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Title Preoperative assessment of cardiovascular risk in non-cardiac surgery and preoperative management of current disease

Summary

- This chapter describes risk assessment and cardiovascular disease management before elective and scheduled surgery.
- The aims are to help patients decide whether to proceed with surgery and to help clinicians minimise risk.
- I have used death as the outcome to illustrate the calculation of non-operative and operative risk.
- I have described a step-by-step sequence to determine risk.
- Patients should perform a fitness test to increase the precision of the risk estimate.

Introduction

I work as a consultant anaesthetist in a district general hospital in the south west United Kingdom. Torbay hospital serves about 260 000 people in the winter plus 100 000 temporary residents in the summer. Most surgical patients are referred by primary care physicians ('GPs'). Local GPs have usually assessed risk and adequately treated cardiovascular disease. Medical history and ongoing management has usually been documented for all but emergency patients.

In this chapter I have concentrated on the assessment and management of patients before planned surgery. The assessment and management of unprepared patients – irregular,

inadequate or non-existent primary care – is slightly different, as is the preparation of emergency cases, but the principles are the same.

Good surgical outcomes depend upon efficient and effective communication in a well-organised perioperative system. You will help your patients more by developing a good working relationship with surgeons and other colleagues than you will with a stethoscope, echocardiogram, myocardial perfusion scan or exercise test. Please read the chapters on organising a preoperative service.

Deciding for or against surgery

Once a patient knows the likelihood for good and bad outcomes, both with and without surgery, he or she can decide what to do.

I would probably choose surgery if it would make my life longer and better. I would definitely decline surgery that would shorten and worsen my life. But surgical outcome, as with any other aspect of life, is uncertain. I have to make my decision based upon the 'average' duration and quality of life for people with my condition who have had surgery, compared to those that have not had surgery. This information is not available for most operations.

We should be embarrassed that we don't know whether most operations are good or not. We should also be embarrassed if we do not clearly explain the chances for good and bad outcomes (with and without surgery), and the uncertainty of these estimates. Few if any of us have been trained to understand and communicate risks. Yet the validity of consent depends upon understanding the arithmetic of risk and chance, expressed simply and accurately, using verbal, written and pictorial information.

I think that we would benefit the people we serve more by improving our understanding and explanation of chance than we would by increasing the accuracy of the prognostic indicators that we already have. Again, before you read this chapter, spend your time reading other texts and help your patients understand **risk**.

Assessing risk

The risk assessment described in most textbooks and articles does not provide patients with the information necessary to make an informed decision (whether or not to have surgery). The most significant information omitted is the risk of good and bad outcomes without surgery (see below). Information about risks following surgery is also usually inadequate. Risks are usually confined to the chance of bad outcomes in the 30 days following surgery. Beyond that, quoted risks usually include specific surgical events (for instance infection, dehiscence, recurrence) but omit other events, the risks of which surgery may still change (for instance death from any cause, clots in legs, heart or brain).

I will do my best to provide you with a template for risk assessment. This template is based upon a logical sequence of steps for risk calculation. I will use death (from any cause) to illustrate this sequence, but you could use this sequence to determine the risks for other specific outcomes (such as myocardial infarction, cardiac arrest, cardiac death, deep vein thromboses). I develop a risk estimate for an individual by starting with average risks for the population. I do this because population risks are the most certain. Each step in the sequence introduces more uncertainty. Most texts quote postoperative rates for cardiovascular death and morbidity in specific patient groups (for instance vascular surgery), but do not assess the risks of these events without **surgery**. Although these studies include a few thousand patients there are larger studies of people not having

surgery that provide more precise risk **estimates**. The last step in my sequence is the effect of a particular operation on a person's risk (of death). This step introduces the most uncertainty in the whole sequence. Once you have followed the steps and calculated the risk, you can compare it with the risk quoted by studies of surgical patients. If the two estimates are particularly different you should recalculate and try and determine whether your patient is particularly different to the patients used to calculate the risks in the surgical studies, and check that you are calculating the risk for the same outcome.

Calculating the risk of dying

The importance of fitness

To predict survival as best you can you need to measure fitness as best you can. The three variables that together predict survival in people without disease are: age, sex and **fitness**. These three variables also predict survival very well for people with systemic disease, although a few factors can sometimes improve prediction **precision**. If you assess fitness by history alone you will be unable to predict survival as well as you could if you measure fitness directly, no matter how many other variables you take into account. If you do not assess fitness at all, your risk estimates will be even less precise.

