As more and more geriatric patients undergo surgery, the anesthetist has to become better acquainted with the special problems these patients present. The author focuses on the cardiovascular aspects of these cases, placing emphasis on the clinical significance of the altered physiology in relation to anesthesia.

There are now 20 million Americans over 65 years of age; and by the year 2000, it is possible that one-quarter of the total U.S. population will consist of the elderly. Life expectancy continues to increase; and as a consequence, surgical intervention in the geriatric patient has become necessary on a more frequent basis.

The states of California and Florida are both distinctly recognized as meccas for the elderly. Approximately 8,000 patients undergo operations annually at Saint Mary’s Hospital in West Palm Beach, Florida. The average age of the 50-year and over group of patients is 70, and the average hospital stay is 15 days for major operative procedures. Our remarkable success with the elderly has resulted from a clinically sound pyramid of care, based on a concentrated team effort. A body of knowledge and clinical experience now has emerged that provides a sound framework for the development of the goals and principles of anesthesia in the elderly.

Cardiovascular dysfunction is frequently encountered in the surgical geriatric patient and may result in the development of serious problems during anesthesia. In this article, we will briefly review some of the physiologic alterations and subsequent problems encountered in our patients. The aging respiratory system, which is integral to cardiopulmonary function, is beyond the scope of this article, and will not be covered.

Normal cardiovascular changes in aging

Physiologic changes in aging are well established. There is a 30-40% reduction in cardiac output between the ages of 25 and 65 years. Increased work loads lead to hypertrophy of cardiac muscle, and the heart becomes less distensible with a decrease in contractility and a reduction in stroke volume. The ability of the heart to increase its rate in response to stress diminishes, and more time is required for return to resting levels. Variable loss in sympathetic tone with vagal dominance contributes to the slow pulse normally observed in the elderly. Energy expenditure is greater and oxygen consumption increases in proportion to loss of cardiac power for a given amount of work performed.

The arteries lose elasticity and become progressively more rigid and less compliant. Total peripheral vascular resistance increases (although cardiac output falls), and a steady rise in systolic and diastolic pressure occurs with ad-
The primary problem: stability of circulation

The primary problem during anesthesia in all geriatric cardiovascular dysfunction is to maintain a stable circulation so as to maximize tissue perfusion. Stability of circulation depends on a good cardiac output, adequate circulating volume, and a compliant vascular bed.

Functional reserve in all organ systems diminishes with aging, and risk increases. Variations in myocardial performance can be related to a fall in cardiac reserve, which in turn, decreases the capacity of the heart to respond satisfactorily to a work stress. Superimposed disease adds to the detriment by further reducing the functional capacity with a greater impairment of cardiac performance and hemodynamics.

In a normal state, the heart is able to increase cardiac output by a reflex increase in heart rate to supply metabolic demand. This response is not sufficient to increase cardiac output when ventricular function is compromised. The increase in cardiac output must be achieved by increasing the stroke volume. The determinants of stroke volume are: (1) the preload, (2) the afterload, and (3) contractility.

The preload (end diastolic volume or pressure) can be equated to the total intravascular volume as determined by ventricular filling pressure. According to the Frank-Starling relation, the force of myocardial contraction is a function of end diastolic volume and cardiac muscle fiber length. End diastolic volume depends chiefly on venous return. A multitude of factors affect venous return both during anesthesia and the operation and, therefore, increase or decrease the preload to alter stroke volume and cardiac output.

The afterload (aortic or pulmonary artery pressure) is the resistance which must be overcome to eject blood. Sympathoadrenal discharge in response to changes in volemic states during anesthesia cause a compensatory increase in afterload, so as to restore circulating volume and maintain tissue perfusion. An excessively high afterload may result from uncontrolled hypertension of any origin. On the other hand, the use of vasodilators reduces excessive afterload and results in improved cardiac performance.

Myocardial contractility depends on the Frank-Starling mechanism and the inotropic mechanism. Anesthetic drugs depress myocardial contractility, and the reduction in contractility is directly proportional to the concentration of drug. Factors during anesthesia that exert a negative inotropic action, such as hypoxia, acidosis, or alkalosis, drug toxicity, and electrolyte imbalance, will alter cardiac dynamics.

The relationship between the heart and circulation is not that of master-servant but rather one of complete interdependence. Blood pressure, which is a correlate of cardiac output and peripheral resistance, reflects the complex interplay between the heart and circulation. This interplay is modulated by the balance between the sympathetic and parasympathetic nervous systems.

