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CONTENTS

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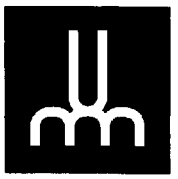

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The CME section provides concise summaries, in a variety of formats, of clinically relevant information intended for all anaesthetic practitioners. Each Journal issue contains a major CME segment, provided, in rotation, by participating Canadian University Departments of Anaesthesia.

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Anaesthesia and myocardial infarction

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Epidemiological studies of the American adult population show an annual incidence of new major coronary events (myocardial infarction (MI), sudden death) of 6.7 per cent.[1] Assuming an even distribution throughout the year, this represents a rate of 0.129 per cent in a period of seven consecutive days. Put another way, 129 major coronary events can be expected per 100,000 adults each week.

Considering that about 1,500,000 anaesthetics are given to adults each year in Canada, and by extrapolating from known US data for patients with and without previous MI,[2,3] one could expect an annual incidence of 3,000 myocardial infarctions (MI) to occur in the seven-day period starting from the day of operation. Forty per cent of these MIs would occur in patients with a MI-free history. Half of all the affected patients will be expected to die within 48 hours. Perianaesthetic MIs may occur in completely asymptomatic patients not previously known to have coronary artery disease (CAD).

Indeed, Tarhan *et al.*[1] found a 0.13 per cent incidence of perioperative (up to seven days postoperatively) MIs in 32,455 patients without a history of previous MI. Thus it would appear from these statistics that anaesthesia and surgery as such do not increase the risk of major coronary events in this population.

However, Tarhan's study also revealed that in patients with a history of previous MI the incidence of perioperative events increased 50-fold on the average (range 30- to 300-fold) depending on the time interval between the MI and the surgery.[4] This incidence is considerably greater than the expected incidence from the natural history of the disease.

These figures make every anaesthetist aware of the devastating effects of perianaesthetic MI for the patient, and of the medico-legal aspects for the physician.

The natural history of myocardial infarction

MIs at large

In 45 per cent of patients with CAD, a MI is the first manifestation of the disease, and for another nine per cent sudden death is the initial event.[5,6] MIs often happen suddenly, without prodromal symptoms or signs. The clinical manifestations are often unrelated to the severity of the coronary lesions. Twenty per cent of all MIs are asymptomatic or silent.

Thirty per cent of the affected patients die before reaching hospital; 15 per cent die in hospital. Fifty-five per cent survive.

Two-thirds of coronary deaths occur suddenly and half of these deaths happen within one hour of the beginning of symptoms.[7] Three-quarters of deaths are due to ventricular fibrillation (VF), with or without evidence of necrosis.[8] Half of the VF episodes occur in patients with good ventricular function and a negative stress test.[9] About 10–25 per cent of deaths are due to myocardial rupture.[10–12]

The mortality among survivors is ten per cent in the first year and three to seven per cent annually thereafter.[13] The main factors determining long-term prognosis include age, left ventricular dysfunction, ventricular arrhythmias, residual ischaemic myocardium, presence and severity of known risk factors, and peripheral atherosclerosis.

Subendocardial MI carries a lower in-hospital mortality rate than transmural MI. However, in

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subendocardial MI the risk of early transmural recurrence is higher and such recurrences are associated with a high mortality rate.[14] Concerning the coronary lesions themselves, left main coronary artery stenosis and triple vessel disease have a serious prognosis, with an annual mortality rate of 12–15 per cent.

Perioperative myocardial infarction

The often quoted 1972 study by Tarhan *et al.*[1] from the Mayo Clinic reported a perioperative MI incidence of 0.13 per cent in patients without a prior history of MI. Among the 422 patients with a previous MI, 6.6 per cent reinfarcted within the first postoperative week. When this last subset of patients was divided according to the age of the infarct, it was found that the reinfarction rate was 37 per cent if the surgery was performed within three months of the infarction; 16 per cent if done within three to six months of the infarction; and five per cent when done more than six months after infarction. The mortality rate of perioperative reinfarction was 54 per cent, 80 per cent of which occurred within 48 hours from the onset of infarction.