Step one: age and sex

In the United Kingdom all deaths are recorded and used to calculate age-specific and sex-specific survival. These figures are updated every three years. You can download Excel survival spreadsheets for men and **women**. Table 5.1 lists examples of mortality risk and median life expectancy for men and women. These data provide a precise but average risk of dying. **The risk of death doubles about every seven years.** For instance, the risk that

a 58 year-old woman will die in the next month is 1 in 2500, the risk that a 65 year-old woman will die is 1 in 1250. **The risk that a man will die is 1.7 times the risk that a woman will die.** Steps two onward determine how average your patient is.

Table 5.1 Examples of United Kingdom mortality risk by age and sex

Risk of dying per month	Chance of surviving	Risk of dying per month	Chance of surviving	Female Age (life expected)	Male Age (life expected)
1 in 10 000	9999 in 10000	1 in 10 000	9999 in 10000	42 (40)	35 (36)
2 in 10 000	9998 in 10000	1 in 5000	4999 in 5000	49 (33)	45 (30)
4 in 10 000	9996 in 10000	1 in 2500	2499 in 2500	58 (25)	53 (22)
8 in 10 000	9992 in 10000	1 in 1250	1249 in 1250	65 (19)	60 (17)
16 in 10 000	9984 in 10000	1 in 625	624 in 625	71 (15)	67 (12)
32 in 10 000	9968 in 10000	1 in 313	312 in 313	77 (11)	73 (9)
64 in 10 000	9936 in 10000	1 in 157	156 in 157	84 (7)	80 (6)
128 in 10 000	9872 in 10000	1 in 79	78 in 79	90 (4)	87 (5)
256 in 10 000	9744 in 10000	1 in 40	39 in 40	97 (3)	95 (3)

Step two: income

Poor people are twice as likely to die as rich people. The association with mortality is reasonably consistent using occupation, educational qualification or income as the measurement of **poverty**. Poverty and the risk factors I have listed below account for differences in all-cause mortality between ethnic groups. There is no need to adjust for the ethnicity of your patients. You can download the mortality figures for your region of the UK and this will account for some geographical variation in income. For patients from the lowest socioeconomic class in your area you could multiply the average mortality rate by

1.5, and for patients from the highest socioeconomic class multiply the average mortality rate by **0.7**.

Step three: smoking, diabetes, blood pressure and cholesterol

Smoking, diabetes, hypertension and hypercholesterolaemia increase the risk of death, mainly through atherosclerotic disease. I have simplified the relationship between these variables and death. There is no blood pressure or cholesterol 'threshold' at which the risk of death suddenly increases. The relationship is a gradual increase in risk with higher values, or a decrease in risk with lower values.

Smoking

About 1 in 4 adults smoke in the United Kingdom. Someone who smokes (or who has stopped in the last 5 years) is about **1.5** as likely to die as average. Someone who has not smoked in the last 5 years is about **0.8** times as likely to die as average. The risk of dying increases by **1.2** for every 10 pack-**years**. One pack-year is smoking 20 cigarettes each day for one year or ten cigarettes each day for two years (and so on).

Diabetes

About 1 in 20 adults in the UK have a diagnosis of diabetes, of whom 10% have type one diabetes and 90% have type two diabetes. Type one diabetes increases the risk of dying **3** times, type two diabetes increases the risk of dying **2** times. Non-diabetics have about an average risk of dying.

Hypertension

About 1 in 3 adults are either already treated for hypertension, or consistently have a systolic blood pressure (SBP) above 140 mmHg when it is measured in primary care. Adults treated for hypertension, or who have a SBP in primary care above 160 mmHg, are about **1.5** times as likely to die as average. Untreated adults with systolic blood pressures in primary care below 130 mmHg are about **0.7** times as likely to die as average. Adults with SBP between 130 mmHg and 160 mmHg have an average risk of dying.

Use the blood pressure recorded in primary care to guide your risk calculations. Do not use blood pressure measured in hospital to calculate risk. Blood pressures measured in hospital clinics are usually higher and do not correlate with outcome as well as those measured in primary care.