During anesthesia, there are an indeterminate number of factors that interact to alter myocardial contractility, venous return, and total peripheral resistance. As a result, excessive loads may be placed on the heart by an increase in either preload or afterload and altered contractility. In the aging heart, and especially where superimposed disease has further impaired myocardial function, one or more compensatory mechanisms may fail. Most important then, is a steady level of anesthesia—balanced, so that there is amnesia, analgesia, relaxation, and reflex inhibition. There should not be cardiovascular depression or sympathetic overactivity during a well conducted anesthesia.
An adequate circulating volume is critical to the stability of circulation. A frequently overlooked factor in the elderly is that of venous stasis with incompetency of venous valves as a result of deep thrombophlebitis. This affects the venous capacity. These patients normally have a stagnant venous pool which can significantly increase to the detriment of the circulating volume under stress. Loss of sympathetic support and other compensatory mechanisms during anesthesia may result in increased capacitance and massive pooling with delay in venous return.

Blood volume, electrolytes, and nutrition are frequently deficient in the elderly, and appropriate preoperative replacement in type and quantity is essential for stability. The anemic patient, particularly with limited cardiopulmonary reserve, responds favorably to gradual administration of packed cells. Chronic visceral and malignant diseases are accompanied by a decrease in blood volume and a low albumin. The replacement of albumin is essential, since plasma-colloid-oncotic pressure is important both in transcapillary exchange and the maintenance of intravascular volume.

An expanded extracellular fluid volume is characteristic in chronic cardiac or renal disease. Dilutional hyponatremia may be present and must be differentiated from salt depletion. Fluid restriction and diuretics are indicated for dilutional hyponatremia, whereas isotonic saline containing sodium 154 mEq/L is administered for true salt depletion. The patient in congestive heart failure with an expanded extracellular fluid volume is especially prone to hyponatremia, hypokalemia, and alkalosis.

**Clinical cardiovascular problems**

The major cardiovascular problems encountered in the geriatric surgical patient in our experience can be categorized as follows:

1. Impairment in myocardial performance, mainly by congestive heart failure, myocardial infarction, arrhythmias, and conduction defects.
2. Critical changes in circulating volume and peripheral vascular dynamics characterized by hypotension or hypertension and manifested as crises and/or hypoperfusion.
3. Drug toxicity and adverse effects of drug actions and interactions on the cardiovascular system.
4. Pulmonary emergencies such as pulmonary edema, pulmonary embolism, and pneumothorax.
5. Cardiovascular dysfunction secondary to chronic disorders of the lung—primarily chronic obstructive lung disease (emphysema with or without cor pulmonale).

The discussion that follows will be limited to some of the considerations in the first three categories that are pertinent to anesthesia. Categories 4 and 5 are beyond the scope of this presentation.

**Congestive heart failure**

Congestive heart failure is a clinical syndrome that is commonly observed in the aged. Those patients with ischemic heart disease, hypertension, outflow obstruction resulting from mitral or aortic valvular lesions, and severe chronic obstructive lung disease are prone to develop heart failure.

Patients with incipient and evident congestive heart failure should be fully digitalized preoperatively. Underdigitalization may control the cardiac rate at rest but will not prevent a rise of considerable magnitude during intraoperative stress. A persistent tachycardia above 90, gallop rhythm, hypotension, narrow pulse pressure, decreased lung compliance, and elevated venous pressure are indications of inadequate digitalization. In this instance, an incremental dose of digoxin may be indicated during anesthesia, even though the serum level is in the therapeutic or toxic range. The elderly will often respond favorably to an additional minimal dose of digoxin ranging from 0.0625-0.25 mg intravenously.
Contractility, be it a primary or secondary impairment, is the basic underlying defect in congestive heart failure. The combination of anesthesia and the operation may lead to marked impairment of the contractile state with a rise in pulmonary venous pressure and progression to frank pulmonary edema. The patient with valvular heart disease, particularly those with a limited output (mitral or aortic stenosis), will suddenly develop pulmonary edema when challenged with rapid and excessive volume loading.