As seen in the previous section, the short-term mortality from MIs at large is 45 per cent, most of which occurs before the patients reach hospital; the perioperative MI mortality of 54 per cent is disturbingly high, given that the perioperative MI happens in the hospital.

It is noteworthy that the highest incidence of perioperative infarction occurs on the third postoperative day (33 per cent compared to 18 per cent on the first day).

A similar study by the same group reported in 1978[2] showed identical results, which was surprising considering that anaesthetic techniques and agents had improved and that there was a better understanding of cardiovascular disease and an acute awareness of the dangers.

On the other hand, two more recent studies have shown much lower perioperative MI rates. Wells and Kaplan reported no reinfarction in 48 patients with an infarct of less than three months.[15] However, 15 per cent of these patients had cardiac dysrhythmias. In another 750 patients without a history of recent MI but with clinically significant ischaemic cardiac disease, the perioperative infarction rate was 1.3 per cent. Rao *et al.* reported similar data.[16] In both these studies the patients had a more thorough preoperative evaluation, more complete perioperative monitoring, and more prolonged postoperative ICU care.

The former studies from the Mayo Clinic made us aware of the problems associated with anaesthesia in CAD whereas the latter two studies incite us to conduct a more aggressive perioperative management. From these studies, the following factors are now recognized to be associated with increased risk of perioperative MI:

- History of a preoperative MI within three to six months
- Presence of congestive heart failure or dysrhythmias;
- Type and site of surgery (increased risk with emergency and major thoracic or upper-abdominal surgery);
- Length of the procedure (reinfarction rate eight times higher in procedures lasting five hours or more);
- Large intraoperative variations in blood pressure and/or heart rate.

Pathogenesis of myocardial infarction

The oxygen equilibrium

Myocardial ischaemia results from the disruption of the equilibrium between myocardial oxygen delivery and requirement. When oxygen delivery cannot meet oxygen requirement, angina results along with ventricular dysfunction and electrical instability. Infarction may be the endpoint of this process. Factors determining the oxygen balance are well known and are summarized in Figure 1. Normally oxygen balance is readily maintained because autoregulation of the myocardial blood supply provides ample reserve to cover a wide range of myocardial oxygen demands. However, when oxygen delivery is limited by coronary artery obstruction, oxygen consumption will be equally curtailed. In this case, when oxygen requirements exceed oxygen delivery, an oxygen debt occurs which produces myocardial ischaemia with possible progression to necrosis.

In practice, MIs always result from some degree of coronary obstruction, temporary or permanent, partial or complete. If coronary obstruction is incomplete, infarction can result from increased

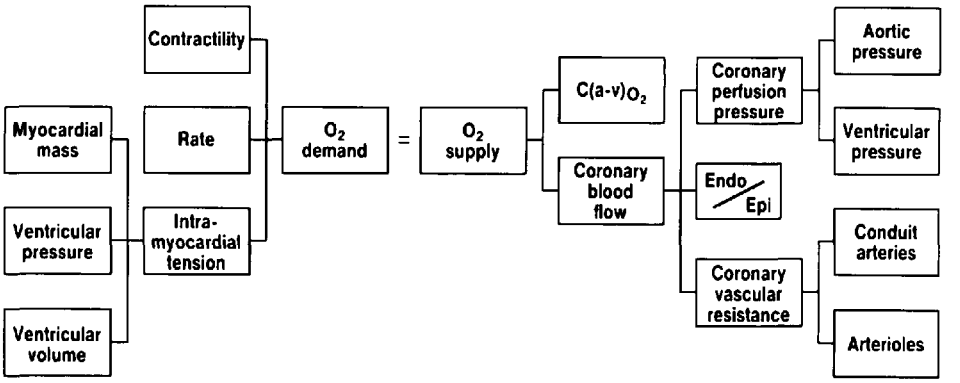

FIGURE 1 Myocardial O_2 supply/demand.

energy demands by the heart. If the obstruction is complete or almost complete, infarction can occur at rest especially in the absence of adequate collateral circulation.