Hypercholesterolaemia

The average adult cholesterol level is about 5.5 mmol/L (220 mg/dL). Lipidaemic atherosclerotic risk is more accurately measured as the ratio of total cholesterol to high density cholesterol, the average of which is about 4.5. People with total cholesterol concentrations at least 8 mmol/L (350 mg/dL), or total:HDL ratios at least 7, are **1.25** times as likely to die as average. People with total cholesterol concentrations less than 4.1 mmol/L (150 mg/dL), or total:HDL ratios less than 3.1, are **0.8** times as likely to die as average. The risk of dying is about average for values in between these.

Lipidaemic atherosclerotic risk is calculated on more than one blood sample. So, as with blood pressures, prognosis should use serum lipid measurements taken in primary care.

Step four: heart, brain, kidneys and legs

People who have had transient ischaemia to heart (angina), brain (TIA) or legs (claudication), or tissue death (myocardial infarction, stroke, amputation), are more likely to die than people who have not. Mortality risk is particularly high immediately after tissue death, but for uncomplicated cases the risk falls to a stable level within a few weeks. These patients are also more likely to have the risk factors listed in steps two and three than people who do not have symptomatic atherosclerosis.

Histories of peripheral vascular disease (PVD), stroke, heart failure, myocardial infarction or renal failure (creatinine concentration more than 150 µmol/L) each independently increase the risk of dying by about **1.5**. Temporary ischaemia, instead of tissue infarction, in the heart (angina) or brain (TIAs) increases the risk of dying by **1.2** rather than 1.5. The elevated risk following infarction is maintained over decades, whilst the lesser risk from stable temporary ischaemia falls over time.

Step five: aerobic fitness

Fit people without systemic disease are more likely to survive than unfit people without systemic disease. Fit people with systemic disease are more likely to survive than unfit people with systemic disease. The risk factors listed in steps one through four – sex, age, socioeconomic state, smoking, diabetes, primary care blood pressure and cholesterol concentration – can often be assessed from existing medical records. Fitness cannot usually be reliably assessed from medical records. Traditional fitness assessment relies upon the maximum work (power) that someone says they can achieve, usually in terms of day-to-day activities, like walking or going upstairs. The approximate power required to achieve these activities is listed in tables as the number of METs (metabolic equivalents).

One MET is the internal power used at rest. The MET scale of daily activities was not intended to be a prognostic measure and imprecisely stratifies risk.

We need to oxidise food to provide power. The food metabolised to provide one MET is widely-reported to require 3.5 millilitres of oxygen every minute for every kilogram of body mass (3.5 ml O₂/kg per min). This is the value most MET scales have used to rate the power required to perform day-to-day activities. Unfortunately this value is incorrect. The correct average adult resting oxygen consumption is 2.6 ml O₂/kg per min, with 95% of adults consuming between 2.0 and 3.4 ml O₂/kg per min. Tests that directly measure oxygen consumption during exercise are usually reserved for assessing patients whose risks of dying may be high (for instance vascular surgery) and where resources are limited (for instance cardiac transplantation, critical care facilities). One can estimate oxygen consumption during graded exercise, rather than measure it directly. See 'using a simple incremental exercise test' for details.

Fitness testing replaces most of the prognostic information provided by step three and step four and improves the precision of risk estimation. However, the risk factors for atherosclerosis listed in steps three and four can help one decide whether to measure fitness using a simple test or a more complex test (see below). The cardiopulmonary exercise test is more complex than the incremental shuttle walk test. It provides the most precise prognostic information for patients with (or without) ischaemic heart disease and heart failure, but it is too labour-intensive a method to use in every preoperative patient.

Measuring fitness, part one: using a simple incremental exercise test

You can measure fitness by watching someone exercise. This provides more accurate prognostic information than relying on what people tell you they can do. But most simple

