Pre-operative assessment of cardiac patients for non-cardiac surgery

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Summary

Non-cardiac surgery presents significant risks to patients with cardiac diseases. With the improvement in anaesthetic techniques and intensive care facilities, many cardiac patients who in the past would have been considered as being at too great a risk are now being considered for non-cardiac surgery. Smaller centres must still practise strict selection of patients if they do not possess an intensive care unit with facilities for full haemodynamic monitoring. We present a review of the recent literature and current practice at our hospital to assist clinicians in assessing these patients for anaesthesia. However, the final decision whether the patient is fit for anaesthesia still rests with the anaesthetist.

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General considerations

Danger of anaesthesia

The properties of some anaesthetic agents and the stress of surgery could lead to an increased demand for oxygen by the myocardium. Some of the mechanisms involved are tachycardia, hypoxia, drug actions, decreased cardiac output, volume overload, shivering, hypotension and blood loss. In the presence of coronary artery stenosis the increased oxygen demand may lead to myocardial ischaemia and decreased cardiac pump function. Likewise, if there is a fixed valvular stenosis tachycardia could lead to decreased cardiac output and pulmonary oedema. Some anaesthetic agents, such as halothane, have a direct depressant efect on the myocardium causing a fall in blood pressure and cardiac output. Ventricular arrhythmias may arise as a result of hypercarbia and increased sympathetic tone caused by hypoxia, hypovolaemia and acidosis.[1,2]

Spinal and epidural anaesthesia

Spinal and epidural anaesthesia is not necessarily a safer procedure for patients with cardiac disease (other than heart failure) than general anaesthesia.[1,3] It can produce hypotension. This can readily be reversed by α-stimulants, but these agents have their own dangers because they increase afterload and may increase oxygen demand, resulting in myocardial ischaemia and heart failure.

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Non-cardiac risk factors

Non-cardiac risk factors associated with an increased risk of cardiac morbidity and mortality after general surgery include the following: (*i*) emergency operation;[3,4] (*ii*) poor general health — viz. respiratory failure, renal failure, hepatic dysfunction and malnutrition;[1,4] (*iii*) advanced age (over 70 years);[1,4] (*iv*) intra-operative hypotension — a fall in systolic blood pressure of more than 30% lasting for more than 10 minutes in a patient with a previous infarct is associated with a 15% re-infarction rate postoperatively;[3,5] (*v*) site of surgery — intrathoracic, abdominal and aortic procedures are associated with an increased risk of postoperative infarction and heart failure;[3-6] and (*vi*) duration of anaesthesia — in a patient with ischaemic heart disease the risk of re-infarction is increased after a procedure lasting more than 3 hours.[3-5]

Ischaemic heart disease

Previous myocardial infarction

Recent myocardial infarction is associated with an increased risk of re-infarction postoperatively. The more recent the infarction, the greater the risk of re-infarction and the higher the mortality rate. Postoperative mortality is also related to age; the older the patient, the higher the mortality after re-infarction.

Knapp *et al.*[7] studied 8 984 male patients over the age of 52 years and found that if there had been a recent infarction the re-infarction rate was 6% and the mortality rate 59%. With no previous history of infarction the chance of postoperative infarction averaged 0,7% and the mortality rate 19%. It was concluded that the operation would be safer if performed 2 years after the infarction. The type of anaesthetic given and the type of surgery did not statistically contribute to postoperative cardiac morbidity and mortality.[7]

A similar study of 12 712 patients over the age of 50 years by Topkins and Artusio[8] showed that in the absence of pre-operative infarction the postoperative infarction rate was 0,66% and the mortality rate 26,5%. If there had been an infarction pre-operatively, the postoperative re-infarction rate was 6,5% and the mortality rate 70%. They drew attention to the following: (*i*) if the infarction had occurred less than 6 months before current surgery, the re-infarction rate was an unacceptably high 54%; (*ii*) if the infarction had occurred 6-24 months previously, the re-infarction rate was 20-25%; and (*iii*) if the infarction had occurred more than 2 years previously, the re-infarction rate was 5,9%.

