FAILURE OF THE PERIPHERAL VASCULAR CIRCULATION

KENNETH SUCHOKA, M.D., AND DORIS C. GROSSHEUTZ, M.D.

Cardiovascular collapse in the postoperative period requires immediate diagnosis and correction of the causative factors to minimize mortality and morbidity. The multiplicity of factors involved may lead to confusion and inept treatment if knowledge of the preoperative condition and operative process is not considered.

Although complete division of central and peripheral causes of circulatory collapse is impossible, this article will be limited for the most part to the peripheral vascular system. A provisional classification of causes of postoperative hypotension is offered.

VENTILATORY FACTORS IN POSTOPERATIVE CIRCULATORY COLLAPSE

Hypoxia and Hyperoxia. As will be shown later, hypoxia exerts an indirect but powerful influence on the peripheral circulation. Its direct influence is mainly on the capillaries causing increased permeability, dilation, and anoy the smooth muscle components with the final result being irreversible circulatory collapse. This has been discussed and documented by many investigators. The importance of prevention and treatment of circulatory failure due to hypoxia during the anesthetic and postanesthetic periods is obvious.

A discussion of the use of oxygen in the treatment of circulatory collapse would not be complete without mention of the fears of hyperoxia. As shown by Lambertsen, there is little cause to fear any outward effects of oxygen given by ordinary means. One investigator, however, decries the use of oxygen in hemorrhagic circulatory collapse because he believes that the increased cardiac output as a result of better oxygenation will increase the bleeding.

Hypercarbia and Hypocarbia. As early as 1906, Couch suggested that some of the complications during and following "etherization" were due to carbon dioxide retention. In 1947, Dripps and Comroe demonstrated a drop in blood pressure following termination of a period of inhaling air with 10 per cent carbon dioxide. This effect was investigated further by Price, who concluded that the postoperative hypotension that occurs after a patient has been hypercarbic during anesthesia is due to the termination of intense sympathoadrenal discharge. The increased sympathetic tone is attributed to an increased carbon dioxide tension causing a rise in circulating catechol amines with attendant vasoconstriction and increased cardiac output. Opposing this effect is the action of carbon dioxide favoring capillary dilatation, peripheral pooling of blood with decreased venous return, and reduced cardiac output probably as a result of acidosis as is discussed later.

When the hypercarbia is relieved, the sympatho-adrenal stimulation is removed immediately; whereas, the factors that tend to cause circulatory collapse as outlined above are allowed to predominate with the resultant postanesthetic hypotension. The treatment for this condition is, obviously, prevention of hypercarbia during the anesthetic period. However, Price suggests that if hypercarbia was present during anesthesia, the abrupt hypotension that follows might be avoided by gradually "washing out" the high carbon dioxide concentrations and thus avoiding sudden loss of sympathetic activity.

Hypocarbia has never been implicated in postoperative hypotension, although decreases in cerebral circulation and cerebral oxygenation, and increases in the circulation of extremities have been noted.

CHANGES IN THE VOLUME AND COMPOSITION OF BLOOD WHICH INFLUENCE PERIPHERAL CIRCULATION

Blood Loss. The most frequent cause of hypotension in the postoperative period is inadequate circulating blood volume. After
extensive surgical procedures, the judgment of the surgeon and anesthesiologist is often not accurate as concerns blood loss, and the amount lost is usually greater than estimated. The importance of blood replacement during surgery cannot be overemphasized, especially in the seriously ill or in the pediatric patient. The chronically ill or debilitated patient has a decreased blood volume and is already in a state of vasoconstriction. Loss of even a small amount of blood can thus lead to circulatory collapse, since this type of patient is unable to further decrease the size of his vascular bed.

