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Mini-symposium: The post-operative period

Post-operative Cardiovascular Complications

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Cardiovascular complications in the post-operative period are a significant cause of morbidity and mortality. In the recent Confidential Enquiry into Perioperative Deaths[1], three of the most common clinical causes of death were cardiovascular (13.5% bronchopneumonia, 10.8% congestive cardiac failure, 8.4% myocardial infarction, 7.8% pulmonary embolism, 6.5% respiratory failure).

The literature on cardiovascular complications after cardiac and vascular surgery is considerable. However, complications after non-cardiac surgery only will be considered in this review, and published work on this topic is limited.

Myocardial infarction

Incidence

Post-operative myocardial infarction (MI) is a leading cause of mortality and morbidity after non-cardiac surgery. The incidence of peri-operative MI in patients without previous infarction undergoing non-cardiac surgery is between 0.1 and 0.65%.[2] The overall incidence of MI in patients with a history of previous infarction is approximately 5% but this is dependent largely on the interval between the previous MI and surgery. Several studies have described this relationship and data from a retrospective study by Rao and colleagues[3] are shown in Table 1. The incidence of re-infarction was very high if surgery was performed in the first 3 months after MI (36%). After this time, the incidence declined but there was no improvement after 6 months (5%). Although these data include MIs occurring intra-operatively, the peak incidence of MI in this study was on the third post-operative day.

In a study of 232 patients (90% of whom had diabetes or hypertension), MI after non-cardiac surgery occurred in 3.8%.[4] Almost half of these were clinically silent and diagnosed only by serial electrocardiography (ECG) and cardiac enzymes. In this study, post-operative MIs occurred more frequently on the first day.

The mortality rate after peri-operative MI has been reported as 36–70%.[5]

Diagnosis

Patients who develop post-operative MI may present with the typical history, physical signs and ECG changes of infarction. However, diagnosis is notoriously difficult and over 60% may be silent.[2] Analgesics administered for post-operative pain and the effects of general anaesthesia in the immediate post-operative period often disguise chest pain. Associated cardiovascular signs, such as hypotension or heart failure, may be attributed to other common post-operative problems.

Classical ECG changes may occur (ST-elevation, T-wave inversion, Q-waves) but many MIs are subendocardial and may produce changes in T-wave morphology only. The classical pattern of raised serum enzyme concentrations is often diagnostic (Fig. 1). However, creatine phosphokinase released from other tissues, e.g. muscle, may be raised after surgery. Therefore, measurement of the isoenzyme creatine kinase-Mb, which is found in cardiac muscle only, is useful in the post-operative period. Creatine phosphokinase reaches a peak after 24h and returns to normal after 2–3 days.

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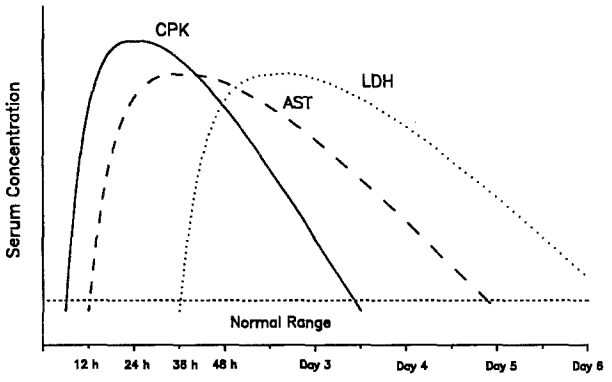

Fig. 1 — Pattern of raised serum enzyme concentrations after myocardial infarction. CPK = creatine phosphokinase; AST = aspartate amino-transferase; LDH = lactic dehydrogenase.

Myocardial infarction may be diagnosed also by radionuclear imaging techniques and trans-oesophageal echocardiography. These are useful techniques, particularly in the diagnosis of subendocardial MI, but are not available generally.

Myocardial infarction must be suspected if any cardiovascular problem occurs in the post-operative period.

Causes of post-operative MI

The most important underlying cause of MI after surgery is pre-existing ischaemic heart disease (IHD). One quarter of middle-aged men in the UK have some evidence of IHD.[6]

Several factors which may be present in the post-operative period increase the likelihood of MI. Rapid and marked fluctuations of blood pressure, heart rate or circulating blood volume may increase myocardial oxygen demand or decrease coronary artery blood flow. Cardiovascular changes are exacerbated by uncontrolled post-operative pain and anxiety. Other factors include increased platelet aggregation and changes in haemostatic mechanisms, increased plama catecholamines and coronary artery vasospasm.[5]

It is now recognised that hypoxaemia occurs well into the post-operative period. Work is now in progress to investigate the relationship between hypoxaemia and cardiac events. It is established that periods of hypoxaemia are associated with tachycardia, and recent preliminary work has shown a relationship between hypoxaemia and ST-segment depression in some patients after surgery.[7] It is likely that unrecognised post-operative hypoxaemia is an important factor in the aetiology of MI.

