>ALWAYS 50-60% of toxic dose</th>
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<tbody>
<tr>
<td></td>
<td>STARTS</td>
<td>MAXIMAL</td>
</tr>
<tr>
<td>CEDILANID</td>
<td>10-30 min.</td>
<td>1-2 hrs.</td>
</tr>
<tr>
<td>DIGOXIN</td>
<td>5-30 min.</td>
<td>11/2-5 hrs.</td>
</tr>
<tr>
<td>DIGITOXIN</td>
<td>and</td>
<td>25 min. to 2 hrs.</td>
</tr>
<tr>
<td>DIGITALIS</td>
<td></td>
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</table>

| Table III |
Acidity increases the incidence of arrhythmias.
Calcium should be given with extreme caution to the digitalized patient, since there may be a possible synergistic effect of the involved drugs. However, calcium gluconate may improve cardiac function when indicated.

Marked carbon dioxide retention may be present in the absence of hypoxia, especially if the anesthetic mixture contains a high percentage of oxygen with positive pressure. During the progression of a state of uncompensated acidosis, the marked increase in hydrogen ion concentration reflects severe metabolic alterations which eventually lead to irreversible cellular damage. Severe cardiac, hepatic, nephritic and pulmonary dysfunctions may be supplemented with the use of an amine buffer.

Both phenothiazine and reserpine reduce blood pressure and increase the risk of superimposed sympathetic depression. The knowledge of all anesthetic agents is beneficial and must be taken into consideration as the best insurance against fatal effects.

**PLAN**

The ultimate goal should be as with all anesthesia, proper assessment of the patient's physical condition and striving toward good alveolar ventilation with the least amount of anesthesia.

**REFERENCES**


**PART II**

A total understanding of the anatomical and physiological changes of the aging process is becoming more important in this day of increased longevity. There are certain occurrences that are inevitable as one grows older. In some these changes are more pronounced than in others.

Some of the most common diseases encountered by the anesthetist are atherosclerosis, arteriosclerosis, left ventricular hypertrophy, cardiac arrhythmias, emphysema, pneumonia and dehydration. When these conditions are present at the time of surgery, it is obvious that the two main concerns of the anesthetist are control and maintenance of the blood pressure and control of the airway with proper ventilation.

There are five factors that influence blood pressure. (Table IV) They are: cardiac output, total blood volume, peripheral resistance, blood viscosity and elasticity.

**FIVE FACTORS INFLUENCING BLOOD PRESSURE**

<table>
<thead>
<tr>
<th>1. Cardiac output</th>
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<tr>
<td>2. Total blood volume</td>
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<tr>
<td>3. Peripheral resistance</td>
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<tr>
<td>4. Viscosity of blood</td>
</tr>
<tr>
<td>5. Elasticity</td>
</tr>
</tbody>
</table>

**Table IV**

Cardiac output is influenced by three factors:

1. **Venous return to the heart.** This is the amount of blood returning to the right side of the heart, and may be decreased due to hypovolemia, abnormal peripheral vascular function, drugs, anesthetic agents and position of the patient.

2. **The force of contraction of the cardiac muscle.** The normal heart has the ability to stretch and accommo-
date an increased venous return as seen in exercise. The aged heart in many cases has been overworked or damaged by disease and cannot accommodate by increasing its force of contraction. This situation can be remedied either by decreasing the work load of the heart itself or strengthening the myocardium with drug therapy.

3. *The rate of contraction.* The faster the rate the less time the heart has to fill properly. A fast rate leads to a decrease in output. On the other hand a rate too slow allows the heart to fill but decreases the output by a decrease in contraction. Anything that creates tachycardia in the geriatric patient may cause an increase in the work load and a decrease in output. Patients receiving digitalis preparations to slow and strengthen myocardial contraction will many times maintain better stroke volume if a vagolytic drug such as atropine is not used as a premedicating agent.

**TOTAL BLOOD VOLUME**

The vascular system should be in slight overdistension under normal conditions. A decrease in blood volume may result from hemorrhage and trauma, emaciation, anemia, and dehydration. These conditions should be corrected before surgery, if time allows, with such agents as plasma, dextran, and whole blood.