The increased extracellular fluid volume associated with congestive heart failure is not a mobile functional pool, and therefore, cannot compensate for a loss of vascular tone and hemorrhage. Aggressive replacement of fluid and blood to maintain blood pressure and tissue perfusion may result in a hypervolemic state upon the patient's recovery from anesthesia and a return of the vascular reflex response. In our experience, the hypervolemic state is often safer for the patient and easier to manage than that of hypovolemia. Pulmonary edema responds much more readily to aggressive treatment than does cardiovascular collapse.

Guidelines to fluid replacement are not distinct. Careful assessment of volume status by appropriate monitoring is essential. Minimal and slow volume restoration, based on the blood pressure, pulse, pulse pressure, renal output, and central venous pressure or pulmonary wedge pressure, is indicated. The Swan-Ganz flow-directed pulmonary artery catheter measurements serve as a reliable index to left ventricular function and guide volume repletion in acute states.

Myocardial infarction

The most frequent pathology encountered in the elderly is ischemic heart disease and arteriosclerosis. Coronary artery disease is prevalent in the surgical candidate and is a main factor in morbidity and mortality. The clinical patterns of myocardial infarctions in the elderly are atypical. The absence of angina may indicate good collateral circulation or self-imposed limited activities at less than stress level. The majority of these patients exhibit an abnormal preoperative electrocardiogram.

A history of previous myocardial infarction deserves careful study since hemodynamic abnormalities may be present. Anterior infarctions from occlusion of the left anterior descending coronary artery may result in destruction of a large area of muscle mass. Subsequent transmural scar formation with paradoxical contraction impairs the functional capacity of the left ventricle. The potential for heart failure is increased in these patients and may be precipitated by acute ischemic insult with prolonged oxygen debt during anesthesia.

Inferoposterior infarction results from occlusion of the right coronary artery; and these patients are usually not at high risk. Heart block is associated more frequently with inferior than with anterior infarction. Serious or fatal heart block may occur with anterolateral infarction due to extensive myocardial injury involving the peripheral branches of the Bundle of His. The common types of intraventricular conduction disturbance, such as bundle-branch and fascicular block, have little clinical significance for anesthetic management. They do not alter contractility, nor do they induce ventricular fibrillation.

The sinus node artery arises from the right coronary in 55% of the patients and from the left circumflex coronary artery in 45%. Occlusive disease proximal to or in the sinus node artery may result in sinus bradycardia or instability of the sinus mechanism. This may be a contributing factor to the slow pulse observed in the elderly. Ventricular irritability may arise during anesthesia if the pulse rate becomes too slow. The potential for atrial tachyarrhythmias is also increased in the presence of bradycardia. Increas-
ing the pulse rate with atropine will frequently eliminate irritable foci.

Silent myocardial infarctions may occur during anesthesia or in the early postoperative period. Atheromatous plaques do not occur at the subendocardial level, but the subendocardium is extremely sensitive to lack of oxygen from perfusion deficit. Changes in coronary blood flow during anesthesia may lead to ischemia and necrosis in the subendocardium, which may later progress transmural.

Myocardial depression from drugs, acute blood loss, anemia, dehydration, electrolyte depletion, hypotension, severe hypertension, acidosis or alkalosis, or any acute derangement from the patient’s normal basal level, may compromise the coronary blood flow and precipitate subendocardial infarction. An existing subendocardial infarction preoperatively may be aggravated by the above mentioned factors and progress transmural. RS-T and T-wave changes during anesthesia indicate an ischemic myocardium and are suggestive of subendocardial injury.

Arrhythmias and conduction disturbances

The incidence of arrhythmias and conduction disturbances is high with advancing age. In our experience, atrial fibrillation, atrial flutter, premature atrial contractions, ventricular premature beats, paroxysmal atrial tachycardia, and varying degrees of heart block are the most frequent patterns observed in the geriatric patient preoperatively.

The elderly patient is more vulnerable to potentially lethal complications from arrhythmias in the intraoperative period. Alteration of rate and stroke volume reduce cardiac output, followed by hypotension and inadequate perfusion. Premature ventricular contractions may deteriorate into ventricular tachycardia and fibrillation. Premature atrial contractions may be a forerunner of atrial fibrillation. Thrombosis formation with embolization may result from atrial fibrillation. Loss of the atrial contribution to ventricular filling from asynchrony during atrial fibrillation may result in severe hypotension.

Abrupt changes in circulating volume, as in severe hemorrhage, will produce myocardial ischemia. The rapidity of development of serious arrhythmias in an ischemic myocardium is directly proportional to the degree of myocardial hypoxia. Myocardial oxygen consumption ($MVO_2$) is determined by the dynamic interplay between intramyocardial tension, contractility, and heart rate.

