THE CIRCULATION IN ANAESTHESIA*

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ALL-INCLUSIVE REVIEWS of the effects of anaesthesia on the circulation are available^{1 2} This presentation focuses on clinically observed effects of anaesthesia and their significance and management

BASIC CONSIDERATIONS

The circulation of blood, and consequently the viability of body tissue, is controlled by three major factors myocardial action or cardiac output, peripheral vascular resistance, and blood volume (Fig 1) Circulatory failure occurs when one or more of these factors is altered without sufficient compensation by the others For instance, if peripheral resistance over a large part of the body is lowered by a high level of spinal anaesthesia, the circulation may fail

FIGURE 1 Components and measurements of the circulation

The parameters by which we monitor the circulation are arterial blood pressure, pulse rate and volume, capillary refill time, and appearance of the skin, pupils, and operative site Another variable that is simple to measure is venous pressure, which reflects the integrated function of the three factors that control the circulation Monitoring venous pressure can give valuable clues as to the causes of a circulatory problem and to its intelligent treatment

GENERAL EFFECTS OF ANAESTHETIC AGENTS ON THE CIRCULATION

The effect of an anaesthetic agent on the myocardium per se is of secondary interest to the effect on the whole circulation More important yet is the net

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effect of an anaesthetic agent on the entire cardiovascular system and the response of the body's compensatory mechanisms. Factors influencing the circulatory effects of an agent are rate of administration of the agent, depth and duration of anaesthesia, ventilation, and preoperative pathophysiologic state of the heart and circulatory system.

Agents that produce general anaesthesia decrease myocardial contractility in direct relationship to the concentration of the agent in the arterial blood (Fig 2). Reduced cardiac output, with or without change in heart rate, ensues. The exception to this is nitrous oxide, which has no demonstrable effect on the heart when oxygenation is normal.

FIGURE 2 Effects of anaesthesia

The peripheral vascular bed enlarges with deepening anaesthesia. A larger proportion of the blood volume is accommodated peripherally. Venous return is reduced and cardiac output decreases secondarily. Vessels in the skin, muscle, and subcutaneous tissue beds are dilated primarily at the expense of the renal and splanchnic vascular beds, which are constricted. In addition, after induction of anaesthesia, a large volume of blood migrates from the pulmonary vascular "reservoir" to the peripheral vessels.³

The basic pattern of myocardial depression and peripheral vascular dilatation is modified in some instances. In light planes of anaesthesia, autonomic reflexes and release of hormones (epinephrine, 17-hydroxycorticosteroids) may partially compensate for the detrimental effects of the agents on the circulation.

EFFECTS OF INDIVIDUAL AGENTS

Diethyl Ether

Investigators disagree on the effects of ether on the circulation. Jones and associates⁴ found that, without premedication, in surgical levels of anaesthesia, heart rate and cardiac output increased, arterial pressure and peripheral resistance decreased, and venous pressure changes were variable. Kubota and co-workers⁵ found that, after light premedication and surgical anaesthesia, there were no "statistically important" changes in cardiac output, peripheral resistance, arterial pressure, or heart rate, but there was a definite increase in venous pressure. Other reports⁶⁻⁸ also show lack of agreement, probably because of slight differences in the conditions of study.

Cardiac output		
Peripheral resistance		No change
Blood volume		Falls

Blood pressure	Unchanged
Venous pressure	Rises
Pulse rate	Rises

FIGURE 3 Effects of ether

It appears likely that ether does depress myocardial contractility, as evidenced by the increase in venous pressure generally found (Fig 3). As a result of an increase in heart rate and stimulation by endogenous epinephrine and norepinephrine, cardiac output and blood pressure are well maintained until deep anaesthesia is reached. The peripheral vascular resistance is lowered.

Ether is still one of the safest agents because of its small detrimental effect on the circulation, its relative slowness in causing changes in levels of anaesthesia, and its stimulation of spontaneous respiration.