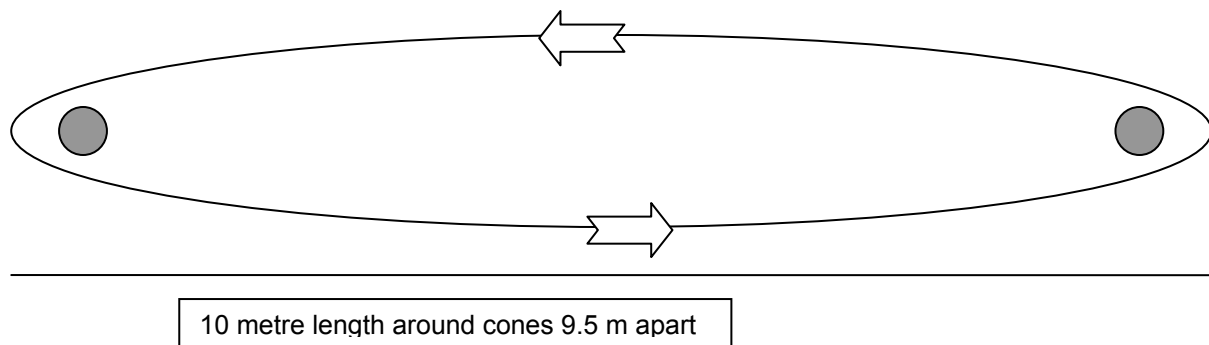
tests, such as the distance walked in 6 or 12 minutes, or the number of stair flights ascended, do not require a person to expend increasing power. These tests permit people to vary the power they work at in any pattern they wish. This means that these tests have limited ability to help you assess fitness and risk.

The most important feature for a fitness test is that it should demand increasing power for at least five minutes. This is because even unfit people can produce brief bursts of high power, fuelled without burning oxygen. Power maintained for at least five minutes depends upon oxygen, and it is the power fuelled by oxygen (aerobic fitness) that best predicts survival.

The 'incremental shuttle walk test' (ISWT) is one fitness test that requires someone to exercise at increasing power. A similar sporting test is the 'shuttle' or 'bleep' test that some of you may have done. In the ISWT you have to keep travelling to and fro 10 metres, around two cones 9.5 m apart (Figure 5.1). In the first minute of the original ISWT you have to complete three 10 m lengths, four lengths in the next minute and so on. In Table 5.2 I have illustrated the power levels that men and women of different ages should complete. I have introduced two additional low levels before the first of the levels that were originally described. These two (new) levels allow the elderly and infirm to start with one length in the first minute, two lengths in the second minute and so on. I have suggested starting levels for men and women of different ages who you think are of average fitness. I have assumed that people start to jog above a speed of 130 m/min when I calculated oxygen consumptions and METs: the relationship between speed and work is different for walking and running. From Table 5.2, a 60 year-old man of average fitness would start at new level five and would be expected to reach new level 17 (a black box). A 60 year-old man who continued to new level 24 (an unshaded box) would have **half** the average risk of

dying. A 60 year-old man with **double** the average risk of dying would stop at new level 11 (a box bracketed by two lines) having also started at new level five. A 60 year-old man with four times the average risk would not exceed new level 3 (another unshaded box). You may need to start patients at a lower level than I have indicated if you think they are particularly unfit, so that they walk at least five minutes before stopping.

Figure 5.1 Incremental Shuttle Walk Test course



The expected peak power (in old METs) for men is $18.4 - (0.16 \times \text{age})$, and for women is $14.7 - (0.13 \times \text{age})$. The risk of dying is greater for less fit people, so the average risk is multiplied by **1.19** for every old MET short of the expected. The risk of dying is less for people who are more fit, so for each MET a patient achieves beyond the expected you multiply the average risk by **0.84**.

Measuring fitness, part two: cardiopulmonary exercise testing

With the ISWT only the distance (or time) walked is recorded. This is used to estimate the peak power and oxygen consumption. How well someone's body copes with all the exercise they have done up to the time they stop is not measured. Therefore all this information that could improve prognostication is not used.

Cardiopulmonary exercise testing (CPX or CPET), unlike the ISWT, directly measures the aerobic capacity of someone whilst they exercise, usually either on a treadmill or bicycle. The CPX test measures the breath volume, oxygen and carbon dioxide contents of someone exercising. An electrocardiogram with ST segment analysis and pulse oximetry is recorded throughout, along with intermittent non-invasive blood pressures. Rarely arterial blood gases are sampled. Like the ISWT power increases throughout CPX until the person can no longer continue exercising. The incremental exercise should last at least five minutes, and preferably ten minutes, to accurately gauge prognosis.