Atherosclerosis

MI usually occurs in a region whose blood supply is restricted by coronary atherosclerosis.[17] Atherosclerosis develops early in life, as was revealed by autopsies performed on American soldiers killed in action in Korea and Vietnam.[18–19] Complete obstruction of one coronary artery was found in five per cent of all soldiers dying in the field in Vietnam. The incidence of coronary atherosclerosis varied from 45 to 70 per cent.

The pathological changes of atherosclerosis are first apparent in the subendothelial area of the arteries and the process silently progresses over many years. By the time the first clinical manifestations appear, the vascular damage is already severe and often multifocal. The affected vessel shows cholesterol-loaded atheromatous plaques that have developed between the intima and the media destroying elastic and muscular elements. In the coronary arteries the endpoint is progressive obstruction accompanied by endothelial lesions, where ulcerated plaques are believed to be the triggering site of thrombus formation.

The thrombotic phenomenon

Recent studies have shown intra-arterial fresh thrombotic plugs at the proximal end of the atheromatous stenosis in the coronary artery irrigating an infarcted area.[20,21] Post-mortem angiography and microscopic pathology done within a few hours of an MI show thrombosis in 90 per cent of the cases.

The thrombotic mechanism is also confirmed by the efficacy of thrombolytic agents in reopening recently obstructed coronary arteries.[22,23]

However, there are more questions than answers about the when, why and how a thrombus is generated.[24]

Spasm

Rare patients with angiographically normal coronaries may develop MI (three per cent of MIs) secondary to arterial spasm. This is known as Prinzmetal's phenomenon.[25–27] However, there is speculation that coronary spasm may be part of the events leading up to MI in a large group of patients with atherosclerosis.[28] In support of this hypothesis, Maseri *et al.*[29] have reported data suggesting that preinfarction angina is associated with vasospasm in the early phase of events leading to infarction.

The following triggering events have been shown to induce coronary spasm in susceptible patients: exercise, the cool pressure test, alkalosis, and drugs such as ergotamine, alpha-adrenergic agonists and calcium chloride.[30,31] Can drugs used during anaesthesia trigger coronary spasm in certain patients? It is also known that coronary spasm can be induced by mechanical stimulation. Can surgical manipulation induce vasospasm?

The precise mechanism of coronary vasospasm is still not completely understood and is probably

multifactorial including vascular, neuro-humoral, and platelet-related factors.[32]

Platelets

The modified or damaged coronary endothelium at the site of atheromatous plaques is the nidus for platelet aggregation, which initiates the cascade of events that may produce MI.[33] Intact arterial endothelium synthesizes and releases prostacyclin which prevents platelet adhesion and subsequent thrombi formation; damaged endothelium is unable to produce this antiplatelet factor. Adherent platelets release ADP, which causes them to aggregate: the thrombotic process is thereby initiated. Also the affected platelets degranulate, secreting coagulation factors and vasoactive substances such as serotonin, ADP and thromboxane A_2.[24] The latter can induce or aggravate vasospasm. Thrombosis can therefore develop at the site of a vasospasm and/or of an atheromatous plaque. The induced arterial vasospasm can lead to intimal rupture adjacent to an atheromatous plaque and induce the cascade of thrombotic events leading to coronary occlusion. In addition, decreased blood flow at the site of atheromatous stenosis may contribute to blood clot formation.

Pathophysiology of myocardial infarction

In the presence of critical coronary obstruction oxygen delivery cannot meet myocardial requirements, the subsequent cellular hypoxia results in anaerobic metabolism, with the depletion of glycogen and production of lactic acid.[12] The myocardial cells lose their contractile properties and regional myocardial dysfunctions appear[24] with paradoxical passive stretching of the ischaemic myocardial segments which cannot sustain the tension generated by the nearby healthy myocardial segments.