The gravimetric method of determining blood loss during surgery gives only a rough estimation of the amount of blood needed for replacement. Twenty-five per cent more should be added to cover that lost into tissues and in the vessels proximal to ligation, as well as that lost into the specimen which was removed. The time-honored hematocrit and hemoglobin determinations are of little value in determining blood volume unless serial recordings are made. These may be normal after acute hemorrhage, since hemodilution usually takes place only after several hours. In the well-hydrated patient, a compensatory increase in plasma occurs within 24 hours, leaving a hemoglobin deficit which can be best treated with packed red cells rather than with whole blood.

The only reliable method of determining blood loss is that of directly measuring blood volume before and after surgery. The measurement of plasma volume by Evans blue dye (T1824) or by radioactive iodinated serum albumin is relatively simple, but is believed by some to be inferior to the determination of the red cell volume by radioactive phosphorus or chromium. Determining both cell and plasma volumes is considered far more accurate than the measurement of one volume and calculating the total from the hematocrit value.

Because of the unpredictability of normal blood volume, solitary preoperative measurement of volume is not as valuable as serial determination during and after surgery. This will help to rule out hypotension due to cardiac or peripheral vascular failure. Postoperative hypotension due to blood loss is common after radical procedures and where there may be bleeding into damaged musculature as may occur in extensive orthopedic surgery. Not noticed as often, but nevertheless common, is bleeding after thoracotomies, and totallectomies and loss of plasma as exudate.

Position. Although total blood volume may be normal, effective circulating volume may be decreased by injudicious movement of the patient postoperatively. The adverse effect of gravity on blood flow to and from the heart can be exaggerated by any mechanism which paralyzes the vasomotor and locomotor system. Postoperatively, residual anesthesia may continue to depress autonomic function and compensatory reflexes to the point where movement may precipitate hypotension. This may also occur if large doses of narcotic or antihypertensive drugs have been given preoperatively, or if surgical or chemical sympathectomy, or spinal anesthesia, has been performed before surgery.

Following perineal procedures, such as transurethral resections and vaginal operations, changes from lithotomy to supine position have been reported to precipitate hypotension of a much as 60 mm. of mercury in 50 per cent of the patients. Blood may be pooled in the legs when they are lowered from the lithotomy position, and more may be removed from effective circulation because of increased hydrostatic pressure on the veins and loss of plasma through capillary walls. This is especially true after long procedures when hypoxic changes in the leg vessels predispose to vasodilatation and increased permeability.

This type of hypotension does not correlate with different anesthetic agents or age groups. It can be prevented to some extent by pressure bandages on the legs, lowering the legs gradually from the lithotomy position, gentle handling and insuring dissipation of the effects of muscle relaxants and anesthetic agents. Treatment consists of raising the legs to provide an autotransfusion, Trendelenberg’s position, vasoconstrictors or blood transfusions.

Transfusion. Blood transfusion in itself can produce hypotension. Administration of incompatible blood, may result in a hemolytic reaction that can go unrecognized during anesthesia. The usual sensaOn of precordial oppression, fullness of the head, and backache
may be masked by the anesthesia. Hypotension is the most common and frequently the only sign of a transfusion reaction while the patient is anesthetized, making diagnosis difficult if other symptoms of cardiovascular collapse are present. A diagnosis can be made by determining the presence of hemoglobin in venous plasma or in the urine, although urinary excretion of hemoglobin may not always be found early in the reaction. This hypotension may be severe and prolonged and may require administration of norepinephrine or phenylephrine. Blood, fluid, and electrolyte replacement must be made cautiously to prevent overloading the circulation if the kidneys are impaired. Sodium bicarbonate and sodium citrate are of doubtful value.

Overtreatment with blood transfusions, especially in the patient with a lowered red blood cell mass and a normal blood volume, can result in hypervolemia and hypotension. This may be particularly true in the patient with impaired cardiac reserve and is probably due to myocardial overload and increased blood viscosity.

Recently, ACTH and cortisone have been advocated in the treatment of hemolytic transfusion reactions on the basis that these steroids, by blocking antibody formation, might lessen the hemolysis.

Fluid and Electrolyte Imbalance. The most common disturbance of electrolyte imbalance seen postoperatively is acidosis of respiratory origin as a result of carbon dioxide retention. This carbon dioxide effect is in addition to its other effects as discussed earlier.