An increase in pulse rate will increase myocardial oxygen consumption and widen any pre-existing disparity in the ratio of oxygen demand to supply in an already-compromised, aging myocardium. Critically ischemic areas of the myocardium will be further deprived of oxygen, and arrhythmias will develop, or the heart will fail.

An effective heart rate is a factor in the maintenance of cardiac output. Differences of opinion exist as to how much of an increase in rate above the basal resting level can be safely tolerated. The basal rate in the majority of our patients is between 60 and 85. Our clinical impression is that, at this basal level, persistent heart rates above 100 during anesthesia are harmful and threaten the myocardium. Tachycardia shortens diastole, which results in inadequate coronary filling and an ischemic myocardium. Atropine is omitted from the premedication in patients with an existing tachycardia; and the use of pancuronium for relaxation in such instances is debatable. These are empirical decisions, based on the preanesthesia evaluation and the clinical experience of the anesthetist.

Nodal tachycardia accompanied by a significant decrease in blood pressure has been observed with the use of halogenated agents, pancuronium, and in digitalis intoxication. Hypotension results from the faulty dynamics produced by asynchronous contraction. The atrial contribution to ventricular filling is lost because of regurgitant flow from improper closure of the atrioventricular
valves; and as a result, the stroke volume falls. If spontaneous reversion fails to occur, the administration of atropine or mephentermine (Wyamine®) may stimulate the sinoatrial node to fire.

**Cardiac pacing**

When temporary or long-term pacing is indicated, the transvenous approach is the most common modality used for our patients. The most common indications for cardiac pacing in our elderly patients are: second or third degree block, sick sinus syndrome (SSS), and bilateral bundle branch block. Symptomatic criteria for pacing are: marked sinus bradycardia with hypotension, a single documented episode of fainting, Stokes-Adams attacks, angina pectoris, congestive heart failure, and any evidence of cerebral insufficiency (lightheadedness, dizziness, and memory failure).18 29,45

Degenerative disease of the conduction system, rather than arteriosclerotic heart disease, is the major cause of chronic heart block in the aged.8,37,42 Lesions that involve the conduction fascicles have been described by Lenègre and Lev.23,42,44 The characteristic lesion in Lenègre’s disease is a sclerodegenerative process in the conduction system. In Lev’s disease, there is fibrosis and calcification of the fascicles as a result of invasion from the diseased fibrous structures that are adjacent to the conduction system.

Sick sinus syndrome encompasses the bradycardias from all causes. In these patients, the rate does not respond to provocative tests (atropine, isoproterenol, and atrial pacing). Cardiac output is low because of the severe bradycardia. In addition, ventricular ectopic beats escape or tachyarrhythmias appear, so that the net result is hypoperfusion of critical regional circulations.15

Unipolar demand pacemakers are more frequently implanted than fixed-rate units for our patients. The fixed-rate unit may compete with intrinsic cardiac rhythm for capture of the ventricular beat. The R-wave of the pacemaker beat may fall on the upstroke of the T-wave of the previous natural beat and precipitate ventricular fibrillation.8,45 Competition is less apt to occur in chronic block. Because there is no sensing device in the fixed-rate pacemaker, it is highly resistant to electrical interference.

Demand pacemakers are standby units which sense spontaneously occurring cardiac activity and suppress their generators upon this signal.18 The risk of sensing stimuli from other electromagnetic fields, such as the electrocautery, is certain—thus, pacing may cease.

In our practice, few problems have been encountered in patients with pacemakers. Local anesthesia is used for implantation of a pacemaker, using transvenous leads and for all battery changes. General anesthesia for thoracotomy and placement of epicardial leads remains a calculated risk, unless transvenous endocardial pacing can be established preoperatively. In our setting, it is unlikely that this modality will be realized, since epicardial leads are placed only when transvenous pacing ultimately fails. A careful, deliberate induction and precise anesthetic management, along with pharmacologic support and a cardiologist in attendance, has minimized complications.

The electrical hazards associated with pacemakers can be minimized by appropriate grounding of all equipment. Monitoring electrodes should be placed at a distance from the pacemaker unit and wires. The indifferent electrode of the electrocautery should be placed at the maximum distance. A distance of six inches from the pacemaker is considered a safe zone for use of the electrocautery.18 Temporary exteriorized pacer units and exposed wires should be protected with material, such as Saran Wrap, which serves as a resistor.