Evidence for the importance of adverse peri-operative factors is provided in the study by Rao and colleagues described previously.[3] The retrospective incidence of MI was decreased considerably in a prospective study in which patients were monitored invasively and changes in cardiovascular parameters treated aggressively (Table 1). For example, the incidence of reinfarction after surgery during the first 3 months after MI was reduced from 36% to 5.8%. However, the design of this study has been criticised and further work is needed to confirm these data.

Myocardial ischaemia

There has been considerable interest in myocardial ischaemia occurring intra-operatively and the importance of myocardial ischaemia in patients after cardiac surgery is recognised. The ECG is monitored continuously after surgery in these patients. However, myocardial ischaemia occurring in the post-operative period in patients after non-cardiac surgery has only recently been investigated.

Myocardial ischaemia on the ward cannot be diagnosed reliably by occasional standard 12-lead ECGs. However, Holter monitoring (recording of the ECG on magnetic tape and subsequent analysis) or computerised ambulatory monitoring (continuous analysis of ECG by computer) can detect reliably the incidence, severity and duration of ischaemia. These techniques are being applied to patients after non-cardiac anaesthesia but few results have been published to date. The available data suggest that 27–40% of patients with known IHD or at least two pre-operative cardiac risk factors who underwent non-cardiac surgery had significant episodes of ST segment depression.[8,9] In both these studies, most episodes were clinically silent and many were associated with tachycardia. Episodes continued to occur up to 7 days after surgery. One patient developed MI on the fourth post-operative day and this was associated with ST depression which became progressively worse from day 1 to day 3. This was not detected by routine daily 12-lead ECGs.[8]

The incidence of clinical angina in patients with IHD after non-cardiac surgery is considerably less than that of silent ischaemia. The relevance of silent myocardial ischaemia is still to be established. Associated factors need to be identified and the effect of controlling these factors requires assessment. For example, myocardial ischaemia has been linked already to tachycardia and hypoxaemia. Finally, does silent myocardial ischaemia influence mortality and morbidity and will its prevention improve patient outcome ? The answers are awaited.

Table 1 — Incidence of peri-operative MI after previous MI in groups of patients studied either retrospectively, with standard management, or prospectively with active intervention.[3]

	Retrospective	Prospective
0–3 months	36%	5.8%
4–6 months	26%	2.3%
7–12 months	5%	1.0%
>12 months	5%	1.6%

Table 2 — Common causes of post-operative sinus tachycardia

Pain and anxiety
Hypovolaemia
Pyrexia
Hypoxaemia
Hypercapnia
Heart failure
Drug effects
Urinary retention

Arrhythmias

The problems associated with the detection of arrhythmias in the post-operative period are similar to those of myocardial ischaemia. Intermittent 12-lead ECG recordings are of little benefit; constant recording of ECG is required.

Several studies have described the incidence of intra-operative arrhythmias but there has been little published work on the post-operative period. Consequently, their incidence and importance are unknown.

Sinus arrhythmias

Sinus tachycardia (>100 beats/min) is by far the most common arrhythmia in the post-operative period. It is important to recognise and treat, as it is likely to be associated with adverse post-operative outcomes such as myocardial ischaemia and infarction (see above) and can precipitate cardiac failure. Heart rates of 170 beats/min may result from sinus tachycardia and there may be diagnostic confusion with supraventricular tachycardia. Identification of P waves and failure to respond to carotid sinus massage help to identify sinus rhythm.

The causes of sinus tachycardia include pain, anxiety, hypovolaemia, pyrexia, drug effects, hypoxaemia, hypercapnia and heart failure (Table 2). In the management of post-operative arrhythmias, it is important to identify and treat any underlying cause. It may be necessary to use beta-blockers, particularly in patients with known IHD or if the ST segments become depressed.

Bradycardia (<60 beats/min) occurs commonly in the immediate post-operative period. It is usually caused by the residual effects of neostigmine, anaesthesia or concomitant medication. It is less common if the muscarinic effects of neostigmine have been antagonised by glycopyrrolate rather than atropine.[10] Hypoxaemia must be excluded in all cases of bradycardia.

Other arrhythmias

Supraventricular and ventricular ectopic beats are common after anaesthesia but their significance after non-cardiac surgery is uncertain. Twenty years ago, the incidence in the first 2–3 h of recovery after a wide variety of surgical and anaesthetic procedures was reported as 48%.[11] Supraventricular arrhythmias were more frequent than ventricular but little further information could be gleaned from the study.