The study of Tarham *et al.*[9] in 1972 showed a postoperative re-infarction rate of 37% if surgery was performed during the 3-month period following myocardial infarction, a rate of 16% in the 3-6-month period, and a rate of 6% beyond 6 months. Their overall mortality rate was 50%.

Steen *et al.*[5] showed an average re-infarction rate of 6%, with a mortality rate of 69%. They concluded that if the infarction had occurred less than 3 months previously there was a 27% re-infarction rate, while if it had occurred more than 6 months earlier the re-infarction rate was 4-5%.

Other interesting points to emerge from the study were as follows: (*i*) there was no difference in the re-infarction rate for

males and females; (*ii*) uncontrolled hypertension added significantly to morbidity — re-infarction rates were 9,4% for hypertensive patients and 4,7% for those who were not hypertensive; (*iii*) diabetes mellitus had no influence; (*iv*) stable angina pectoris also made no difference; (*v*) the position of the primary infarct played no role in the incidence of re-infarction; (*vi*) the type of anaesthetic did not contribute significantly to postoperative re-infarction or mortality; (*vii*) intrathoracic and upper abdominal procedures were associated with an increase in the incidence of postoperative infarction; (*viii*) the duration of surgery was important — in the case of a procedure of under 3 hours the re-infarction rate was 5,9%, while for a procedure of over 3 hours there was a re-infarction rate of 15,9%; and (*ix*) intra-operative hypotension was associated with a 15,2% re-infarction rate after the operation (when no hypotension occurred, the rate was 3,2%).

Finally Knapp[10] found that if infarction had occurred more than 3 years before current surgery, the re-infarction rate was 1%. This was the same as the primary infarction rate for the male population of over 50 years of age in the study area.

The following conclusions can be drawn from the studies mentioned: (*i*) surgery within 6 months of an infarct is associated with an unacceptably high incidence of postoperative re-infarction and mortality; (*ii*) 3 years after infarction the risk of postoperative infarction is almost the same as that of a patient who has not had a previous infarct, providing he is well and without symptoms or complications; and (*iii*) the method of anaesthesia and the agent used do not affect the re-infarction rate.[5]

The majority of postoperative re-infarctions do not occur during surgery but during the first 5 postoperative days. A high percentage of postoperative re-infarctions (up to 50%) are silent and manifest with extrasystoles, pulmonary oedema or hypotension. The current practice at our institution is as follows: (*i*) elective surgery is postponed for as long as possible following myocardial infarction — a minimum period of 6 months is recommended; (*ii*) for a high-risk operation, e.g. aortic surgery within 1 year of infarction, careful monitoring intra-operatively and for the first 5 postoperative days, with special attention to systemic blood pressure, pulse rate and rhythm, pulmonary wedge pressure, systemic vascular resistance and cardiac output, is advised; (*iii*) when an emergency procedure is required within 6 months of a myocardial infarction full haemodynamic monitoring and manipulation is indicated, combined with a stress-free anaesthetic; and (*iv*) patients requiring major surgery such as intrathoracic and upper abdominal surgery within 1 year of the primary infarction should be transferred to a specialist centre with full intensive care facilities.

Unstable angina

It is unsafe to operate on patients with unstable angina.[11] The patient should be stabilized medically before elective surgery. If necessary, a coronary artery bypass graft may first be required to relieve angina and protect the myocardium. This can be followed later by the non-cardiac procedure. If an emergency procedure is unavoidable, however, we would recommend the following: (*i*) transfer of the patient to a large centre with intensive care facilities; (*ii*) adequate sedation and analgesia; (*iii*) liberal doses of nitroglycerin, ß-blockers and/or a calcium antagonist, with a 6-hourly increase in the dosages of these drugs while the condition remains unstable; (*iv*) vigorous treatment of existing hypertension, infection, heart failure, arrhythmias or any other factor contributing to the unstable state; and (*v*) full haemodynamic monitoring with a flow-directed pulmonary artery catheter to guide fluid therapy and drug administration, particularly if there is any doubt about left ventricular function.

