Coronary artery disease: Anesthetic considerations

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The author reviews coronary circulation and coronary artery disease, and discusses the effects of inhalation and intravenous anesthetic agents and muscle relaxants on coronary blood flow. Also discussed are the effects of hypotension and hypertension on the diseased coronaries as well as anesthetic techniques and drug treatments for coronary circulatory problems during general anesthesia.

In discussing the effects of various anesthetic agents and techniques on patients with coronary artery disease, this article will focus on three main areas: (1) a description of coronary circulation and coronary artery disease, (2) a list of anesthetic agents and techniques related to coronary artery disease, and (3) treatments for coronary circulatory problems during anesthesia.

Coronary circulation

The left and right coronary arteries supply blood and nutriments to the entire heart. These arteries are the first branches of the aorta, originating at small orifices behind the flaps of the aortic valve. Coronary blood returns to the heart through the coronary veins or small thebesian veins in the myocardium. The left coronary artery has a short common stem (0.5-2 cm long), that divides into the anterior descending branch (which supplies left and right ventricles), and the circumflex branch (which supplies the left atrium and left ventricle). The anterior descending branch also subdivides to anastomose with branches from the right coronary artery. These anastomoses are normally small but may enlarge greatly in patients with coronary vessel disease (i.e., atherosclerosis).

The right coronary artery divides into the posterior descending branch (which supplies left and right ventricles) and the marginal branch (which supplies the right atrium and right ventricle). Branches of the right coronary artery also supply the sinoatrial node (SA) and the atrioventricular node (AV) in most human hearts.

Normal resting coronary blood flow is about 225 cc/min, or 4.5% of the total cardiac output. This flow can increase four to five times the normal rate during strenuous exercise. Oxygen consumption is about 12 cc per 100 cc of blood flow. Sixty per cent of the oxygen is extracted from each cc of blood flowing through the coronary circulatory system. This percentage of extraction remains fairly constant. Inflow of blood into the coronary arteries occurs during ventricular diastole.

Heart rate and contractility are controlled by several factors. The cardiac impulses which initiate the rhythmical contractions of the heart arise spontaneously from within the heart tissue itself and can function without extrinsic nervous control. However, the autonomic nervous system does regulate the rate of firing and strength of cardiac contraction through two sets of nerves: the parasympathetic (mainly through the vagus nerve) and the sympathetic.
The fibers of the vagus are found in the SA node, atrial muscle fibers, and the AV node. The neurotransmitter is acetylcholine. The fibers of the sympathetic nerves supply all regions of the atria and ventricles. The neurotransmitter is norepinephrine. Normal heart rate is maintained by a balance between parasympathetic and sympathetic regulation.

Parasympathetic (vagal) stimulation causes decreased SA node firing rate, decreased atrial contractility, and decreased impulse speed through the AV node (which lengthens the P-R interval and can produce heart block in patients with heart disease). Thus, cardiac output is reduced.

Sympathetic stimulation causes increased heart rate, increased contractility of both atria and ventricles, and increased impulse speed through the AV node. This normally increases cardiac output.

The control of the autonomic nervous system on the heart is modulated by the central nervous system (i.e., in pain or fright) or by reflex mechanisms caused by stimulation of pressoreceptors, located in the aortic arch and carotid sinuses. For example, a sudden increase in aortic blood pressure stimulates the pressoreceptors. This stimulates the cardioinhibitory center in the medulla, thus inhibiting the cardiac accelerator center and decreasing the heart rate.

On the other hand, a sudden decrease in aortic blood pressure causes less stimulation of the pressoreceptors; consequently, the cardioinhibitory center is not stimulated and the cardiac accelerator center is not depressed. This causes a reflex increase in heart rate.

These pressoreflexes are the primary control mechanisms of heart rate. Secondary factors include blood oxygen levels, blood carbon dioxide levels, drugs, electrolyte values, and degrees of alkalinity or acidity.

The primary control of coronary blood flow is as follows:

“Blood flow through the coronary system is regulated almost entirely by local blood flow regulation in the heart tissue itself in response to the needs of the cardiac musculature for nutrition. This mechanism works equally well when the nerves to the heart are intact or are removed. Whenever the vigorous of contraction is increased, regardless of cause, the rate of coronary blood flow simultaneously increases, and, conversely, decreased activity is accompanied by decreased coronary flow.”