**Hypertension**

Hypertension is so common in the aged that optimal levels of blood pressure during anesthesia can only be determined by relating it to myocardial...
performance and tissue perfusion. The severity of arteriosclerosis in aging is related to the degree and duration of hypertension.22

Hypertension presents problems in the surgical patient, since it alters critical regional flows, increases cardiac work, and precipitates heart failure.12 Control of blood pressure at optimal levels with appropriate antihypertensive and other measures prior to an elective operative procedure decreases risk. Vast experience has proven it best to continue antihypertensive drugs until the day of operation. Uncontrolled hypertension may lead to cerebrovascular accident, renal failure, myocardial failure, and reduction in coronary flow leading to myocardial ischemia with subsequent serious cardiac arrhythmias or myocardial infarction.18,22

Nitroprusside is a valuable drug for the control of hypertensive crisis in the intraoperative period. The drug is potent, rapid in action, short in duration, non-toxic in clinical doses, and is ideally suited for emergency use because of its predictable action and ease of control.86 Ventricular function and tissue perfusion improve, since vasodilators unload the ventricle by reducing excessive afterload and, thereby, decrease cardiac work and myocardial oxygen consumption. In severe cardiac dysfunction with hypoperfusion of vital tissues, the use of nitroprusside lowers ventricular filling pressure and results in an increase in cardiac output.19

Digoxin

Digoxin, which is a pure glycoside, is widely used in the geriatric patient mainly for congestive heart failure and atrial fibrillation. The positive inotropic action of digoxin enhances cardiac performance and reduces overall myocardial oxygen consumption.1,82 Prophylactic digitalization is never used on the basis of age alone in our patients. A valid indication must exist to justify prophylactic digitalization preoperatively. The dosage of digitalis must be adjusted, since the volume of distribution is decreased in the elderly as a result of a reduction in lean body mass and plasma water, causing higher serum concentrations.82 The age-related decrease in renal function is also an indication for a smaller dose.

Rapid digitalization for acute intraoperative emergencies must be undertaken with extreme caution, since a therapeutic level at one point may later fall in a toxic range in the labile patient.9 The maximum dose of digoxin that we usually administer intraoperatively is 0.5 mg. In our experience, rapid total digitalization has led to electrolyte derangement and arrhythmias in the postoperative period. Unless there is an absolute indication for digitalis (such as, congestive heart failure or atrial fibrillation), our choice for conversion of arrhythmias would be cardioversion, lidocaine, or propranolol. The electrocardiograph pattern, serum potassium level, and arterial blood gases should be monitored when digitalis is administered during anesthesia.

Ventricular response is the best guide to determine an adequate initial dose of digitalis administered intravenously to patients who require it during anesthesia. Intravenous doses of digoxin initially range from 0.1-0.5 mg, depending on the heart rate. Suggested dosages are 0.125-0.25 mg for a 110-120 rate; 0.25-0.375 mg for a 140-160 rate; and 0.375-0.5 mg for heart rates over 160.49

The patient with a chronic atrial fibrillation who presents for anesthesia with a rapid ventricular rate must be carefully evaluated before more digitalis is administered. If the R-R interval is regular, it would indicate an A-V block with a junctional or ventricular tachycardia that is most likely due to digitalis intoxication. On the other hand, an irregular R-R interval with a rapid ventricular rate is an indication for an additional dose of digitalis.

Serum levels of digoxin serve as a guide to tolerance. Therapeutic levels range from 0.7-1.4 ng/ml, and levels
above 2 ng/ml presume toxicity. Tachyarrhythmias may result from an overdigitalized heart, and the problem is enhanced in the presence of hypokalemia, acidosis or alkalosis, and poor renal function. Development of digitalis sensitivity and arrhythmias in the presence of hypokalemia is well recognized. A serum potassium level below 3.5 mEq/l is a contraindication to induction of anesthesia in our practice. By the time the serum level of potassium reaches 3.0 mEq/l, there is probably already a total deficit of 400 milli-equivalents. Potassium can be safely administered intravenously at a rate of 1 mEq/kg/hr if urgently needed, especially if the level is below 3.0 mEq/l. Electrocardiographic monitoring is the best guide to detect early changes associated with high or low serum potassium levels. There is an inverse relationship between the potassium level and the pH. As the pH rises, the potassium falls; therefore, the pattern of ventilation becomes significant—and hyperventilation should be avoided. In alkalosis, the hydrogen ion in the cell falls and potassium shifts from the extracellular fluid into the cell. Conversely, the potassium ion shifts from the cell into the extracellular fluid in acidosis. Physiologic levels are least disturbed in the elderly when ventilation is directed toward maintaining arterial blood gases in a normal range.