Stable angina

It is our experience that patients with stable angina usually do quite well, provided their anxiety and pain are adequately managed. It is strongly advised that ß-blockers be continued up to and immediately after surgery, since their withdrawal is known to precipitate serious ischaemia.[12,13] Patients with triple-vessel disease have a risk equal to those with a history of a previous infarction.[6,13] Should angina occur shortly before surgery, we recommend that the procedure be postponed for at least 4 hours while the angina is treated and the ischaemic myocardium is allowed to recover.

Other conditions

Coronary artery bypass graft. Non-cardiac surgery is well-tolerated in these patients. The risk of postoperative myocardial ischaemia is less than that in patients with ischaemic heart disease.[14] Prophylactic antibiotics are not required.[15]

Nonspecific ST- and T-wave changes. These do not appear to be associated with an increased risk of postoperative myocardial infarction. They do, however, appear to be associated with an increased risk of postoperative cardiac death (arrhythmias and pulmonary oedema).[3] Our policy is to exclude ischaemic heart diseases by means of a pre-operative exercise ECG or an exercise thallium scan.

Prolonged Q-T interval.[11] Prolongation of the Q-T interval is associated with ventricular arrhythmias, syncopal attacks and sudden death. It may be congenital, due to decreased serum K^+, Mg^{2+} or Ca^{2+} values, or associated with drugs (e.g. quinidine, procainamide, phenothiazines and tricyclic antidepressants). Hypothermia and neck surgery have been reported to prolong the Q-T interval. We suggest that the reversible factors be corrected before surgery.

Factors not associated with a significant risk of cardiac death

Factors not associated with a significant risk of cardiac death[3] are: (*i*) hypertension without organ damage; (*ii*) S_4 gallop; (*iii*) systolic ejection murmur due to aortic sclerosis; (*iv*) symptomatic peripheral vascular disease; (*v*) controlled diabetes mellitus; and (*vi*) hyperlipidaemia without ischaemic heart disease.

Cardiac failure

Adequate cardiac pump function with a normal reserve is important, as the patient needs an increased cardiac output to cope with the demands associated with the haemodynamic stress of surgery. Cold and shivering postoperatively may increase the cardiac output three to four times.[1] Anaesthetic agents also depress myocardial pump function and may precipitate cardiac failure in a patient with borderline cardiac function. Patients in frank heart failure are extremely poor operative risks. Operating in the presence of a third heart sound or elevated jugular venous pressure carries the same risk of postoperative cardiac morbidity and mortality as operating on a patient within 6 months of myocardial infarction.[1] Patients with class III or class IV symptoms (New York Heart Association Classification) but without a third heart sound or elevated jugular venous pressure also have an increased risk of postoperative pulmonary oedema.[1,3,4] In one study[3] 40% of patients with postoperative pulmonary oedema were reported to have died of cardiac complications.

Patients with controlled heart failure receiving medication for class I and II symptoms have only a slight risk of post-operative pulmonary oedema. However, more than 50% of

patients who develop pulmonary oedema due to acute left ventricular decompensation postoperatively have no previous symptoms of heart failure.[3]

A previous myocardial infarction does not increase the risk of postoperative pulmonary oedema provided the patient is symptom-free.[3]

For the patient in heart failure we would advise the following: (*i*) postpone elective surgery until adequate control is ensured, give full treatment with digoxin and/or vasodilators and diuretics, and correct factors such as anaemia and hypokalaemia; (*ii*) if the surgery is of an urgent nature transfer the patient to a major centre where careful invasive monitoring with pulmonary artery catheter, arterial line and central venous pressure monitoring should be utilized to control preload, left and right ventricular stroke work and afterload — these monitors should be used to guide fluid and drug administration to manipulate the haemodynamic situation;[16] and (*iii*) give prophylactic digitalis to patients with a previous history of cardiac failure, especially when halothane or enflurane is to be used.[1,17,18]