**PERIPHERAL RESISTANCE**

There is resistance to the flow of blood by the small arterioles, capillaries, and venules. The small vessels have the ability to contract and shunt large quantities of blood from one part of the body to another. Conversely any dilatation of these vessels can cause a pooling of large amounts of blood. When this occurs there is a decrease in venous return to the heart followed by a drop in blood pressure due to decreased cardiac output. In the young this condition can quickly be corrected with the use of vasoconstrictor drugs, but in the elderly the vessels have become sclerosed and will not respond. When there is a decrease in total circulating blood volume, whether it is due to blood loss or decreased peripheral resistance, the body has a regional compensatory mechanism that attempts to maintain blood pressure and flow to the heart and brain. This maintained flow may be at the expense of the kidneys, and vasopressor drugs only serve to add insult to injury.

**VISCOSITY**

The thickness of the blood is measured against viscosity of water which is used as the unit by which all other liquids are measured. Blood is five times more viscus than water. The high viscosity of blood creates its own resistance in the peripheral vascular system. The two components mainly responsible for this viscosity are the corpuscles and the plasma proteins. The viscosity is lowered by a decrease in proteins due to diet deficiency and some anemias. A false increase is seen in patients who are in a state of anhydremia. These conditions can be aided by high protein diets preoperatively and the use of whole blood when indicated.

**ELASTICITY OF ARTERIAL WALLS**

During systole or contraction of the ventricle, blood rushes out and overdistends the arteries. During diastole or relaxation of the ventricle the arteries contract back to their normal size thus pushing the blood through the vascular system. If the arteries were rigid and had no elastic ability there would be no diastolic blood
pressure. In other words this elastic ability of the arteries acts as a secondary pump to move the blood forward between heartbeats.

With a basic understanding of the factors that influence blood pressure we will discuss the diseases most frequently encountered.

ARTERIOSCLEROSIS

The vessels of the brain, kidneys and coronary arteries become very hard and brittle due to atrophy of the muscular layer. The muscular layer is then replaced with salts of lime. Due to their loss of elasticity these diseased vessels cause a deficiency in circulatory compensatory mechanisms. The arteriosclerotic patient will have monumental changes in blood pressure due to abrupt changes in position and will not respond readily to peripheral vasoconstrictors. They must be handled with great care and must not be allowed to experience severe hypotension for prolonged periods.

ATHEROSCLEROSIS

There are fatty changes that involve the intima of the vessels. Patches of the lining break down and are replaced with lime salts which tend to spread and strip the inner lining. This may cause a weakening of the vessel and precipitate such emergencies as cerebral hemorrhage or coronary thrombosis. If the plaque becomes enlarged it may completely occlude the vessel. Due to the irregular inner surface of the vessel, clotting is encouraged. These emergencies may arise at any time and necessitate careful and constant vigilance in monitoring and controlling blood pressure.

LEFT VENTRICULAR HYPERTROPHY

This condition results when there is an increase in the work load of the heart. It may be due to a disease of the heart itself, or to increased peripheral resistance. When the compensatory mechanisms allow the heart to meet this stress without failure it is called compensation. The heart uses its reserve power to meet the strain of aging and therefore has difficulty responding to additional strain such as anesthesia and surgery. When added strain does occur the patient may develop decompensating heart disease. This is demonstrated clinically by decreased cardiac output, a drop in systolic blood pressure and an increase in capillary and venous pressure. A patient who is suspected of decompensation, increased circulating volume, or decreased circulating volume can be easily monitored with a venous line.

There are many sophisticated venous monitors on the market, but the necessary equipment is available in most situations: an intravenous infusion of normal saline (preferably), a spinal manometer, intravenous tubing, a three-way stopcock and an intracath with an extension tube. The
venous pressure can be monitored through a peripheral vessel providing a large needle or intracath is used and there is no compression of the limb in which it is placed. (Table V) The most accurate reading will be obtained through a central venous pressure line. The vein we most often use for this is the external jugular vein. The intracath can then be placed almost at the right atrium. Normal venous pressure values are expressed either as 8-12 centimeters of water pressure or 80-120 millimeters of water pressure.