Causes of supraventricular and ventricular arrhythmias after anaesthesia and surgery include pre-existing cardiac disease, drug effects, hypoxaemia, electrolyte imbalance (especially potassium), myocardial infarction or ischaemia, hypercapnia, metabolic acidosis and alkalosis and poorly positioned central venous catheters.

Treatment

Treating the cause of the arrhythmia is often more important than anti-arrhythmic therapy. The decision to use specific therapy depends on the severity and type of arrhythmia and its effect on cardiovascular function, e.g. blood pressure, cardiac output, myocardial ischaemia. For a full description of the diagnosis and management of cardiac arrhythmias associated with anaesthesia the reader is referred elsewhere.[12]

Hypertension

Hypertension in the post-operative period occurs frequently and may be due to pain, severe anxiety, hypoxaemia, hypercapnia, fluid overload, urinary retention or acute withdrawal of medication (Table 3).

Myocardial oxygen consumption is increased during periods of hypertension because of increased ventricular wall tension. This may lead to myocardial ischaemia, failure or infarction, especially when associated with IHD.

In a large study (1844 patients) of changes in blood pressure after a wide variety of elective surgical and anaesthetic procedures, 60% of those who were hypertensive in the immediate post-operative period (3.3%) had a history of hypertension.[13] Complications such as arrhythmias, cardiac failure and cardiovascular accidents were associated with periods of hypertension lasting more than 3 h.

Many patients undergoing anaesthesia are receiving concurrent oral medication. In a retrospective study of 57,176 patients and a prospective study of 216 patients, 24–32% were receiving medication before surgery[14]; 10–16% of patients were taking medication for cardiovascular disease. Acute withdrawal of antihypertensive agents is associated with rebound hypertension in the post-operative period.

Table 3 — Common causes of post-operative hypertension

Pain and anxiety
Hypoxaemia
Hypercapnia
Fluid overload
Acute withdrawal of chronic antihypertensive medication
Urinary retention

Table 4 — Common causes of post-operative hypotension

Reduced preload
Hypovolaemia
Pulmonary embolism
Reduced cardiac contractility
Drug effects
Pre-existing disease
Myocardial infarction
Septicaemia
Reduced systemic vascular resistance
Septicaemia
Drug effects

There is considerable knowledge of the incidence and effect of post-operative hypertension after cardiac and vascular surgery. Invasive arterial pressure monitoring is routine in these cases and provides reliable and continuous data. However, after non-cardiac surgery, blood pressure is often measured only at 4-h intervals initially, and even less frequently during the night and on the 2nd and 3rd post-operative days.

The availability of automatic non-invasive blood pressure monitors may increase the detection rate of hypertension on general wards after surgery.

Treatment

In common with all cardiovascular complications after surgery, the treatment of the underlying cause of hypertension is an important factor in the management.

Patients with pre-existing cardiovascular disease, such as IHD or hypertension, are particularly at risk and concomitant medication should be continued. This will often require intravenous administration of drugs, as gastric emptying and gastrointestinal absorption are impaired after surgery, particularly in the presence of paralytic ileus or opioid analgesia.

If potent antihypertensive agents are used, it is important to monitor blood pressure continuously, preferably with an intra-arterial cannula. Measurement of central venous pressure (CVP) is preferable also, particularly in patients with pre-existing cardiac problems. Many episodes of post-operative hypertension last for only a few hours.[13] Therefore, the use of drugs with a short duration of action is preferable in the first instance. Patients may be particularly sensitive to these agents after anaesthesia and surgery and all should be given slowly and with particular care.

Sodium nitroprusside is a potent hypotensive agent. Infusion rate should be titrated carefully according to its effect and the starting dose should be small (0.3 μg/kg/min). In order to prevent undesirable plasma cyanide ion concentrations, the maximum recommended dose for use over a period of several hours is 8 μg/kg/min.

Glyceryl trinitrate (10–200 μg/kg/min) may be a more appropriate choice in patients with IHD but may be ineffective in some patients. Hydralazine (initial dose 5–10 mg slowly i.v.) is useful, but its onset of action is delayed (15–30 min) and a further dose should be given only after this time. Sodium nitroprusside, glyceryl trinitrate and hydralazine may be accompanied by an undesirable compensatory tachycardia.

Beta-adrenoceptor blockade is a useful method of controlling hypertension, especially when accompanied by tachycardia. Labetalol is particularly useful because of its α-and β-blocking properties. There is a variable dose-dependent fall in arterial pressure, which may last from 6–16 h, after intravenous administration. The recommended dose is 50 mg given over 1 min which can be repeated every 5 min to a maximum of 200 mg. Intravenous infusion (starting dose 2 mg/min) produces a more controlled response.