Hypertension[19,20]

It was previously recommended that antihypertensive treatment should be stopped before surgery, but today it is felt that:

1. Patients must be maintained on their drugs up to and directly after surgery.[11,17] Beta-blockers need not be withdrawn.[12,21,22] If one wishes to stop a ß-blocker for another reason, it should be tapered off and not withdrawn abruptly.[23] When clonidine has to be withdrawn one can counter the problem of rebound hypertension by pretreating the patient with reserpine to deplete the catecholamine stores.[24] Another way of withdrawing clonidine is to taper the dose off for 4-5 days. If the patient is on a diuretic, it is important to ensure normal serum potassium levels.

2. A diastolic blood pressure of 90-100 mmHg does not imply an increased risk to the postoperative patient.[25] There is good reason to accept that the blood pressure should not exceed 160 mmHg systolic and 110 mmHg diastolic in the patient scheduled for surgery.[11,25] Today the important aspect is adequate control of high blood pressure and not the specific drugs used for treatment.

3. It is important to exclude causes of secondary hypertension before surgery. For example, unknowingly operating on a patient with a phaeochromocytoma is associated with a mortality rate of 50%.

4. Operating on patients with known cerebrovascular disease does not carry an increased risk, provided that excessive hypotension does not develop during the operation, but a previous cerebrovascular accident does increase the risk of postoperative stroke,[10] which carries a poor prognosis (50% mortality).[1,11,26,27]

Conduction abnormalities

A temporary pacemaker is required for the following conduction disturbances:[1,28,29] (*i*) complete heart block; and (*ii*) Mobitz II block.

In addition, we insert a temporary pacemaker in the following situations:[30–32] (*i*) first-degree atrioventricular block plus complete left bundle-branch block;[30] (*ii*) first-degree atrioventricular block with right bundle-branch block and left anterior hemiblock;[30] and (*iii*) right bundle-branch block and left posterior hemiblock.[31]

The need for pacing asymptomatic patients with the above-mentioned three conditions is debatable. Should problems arise, an anaesthetist experienced in rapid insertion of a temporary pacemaker might not insist on pre-operative pacing for these conditions. First-degree atrioventricular block, Mobitz I block,[33] right bundle-branch block and the bifascicular blocks (right bundle-branch block and left anterior hemiblock or left bundle-branch block) do not require temporary pacing, provided the patient has never experienced syncopal attacks.[11,31,33–38] Pre-operative insertion of a temporary pacemaker for congenital complete heart block would depend on whether the patient was asymptomatic and had an adequate pulse rate response to exercise.[39] The temporary pacemaker should be inserted 36-48 hours before surgery as the electrode needs a minimum of 24-36 hours to be covered with fibrin and to become fixed to the endocardium. If the anaesthetist is experienced with the workings of a pacemaker, however, this can be inserted shortly before operation provided that the threshold is checked and the pacemaker functions effectively before induction of anaesthesia.

Care should be taken not to touch electrical apparatus while handling non-insulated parts of the pacemaker or electrode, since current leaks of small amplitudes may be conducted to the patient's heart and cause ventricular fibrillation.[37]

Permanent pacemakers

The function of the pacemaker should be checked pre-operatively. The main problems associated with electrocautery are well known — it may give rise to ventricular fibrillation or inhibition of the pacemaker. If electrocautery is to be used it is suggested that the indifferent diathermy electrode be placed as far away from the pacemaker as possible and that good contact between the patient and diathermy plate be ensured. The use of diathermy should be limited to 1-second bursts, as far apart as possible. It is advisable to convert the pacemaker to a fixed mode with a magnet before diathermy is used.[37] Attention should be given to the threshold as this may rise during hypokalaemia, sepsis and after administration of suxamethonium.[28] Should this occur, one must consider the use of catecholamines to lower the threshold again. Antibiotic prophylaxis is not required.[15]

Arrhythmias

Sinus rhythm is the only rhythm not associated with an increased risk of postoperative morbidity or mortality.[3] The cause of sinus tachycardia must be sought, the most important being congestive heart failure. Occurrence of more than five ventricular extrasystoles per minute is associated with an increased risk of postoperative myocardial ischaemia and infarction.[3] If pathological extrasystoles are noted before surgery the cause should be defined and treated if possible.