**Propranolol**

Propranolol (Inderal®) is a very useful drug in the management of life-threatening arrhythmias, angina pectoris, outflow tract obstruction in valvular heart disease, and hypertension in many elderly patients. Arrhythmias are controlled by the beta-adrenergic blocking properties of propranolol and by its direct effect on the cell membrane, similar to that of quinidine and procainamide. The drug is used alone or in combination with digitalis and/or quinidine for control of ventricular arrhythmias. This combination has been very effective in both the immediate and long-term reduction of the rapid ventricular rates in some patients with atrial fibrillation.

The use of propranolol decreases myocardial oxygen consumption by decreasing the heart rate, left ventricular work, and the force of myocardial contraction. However, the drug prolongs the systolic ejection period and increases end-diastolic volume and pressure, so that intramyocardial tension increases. Since myocardial wall tension is a determinant of oxygen consumption, these opposing forces must remain in balance to achieve the optimum effect from propranolol. The beneficial effects of a decrease in heart rate and contractility can be undone by a dilated ventricle with excessive wall tension, so that ischemia persists and/or intensifies and the heart will fail under an increased workload.

The hypertensive patient exemplifies the use of multiple drugs and their interactions. Propranolol is used in combination with antihypertensive drugs to achieve a specific action. It is particularly useful in the elderly, since beta adrenergic blockers do not induce postural hypotension. Propranolol lowers blood pressure by decreasing cardiac output, inhibiting renin release, and depressing sympathetic activity. When used in combination with vasodilators, as hydralazine, propranolol blocks the reflex tachycardia. Vasodilators decrease cardiac output, and compensation occurs by retention of sodium and water to increase the circulating volume. Diuresis is induced by the administration of thiazides, which increase the renal excretion of sodium and chloride with an accompanying volume of water. At the same time, potassium excretion occurs in amounts sufficient to produce hypokalemia.

None of the propranolol/antihypertensive drug combinations just mentioned are discontinued in our patients, since the uncontrolled underlying con-
dition poses a greater threat. However, the problem during anesthesia may well be that of vasodilatation, hypovolemia, hypokalemia; and, further, the reflex effects of increased sympathetic stimulation of cardiac output are blocked by propranolol.1,8,8

The patient is predisposed to acute myocardial failure, unless the management of anesthesia is directed toward correction of deficits preoperatively along with light levels of anesthesia with appropriate pharmacologic support intraoperatively. Propranolol is a competitive antagonist; therefore, it is reversible. Thus, the drug of choice is isoproterenol (Isuprel®), since it has a selective effect on beta receptors.

Conclusion

A brief survey of some of the geriatric cardiovascular problems that the anesthetist encounters has been presented. A thorough preanesthesia evaluation and establishment of baselines are essential, so as to determine potential problems that might develop during anesthesia. Appropriate monitoring of critical parameters of cardiocirculatory function will aid in the early detection of significant changes in myocardial performance and tissue perfusion.

Our group has used all agents and techniques, and predicates anesthetic management on the assumption that every elderly patient has an ischemic myocardium. By this approach, the occurrence of problems during anesthesia can be minimized or eliminated. Anesthesia management for the geriatric patient remains a challenge, as it poses numerous complex problems, unlike those of other patients.

REFERENCES


December/1976


AUTHOR

M. Del Portzer, CRNA, is engaged in group practice at Saint Mary's Hospital in West Palm Beach, Florida. She holds a BS degree from Western Reserve University, Cleveland, Ohio. She received her anesthesia training from Cincinnati General Hospital School of Anesthesia.

This paper was presented at the American Association of Nurse Anesthetists 43rd Annual Meeting, Clinical Session, and Postgraduate Course held in San Francisco during August, 1976.

ACKNOWLEDGEMENT

The author wishes to thank John D. Rodgers, MD, and Margaret Villi, CRNA, for their suggestions in the preparation of this paper; and Darline M. Joannides from Medical Records for the statistical data.