Cyclopropane

Jones and associates⁹ found that cardiac output, arterial and venous pressures, and peripheral resistance all were increased during anaesthesia with cyclopropane. In their study, the patients were not premedicated and were kept lightly anaesthetized for 2 hours with steady alveolar concentrations of cyclopropane. Respiration was spontaneous but with normal arterial carbon dioxide tensions. In deeper anaesthesia, with end-expired concentrations of cyclopropane above 20 volumes per cent, cardiac output decreased in direct relationship to the increase in concentration of anaesthetic agent. These workers also found, as did Li and Etsten,¹⁰ that, after preoperative administration of morphine, cardiac output and heart rate decreased even during light anaesthesia. Arterial and venous pressures and peripheral resistance still were increased (Fig 4).

Cardiac output	Falls
Peripheral resistance	Rises
Blood volume	Falls

Blood pressure	Rises
Venous pressure	Rises
Pulse rate	Falls

FIGURE 4 Effects of cyclopropane

The increases in blood pressure and peripheral tone are likely due to sympathetic stimulation. There also must be a parasympathetic response to give a normal, or slower, heart rate. The maintenance of good haemodynamics in light anaesthesia makes cyclopropane a popular choice for the poor-risk patient.

Arrhythmias are common with cyclopropane, particularly with high concentrations of it and increased tensions of carbon dioxide.¹¹ It must be concluded that cyclopropane has some specific effect on the myocardium, which causes displacement of the pacemaker, interference in the bundle of His, or increased local excitability.

Cyclopropane shock is probably related to carbon dioxide retention.¹² Both cyclopropane and carbon dioxide are stimulants of the sympathetic system. It is reasonable that, after eliminating either of them, hypotension occurs from cessation of the high sympathetic activity.

Halothane

Deutsch and associates¹³ report that, in normal persons without premedication, cardiac output, peripheral resistance, and arterial pressure were significantly reduced after 30 to 60 minutes of exposure to 1.5 per cent halothane (Fig 5).

Cardiac output	Falls
Peripheral resistance	Falls
Blood volume	Rises
Blood pressure	Falls
Venous pressure	Rises
Pulse rate	Falls

FIGURE 5 Effects of halothane

After about 2 hours, the cardiac output returned to normal with an increase in heart rate. The increase in blood volume found during halothane anaesthesia¹⁴ would aid this process.

Many investigators have found, as did Wenthe and co-workers,¹⁵ that halothane causes reduced cardiac output, by myocardial depression, with increased venous pressure. The decreased blood pressure is partially due to lowered peripheral resistance from arteriolar dilatation. Halothane elicits little sympathetic response but the bradycardia denotes parasympathetic excitation.

The proponents of halothane contend that, because of the peripheral vascular dilatation, body tissues are adequately perfused in spite of the decreased blood pressure. There is no question but that halothane can be used safely in low concentrations even in poor-risk cardiac patients.^{16,17}

Barbiturates

Little change in circulatory dynamics occurs when the intravenous administration of barbiturates is used prudently in combination with nitrous oxide and

Cardiac output		Falls
Peripheral resistance		Falls
Blood volume		No change

Blood pressure	Falls
Venous pressure	Rises
Pulse rate	Falls

FIGURE 6 Effects of thiopental

relaxants. However, rapid induction with a large dose of thiopental sodium causes a severe depression in cardiac output and arterial pressure¹⁸ (Fig 6). This may be due in part to a direct effect on the heart, but peripheral pooling of blood because of venous dilatation,¹⁹ with lessened venous return, is largely responsible.

The patient with abnormal or unstable circulation, as in hypertension or after gross haemorrhage, reacts poorly to barbiturates. Doses must be reduced and given less often.

Spinal Anaesthesia

The circulatory effects are primarily due to a decrease in peripheral resistance because of preganglionic sympathetic block.²⁰ Arteriolar and venous tones are lessened. The hypotension depends on the height of the block and is not significant until sensory loss is above the costal margin.