The patient should be monitored closely during surgery, and intravenous lignocaine in adequate doses should be used when necessary for ventricular arrhythmias. If a single bolus of lignocaine is unsuccessful in suppressing the ventricular extrasystoles the bolus should be repeated and a constant infusion of lignocaine should be started.

In the case of the patient who is on anti-arrhythmic drugs pre-operatively we recommend keeping the patient on the drugs; however, the use of lignocaine intra-operatively is preferred. Care should be taken because most drugs used to treat arrhythmias may depress myocardial function.

The heart rate of a patient with sinus bradycardia while on a ß-blocker or digitalis can usually be increased with atropine. If the patient on a ß-blocker does not respond to atropine we recommend exercise in bed to evaluate the degree of ß-blockade. If this does not increase the heart rate, an infusion of isoprenaline is started to try to speed it up. If the degree of ß-blockade is so extensive that even this measure does not help, we usually postpone surgery and decrease the dose of ß-blockers gradually until an adequate response is obtained. This, however, is seldom required. In larger centres a temporary pacemaker can be inserted.

In the absence of a clear reason for sinus bradycardia one should keep in mind drugs and metabolic causes, of which hypothyroidism is the most dangerous as far as surgery is concerned.

Finally, the sick-sinus syndrome should be excluded as this is a definite indication for insertion of a pacemaker pre-operatively.[1,11,33]

Valvular heart disease

If the patient with valvular heart disease is asymptomatic or only mildly symptomatic[38] and has a normal heart size on the chest radiograph and a normal ECG, there is no need to anticipate problems during anaesthesia. Aortic stenosis, severe aortic incompetence and mitral stenosis are the most dangerous of the valve lesions during anaesthesia.[40] If elective surgery is considered in a symptomatic patient with aortic valve disease or in one who has marked left ventricular hypertrophy or dilatation, he must first be assessed for valve replacement before non-cardiac surgery;[41] likewise the symptomatic patient with mitral stenosis and gross changes on the ECG and chest radiograph should be considered for valvotomy first. Some authorities feel that patients with asymptomatic mitral stenosis should be digitalized prior to anaesthesia as sudden atrial fibrillation with a rapid ventricular rate may result in pulmonary oedema.[42-44] Mitral incompetence without cardiac failure is the best-tolerated of all the lesions.[45] However, if a patient requires mitral valve replacement this should be done before the operation. If a patient with symptomatic valvular disease requires emergency surgery, he should be referred to a large centre where haemodynamic monitoring can be performed.

Patients with hypertrophic obstructive cardiomyopathy, bicuspid aortic valve and prolapsing mitral valve also require antibiotic cover for all dental, upper respiratory tract, urogenital and lower gastro-intestinal tract manipulations as well as for any other invasive procedures.[15,46,47] Parenteral antibiotics give more predictable blood levels and are preferred in the following situations:[15,47]

Dental and upper respiratory tract procedures: (*i*) aqueous crystalline penicillin G (1 million U) intramuscularly with procaine penicillin G (600 000 U) intramuscularly 1 hour before, followed by penicillin V 500 mg orally 6-hourly for 8 doses; (*ii*) penicillin V (2 g) 30 minutes before, followed by 500 mg orally 6-hourly for 8 doses; and (*iii*) penicillin allergy — erythromycin 1 g orally 1½ - 2 hours before, followed by 500 mg orally 6-hourly for 8 doses.