In the presence of normal kidney function and the absence of prolonged hyperventilation, this acidosis is compensated for rapidly so there may not occur marked or prolonged pH changes. However, a fall in pH, if of any magnitude, causes decreased myocardial contractility and peripheral vasodilatation.

Postoperatively, there may also occur metabolic acidosis, alone or in conjunction with respiratory acidosis. If this is not compensated for by the respiratory system and kidney, peripheral and central vascular collapse may occur. Indeed, Clowes states that probably the most common cause of death after extracorporeal circulation is uncompensated acidosis. Severe hypoxia can invoke marked changes in the electrolyte composition of blood and the effect of prolonged hypoxia is that of severe acidosis which further compromises the peripheral circulation.

The cure for respiratory acidosis is, obviously, more adequate ventilation; but in some cases, the cure is difficult, if not impossible, to apply. The same holds true for the treatment of hypoxia. Some hope for the treatment of respiratory acidosis when increased ventilation is impossible may be found with 2-amino-2-(hydroxymethyl)-1,3-propanediol (THAM).

An uncommon and perhaps an overlooked cause of postoperative hypotension is hypopotremia, the mechanism of which is discussed by Welt in his text on this subject. Rabenko found that postoperative circulatory collapse as a result of sodium loss before, during, and after surgery occurred frequently in the aged. Both he and Bakaleinik believe that postoperative hypotremia is often unrecognized because the serum concentration is kept fairly normal by dilution or concentration of plasma, with, however, detrimental effects on the circulation as manifested by hypotension. Other electrolytes exert their effect only indirectly, such as the effects of hyperkalemia in causing disturbances of conduction and/or ventricular arrest after a period of hypercapnia. That dehydration, during, or after surgery can cause circulatory collapse has been well documented.

The mechanism is based on diminished blood volume, increased blood viscosity, capillary stasis and tissue hypoxia. Similar changes may occur in diabetic coma and acidosis. Treatment of fluid and electrolyte balance has been well defined; but overtreatment can also be deleterious to the circulation.

Trauma. Traumatic shock is a common problem preoperatively that can be controlled before operation is undertaken. Occasionally it is a problem in the postoperative period due to a perforation of a viscus, such as might occur during a transurethral resection and is sometimes seen after extensive operations. Needless to say, blood loss is the major problem, but loss of extracellular and intracellular fluid as well as electrolyte disturbances must be considered. It has been shown that a 30 to 50 per cent blood loss can cause a
reduction of 1% to 2 liters of interstitial fluid. Intravenous salt can thus play an important role in recovery from traumatic shock, although some investigators have objected to its use because of the inability of these patients to excrete sodium in the urine. Hinger's lactate solution is probably preferable to saline in treating this condition.

The increased adrenocortical activity following tissue trauma, which will be discussed later, must be considered if prolonged operations are performed on debilitated patients.

The prolonged vasoconstriction which results as compensation for hemorrhage and fluid loss has been found to be detrimental to the shocked organism and may contribute to the irreversibility of circulatory failure. The use of decongestive agents and ganglionic blocking agents have been reported to be beneficial in the treatment of laboratory animals subjected to trauma, but this has not proven clinically superior to circulatory volume replacement.

EXOGENOUS SUBSTANCES AS FACTORS IN PERIPHERAL CIRCULATORY FAILURE

Bacteremia. Peripheral vascular collapse due to bacteremia should be suspected if hypotension occurs after instrumentation of the genitourinary tract, abdominal or perineal operations in the presence of peritonitis, and after wound dressing of large burned areas. Hypotension due to bacteremia may also occur if contaminated fluids or blood are given intravenously. The onset is abrupt, associated with hyperpyrexia and bradycardia and may be confirmed by positive blood culture. This hypotension is not due to heart failure, but is due to peripheral circulatory failure. Bacteria or bacterial endotoxins paralyze the peripheral vascular bed and alter the elastic properties of the small vessels. This results in greater distention of capillaries either as a result of direct damage or by paralysis of vasoconstrictor nerves. The consequences are peripheral pooling of blood, a decrease in venous return and a fall in cardiac output. There also occurs increased capillary permeability with the loss of plasma into extracellular spaces, decreased total effective blood volume and hemocoagulation.