**CARDIAC ARRHYTHMIAS**

The two most common arrhythmias seen in the senescent patient are auricular fibrillation and heart blocks of varying degrees.

Any disease causing ischemia or hypertrophy of the atrium can produce atrial fibrillation. This is not a benign disease. Clots may form in the atrium and later cause emboli that may cripple or kill. Atrial fibrillation can sometimes be recognized without an electrocardiogram. Since all the impulses from the atrium are not able to travel through the atrioventricular node, the ventricular rate is usually very irregular. Many that do travel through cause the ventricles to contract before they have properly filled. Thus the output by the ventricles is so decreased that it is impossible to palpate this ejection as a peripheral pulse. This situation produces a pulse deficit, or a difference in the number of beats counted at a peripheral pulse and those heard at the apex with a stethoscope. Before an attempt is made to treat this an electrocardiogram should be taken to prove the condition. Obviously these patients can not tolerate any drugs that will cause tachycardia or hypotension. If they have been treated pre-operatively they usually will come to the operating room on such drugs as digitalis or quinidine. A thorough understanding of the mechanism of this disease and of the drugs are necessary for the anesthetist to choose the proper anesthetic agents.

Reports of first, second and third degree heart block may be seen in patients who have had electrocardiograms preoperatively. In first degree heart block all impulses travel from the atrium to the ventricle but at a slower rate than normal. In second degree block only some of the impulses travel through the atrioventricular node. These impulses usually travel through at the same ratio and produce anything from 2:1 to 8:1 heart block. In other words, in 2:1 heart block, for each two beats in the atrium only one impulse travels through the atrioventricular node. In third degree block, no impulses are able to traverse the A-V node. Heart block can be a transient occurrence and may be caused by such things as severe hypotension or anoxia causing an infarction, electrolyte imbalance, mainly hyperkalemia, digitalis effect, coronary sclerosis, and hypertensive heart disease. Many of these patients will come to the operating room on such drugs as the Rawolfias, diuretics, digitalis and isuprel. Here again it is profoundly important to know and understand the action of these drugs.

Emphysema is recognized as the most common disabling diseases of the respiratory system. The alveoli of the lung and the lung itself lose their elasticity and remain in a state of inflation. There is a partial obstruction to a bronchus which permits air to enter more freely than it can leave. There is no single causative factor but various pathogenic mechanisms play a
role: (1) chronic bronchiolar infection or allergic edema of the mucosa; (2) severe recurrent paroxysmal coughing which results in air trapping; (3) disorders of the pulmonary or bronchial circulation; (4) structural defects of the thorax; (5) changes in hormonal influence.

Some of the clinical signs manifested by the emphysematous patient are history of asthma, chronic cough and expectoration, dyspnea without orthopnea, and an increase in the anteroposterior diameter of the chest. Expiration is prolonged and often accompanied by rhonchi and wheezes at the end of expiration. There is a tremendous increase in the hematocrit, hemoglobin and red blood cell count, as an attempt to compensate for a decrease in the exchange of carbon dioxide and oxygen over the alveoli. This creates a corresponding increase in cardiac work, without corresponding improvement in blood oxygenation.

The emphysematous patient depends on a degree of hypoxia to support his respiratory drive. If you remove this drive by the administration of oxygen you are obligated to maintain proper ventilation with a slow rate and prolonged inspiratory phase. An attempt to control emphysematous respirations, as you would those in a patient with normal lungs, will only lead to cardiovascular disturbances of hypotension and eventual heart failure.

The use of such units as IPPB both preoperatively and postoperatively is of great value. Oral expectorants may be used to aid in the elimination of tenacious secretions. Drug therapy may include aminophyllin, isuprel, ephedrine and steroids.

REFERENCES


PART III

Since the geriatric patient is so often affected by acid-base balance, it seems impractical to speak of the care of the aged under anesthesia without some understanding of the subject. This is a subject surrounded by much controversy and confusion in the search for new definitions. In spite of the complicated picture, we will try to present a basic workable concept to be used in the clinical situation. As a starting point, we should be familiar with the normal values of the basic chemistries. (Table VI) The normal values will vary somewhat from laboratory to laboratory but are usually within the figures given here.