This intrinsic local control of coronary blood flow is mainly a direct function of the metabolic need for oxygen by the cardiac muscle; as the heart requires more oxygen, the coronary blood flow increases.

A lack of oxygen dilates coronary arterioles. The exact mechanism of this regulation is not fully understood, although oxygen consumption is thought to be proportional to myocardial muscle tension. For example, an increase in arterial blood pressure, which causes an increase in myocardial muscle tension, in turn causes a greater need for oxygen as oxygen consumption increases.

The three factors which determine myocardial oxygen consumption are heart rate, systolic blood pressure, and contractility. Therefore, increased work of the myocardium increases oxygen need. Myocardial oxygen lack may also result from anemia, systemic hypoxemia, or increased blood viscosity.

Secondary control of coronary blood flow is through stimulation of the autonomic nervous system. This regulation is both indirect and direct. Indirectly, sympathetic stimulation increases heart rate and contractility, thereby increasing the metabolic rate and myocardial oxygen consumption. This increased oxygen need sets off the local flow regulatory mechanism previously described. Directly, sympathetic stimulation weakly dilates the coronary arterioles themselves through the action of acetylcholine and norepinephrine, thus further increasing coronary blood flow.

Norepinephrine increases coronary flow by causing coronary dilatation and by increasing blood pressure. Epinephrine has the same effect due to two mechanisms: (1) increased duration of diastole (in increasing heart rate within the physiological range, epinephrine shortens systole more than diastole), and (2) a coronary dilator effect from local metabolites (i.e., adenosine) produced by the relative myocardial hypoxia when strength of contraction is increased. However, epinephrine also produces a mild direct coronary arteriolar vasoconstriction (from the stimulation of coronary vessel alpha-receptors) and increased myocardial work and oxygen consumption.

Other possible direct coronary vessel dilators are adenosine 5'-diphosphate (ADP), adenosine triphosphate (ATP), bradykinin, xanthines, potassium, hydrogen ion, and carbon dioxide. Nitroglycerin and the nitrites dilate coronary vessels, as does a reduction in pH, an increased PaCO₂, or a lactic acidemia. Conversely, angiotensin and vasopressin constrict coronary vessels. Increased vagal tone, which slows the heart rate, can indirectly decrease coronary blood flow.

Coronary blood flow is also regulated mechanically by two factors. The aortic diastolic blood
pressure is the effective filling pressure for the coronary vessels. A decreased diastolic pressure, or a greatly shortened diastole (i.e., during tachycardia), reduces coronary blood flow. Coronary flow is also directly proportional to cardiac output.5

Coronary artery disease (also called arteriosclerotic heart disease) is the leading cause of death in the Western world. It is characterized by irregular intimal thickening in the arteries and deposits of fatty substances (especially cholesterol), causing plaque formation. These developing plaques disturb the arterial endothelium, causing platelets and fibrin to be deposited on the surface of the plaques, and small blood vessels to grow into the fibrous lesions, thereby further increasing their size.

These lesions may suddenly obstruct the arterial lumen by rupture of blood vessels within the plaque. The lesions may enlarge slowly through further depositions of fat and scar tissue and a small clot may form, obstructing the vessel lumen when the intima covering a plaque breaks. The plaque itself may embolize and occlude vessels "downstream." Finally, the atherosclerosis can weaken the coronary arterial wall and predispose it to aneurysm formation.

The atherosclerotic process most frequently affects the left anterior descending branch, the right coronary artery next. The circumflex branch is least affected. Arteries which are obstructed by these lesions may not allow sufficient blood flow during conditions of increased demand (for example, stress, emotion, cold, physical activity, heavy eating).8

Inadequate oxygenation of the heart, with resulting myocardial ischemia, injury, or infarction, can be precipitated by three factors: (1) a decrease in oxygen supply due to an obstructed coronary blood flow; (2) an increase in cardiac work and thus oxygen consumption; or (3) a reduction of arterial oxygen content (i.e., from hypoxia or abnormal ventilation). Furthermore, in patients with coronary artery disease, any mechanism which increases heart rate, systolic blood pressure, or myocardial contractility may cause insufficient myocardial oxygenation.