Some investigators believe that the bacterial action may be responsible for the failure of blood replacement therapy in various types of traumatic shock. Bacteremia can result not only from direct bacterial contamination, but also from invasion across previously intact barriers. In dogs, recovery rates from hemorrhagic shock were reportedly increased by the use of parenteral antibiotics.

The early recognition of bacterial shock and prompt treatment cannot be overemphasized. Although hemocoagulation and decreased blood volume are not prominent; nevertheless, a small blood loss will lower blood pressure and transfusion is often required. Fluids are needed to replace those lost into the tissue through damaged capillary walls. Broad spectrum antibiotics injected intravenously in massive doses should be employed until sensitivity studies indicate the specific antibiotics that will be the most effective. Mortality in one series decreased from 91 to 30 per cent with appropriate antibiotics.

Vasoconstrictor drugs may be needed as adjunctive therapy to maintain blood flow to vital areas, and to preserve urinary function until the infection is controlled by the antibiotics. Ephedrine, phenylephrine, epinephrine, and methoxamine maintain blood pressure, but the increase is not sustained. Norepinephrine is the most effective vasoressor, but its effect is transient. Metaraminol, although less effective than norepinephrine, has a more prolonged action and also shows selective effect on the venous vasculature. This results in dilution of the hepatic veins and constriction of the splanchic bed, which tends to counteract the massive pooling in that area.

Adrenal insufficiency does not seem to play a prominent role in bacteremic shock and cortisone gives no protection against the hypotension resulting from the injection of endotoxin in experimental animals. The use of ACTH, cortisone, and adrenal cortical extract have been of questionable benefit in humans. Corticosteroids can minimize some of the symptoms of septicemia, lower metabolic rate, and aid in conserving energy. Objections to their use are that infection may spread more
readily, and the existing electrolyte and fluid imbalance may be aggravated.\textsuperscript{24}

The advantages of hypothermia in the treatment of bacteremia have recently been reported. The purpose of hypothermia is to reduce metabolism and minimize injury caused by inadequate blood flow until specific treatment becomes effective.\textsuperscript{100, 101}

Drugs. Drugs used in the preoperative period may have prolonged effects and influence the peripheral vascular tone postoperatively. Among them are: the corticosteroid group (which will be discussed later)—the Rauwolfia derivatives—the ataractic drugs—the narcotics—and miscellaneous other drugs—that may influence the cardiovascular system.

Rauwolfia, which alters the sympathetically-parasympathetic balance by suppressing sympathetic activity at the hypothalamic level, can cause postoperative hypotension which is often difficult to treat.\textsuperscript{102, 103, 104} This autonomic imbalance can often be corrected by the use of atropine or oxyphenonium.\textsuperscript{105}

Rauwolfia causes disappearance of all but a minor fraction of catechol amines from the tissues, thus preventing a compensatory rise in these amines during anesthesia and operation.\textsuperscript{106, 107} Ephedrine, methamphetamine, and mephenteramine, which supposedly act by facilitating the release of catechol amines, are ineffective in treating hypotension due to medication with Rauwolfia derivatives.\textsuperscript{108, 109, 110} Norepinephrine, phenylephrine, and methoxamine are indicated in the treatment of this type of hypotension. However, an ounce of prevention by the discontinuance of therapy with Rauwolfia derivatives for a period of one to two weeks prior to operation is probably more desirable.\textsuperscript{111}

Patients who are treated with the ataractic drugs may develop postoperative hypotension.\textsuperscript{112-116} Promethazine, which is considered the most innocuous of these drugs, has, nevertheless, been implicated in postoperative hypotension after sudden changes of position.\textsuperscript{117, 118}