Cardiac output		Falls
Peripheral resistance		Falls
Blood volume		No change

Blood pressure	Falls
Venous pressure	Falls
Pulse rate	Falls

FIGURE 7 Effects of spinal anaesthesia

The peripheral pooling of blood with less return to the heart secondarily decreases cardiac output and blood pressure. Venous pressure is low (Fig 7). Elevating the feet or giving a vasoconstrictor, such as mephentermine (Wyamine), will increase venous return, central venous pressure, cardiac output, and arterial pressure.

Bradycardia, often seen with spinal anaesthesia, is attributed to block of the

preganglionic cardiac accelerator fibres in the T1 to T4 region. It also seems related to the severity of the hypotension.

Methoxyflurane

At light levels of anaesthesia, methoxyflurane (Penthrane) has been found to exert effects similar to those of halothane.²¹ Values for cardiac index, arterial pressure, stroke volume, and peripheral vascular resistance all were lower than control values. Heart rate increased slightly. The hypotension was concluded to be primarily due to the decrease in cardiac output, which probably was due to decreased myocardial contractility.

Halopropane

This agent, also a fluorinated compound, has been reported to decrease arterial pressure and peripheral resistance.²² Pulse rate and venous pressure were increased while cardiac output was unchanged.

OTHER FACTORS AFFECTING THE CIRCULATION

State of Ventilation

All agents commonly used for general anaesthesia, with the exception of nitrous oxide and ether, depress the respiratory centre. Hypoventilation is likely with spontaneous respiration. Tachycardia and augmented cardiac output and arterial pressure result from mild hypoxia and respiratory acidosis. When these conditions are severe, the heart becomes ineffective because of hypoxia and its rate slows. Cardiac output and arterial pressure decrease, venous pressure increases, and the circulation soon fails completely.

With controlled respiration, hyperventilation is usual. Oxygenation is adequate but the positive-pressure breathing has a deleterious effect on cardiac output.²³ The higher mean pressure in the airway inhibits venous blood flow into the thorax and through the lungs. Less blood reaches the left ventricle and its output falls.

Relaxants

No direct effect on the heart has been shown.²⁴ Gallamine (Flaxedil) does increase heart rate and repeated doses of succinylcholine can decrease it severely, especially in children. Extreme alteration of rate in either direction decreases cardiac output and blood pressure.

In addition, loss of external tone by vessels because of muscle paralysis allows them to dilate and causes more peripheral pooling. Tubocurarine may produce ganglionic blockade with vasodilation if a large dose is given rapidly.

Acidosis

A mild degree of metabolic acidosis, with a decrease of the standard bicarbonate and buffer base, occurs during general anaesthesia and surgery.²⁵ If pre-existing acidosis, as in diabetes mellitus, has been present, or if tissue perfusion is inadequate, as in poor circulation during operation, moderate to severe

metabolic acidosis will occur. The subnormal blood pH depresses the myocardium and cardiac output diminishes. Venous pressure is increased and arterial pressure is decreased.

DETERMINING THE CAUSES OF INADEQUATE CIRCULATION

When the available data, primarily arterial pressure values, indicate that the cardiovascular status is unsatisfactory, successful restoration of circulation can be accomplished rationally if one first determines the likely causes. If the venous pressure also is low or is becoming low, the cause is not primarily myocardial. The problem is either a low circulating blood volume in relation to the capacity of the cardiovascular system or peripheral vasodilatation. The blood volume can be increased safely by giving blood or plasma-volume expanders. This will increase cardiac output and arterial pressure in accordance with Starling's law. Stroke volume is increased when the atrial pressures or end-diastolic ventricular filling pressures are increased up to a central venous pressure of about 15 mm of mercury. Further increases in atrial pressure will cause the heart to fail.

Venous pressure is a helpful indicator in the situation of rapid blood loss and replacement. When hypotension persists, a high venous pressure indicates that the problem has shifted from one of insufficient blood volume to one of myocardial inefficiency resulting from administration of hypothermic, acidotic ACD blood containing more than normal amounts of potassium.