During episodes of ischemia, coronary blood pH falls, lactate is produced by anaerobic metabolism, potassium efflux from the myocardium occurs, and the electrocardiogram becomes abnormal (i.e., ST segments are depressed).6,9

The effects of anesthesia on heart function

The effects of various anesthetic agents, muscle relaxants, and other chemicals on coronary circulation and heart function are as follows.

Nitrous oxide promotes a stable hemodynamic state. It has no significant effect on heart rate or cardiac output. It does not depress the myocardium, nor does it sensitize it to catecholamines.6 However, nitrous oxide has been shown to depress myocardial function in patients with coronary artery occlusion.10

Halothane is a direct depressant of the myocardium due to its negative inotropic effect. Cardiac output and blood pressure are reduced according to the dosage amount. Furthermore, halothane causes a vagal slowing of the heart rate. These factors could precipitate an ischemic episode in a patient with coronary artery disease.

Halothane also directly affects the pacemaker by displacing it downward or causing a shift to ectopic foci. This predisposes the heart to nodal rhythms, first degree block, or premature ventricular contractions. The myocardium is thus sensitized to catecholamines.5 Nevertheless, recent studies with non-failing canine hearts suggest that by reducing myocardial oxygen demand, halothane decreases the severity of myocardial ischemia, and may be an appropriate anesthetic when coronary blood flow is limited.11

During the administration of enflurane, most patients maintain a stable cardiac rhythm and rate with a minimal incidence of arrhythmias. There is only a slight depression of myocardial contractility, and cardiac output is not significantly affected in eucapnic patients.5 There is an increase in coronary blood flow under enflurane anesthesia, along with decreased coronary resistance.12

Hypercarbia with enflurane can result in increased cardiac output and mean blood pressure. However, arterial hypotension accompanies enflurane anesthesia, especially during induction. This effect could be hazardous in patients with compromised coronary circulation. Furthermore, the myocardium is sensitized to catecholamines by enflurane, but to a lesser degree than by halothane.5

Methoxyflurane decreases blood pressure, cardiac output, and contractility. Heart rate and rhythm are usually stable although a sinus bradycardia (which responds to atropine) can develop in deep anesthesia. The myocardium is sensitized to catecholamines, but to a lesser degree than with either halothane or enflurane. Ventricular arrhythmias can occur with sympathetic stimulation (i.e., from respiratory acidosis, tracheal intubation, and surgical stimulation during light anesthesia).8
Methoxyflurane also causes a consistent negative inotropic effect. If vasopressors are administered concomitantly, myocardial ischemia and insufficiency can result, especially in patients with coronary artery disease.5

The administration of diethyl ether is characterized by relative circulatory stability. The direct myocardial depressant effect of ether is offset by increased sympathetic activity.6 Cardiac output remains unchanged, although heart rate increases (ether is vagolytic). Blood pressure remains normal during ether maintenance if the sympathetic nervous system continues to function (ether causes an increase in blood levels of norepinephrine).

Ether protects the myocardium against the arrhythmogenic effects of catecholamines. Because of its stabilizing effect on the circulatory system, ether is indicated for patients with coronary artery disease.6 However, a turbulent second stage must be avoided.18

Cyclopropane does not significantly reduce cardiac output or arterial blood pressure; they may even be slightly elevated (at anesthetic concentrations of cyclopropane) due to increased total peripheral resistance. Heart rate usually decreases due to the stimulation of aortic and carotid pressure receptors as blood pressure is increased (cyclopropane is sympathomimetic). There is an increase in vagal tone because cyclopropane is vagotonic.

The excitability of heart muscle, however, is increased. This may precipitate arrhythmias, due to vagal stimulation and bradycardia and displacement of the pacemaker to extrafocal sites. Cyclopropane is probably not the agent of choice in patients with coronary vessel disease.

Isoflurane, a fluorinated ether-type anesthetic, at anesthetic concentrations, sustains cardiovascular dynamics as does diethyl ether. Isoflurane neither depresses the myocardium nor sensitizes it to the action of catecholamines.

As long as the PaCO₂ is kept constant, myocardial function is maintained. However, arterial blood pressure may decrease, especially during induction. Also, isoflurane is a potent respiratory depressant, so respirations must be carefully controlled during anesthesia to prevent hypoxia.