The phenothiazine derivatives suppress sympathetic activity both centrally and peripherally, and the drug of choice in the treatment of hypotension resulting from the use of these ataractic drugs is noradrenaline.\textsuperscript{119, 120}

Preoperative sedation with narcotics can raise the threshold of the respiratory and vaso-
cerebral emboli or surgery affecting the area of the fourth ventricle;121, 125, 127 the persistence of spinal or epidural anesthesia postoperatively which may prevent restoration of normal vascular tone.128 These may be especially operative if postural changes are made that will decrease venous return to the heart.129

Activation of vagal reflexes postoperatively may cause bradycardia and hypotension; for example, with pressure in the carotid sinus areas from hemorrhage or tight surgical dressings.128, 129

Increased intra-abdominal pressure as by replacement of distended loops of bowel as in the repair of hernias may activate a coeliac ganglion reflex which may result in a diminution in pulse pressure with little change in pulse rate.132, 133

Pelviccardiac and rectocardiac reflexes are implicated most frequently in postoperative hypotension involving rectal or pelvic surgery. Pain in the recto-pelvic area or an overstretched bladder can cause hypotension which responds readily to the elimination of the causes of the reflex stimulation. The probably neural pathway is through the afferent recto-pelvic parasympathetic nerves and down the efferent vagus nerves causing vasodilation and bradycardia.134 This pelviccardiac reflex is thought to be the major factor of hypotension seen so often after major vaginal surgery.135 Here again, elimination of the stimulus is desirable, but drugs, such as atropine to treat the bradycardia and sympathimic amines to treat the vasodilation, are helpful.

Pulmonary embolism during and after operation may account for hypotension. Although vagal stimulation and decreased blood flow with a reduction in cardiac output are usually blamed for such hypotension, there also occurs, through pressor receptors in the pulmonary vessels, a reflex peripheral vasodilation.136 In addition to therapy directed at correcting central circulatory failure, vasoconstrictors may be helpful.

Hormones. The literature on the relationship of the adrenal cortical hormones to the maintenance of vascular integrity is voluminous. The contractile responsiveness of vascular muscle to norepinephrine, requires equilibrium between intracellular and extracellular electrolytes which is controlled by desoxy corticosterone or the adrenal mineral corticoids.137-141 Gluco-corticoids sustain vascular tone and reactivity by controlling the carbohydrate metabolism of smooth muscle cells.132 In addition, this steroid is thought to control cell hydration by affecting enzymatic activity necessary for the release of intracellular fluid. Some investigators believe this aspect of cell hydration responsible for the sensitivity of adrenalectomized animals to stress.142 Equally important is the role of the steroids in maintaining normal blood volume by controlling salt and water retention.143 The importance of adrenal cortical steroids during stress and the need for adequate substitution therapy with the use of both glucocorticoids and mineral steroids in surgical patients with Addison's disease or panhypopituitarism is well known.144 It is also recognized that patients undergoing bilateral adrenalectomy or adrenalectomy for Cushing's disease need immediate substitution therapy both intravenously and intramuscularly postoperatively.145 If inadequate dosage is suspected, the immediate response in terms of rise in blood pressure to the intravenous injection of 100-200 mg. of hydrocortisone is an index of dosage.

Patients who have received or are receiving corticotrophic or corticosteroid therapy prior to operation must be adequately treated in the preoperative, peroperative, and postoperative period. This problem is often complicated by the lack of accurate histories describing therapy with specific hormones. Prolonged ACTH therapy interferes with the ability of the antipituititary gland to release corticotrophic hormones needed during stress. Long term corticosteroid therapy also suppresses the normal pituitary-adrenal function and causes atrophy of the adrenal cortex.146-147 Thus, under these conditions, neither gland can respond to the stress of anesthesia and operation and hypotension may develop if adequate therapy is not instituted.148-151 Although there seems to be little correlation between the amount of suppression and the size and duration of dosage, a fair assumption can be made that adreno-cortical depression will follow 30 units ACTH, 75 mg. cortisone or 20 mg. prednisolone if given daily over a period of seven days or more.132 The duration of adreno-cortical suppression after discontinuing
steroid therapy varies from two days to two years depending on the individual case. Prednisolone and prednisone are metabolized more slowly than other steroids and are potent inhibitors of steroid output.