Myocardial cells can sustain ischaemic insults of up to 20 minutes from which they may still recover their integrity. However, beyond that limit irreversible cellular damage occurs and increases with duration of ischaemia. After 60 minutes of ischaemia most cells will be irreversibly damaged with cellular oedema and loss of myocardial function. These time limits observed in animal studies are not directly applicable to humans because of collateral circulation and the use of certain drugs. One must also remember that MI is not a homogeneous lesion: there is a central zone of necrotic cells surrounded by ischaemic cells which are potentially salvageable by certain drugs (β-blockers, calcium antagonists) and by reperfusion techniques (thrombolytic therapy, percutaneous transluminal coronary angioplasty (PTCA), etc.).

After one hour of ischaemia, the affected region is infiltrated by leukocytes and tissue oedema sets in; this is the onset of coagulation necrosis. The oedema peaks by the 36th hour and begins to resorb at about the 48th hour. Arrhythmias most frequently occur during this phase of onset and consolidation of infarction. On the eleventh day necrotic cells are replaced by collagen fibres and on the 18th day scarring is well-defined although there persists inflammatory cells. Scarring is complete five to six weeks following infarction.[12,34]

Anaesthesia, surgery and myocardial infarction

Preoperative stress

Before surgery patients cannot avoid some degree of emotional upset.[15] Through the autonomic nervous system, anxiety can manifest itself by a hyperdynamic cardiovascular system – increased aortic pressure, heart rate and myocardial contractility.[35] These events may tip the scale toward myocardial ischaemia. In patients with CAD, this stress-induced catecholamine release may precipitate angina, MI, and dysrhythmias. According to Lown, emotion is a known transitory risk factor which can cause sudden death in the coronary patient. Haft[37] has shown that stress can affect platelet aggregation. Moreover, there is a theoretical possibility that psychological stress and/or adrenergic stimuli could precipitate coronary vasospasm.[35]

Intraoperative myocardial ischaemia

During anaesthesia, haemodynamic instability affecting the myocardial oxygen balance may bring about episodes of myocardial ischaemia. The study by Slogoff and Keats[38] in patients undergoing coronary artery surgery revealed a postoperative infarction rate three times higher in patients who had shown signs of myocardial ischaemia intraoperatively. Also, postoperative infarction was rare in the absence of intraoperative haemodynamic instability. Important haemodynamic disturbances associated with intraoperative myocardial ischaemia are tachycardia, and hypertensive or hypotensive episodes. Among these, tachycardia is the most

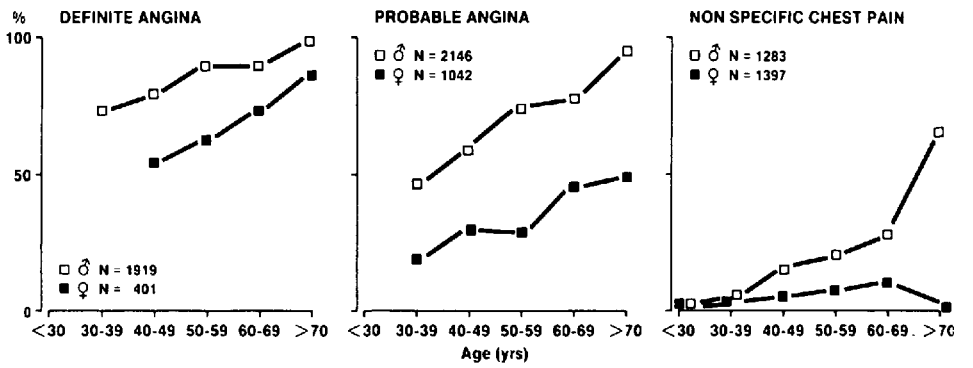

FIGURE 2 Incidence of CAD related to age group for women and men and to presence of anginal symptoms. Unpublished data from the Montreal Heart Institute.

important,[38,39] because of the resulting increased energy requirement and decreased diastolic perfusion time. Prolonged episodes of hypotension may also lead to intraoperative myocardial ischaemia by decreasing oxygen delivery downstream from a coronary stenosis.

Intraoperative ischaemia is suggested by ST-T changes on the ECG and/or increased pulmonary capillary wedge pressure. These events most commonly occur whenever the patient is not sufficiently protected from sympathetic overactivity during stimulation, intubation, manipulation and surgery.