The expected peak powers (and related peak oxygen consumptions) for men and women of different ages, are the same for CPX as for ISWT. Combinations of CPX variables that can further improve prognosis include: the amount of breathing needed to get oxygen in (\dot{V}_E/\dot{V}_{O_2}) and carbon dioxide out (\dot{V}_E/\dot{V}_{CO_2}); the amount of oxygen used to fuel increasing power (\dot{V}_{O_2}/W); and the threshold above which aerobic respiration alone cannot meet

metabolic demand – the ‘anaerobic threshold’. Heart rate during exercise and recovery also supply additional prognostic information. Changes in the ECG complex, for instance ST depression or elevation, do not usually add to the prognostic precision provided by CPX variables.

The prognostic information measured by CPX is largely provided by the peak oxygen consumption. This can be simply estimated by the ISWT. Patients with hip and knee osteoarthritis or claudication who find walking painful may prefer bicycle CPX to either the ISWT or treadmill CPX. The more complete prognostic information provided by CPX tests is probably of use if it:

- Helps clinicians determine whether surgery is more likely to harm or benefit a patient;
- Helps patients determine whether to have surgery;
- Helps determine the efficient use of scarce resources (critical care, transplants);
- Helps determine the likelihood that interventions could improve outcomes.

CPX testing is diagnostic as well as prognostic. Early ischaemic heart disease, heart failure and pulmonary vascular disease can be identified before typical symptoms or signs develop. CPX results can be used to inform exercise and drug prescriptions, with the intention of reducing both long-term and perioperative morbidity and mortality.

Methods other than CPX testing that could help to improve prognostication include dobutamine stress echocardiography and serum concentrations of brain natriuretic peptide (BNP) and n-terminal pro-B-type brain natriuretic peptide (NT-pro-BNP).

How to use steps one to five

If you use either the ISWT or CPX test to measure fitness do not use step three. Use step three if you cannot use step five because you have not yet organised preoperative fitness testing, or if a patient is unable to exercise.

Estimating survival without a fitness test

Step one: determine baseline risk with age and sex;

Step two: multiply baseline risk from step one by **1.5** for poorest, **0.7** for richest;

Step three: multiply risk from step two by diabetes (**3** for type one or **2** for type two), then smoking (**1.5**) or non-smoking (**0.8**), then treated hypertension or SBP more than 160 mmHg (**1.5**) or SBP less than 130 mmHg (**0.7**), then cholesterol high (**1.25**) or low (**0.8**);

Step four: multiply risk from step three by myocardial infarction (**1.5**), then heart failure (**1.5**), then stroke (**1.5**), then peripheral vascular disease (**1.5**), then creatinine concentration more than 150 µmol/L (**1.5**).

Estimating survival with a fitness test

Step one: determine baseline risk with age and sex;

Step two: multiply baseline risk from step one by **1.5** for poorest, **0.7** for richest;

[No step three]

Step four: multiply risk from step two by myocardial infarction (**1.5**), then heart failure (**1.5**), then stroke (**1.5**), then peripheral vascular disease (**1.5**), then creatinine concentration 150 µmol/L (**1.5**).

Step five: multiply risk from step four by the relative risk associated with the peak power achieved in the ISWT or CPX test.

Further adjustments to this risk estimate may be made using other results from a CPX test.

Readers interested in using CPX results should refer to specialist texts.

'What is the risk that a 54 year-old man will die in the next month, who smokes and is treated for hypertension and type II diabetes, and who had a heart attack four years ago?'

Step one: average risk of dying for a 54 year-old man is 1 in 2235.

Step two: estimate unchanged as no socioeconomic information.

Step three: estimate multiplied for type two diabetes (**2**), smoking (**1.5**) and hypertension (**1.5**) = $1 \times 2 \times 1.5 \times 1.5 = 4.5$ in 2235.

Step four: estimate multiplied for previous heart attack (**1.5**) = $4.5 \times 1.5 = 6.75$ in 2235.

In other words, his approximate risk of dying in the next month is 1 in 330, and his approximate chance of survival is 329 in 330.

'What is the risk that a 54 year-old woman will die in the next month, who smokes and is treated for hypertension and type II diabetes, who had a heart attack four years ago, and who can reach new level 10 in the ISWT'

Step one: average risk of dying for a 54 year-old woman is 1 in 3418.

Step two: estimate unchanged as no socioeconomic information.

Step three: no step three because she did a fitness test.

Step four: estimate multiplied for previous heart attack (**1.5**) = 1.5 in 3418.