Esmolol is a new short-acting β_1-selective blocker which has a half-life of 9 min and is metabolised by blood esterases.[15] Consequently, it has a short duration of action. It has been used effectively in the control of post-operative hypertension and the recommended infusion rate is 100–300 μg/min. Steady-state concentrations are reached rapidly because of its short half-life.

It is likely that uncontrolled hypertension contributes significantly to cardiac morbidity after anaesthesia but further work is needed to ascertain its true incidence and importance after non-cardiac surgery.

Hypotension

Hypotension is extremely common in the post-operative period and the causes can be classified into reduced preload (hypovolaemia, pulmonary embolism), reduced cardiac contractility (drugs, pre-existing disease, myocardial infarction, septicaemia) and reduced systemic vascular resistance (SVR) (drugs, septicaemia) (Table 4).

Hypotension should be treated aggressively as it may lead to impaired perfusion and failure of vital organs. Perfusion of surgical anastomoses may be impaired also, leading to subsequent breakdown.

The diagnosis of the cause and subsequent management of hypotension is aided considerably by measurement of CVP. Measurement of pulmonary capillary wedge pressure (PCWP) is useful also, particularly in patients with myocardial disease.

Hypovolaemia is characterised by a low CVP and reduced PCWP (<5–10 mm Hg) and a normal or low cardiac output. The combination of raised CVP, PCWP (>15 mm Hg) and SVR with reduced cardiac output indicates impaired myocardial performance. If septicaemia is suspected, pulmonary artery catheterisation is highly desirable; the characteristic findings are a high cardiac output and low SVR.

Treatment of the cause of hypotension is a vital part of the management. Cardiovascular parameters

Table 5 — Antibiotic prophylaxis against subacute bacterial endocarditis after surgery and anaesthesia

Dental surgery & instrumentation of upper respiratory tract	
Low risk	amoxycillin 3G & probenicid 1G orally 4h before surgery or amoxycillin 1G iv at induction amoxycillin 500mg 6h later
High risk* or received penicillins in the previous month	amoxycillin 1G & gentamicin 120mg iv at induction amoxycillin 500mg 6h later
Genitourinary surgery	
All patients	amoxycillin 1G & gentamicin 120mg iv at induction amoxycillin 500mg 6h later
Obstetric, gastrointestinal & gynaecological surgery	
High risk	Genitourinary protocol
Allergy to penicillin	
all cases	vancomycin 1G iv over 1h & gentamicin 120mg iv

Antibiotics given orally only if gastric emptying not delayed. Ampicillin can be substituted for parenteral amoxycillin.
*see text

should be optimised by the use of appropriate fluid replacement and inotropic agents.

Cardiac failure

After a variety of surgical procedures, including vascular but not cardiac surgery, post-operative pulmonary oedema occurred in 3.6% of patients over 40 years of age.[16] Common causes of heart failure are pre-existing heart disease, fluid overload, myocardial infarction and hypertension. Cardiac failure is often misdiagnosed as chest infection.

Pulmonary embolism

Pulmonary embolism (PE) is a frequent cause of death in the post-operative period (7.8% of CEPOD[1] deaths). Deep venous thrombosis in the leg or pelvis is the usual cause of the embolus. However, PE may originate from the inferior vena cava, right side of the heart or other large veins.

Pulmonary embolus presents with sudden onset of dyspnoea, hypoxaemia and tachycardia, although small emboli may result in less dramatic clinical signs. As with myocardial infarction, chest pain may be absent. Acute right heart failure, hypotension and peripheral vasoconstriction indicate a large PE.

The ECG is often unchanged but may show signs of right ventricular strain (right ventricular T-wave inversion, right axis deviation, right bundle branch block). Chest X-ray is often unhelpful but may show dilation of the hilar vessels proximal to the embolus and reduced vascular markings distal to it. Wedge-shaped opacities and pleural effusion may develop later.

Ventilation-perfusion scanning shows areas of poor perfusion with normal ventilation but this is often difficult to interpret in the post-operative period or after multiple small emboli. Pulmonary angiography is the most reliable method of detecting large emboli.

Bacterial endocarditis

This may occur after surgery in patients with congenital or valvular heart disease and in patients with prosthetic valves. Signs and symptoms develop slowly and it is not likely to present in the first few days after surgery. It is important to be aware of this problem and to ensure administration of appropriate prophylactic antibiotics (Table 5). Patients with prosthetic valves and previous history of bacterial endocarditis are particularly at risk and prophylaxis may differ from those patients considered to be at low risk.

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