Ethylene does not significantly affect blood pressure or cardiac action at anesthetic concentrations, nor does it sensitize the myocardium to catecholamines. Ethylene anesthesia is used alone for superficial operations; combined with other anesthetic agents or relaxants, it can be used for abdominal or chest surgery. It is almost completely free of toxicity and is rapidly eliminated. As such, it may be the agent of choice for elderly, debilitated, or poor-risk patients.

Thiopental has a direct depressant effect on the myocardium, causing decreased cardiac output. Dose-related hypotension follows thiopental injection, especially in patients with hypertension. Thiopental does not sensitize the myocardium to catecholamines, but its depressant effect on the heart limits its use in patients with arteriosclerotic heart disease or coronary insufficiency.

Diazepam, used as a basal hypnotic in anesthesia, causes no significant cardiovascular depression or hypotension, that is, unless other central depressants have been administered previously. Diazepam is also used in cardioversion; thiopental used for the same purpose causes a greater incidence of cardiac irregularities.

Droperidol does not depress the myocardium, and its anti-arrhythmic effect protects the heart tissue against sensitization to catecholamines (due to a mild beta-blocking action). It stabilizes cardiovascular function, although a mild decrease in blood pressure occurs because of droperidol’s alpha-blocking effect.5

Morphine causes a peripheral vasodilatation (probably due to histamine release), reduced afterload, and decreased cardiac output (due to reduced venous return in large doses). Clinical dosages of morphine to produce anesthesia do not cause myocardial depression.14 Administered in large dosages, there is a reduction in ventricular work and decreased myocardial oxygen need.

These effects, coupled with reduced afterload, are of significant benefit to patients in the acute phase of a myocardial infarction. This decrease in oxygen consumption and cardiac work is also advantageous to patients with coronary artery disease.5 However, hypovolemic patients may develop profound hypotension (with resulting coronary insufficiency) with morphine administration. Usually, if proper ventilation is maintained and hypoxia is avoided, blood pressure does not drop significantly.8

Fentanyl has minimal cardiovascular effects. A mild bradycardia occurs (which is relieved by atropine) because fentanyl is vagotonic. There is no myocardial depression and no histamine release. A slight reduction in systolic blood pressure may result; this effect can be significant in patients with severe hypovolemia. Fentanyl is a potent respiratory depressant, and apnea and rigid chest can occur after rapid intravenous injection. Therefore, ventilation must be controlled.

The depolarizing muscle relaxant succinylcholine does not affect the myocardium directly. How-
ever, arrhythmias can occur after its administra-
tion, and bradycardia is frequently encountered. 
Ventricular premature contractions and other car-
diac irregularities are found, especially in children, 
patients with burns or nerve damage, patients with 
high serum potassium levels, and in patients being 
intubated while in a light plane of anesthesia. 

Consequently, patients with coronary artery 
disease should be in a sufficiently deep plane of 
anesthesia before succinylcholine is administered 
and endotracheal intubation is attempted. 

The nondepolarizer curare usually causes a 
dosage-related drop in blood pressure in anes-
ethetized patients as a result of three effects: (1) 
sympathetic ganglia blockage, (2) diminished 
venous return due to muscular relaxation, and (3) 
histamine release from body tissues. Thus, hypo-
volemic patients may develop serious hypotension 
after the administration of curare; in patients with 
compromised coronary circulation, myocardial 
ischemia could result.

Pancuronium causes a slight stimulation of 
the cardiovascular system, usually resulting in a 
10% increase in heart rate and blood pressure. 
The drug is thought to be a direct cardiac chron-
otropic and inotropic stimulant, and it is also vago-
lytic. Pancuronium is often used with patients with 
heart disease and patients with hypovolemic.$

Other factors affecting heart function

The net cardiac effect of increased arterial car-
bon dioxide tension in an awake patient is in-
creased cardiac output.$ This effect is diminished 
in the anesthetized patient. Furthermore, hyper-
carbia increases sympathetic activity and raises 
plasma catecholamine levels. Increased arterial 
carbon dioxide tension also enhances cardiac re-
sponses to vagal stimulation. As mentioned previ-
ously, a $PCO_2$ elevation causes an increase in 
coronary blood flow.