Step five: estimate multiplied by relative fitness. Average peak power (in old METs) expected for a 54 year-old woman is $14.7 - (0.13 \times 54) = 7.7$ old METs. This is new level 15 (or old level 13) in the ISWT. She reached new level 10, or about 5.6 old METs. The difference is $7.7 - 5.6 = 2.1$ METs. The relative risk is $1.19 \times 2.1 = 2.5$. So the new estimate is $1.5 \times 2.5 = 3.75$ in 3418.

In other words, her approximate risk of dying in the next month is 1 in 910, and her approximate chance of survival is 909 in 910.

Step six: the effect of surgery

Estimation of risk associated with an operation is more uncertain than the risk estimations for steps one through five. Most people feel worse for some time after surgery than they did before surgery. Similarly most people are probably more likely to die in the month after elective surgery than if they had not had surgery. Elective operations are performed to prolong life, and make it better, but these effects can only be detected when patients are followed up for longer than one month. How much surgery extends life and improves its quality remains unknown for most operations. Although longer-term quality of life has been assessed in some postoperative patients, particularly after operations for cancer, randomized controlled trials that can determine benefit and harm are largely confined to surgery for coronary artery disease.

I will confine myself to estimating the effect of different surgeries on survival in the first postoperative month, though this is still based upon limited information. No one would choose to have surgery if they just considered available 30-day survival information, because elective surgery decreases someone's chance of being alive in 30 days.

The risk of dying is increased more by major surgery than by minor surgery. Major surgery includes operations on arteries and intra-abdominal and intrathoracic viscera.

Postoperative inflammatory responses increase oxygen demand, acid production and blood coagulability. This response peaks about two days after surgery and, in uncomplicated cases, gradually diminishes over subsequent days and weeks. The risk of dying parallels these changes, although there are additional factors that can increase the

postoperative risk of dying, for instance drug administration errors and hospital-acquired infections.

The risk of dying should be the same as being at home if the body's physiological state is unchanged by surgery, as long as miscellaneous risks from being hospitalized are minimal. Indeed, home is not a risk-free environment and it is easy to imagine someone whose risk of dying may be less having elective surgery than pursuing their normal activities. On average the risk of dying will be unchanged by minor surgery, such as cystoscopy. The risk of dying increases most, about **8** times, in the month following operations such as abdominal aortic aneurysm repair. Other major surgeries, such as anterior resections, increase the risk about **5** times. The risk following elective joint replacement increases about **3** times.

You calculate the absolute risk of dying in the month following surgery by multiplying the figure you have calculated (in the steps I have outlined above) by the relative risk associated with the proposed surgery. It is possible that this risk estimation could be made more accurate by factoring additional patient, anaesthetic or surgical factors, but it is unclear what factors to include and whether they increase or decrease the relative risk of surgery (see below).

Reducing the risk of dying

Reducing the relative operative mortality risk

Most research has tried to reduce the relative risk of a given operation by preoperative, intraoperative and postoperative monitoring, fluid and drugs. Although most research only measured survival up to one month following surgery, it seems reasonable to suppose that

the measured reductions in mortality would have persisted. Preoperative assessment of risk, as described above, can help to identify which patients are more or less likely to benefit from such perioperative interventions.

Reducing the non-operative mortality risk

Reducing the relative risk of operations with intensive methods of risk-reduction uses critical care resources that are scarce and expensive. So they will only be used for the highest-risk cases. There are more low-risk than high-risk surgical patients. Although their individual risks of dying are less, the total burden of postoperative low-risk death is greater than high-risk death. Although reducing the relative risks of major operations saves lives, mortality can be reduced more by making patients fitter before their operations.

Non-operative risk is less for non-smokers, although it may take months or years before the risk of dying falls after stopping smoking. Survival is prolonged by some drugs.

Survival is prolonged predominantly through a reduction in strokes and acute coronary syndromes, but kidney failure, heart failure and some cancers may also be made less likely. It is not clear how long drugs take to reduce the non-operative risk of dying. After myocardial infarction (or other acute coronary syndromes) the risk of dying is reduced within days of starting protective drugs. But drugs further reduce risk (compared to placebo) over the following weeks and months. The relationship between drug administration and risk reduction is probably similar for the primary prevention of cardiac or brain ischaemia. Drugs that prolong survival for both symptomatic and asymptomatic people include aspirin, 'statins' (HMG CoA reductase inhibitors) and angiotensin converting enzyme (ACE) inhibitors. Other drugs that decrease risk in one or other group include beta blockers, calcium channel antagonists, thiazide diuretics, other antihypertensives and spironolactone.