<table>
<thead>
<tr>
<th>SERUM VALUES</th>
<th>NORMAL SERUM VALUES</th>
</tr>
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<tbody>
<tr>
<td>pH Serum</td>
<td>7.37 - 7.45</td>
</tr>
<tr>
<td>Sodium</td>
<td>136 - 145 mEq/L</td>
</tr>
<tr>
<td>Potassium</td>
<td>3.5 - 5.5 mEq/L</td>
</tr>
<tr>
<td>Calcium (as CO&lt;sub&gt;3&lt;/sub&gt;)</td>
<td>4.5 - 5.5 mEq/L</td>
</tr>
<tr>
<td>Chloride</td>
<td>9 - 11 mg %</td>
</tr>
<tr>
<td>Bicarbonate (combining power)</td>
<td>20 - 30 mEq/L</td>
</tr>
<tr>
<td>Protein (total)</td>
<td>6 - 8 gms. / 100 ml</td>
</tr>
<tr>
<td></td>
<td>14 - 19 mEq / L</td>
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Table VI
Let us review the main factors in control of acid-base balance. Normal pH depends primarily upon the relative quantities of carbonic acid and bicarbonate present in the extracellular fluid. Although the blood contains five buffer pairs, we will think mainly of the normal ratio of one part of carbonic acid to twenty parts of bicarbonate when considering disturbance of the system. As a review of how buffers work as a chemical sponge to absorb excess acid or base, think of the addition of hydrogen ions (acid) to the bicarbonate buffer. The chemical neutralization reaction will cause an increase in the carbonic acid part of the system. Since carbonic acid is only weakly ionized, hydrogen ion addition has not altered the hydrogen ion concentration, but the total bicarbonate has been decreased and carbonic acid content has increased. Carbonic acid is easily broken down to water and carbon dioxide which can be eliminated through the lungs by increased tidal volume. Thus both carbonic acid and bicarbonate are reduced in total amount but the ratio remains the same and the pH remains essentially the same. The bicarbonate is replaced by metabolism of lactates and the fixed anions are excreted later from the kidneys.

Acidosis and alkalosis are relative terms as the body never becomes truly acid. Table VII shows the normal pH range and the limits of pH change compatible with life. Now let us look at the primary classification of acid-base disorders. (Table VIII) We find four categories: Metabolic acidosis, respiratory acidosis, metabolic alkalosis and respiratory alkalosis.

<table>
<thead>
<tr>
<th>pH</th>
<th>ACID - BASE BALANCE</th>
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<tbody>
<tr>
<td></td>
<td>METABOLIC ACIDOSIS</td>
</tr>
<tr>
<td></td>
<td>BICARBONATE DEFICIT</td>
</tr>
<tr>
<td></td>
<td>RESPIRATORY ACIDOSIS</td>
</tr>
<tr>
<td></td>
<td>CARBONIC ACID EXCESS</td>
</tr>
<tr>
<td></td>
<td>METABOLIC ALKALOSIS</td>
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<tr>
<td></td>
<td>BICARBONATE EXCESS</td>
</tr>
<tr>
<td></td>
<td>RESPIRATORY ALKALOSIS</td>
</tr>
<tr>
<td></td>
<td>CARBONIC ACID DEFICIT</td>
</tr>
</tbody>
</table>

Table VIII

Metabolic acidosis may be considered as bicarbonate deficit. Four common pathways may lead to metabolic acidosis.

1. **Excessive excretion of sodium bicarbonate.** The loss of biliary, pancreatic, or lower bowel secretions via fistulas or because of diarrhea results in varying degrees of bicarbonate depletion. An impaired ability of the kidney to reabsorb sodium bicarbonate (chronic pyelonephritis) leads to renal tubular acidosis. In patients with ureterosigmoidostomy, chloride reabsorption and bicarbonate secretion by the colonic membrane often produce acidosis.