Catecholamines (norepinephrine, epine-
phrine, isoproterenol) generally raise blood pres-
sure and also affect coronary blood flow.$ Norepine-
phrine and epinephrine are synthesized and 
released in increasing amounts during situations 
of acute stress.$ The actions of norepinephrine 
and epinephrine have been discussed earlier in 
this article. Epinephrine also causes a marked in-
crease in myocardial oxygen consumption.

Isoproterenol is a beta-adrenergic stimulant 
which causes an increase in myocardial contractil-
ity and rate while lowering peripheral vascular re-
sistance.$ It also causes increased coronary blood 
flow parallel to an increase in heart rate.$

Preoperative medications are important in the 
patient with coronary artery disease, principally 
to obtund nervous system activity (i.e., to de-
crease vagal tone and decrease catecholamine 
release).$ Atropine should be administered with 
cautions in patients with compromised coronary 
circulation. If the myocardium is underperfusion 
because of hypotension (due to bradycardia), then 
atropine may be indicated.

However, if the heart rate is increased by 
atropine without a concomitant increase in coro-
nary perfusion pressure, then ischemia will result.$ 
Scopolamine preoperatively may be indicated in 
this situation, because it is an effective vagolytic 
agent and does not cause as much of an increase in 
heart rate.$

Anesthetic techniques

"Anesthesia in patients with coronary ather-
sclerotic heart disease must interfere as little as 
possible with myocardial metabolism in order to 
reduce the incidence of perioperative myocardial 
infarction. The delicate balance between the myo-
cardial oxygen supply and myocardial oxygen de-
mand must be preserved during anesthesia and 
surgery."

Generally, patients with coronary heart dis-
ease will tolerate anesthesia well if overdosage, 
hypoxia, and hypotension are avoided. Further-
more, adequate myocardial perfusion must be 
achieved by maintaining a normal diastolic blood 
pressure since coronary flow occurs during diastole. 
A stormy second stage should be avoided, and 
ventilation must always be adequate. Fluid over-
load must be avoided, as increased congestion 
could develop into heart failure.$ Hypertension 
and tachycardia, accompanied by increased heart 
work and myocardial oxygen consumption, should 
also be prevented.$

Generally, the patient's rate-pressure product 
should be maintained under 12,000-15,000 to avoid 
ischemic changes.$ Patients with coronary artery 
disease must be kept warm, because exposure to 
cold can cause an increase in arterial blood pres-
sure and peripheral vascular resistance, thereby in-
creasing cardiac work and precipitating and ische-
mic episode.$

In general, deep anesthesia is preferred for 
patients with coronary artery disease because of 
better control of pain, apprehension, and spasm 
in the immediate postoperative period when most 
arrhythmias and infarcts are likely to occur.$ An 
adequately deep plane of anesthesia is generally 
associated with decreased myocardial work and 
oxgen need, while light anesthesia, accompanied 
by the stimulation of endotracheal intubation and
surgery, can result in dangerous catecholamine release.

Conclusion

The basic guidelines previously described can help prevent coronary circulatory problems. An acute episode of coronary ischemia during anesthesia can usually be recognized by changes on the electrocardiogram monitor.

Patients with chronic heart disease who are also receiving digitalis may show a pattern with reduced T-wave amplitude and ST segment depression. Digitalization of a patient with heart disease can produce inverted T-waves and marked ST depression.

An acute ischemic episode may manifest itself by flattened or inverted T-waves and depressed ST segments. If the T-waves are already inverted, they may become upright. Lead V₅ is more useful than lead II in determining ischemic ST segment changes. Myocardial ischemia also predisposes the heart to arrhythmias and extrasystoles.

Drug therapy, as treatment for coronary ischemia in the anesthetized patient, is designed to maintain an adequate coronary perfusion pressure while not significantly increasing myocardial oxygen demand. This is achieved through control of hypotension or hypertension. The pressor drugs epinephrine and isoproterenol are available, and have already been discussed.

Excessive hypotension during anesthesia can also be treated by decreasing the depth of anesthesia, increasing the rate of intravenous fluid administration, or repositioning the patient. Tachyarrhythmias can be corrected by the administration of propranolol. Controlled hypotension, with reduction in cardiac work and improved coronary circulation, can be obtained by the judicious administration of a nitroprusside or nitroglycerin infusion.

A hypertensive episode during anesthesia can usually be recognized by changes on the electrocardiogram monitor.

REFERENCES


AUTHOR

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