Hypotension due to inadequate stress response is sometimes not seen until the postoperative period because of the masking effect of anesthesia on traumatic stimuli. Often, it is only when the effects of anesthesia have disappeared that hypotension is fully manifested.

Chronically debilitated patients, such as those with long-standing tuberculosis, may develop hypotension when exposed to excessive and prolonged surgical stress because of depletion of adreno-cortico steroids in spite of adequate therapy in other areas. Indeed, there are those who suggest that steroid insufficiency should be immediately suspected if a patient has postoperative hypotension in the absence of other obvious causes. Hypotension or anesthesia per se may suppress adrenal function by interfering with blood flow to and from that organ. The beneficial effects of cortical steroids in hemorrhagic shock have been widely debated. Early failures of steroids in ameliorating shock have been attributed to too little too late. Some investigators believe that if blood is not immediately available, large doses of hydrocortisone given intravenously may be beneficial in treating hemorrhagic shock.

The relationship of thyroid to vascular reactivity has not been clearly defined, although this hormone limits the activity of amine oxidase, which increases the sensitivity of the vascular system to the action of catechol amines. Thus, a decrease in thyroid as might occur in hypothyroidism could result in a decreased sensitivity to endogenous noradrenaline. Postoperatively, when circulatory homeostasis is probably dependent on response to endogenous noradrenaline, a deficiency in thyroid may be responsible for hypotension in the hypothyroid patient. In addition, a prolonged vasodilatory response to histamine has been demonstrated in the athyroid laboratory animal. Treatment in cases such as these is, obviously, the injection of thyroxin.

Panhypopituitarism or hypophysectomy can result in an unstable peripheral vascular system that could account for postoperative hypotension in these patients. The mechanism is probably the lack of ACTH influencing the adrenal cortex and the lack of somatotropic hormones. The treatment consists of adequate hormonal substitution therapy.

Humoral Factors in Circulatory Collapse

Histamine. As early as 1919, Dale showed that histamine, given parenterally to anesthetized or hypovolemic animals, caused peripheral circulatory collapse; the same amount given to unanesthetized normovolemic animals had only a transient effect. He postulated that histamine was released as a result of injury to tissue; and that in addition to histamine, other “cleavage products of protein” might be released that would affect the circulation. Later investigators demonstrated the role of histamine in causing peripheral circulatory collapse from anaphylactic shock and, possibly, from shock due to trauma or stress or endotoxin. Hanna showed that in addition histamine injection resulted in an increase in circulating potassium and had a biphasic effect on cardiac output consisting first of an increase, then a decrease. Walton and associates injected various plasma substitutes into dogs and noted that polycrystaline pyrrolidine caused a rise in circulating histamine followed by hypotension, and that there was a sharp rise in histamine following dextan infusion, although hypotension was not detected. The suggestion was made that histamine released by meperidine was responsible for the hypotension often seen after the use of this drug. This hypothesis was substantiated by experiments in our laboratory. This may be a prominent factor in hypotension following meperidine. Schayer believes that there is a simultaneous rise in catechol amines and in histamine during stress, and that the action of one opposes that of the other with the possible end result of circulatory collapse. On the other hand, Weller states that stress calls forth an increase in histamine and adrenal cortical hormones simultaneously, and that the two are antagonistic to each other in terms of
affecting the peripheral circulation. Whatever the mechanisms that release histamine and whatever the antagonists or synergists that tend to counteract or enhance histamine effects, it seems that histamine plays a major role in the peripheral circulation.