2. **Excessive production of metabolic acids.** Uncontrolled diabetes mellitus, starvation, severe infectious disease, and ketogenic diet lead to fall in plasma bicarbonate. Lactic acidosis (hypoxic or shock acidosis) is a form
of metabolic acidosis presumed to be a result of severe tissue anoxia. Onset is sudden and the condition is readily recognized — such as cardiac arrest, shock, hyperpyrexia, acute pancreatitis and certain poisonings.

3. Failure to excrete metabolic acids. Renal failure with uremia leads to metabolic acidosis.

4. Excessive ingestion of acid-forming substances. Excessive parenteral administration of a solution of sodium chloride floods the extracellular fluid with chloride ions. Ammonium chloride, ferrous sulfate or methyl alcohol ingestion has the same effect.

Acidosis is produced when ketone, chloride, or organic acid ions replace bicarbonate ions. Characteristically the serum potassium is moderately or severely elevated. Highest values occur in patients with renal failure. Body compensatory action consists of the kidneys’ effort to excrete hydrogen ions and non-bicarbonate anions as well as effort by the lungs to blow off carbon dioxide by hyperactive breathing. The goal of therapy is to restore the level of bicarbonate. This can be done by administering sodium bicarbonate and solutions containing sodium lactate. The lactate ions are metabolized in the liver and converted to bicarbonate ions.

The second type of imbalance, respiratory acidosis, is inadequate pulmonary excretion of carbon dioxide which leads to carbonic acid excess, decrease in the ratio of sodium bicarbonate to carbonic acid, and a fall in blood pH. Renal compensation is achieved by preferential reabsorption of bicarbonate ion to increase plasma bicarbonate. If kidneys are not functioning normally to preserve bicarbonate the fall in pH will be greater.

Since respiratory acidosis is frequently seen and many times caused by or compounded during anesthesia, we need to differentiate between chronic and acute types.

Chronic respiratory acidosis is gradual in development and is associated with diseases such as emphysema or asthma. There is total uptake of body buffer with a blood pH about 7.3. The blood picture shows low serum chloride and high plasma bicarbonate. Acute respiratory acidosis occurs in minutes or hours. It is caused by acute airway obstruction, acute infectious diseases of the lungs, muscle relaxants, narcotic overdosages, CNS disease or anything that causes hypoventilation. The pH change is maximal here (as low as 6.95). Body buffers have not had time to be upset yet as the cellular mass has not entered the picture. The extracellular acidosis is much more severe than the intracellular, consequently high potassium levels are not seen until the acidosis is suddenly relieved.

Treatment for respiratory acidosis is, of course, aimed at improving the ventilation. Find the obstruction, change the soda lime, squeeze the bag harder, get a ventilator, relieve the bronchospasm, or do whatever needs to be done for good ventilation. This is the type of acidosis that is amenable to treatment with tris buffer or other intravenous buffer. In acute respiratory acidosis and in lactic acidosis the hydrogen ion load is exposed and vulnerable circulating in the extracellular fluid. Tris buffer is best used in massive blood transfusions (including heart-lung machines), hypothermia, and acute respiratory acidosis.

Acidosis threatens the patient in many ways — myocardial arrhyth-
mias, fibrillation and circulatory failure unresponsive to norepinephrine. Alterations in cardiac output and peripheral resistance render the patient unresponsive to blood transfusions or vasoconstrictors. The irritability of the heart muscle and walls of the great vessels (which is essential for maintenance of blood pressure) depends on a gradient of cation concentration across the cell membrane.

Now let us look at the opposite side of the acid-base picture — alkalosis. Metabolic alkalosis is characterized by an increase in plasma bicarbonate, a resultant increase in the bicarbonate:carbonic acid ratio and consequently a rise in pH. Hypoventilation which tends to raise the pCO₂ provides limited compensation. The kidneys tend to conserve hydrogen ion and excrete bicarbonate ions. This renal mechanism will fail in the face of continued water loss as the need to conserve sodium supersedes the hydrogen ion loss. An excess of bicarbonate is most frequently caused by loss of chloride and potassium through vomiting, as in pyloric obstruction, high intestinal obstruction, or pernicious vomiting of pregnancy. X-ray therapy or other conditions which encourage potassium loss (such as hyperadrenalism) may also cause bicarbonate excess. Prolonged parenteral use of potassium-free solution may have the same result. Chronic ingestion of sodium bicarbonate usually is compensated for fairly well by the kidneys if there is no sodium or water depletion. However, if the use of alkalies is prolonged, a sizable potassium deficit may develop, renal compensation will be impaired and marked alkalosis will result.