Preoperative services can help primary care practitioners ensure that patients are offered the most effective current drug combination. The risk of dying will be least for patients who have had the longest preoperative time getting fitter, not smoking and taking the best drugs.

Common problems

Hypertension

Hypertension, measured in primary care, is a common risk factor for stroke, heart disease and death. Other non-operative risks that increase mortality by similar amounts are: poverty; smoking; high cholesterol concentration; high creatinine concentration; history of heart attack, stroke or peripheral vascular disease. Being male, seven years older or one MET less fit each carry more risk than hypertension or the other factors I have listed.

Four, or possibly five, of these risk factors can be modified to reduce non-operative risk by similar amounts. I think that it is interesting that preoperative blood pressure has been concentrated on, often to the exclusion of the other risk factors. Blood pressure is the only variable that can be easily measured. But ironically blood pressure is the only variable that systematically overestimates risk when measured in hospital. I think that it is illogical to cancel operations on the basis of blood pressures measured in hospital, often without discussion with the patient or their consent. Rarely are operations cancelled because of smoking, cholesterol concentration or aerobic fitness.

The calculation of the risk incurred by hypertension and its management should be based upon blood pressures measured in primary care. If a patient does not want their blood pressure reduced to limit non-operative long-term risk there is little reason to insist that they have their blood pressure reduced for an operation. When a hypertensive patient has surgery their relative risks probably increase by an average amount. Because their long-term non-operative risks are already increased the absolute risk of death or other cardiovascular complication will be greater than for a patient who is not hypertensive.

Echocardiograms

Fitness, measured by either ISWT or CPX, predicts survival more accurately than does transthoracic echocardiography. Even dobutamine stress echocardiography, single photon emission computerised tomography or stress scintigraphy probably add little prognostic information to CPX results.

Patients with left ventricular outflow obstruction, such as severe aortic stenosis, can be anaesthetised for non-cardiac surgery. One might expect that risk could be estimated by fitness tests, because patients whose cardiac output is limited will reach lower power levels. It is unknown whether fitness tests adequately assess both non-operative and operative risks in patients with undiagnosed aortic stenosis. Because the answer is uncertain, and because tailored intraoperative management may reduce risk, it seems reasonable to use echocardiography to identify patients with moderate or severe aortic stenosis. I cannot guide readers on how useful different echocardiographic strategies are to measure and limit mortality risk. What follows is a strategy that has not been validated: your local policy will depend upon discussions with cardiologists and echocardiographers.

You could request an echocardiogram for a patient with an undiagnosed murmur who has one or more of the following:

- Left ventricular hypertrophy on a resting 12-lead electrocardiogram;
- Unable to achieve 4 old-METS or 5.5 new METs (peak oxygen consumption of 14 mls O₂/kg per min);
- Faints or angina;
- Planned major surgery (if older than 40 years);
- Planned hypotensive anaesthesia (if older than 40 years).

CABG, angioplasty and stents

Coronary artery bypass grafting (CABG) is a palliative procedure to relieve the pain of angina. The main indication for CABG is when a patient wants surgery to try and relieve angina that is inadequately controlled by drugs. Most of the evidence for CABG comes from 2649 patients recruited into seven randomised controlled trials between 1972 and 1984.

Ten years after randomization to CABG or medicine, surgery prolonged the average survival by four months, from 8 years and 5 months to 8 years and 9 months (difference unlikely to be less than 50 days). Hospitalization and subsequent recovery after CABG can take four months, so four months' survival difference is not a good reason to opt for surgery, unless it is accompanied by reduction in symptoms. Some people die after CABG who would have survived. Overall survival is worse in the first year after CABG than it is without. It is therefore not surprising that patients do not benefit from 'prophylactic' CABG before undergoing non-cardiac surgery.