Postoperative MI is therefore closely related to new perianaesthetic ischaemic episodes. In Slogoff and Keat's study, no other preoperative factor was associated more closely with perioperative MI.[38] Their data suggest that the incidence of ischaemic episodes may reflect the ability of the anaesthetist in the management of patients with CAD. On the other hand in the Coronary Artery Surgery Study (CASS),[40] patient age and left main coronary artery lesions were the major predicting factors of perioperative MI.

Preoperative cardiac evaluation

Given the importance of ischaemic cardiac disease on perioperative morbidity and mortality it is essential to search for this disease in all patients presenting for anaesthesia. In 1968 Powers[41] reported that six per cent of the population in the fifth decade had cardiovascular disease; 23 per cent in the sixth decade; 45 per cent in the seventh decade; and 100 per cent of those over the age of 70. Data from our institution shows that the probability of CAD is a function of age, sex and symptoms (Figure 2).

History and physical signs

A history of angina must be sought. Chest pains induced by physical effort, emotion, or meals are highly suggestive of CAD, especially in men. The Table lists the known factors related to CAD. When two or more are present, one must be highly suspicious of the presence of CAD.

During angina-free periods of CAD the physical signs are few and may be limited to a fourth heart sound. During anginal attacks, hypertension and gallop rhythm are frequently observed; signs of left ventricular failure may develop in the more severe forms[42] along with a mitral systolic murmur. Peripheral vascular obstructive disease is often associated with CAD. Finally, certain signs such as arcus senilis, xantholasmus, xanthoma and ear lobe crease are suggestive of atherosclerotic disease.

CAD must be suspected in the presence of hypertensive arterial disease and congestive heart failure.[43] As such, congestive heart failure must be identified as it is known to increase the risk of perioperative MI.

Electrocardiography

A normal ECG does not eliminate severe CAD. However, ECG changes such as signs of left ventricular hypertrophy, bundle branch block, Q-waves and ST-T changes suggest the likelihood of CAD. These signs must not go unnoticed as they

could be the only clue to undiagnosed CAD. Permanent ST segment elevations may indicate ventricular aneurysm causing left ventricular dysfunction.[42] However, it is not possible to determine the age of an MI from an isolated ECG.

Stress test

Suspected but unproven CAD may be revealed as ischaemic changes on continuous ECG recording during exercise (stress test). Although false positive or false negative results may occur, usually a positive stress test represents an index of the severity of ischaemic heart disease. It indicates the limit of physical effort, heart rate, or blood pressure which the patient can tolerate before angina develops. In recent years, radioactive thallium administration during a stress test has helped identify ischaemic areas when clinical and electrical signs were doubtful.

The patient with previous coronary artery surgery

In general, a previous successful coronary artery bypass graft (CABG) reduces the risk of MI[44,45] compared with patients who have not had surgical palliation for their CAD. Cruchley *et al.*[44] reported few ischaemic complications in patients who had previous CABG. However, most venous grafts remain patent only for a limited period of time. Five years after grafting, 20 per cent of venous grafts will be obstructed, most of these obstructions having occurred within the first year post-grafting. After ten years, only 40 per cent of the grafts are still patent of which 70 per cent will have significant stenotic lesions.[47,48] On the contrary, 90 per cent of internal mammary grafts are still patent and free of atherosclerosis ten years after insertion.[49,50] Thus myocardial revascularization surgery does not provide a permanent solution to the problem of myocardial ischaemia and many patients remain at risk, especially when saphenous vein grafts are more than five years old.

TABLE Risk factors for CAD

Non modifiable
Age
Sex
Family history of atherosclerosis
Modifiable
Major
Hyper or dyslipidemia
Hypertension
Smoking
Carbohydrate intolerance
Obesity
Minor
Oral contraceptives
Sedentary living
Personality type
Psycho-social tension