Treatment of metabolic alkalosis is aimed at correction of the underlying disease and simple potassium replacement. Correction of sodium and water deficits and potassium chloride replacement may not be adequate and acidifying solutions such as ammonium chloride may be needed.

The basic defect in patients with respiratory alkalosis is excessive pulmonary excretion of carbon dioxide. Compensation is achieved by two mechanisms: (1) renal excretion of bicarbonate in increased amounts, and (2) increased metabolic production of organic acids. A considerable elevation of pH often occurs early in respiratory alkalosis since the renal mechanism is not immediately effective.

Respiratory alkalosis produces a characteristic syndrome of neuromuscular irritability manifested by hyperreflexia, a positive Chvostek sign, muscular twitching, and at times a generalized convulsion. (Chvostek's sign is a spasm of the facial muscles resulting from tapping the cheek along the branches of the facial nerve.) The commonest causes of respiratory alkalosis are anxiety states, cerebral disease such as thrombosis, hemorrhage or head injury, and acute infections with fever. We can produce this condition in the anesthetized patient by hyperventilation, either with hand and bag or more likely with ambitious use of the ventilator. However, a mild alkalosis (rise in pH to 7.5) of short duration is usually well tolerated by most patients.

Treatment of respiratory alkalosis is directed at finding the cause and correcting the underlying disease. Sedation and rebreathing into a paper bag is helpful in anxiety-induced cases. Patients on ventilators can be adjusted to less pressure and volume if they become alkalotic. When re-
spiratory alkalosis continues for several days, significant potassium depletion may occur and should be treated with about 1 mEq of potassium per kilogram of body weight per day.

We should not consider acid-base balance without some understanding of the place of potassium ions in the body. Potassium is found chiefly in the cells and the body cannot tolerate much fluctuation in the serum level. The kidneys readily eliminate excess potassium. In recent years we are more aware of the important role that potassium plays in fluid therapy. There are three aspects of potassium disorders: potassium depletion, abnormal distribution of potassium, and a combination of the two.

Potassium depletion is seen in simple starvation. May I remind you that many senile patients come to us relatively starving. If they have been sick for awhile or bedfast from a fractured hip and disoriented in a strange place, their food consumption is often very low. Unless an attentive family or conscientious nursing personnel is available to insist on good fluid and food intake, this state of poor nutrition goes unchecked. On-the-spot evaluation of hydration and nutrition in the aged patient may be done by looking at the condition of the skin and mucous membranes. In dehydration the skin is doughy and fails to spring back if picked up and released. The mucous membranes are dry and dull. Is the patient edematous? Edema can be caused by overhydration, cardiac or kidney disease, or protein depletion. Generally speaking, edema occurs when the serum level falls below 5 gm. per 100 cc.

Patients maintained on potassium-free feedings may develop sizable cumulative losses of potassium. If there is no stress reaction or sodium administration, relatively little alkalosis develops; but if a stress reaction is present and sodium is administered, the loss of potassium may be associated with development of marked alkalosis. In potassium depletion due to inadequate intake and without alkalosis the serum potassium usually remains in the normal range until the total deficit is 3-5 mEq per kilogram.

Potassium depletion can be seen in both alkalosis and acidosis. In alkalosis we see severe hypokalemia due to excessive renal loss. Administration of large amounts of potassium are needed over a long time (several days) to correct this condition. The potassium depletion will not be corrected until the alkalosis is reversed because large renal losses continue. In acidosis potassium depletion is nearly always associated with a normal or even markedly elevated serum potassium because of the abnormality in distribution induced by acidosis. The abnormalities in hydration, sodium and acidosis must be corrected and the serum potassium restored to normal before potassium repletion can be undertaken. A rebound hypokalemia is often seen during the recovery phase of severe metabolic acidosis.