The proper treatment for excessive histamine in the body is prevention, if possible. Although there is evidence that pretreatment by an antihistaminic may modify the release of histamine due to stress, once circulatory collapse due to the action of histamine has occurred, further treatment with antihistamines is useless. Vasopressors, especially noradrenaline, will restore peripheral circulatory tone in the histamine-poisoned circulatory system.158

**Polypeptides.** Recently, a great deal of interest has been aroused by the action of certain polypeptides on the peripheral circulation. Injection of these substances in experimental animals causes peripheral circulatory collapse that is not blocked by atropine, but which can be reversed by vasopressors.159 Lewis160 postulated that these vasodilatory substances called plasma kinins were derived from plasma protein acted upon by proteolytic enzymes which appear after trauma from almost any cause.

**DISCUSSION**

Since operation and anesthesia are forms of trauma, if such trauma results in the release of vasodilatory polypeptides, a most attractive explanation for a common final humoral pathway for peripheral vascular collapse appears. With the realization that oversimplification is dangerous, there is, nevertheless, evidence to support the hypothesis that extracorporeal circulation, hypercarbia, acidosis, anoxia, hemorrhage, endotoxins, depressant drugs, surgery, and, indeed, any form of psychic or physical trauma causes a rise in blood levels of proteolytic enzymes such as fibrinolysins.156, 281, 152 These enzymes act on proteins to form polypeptides, or plasma kinins, which cause vasodilatation and circulatory collapse.160 Referring to the action of histamine in causing vasodilatation, Ungar114 suggests that perhaps histamine is in itself a precursor or triggering agent in the formation of plasma kinins.

Having postulated that plasma kinins might be the common cause of circulatory collapse, one is tempted to conjecture further as to the presence of a common anatomic lesion as the final effect of the above-mentioned humoral agents. Chambers,156 Zweifach155 and Irwin and associates160 demonstrated in the tissues of living animals the nature of capillary circulation consisting of A-V shunts with smooth muscled arterioles opening into venules, muscle-free A-V capillaries arising from small arterioles and true capillaries equipped with muscled pre-capillary sphincters. These investigators showed that nerve stimulation or the injection of epinephrine results in the closure of the pre-capillary sphincters and the muscular portion of the arterioles. They were also able to show that histamine and local tissue changes with the release of polypeptides resulted in relaxation of the sphincters and the arterioles with subsequent pooling and stagnation of blood in the capillaries. In addition, Chambers157 withdrew blood from rats subjected to hemorrhage, tourniquet shock, and other forms of trauma and injected it into another rat. He, then, observed relaxation of the sphincter and other smooth muscle components of the capillary system. Price114 and Chambers144 noted that deep anesthesia also caused relaxation of the smooth muscle components of the capillary system.

Based partially on fact and partially on conjecture, one is then tempted to name the action of plasma kinin as the final cause and paralysis of smooth muscle in the capillary system as the final effect. We believe that further investigation will, indeed, dispose logically simple mechanisms that will account for peripheral vascular collapse, even if the factors that trigger these mechanisms are complex.

It is obviously impossible to examine closely all the various factors involved in postoperative peripheral vascular collapse. We have attempted to briefly outline some common causes of peripheral circulatory failure and the contemporary treatment of the effects of these causes.

In addition, some "armchair reasoning" was done in the hopes that the ideas presented might serve as a stimulus for further thought and investigation.
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ASTHMA In the management of the patient with chronic bronchial asthma, it is important to estimate the amount of permanent damage to the lung tissue caused by fibrosis and loss of elasticity. In testing pulmonary function, it has been found most useful to use the mean expiratory flow rate determinations instead of the one-, two- or three-second flow rates, or the maximum midexpiratory flow rate. Evidence has been accumulated to show the presence of some pulmonary dysfunction in the majority of apparently symptom-free asthmatic patients carrying on normal physical activities, even at those times when no signs of pulmonary disease are apparent on routine physical examination. (Chapin, H. B., and Loeb, L.: Pulmonary Ventilation Function in Patients with Asthma, Canad. Med. A. J. 84: 641 (Mar. 23) 1961.)