Preoperative preparation

Medical therapy

As a general rule, all medications required by the coronary patient to remain stable must be continued up to the morning of surgery. The danger of sudden withdrawal of beta-blockers in the ischaemic cardiac patient is well documented.[15,51,52] Likewise sudden withdrawal of calcium antagonists, antihypertensive drugs, nitrates, diuretics, and digitalis may cause undesirable consequences if the plasma level of these drugs is below the therapeutic range at the time of anaesthesia. On the other hand it is essential to recognize that beta-blockers may interact with anaesthetic drugs; the myocardial depressing effect of hypercapnia and especially of hypoxia are potentiated by beta-blockers.[42]

Prophylactic digitalization is still controversial. In its favour are the facts that digitalization minimizes the negative inotropic effects of anaesthetic agents[53] and prevents the perioperative occurrence of rapid ventricular response to atrial flutter or fibrillation. On the other hand digitalis-induced arrhythmias may be difficult to treat.[54] Digitalis is classically indicated for congestive heart failure, rapid atrial fibrillation, and for frequent supraventricular premature beats. It is essential that optimal plasma digitalis and potassium levels be maintained since the association of digitalis and hypokalaemia can cause dangerous dysrhythmias.

Anticoagulants are used in patients with ventricular aneurysms, in patients suspected of having intracardiac thrombi and in patients with prosthetic valves. Such therapy may have to be interrupted a few days before surgery, especially in cases where haemostasis may be difficult or the threat of disastrous bleeding is present, such as in vascular or neurosurgery. When necessary, the oral anticoagulant is replaced by intravenous heparin continued up to the time of surgery.

Patient rapport

Because of the possible grave consequences of stress-induced anxiety in coronary patients, it is essential to establish close contact in a climate of confidence. The anaesthetist should reassure the patient about his condition and explain all the precautions that will be taken in the perioperative period to prevent complications. We believe that this psychological preparation is of the utmost importance.

Premedication

There is a general consensus that coronary patients should receive a heavy premedication to help control their anxiety.[52] A good sleep on the night preceding surgery must be assured by proper medication. With the exception of very short procedures, the combination of an anxiolytic and a narcotic drug in sufficient dosage will produce the desired effects.

Anaesthesia

It has been shown that perioperative infarction occurs mainly in patients following a haemodynamically stormy operative period.[38] Intraoperative haemodynamic instability must be detected and treated effectively: this implies the use of adequate monitoring for the early detection and control of haemodynamic abnormalities.

Monitoring

Except for short procedures (diagnostic procedures, minor surgery of less than 30 minutes duration) or for patients with minimal risk of perioperative infarction, it is imperative to use elaborate monitoring which should be preferably placed under local anaesthesia, before induction of anaesthesia. This monitoring includes:

- An arterial line for continuous on-line beat to beat blood pressure measurement and for drawing blood samples.
- Continuous precordial ECG monitoring from leads II and V_5 (CM_5 or CS_5) to identify dysrhythmias and early signs of myocardial ischaemia, both of which must be managed aggressively.
- In high-risk patients, a pulmonary artery catheter for continuous measurement of pulmonary artery pressure and intermittent measurement of pulmonary artery wedge pressure is most useful. On the one hand a sudden rise in these pressures may reflect a decreased left ventricular compliance from regional or global ischaemic dysfunction.[55,56] On the other hand a sustained increased pulmonary wedge pressure often indicates rising intraventricular end-diastolic pressure or hypervolemic heart failure, which may induce subendocardial ischaemia by interfering with subendocardial blood perfusion. In addition, the use of a thermistor-type catheter allows measurement of cardiac output and calculation of other important data (e.g., systemic and pulmonary vascular resistance); this information can be obtained before induction of anaesthesia and repeated as required to provide information valuable for the management of these patients.

  As a rule the haemodynamic variables should be maintained within 20 per cent of normal pre-induction values. When variations beyond these limits occur, they are treated rapidly and aggressively to limit their duration as much as possible. Cardioactive and vasoactive drugs must be available, including vasodilators, beta-blockers, inotropic drugs, antiarrhytmics, and vasopressors. In particular, appropriate solutions of phenylephrine and nitroglycerin should be ready for immediate use.
- Blood gases, electrolytes, and glucose are closely watched and maintained within normal limits.
- Left and right cardiac filling pressures, haematocrit, and urine flow are equally closely monitored and will guide blood and fluid therapy. Haematocrit should not be allowed to go below 30 per cent.
- Postoperatively the patient must be closely monitored for 48 to 72 hours or longer, should any haemodynamic abnormality have been noted. Early chest physiotherapy is instituted to prevent atelectasis and hypoxaemia. Serial 12 leads ECGs, cardiac enzymes (CK-MB isoenzymes and LDH isoenzymes) are done daily up to the seventh day in suspected cases of perioperative MI.