In many chronic illnesses such as tuberculosis and carcinomatosis, and in severe chronic cardiac failure, cirrhosis and nephrosis, potassium depletion is seen in association with a normal or mildly elevated serum potassium. High levels of aldosterone and diuretic agents combine to cause potassium loss in these patients.

Symptoms of potassium loss are variable and easily overlooked because of the more dramatic nature of the inciting cause of the potassium loss. Briefly the patient with potas-
sium deficiency exhibits progressively anorexia, nausea, muscular weakness, mental depression, lethargy, apathy, mental confusion, shallow respirations, irregular pulse, and runs a "downhill" course. As you will notice, some of these symptoms are "normal" in the elderly patient (especially a premedicated one ready for surgery), and a positive diagnosis cannot be made from them. A serum potassium determination and ECG changes are helpful. Table IX shows a rough correlation between serum potassium and the ECG. In some instances ECG ab-

| Relationship Between ECG and $K^+_{\text{s}}$ |
|-----------------|-----------------|
| 10              | 9               |
| 8               | 7               |
| 6               | 5               |
| 4               | 3               |
| 2               | 1               |

Table IX

ormalities appear before manifest hypokalemia. This is particularly true in digitalized patients where digitalis toxicity may be induced by mild degrees of potassium depletion. In hypokalemia ($K^+_{\text{s}}$ 2-3) we see ST depression, low amplitude T wave or inversion of T wave, and prominent U wave. In hyperkalemia we see first a prolonged P-R interval and then absence of atrial activity (no P wave), tall slender peaked T wave and widening of QRS complex. In hyperkalemia the cardiotoxicity at a given serum potassium becomes more marked by a decrease in serum sodium.

Any catabolic state such as might be induced by trauma, burns, major surgery, or sepsis leads to widespread cell damage and liberation of the intracellular contents. A severe catabolic state might liberate up to 90 mEq of potassium per day that the body must excrete. This may lead to transient hyperkalemia. In these patients it is advised not to use large single doses of succinylcholine chloride. Succinylcholine causes a release of potassium as the muscles depolarize. If it happens rapidly as when marked fasciculations occur, cardiac arrest can result from the momentary high potassium that is released. It is better to choose some other method for relaxation than with succinylcholine for intubation in these cases.

In debilitated geriatric patients it is well to consider awake intubation. Elderly patients and all hypovolemic patients are sensitive to pentothal. It causes a reduced cardiac output with resultant hypotension. The use of muscle relaxants and positive pressure breathing will compound hypotension. This sequence can produce severe circulatory depression. Elderly patients tolerate topical anesthesia well. Small doses are sufficient because they already have obtunded pharyngeal and laryngeal reflexes. They are often easier to intubate because they are edentulous. A transtracheal block is an easy method for good local anesthesia prior to intubation. 1% Pontocaine 2 ml. or 5% Cyclaine 3 ml. are generally used. Another easy method is blind nasal tracheal topical anesthesia. A well lubricated catheter is passed through the nose into the trachea as for a tracheal toilet. A sharp cough indicates passage into the trachea. 1% Pontocaine 3 ml. or
2% Xylocaine 5 ml. is injected through the catheter into the trachea. The resultant cough sprays the cords well. Once the endotracheal tube is in place inhalation anesthesia is easily accomplished with cyclopropane.

Another technique that can be well used in critically ill patients for intra-abdominal analgesia is peritoneal lavage. A large volume of a relatively dilute solution of the local anesthetic agent gives the best results: Pontocaine 0.1% solution gives 1-1½ hours of anesthesia; Procaine 1.0 or 0.5% gives 30-40 minutes of anesthesia; Xylocaine 0.5% gives 30-60 minutes of anesthesia (Xylocaine gives best results).

Up to 200 ml. of solution is poured into the peritoneal cavity. The peritoneum is closed either with a pack or by pulling the edges together. The solution is allowed to remain in the cavity 5-8 minutes and then the excess is suctioned away. Toxic reactions are negligible and the relaxation can be astounding. The intestine shrinks out of the way. A patient undergoing exploratory laparotomy under local anesthesia can be comfortable and relaxed with this technique.

REFERENCES