Patients with prosthetic heart valves: (*i*) aqueous crystalline penicillin G (1 million U) intramuscularly with procaine penicillin G (600 000 U) intramuscularly plus streptomycin 1 g intramuscularly ± 30 minutes before, followed by penicillin V 500 mg orally 6-hourly for 8 doses; (*ii*) pencillin allergy — vancomycin 1 g intravenously over 30 minutes ± 1 hour before, followed by erythromycin 500 mg orally 6-hourly for 8 doses.

Genito-urinary and gastro-intestinal manipulation: (*i*) aqueous crystalline penicillin G (2 million U) intravenously or intramuscularly or ampicillin (1 g) intravenously or intramuscularly, plus gentamicin 1,5 mg/kg (not more than 80 mg) intravenously or intramuscularly or streptomycin 1 g intramuscularly, followed by gentamicin and penicillin/ampicillin at the same dose 8-hourly for 2 doses, or streptomycin and pencillin/ampicillin 12-hourly for 2 doses; (*ii*) penicillin allergy — vancomycin 1 g intravenously over 30 minutes ± 1 hour before, plus streptomycin 1 g intramuscularly, repeated after 12 hours.

Prosthetic valves

If the patient is not in cardiac failure and the valve is functioning well, he can be expected to do well during anaesthesia and postoperatively. If the patient has cardiomegaly and a prosthetic valve but is not on maintenance digitalis, we feel that he should be digitalized pre-operatively as myocardial function is unlikely to be totally normal and the stress of surgery may precipitate pulmonary oedema. Antibiotic prophylaxis is essential for these patients. It is given as outlined above, but in addition, for all upper respiratory and dental procedures, the patient must be covered with an aminoglycoside as well as penicillin.[15,47]

Another problem associated with non-tissue valves is anticoagulation. The necessity for anticoagulation is far greater for mitral or combined mitral and aortic prostheses than for an isolated aortic prosthesis.[48,49] For all the valves we recommend that oral anticoagulation be stopped 3 days before surgery to allow the prothrombin index to rise to normal.[44,50] While some authorities feel that all surgical procedures, other than those involving the eye, liver and central nervous system, can safely be carried out with the patient under anticoagulant cover,[51] most would recommend that anticoagulants be withdrawn pre-operatively and that 12-24 hours elapse after surgery before the patient is started on heparin. If the patient has no bleeding problems, he can restart oral therapy 3 days later. Some feel that the patient with an isolated aortic prosthesis may safely be left without anticoagulation until he is able to resume therapy.[48,49]

Congenital heart disease

Patients with mild pulmonary stenosis, atrial or ventricular septal defects, or a patent ductus arteriosus[41] without heart failure or pulmonary hypertension can undergo elective surgery, usually without any problems.[38] They do require antibiotic prophylaxis. It would seem advisable to repair a coarctation of the aorta before elective surgery. Patients with untreated severe pulmonary stenosis, tetralogy of Fallot or Eisenmenger's syndrome do not do as well during surgery.[38,52] If emergency surgery is required it should only be attempted in a major hospital where invasive monitoring can be carried out.

Asymptomatic carotid bruit

An asymptomatic carotid bruit does not appear to place the patient at an increased risk of postoperative stroke during non-cardiac surgery.[27] Nevertheless its presence should alert the physician to the possibility of carotid artery stenosis which may render the patient susceptible to cerebral damage during intra-operative hypotensive episodes.[11]

Conclusion

We have briefly reviewed the current practice in our institution with regard to cardiac patients admitted for non-cardiac surgery. The concepts outlined are based on world literature and on our experience.

Haemodynamic monitoring and manipulation in operating rooms have made surgery in the presence of cardiac lesions a much safer procedure. This factor, together with programmed hand calculator facilities, has given the cardiac anaesthesiologist a scientific and correct manner in which to treat the more severe cardiac lesions intra-operatively.

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