Choice of anaesthetic agents

As far as anaesthetic agents are concerned, there is no magic "recipe" to prevent perioperative infarction. The choice should be made according to the status of the patient, the proposed procedure, and

the experience of the anaesthetist. No anaesthetic technique has been shown to be irrefutably superior in the management of coronary patients.[1,2,44,57,58] However, Rao *et al.* reported that the highest incidence of perioperative reinfarction occurred in patients anaesthetized with nitrous oxide + oxygen + relaxant + narcotic technique.[16]

The anaesthetic most commonly used is a balanced anaesthetic including a potent narcotic, a muscle relaxant, 50 per cent or greater oxygen concentration in nitrogen (i.e., without nitrous oxide). This basic anaesthetic can be completed as required by the addition of an halogenated inhalation agent.

Fentanyl usually insures haemodynamic stability and, in sufficient doses, provides significant protection against the sympathetic stimuli of intubation, incision, and pain.[59,60] Sufentanil has the same properties, with the added advantage of shorter duration of action.

The choice of muscle relaxant depends on the condition of the patient. Pancuronium, because of its sympathetic stimulating properties, is not the best choice in hypertensive and/or tachycardic patients.[61] Atracurium may cause significant blood pressure drops, as may occur with curare and metocurine. Vecuronium,[62] pancuronium-metocurine[63] or pancuronium-atracurium[64] combinations are acceptable.

Nitrous oxide is not desirable especially because of its peripheral systemic and pulmonary vasoconstrictive effect.[65] Oxygen, with or without air, is administered according to the desired PaO_2.

Enflurane and methoxyflurane are powerful myocardial depressants in the presence of beta-blockade, whereas halothane and isoflurane are relatively free of this effect.[42] Isoflurane is a peripheral and coronary vasodilator[66] and a weaker myocardial depressant than halothane at equipotent doses.[67] Whether isoflurane-induced coronary vasodilatation is useful or harmful in ischaemic myocardial regions is controversial. For cardiovascular stability, isoflurane when carefully administered as a supplement to a narcotic-based anaesthetic, appears to be a very useful agent.

Detection and diagnosis of perioperative myocardial infarction

Obviously, in the perioperative period, angina may be absent or masked and may be confounded with postoperative pain. Often infarction will be suspected by its effects on the general condition of the patient or by the presence of its complications. On clinical examination, non-specific signs such as pallor, diaphoresis, dyspnoea should make one suspect the onset of infarction. A vagal bradycardia may be a sign of an inferior infarction.

The electrocardiogram

Initially the ECG may be normal or difficult to interpret and serial ECGs may be required in order to detect the classical signs of ischaemia or necrosis. The deep Q wave of necrosis, lasting more than 0.04 seconds, diagnostic of myocardial infarction, may not appear until the second or third day postinfarction. Electrical signs may be limited to ST segment changes indicating necrosis or subendocardial ischaemia. High voltage T waves may be a sign of acute ischaemia.

Biological signs

Elevated CK-MB fraction is the most specific indicator of myocardial necrosis.[68,69] Normally this isoenzyme has minimal blood levels and rising blood levels in the six to eight hours postinfarction are diagnostic. The peak value varies with the size of the infarct.

Serial determination of LDH blood levels show an onset of elevation at the sixth hour and this persists up to the sixth day after infarction. Of the five LDH isoenzymes, fraction 1 is the most specific for MI and the LDH_1/LDH_2 ratio becomes greater than 1.