Administration of a vasoconstricting drug is indicated when the cause of inadequate circulation appears to be dilatation of the vascular bed. This type of cardiovascular problem may occur with high spinal anaesthesia or overdosage with ganglion-blocking agents such as trimethaphan (Arfonad).

The cause is primarily poor myocardial contractility if the venous pressure is more than 15 mm of mercury or is increasing rapidly. The heart is having difficulty pumping the volume of blood being returned to it, regardless of the blood volume at that time. In this case, several factors must be investigated. Excessive concentration of anaesthetic agent is the first consideration. The possibility of myocardial failure because of previous disease or of hypoxia from any cause also must be considered; the alveolar and arterial tensions of oxygen should be increased. Disorders of cardiac rate and rhythm may be present. Atropine will abolish sinus bradycardia. Isoproterenol will increase the ventricular rate in complete heart block. Intravenous administration of lanatoside C (Cedilanid) or digoxin (Lanoxin) is indicated for a circulation that is failing because of atrial fibrillation or flutter and for myocardial failure with tachycardia. Administration of sodium bicarbonate will help if significant metabolic acidosis is thought likely to be present.

Another cause of high venous pressure is overtransfusion of blood or fluids. Even during operation, phlebotomy or limb tourniquets are possible and effective. Vasopressors, given in the presence of high venous pressure, theoretically will force more blood centrally and will further embarrass heart action.

Knowing the venous pressure is valuable in that it allows one to differentiate between a problem necessitating treatment of a poorly functioning heart and a

problem in which the blood volume can be safely increased to improve the circulation. It allows one to decide which of the three components that control the circulation is the major factor at fault.

Simple Means of Measuring Venous Pressure

A system similar to that for measuring the pressure of cerebrospinal fluid functions satisfactorily. A glass or disposable plastic manometer and a three-way stopcock are fixed to the intravenous stand. The three outlets of the stopcock are connected (1) to a vein, (2) to the manometer, and (3) to an intravenous set so that the vein can be connected either to the manometer or to the intravenous set by turning the stopcock. Another method is to use an intravenous set with a built-on sidearm tubing which is used as a manometer. Zero on the manometer must be placed at mid-chest level at all times to obtain a meaningful reading. Fluctuation of the fluid in the manometer with respiration indicates a clear system.

It is preferable to measure central venous pressure by a small catheter advanced into the chest. A needle in the external jugular vein, however, will transmit accurately a pressure close to that in the right atrium.

SUMMARY

The integrity of the circulation depends on the summation of the effects of the cardiac output, the peripheral vascular resistance, and the blood volume. Agents producing general anaesthesia affect the circulation by decreasing cardiac output and peripheral resistance, this effect is in proportion to the concentration of agents in the blood. Other factors which may alter the circulation in anaesthesia are the status of ventilation, the degree of hypoxia, the administration of relaxants, and the occurrence of metabolic acidosis. Along with the usual clinical means of evaluating the circulation, venous pressure values are helpful data. These values permit differentiation between a problem in which myocardial function needs improvement and one in which blood volume can be safely increased to treat an inadequate circulation.

RÉSUMÉ

L'intégrité de la circulation dépend de la somme des effets du débit cardiaque, de la résistance périphérique et du volume sanguin. Les substances qui produisent l'anesthésie générale affectent la circulation en diminuant le débit cardiaque et la résistance périphérique, ces effets sont directement proportionnels à la concentration de ces substances dans le sang. Les autres facteurs qui peuvent modifier la circulation, au cours de l'anesthésie, sont le volume de la ventilation, le degré d'hypoxie, l'administration de myorésolutifs et l'apparition d'acidose métabolique. En plus des signes cliniques usuels sur lesquels nous nous basons pour évaluer la circulation, la pression veineuse peut fournir de précieux renseignements. Ses données permettent de faire le diagnostic entre une situation où le myocarde a besoin de support et une situation où le volume sanguin peut être augmenté en toute sécurité pour corriger une circulation inadéquate.

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