Percutaneous coronary angioplasty and stenting, collectively known as 'percutaneous coronary intervention' (PCI), has also been used to treat angina since the CABG studies were published. PCI is less invasive than CABG, but relief from angina is less prolonged because endothelium grows through the stent mesh to occlude the coronary artery. Cytotoxic 'drug-eluting stents' (DES) inhibit endothelial overgrowth, but leave the thrombogenic stent in direct contact with blood. Mortality (due to clotting inside stents) is increased in the first three to six months after DES, despite the use of antiplatelet agents. The risks of stent thrombosis, myocardial infarction and death may remain increased beyond six months if antiplatelet drugs (particularly clopidogrel) are stopped. Long-term survival after PCI is not better than after CABG, and may be worse. There is a lot of research being published, so I have not tried to quantify the risks and benefits of PCI. The reader will have to establish current figures to help patients decide how long to delay elective surgery following PCI.

If a patient you assess for non-cardiac surgery is unhappy with their angina you could refer them to a cardiologist (for review of anti-anginal medication, PCI or CABG). There is little a cardiologist can do if a patient is already on best current medical treatment, and either does not want PCI or CABG, or whose angiography has already shown anatomy that is not amenable to either technique.

Most patients will not experience angina during preoperative exercise testing, including those with ischaemic heart disease and a history of angina. But if any of the following scenarios happen you could consider referral to a cardiologist. The aim is to identify patients more likely to die than the average with ischaemic heart disease. The seven trials I mentioned above found that CABG increased median survival by 9 months, from 7 years six months to 8 years 3 months (difference unlikely to be less than 3 months) in the third of

patients with higher risk, and by 19 months, from 6 years 9 months to 8 years 4 months (difference unlikely to be less than 5 months) in patients with left main stem coronary artery stenosis of at least 70%.

Current guidelines recommend angiography (after further exercise ECG test such as CPX):

a) If a patient doing an ISWT **stops** because of unexpected angina and:

- does not achieve 4 old METs;
- has at least 1 mm ST depression but does not achieve 6.5 old METs;
- has at least 2 mm ST depression but does not achieve 9 old METs;
- has at least 3 mm ST depression but does not achieve 13 old METs;
- has at least 4 mm ST depression but does not achieve 17 old METs.

b) If a patient doing an ISWT experiences angina but does not stop because of it, and has:

- at least 1 mm ST depression but does not achieve 5 old METs;
- at least 2 mm ST depression but does not achieve 7 old METs;
- at least 3 mm ST depression but does not achieve 10 old METs;
- at least 4 mm ST depression but does not achieve 13 old METs.

c) If a patient doing a CPX test does not experience angina, and has:

- at least 1 mm ST depression but does not achieve 2 old METs;
- at least 2 mm ST depression but does not achieve 5 old METs;
- at least 3 mm ST depression but does not achieve 8 old METs;
- at least 4 mm ST depression but does not achieve 11 old METs.

Again, if the patient has had coronary angiography that has shown that PCI and CABG would not be helpful, there is little to be gained by cardiological review.

Unprepared elective or scheduled patients

I have concentrated on risk assessment and disease management in patients presenting for elective or scheduled surgery who are under the care of a primary care physician. The principles that I have outlined also apply to patients who are either not registered with, or who have not attended, a primary care service. To assess non-operative risks you will need to spend more time either directly assessing risk or liaising with a primary care service. Unprepared patients are more likely to benefit from delaying surgery to allow risk factors to be assessed and reduced, but perhaps are also less likely to want to do so.

The principles of assessment and preparation of patients for emergency surgery are also the same. But risk reduction will depend upon reducing the relative risk of surgery as there will be no time to reduce non-operative risk. See chapters 16 and 20.

Conclusion

Preoperative services have a huge unrealized potential to reduce postoperative mortality and morbidity by promoting preoperative health and fitness. Both clinicians and patients should consider the preoperative period a training opportunity. It is not a passive wait. It is an active schedule of exercise for survival, both perioperative and long-term. In the context of a well-organised preoperative service, policies that solely focus on reducing the preoperative waiting time deprive patients of the opportunity to decrease perioperative risk, leaving them exposed to avoidable damage and death. I think that the option of delaying surgery to permit an improvement in fitness should be entertained for every patient having

elective or scheduled surgery. In some patients cancers will spread and aneurysms will rupture, whilst in other patients fitness will improve so that they survive to benefit from surgery that would otherwise have killed or maimed them. The unanswered question is “What type of training will produce how much risk reduction in which patients?”

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Internet Resources

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