Scintillation counting

Over the past few years myocardial scintillation counting has made an important contribution toward the diagnosis and evaluation of MI.[70] Two techniques are most useful. On the one hand certain tracers, such as thallium, are picked up by normal myocardial cells; the labelling of tissue is closely knit to its perfusion. The ischaemic or necrotic regions will show up as tracer-free areas compared to the surrounding healthy myocardium. On the other hand other markers, such as technetium 99M pyrophosphate which is most frequently used, will fix on infarcted myocardial tissue. Cells irreversibly affected will preferentially pick up the technetium while injured cells have less affinity for this marker. This preference is due to the abundance of free calcium in necrotic cells.

Myocardial scintillation counting with thallium will detect an MI almost immediately but with pyrophosphate, there will be a delay of 24 to 72 hours before detection of necrosis. New tracers are presently under study.

Complications of myocardial infarction

Lethal ventricular dysrhythmias in the early hours of an MI have already been mentioned. Later, infarction can induce dysrhythmias and conduction defects which require close monitoring and continuous ECG surveillance until the phase of stabilization of infarction.

Left ventricular insufficiency is present in 20 to 35 per cent of cases of infarction and is the most threatening complication.[10] The ensuing decreased cardiac output reduces coronary blood flow and worsens the ischaemia, extending the necrosis, and may lead to other complications, such as cardiogenic shock.

Ten to 15 per cent of acute infarcts degenerate into cardiogenic shock in which case the prognosis is grave,[10] short-term survival being rarely greater than 35 per cent. In the absence of an associated mechanical defect, at least 40 per cent of the myocardium must be destroyed for cardiogenic shock to occur.

Myocardial rupture is found in up to 25 per cent of in-hospital MI deaths.[10–12] It is the result of elongation and thinning of ischaemic fibres and may be precipitated by a hypertensive crisis. Complete rupture of the free wall of the left ventricle brings on immediate death by haemopericardium and acute tamponnade. If the rupture is incomplete or subacute it can lead to pseudoaneurysm formation. Septal rupture produces a ventricular septal defect with a left to right shunt.

Papillary rupture or dysfunction may be the result of papillary muscle necrosis and can cause mitral valve regurgitation.[71] The diagnosis is made by the detection of a holosystolic murmur at the apex and by the appearance of giant V waves on the pulmonary wedge pressure tracing. As in septal rupture, this type of mitral regurgitation often requires surgical treatment which is best undertaken after a period of mechanical support (intraaortic balloon counterpulsation) and medical therapy to stabilize the haemodynamic condition.

A pericardial reaction often accompanies an infarction: in most cases the evolution of this pericarditis is benign and requires no therapy.[72] However, a syndrome described by Dressler in 1959,[73] characterized by pericardial and pleural inflammation, occurs in three to four per cent of cases. It is accompanied by prolonged and recurring fever and may be caused by antimyocardial antibodies.

Conclusion

The overall incidence of CAD is decreasing in North America; the preventive, diagnostic and therapeutic measures have improved in the last decades as have progressed our knowledge of the disease. Despite these facts, the problem of the patient with CAD presenting for anaesthesia is still serious and likely to increase as the population ages. Therefore, the vigilant anaesthetist must seek out and protect patients at risk of developing perioperative MI.

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Résumé

L'infarctus du myocarde (IM) peut survenir en période périopératoire comme en tout autre temps, et cette éventualité est encore plus grande chez les malades avec une histoire d'infarctus. Le taux de mortalité des IM périopératoires est élevé. Les incidents entourant l'intervention influencent la survenue et la gravité de ces infarctus. L'athérosclérose déjà installée est habituellement le terrain sur lequel se greffe un thrombus et/ou un vasospasme. L'ischémie myocardique résultant de certaines perturbations périopératoires peut conduire à un infarctus du myocarde.

Tous les adultes devant subir une chirurgie doivent être investigués en vue de la détection d'une maladie coronarienne, d'hypertension artérielle et de signes d'athérosclérose. Lorsqu'une pathologie coronarienne est pressentie, un monitoring approprié est nécessaire pour la période per et postopératoire. Tout indice d'infarctus périopératoire mérite une investigation poussée et l'infarctus confirmé doit être traité vigoureusement pour en éviter les complications redoutables.