CHAPTER 1

Preoperative Assessment of Cardiac Risk

Deepak K. Tempe

The cardiac disease continues to be a major problem all over the world. In the developing countries, valvular heart disease (VHD) of rheumatic origin continues to constitute a major cardiac health care problem. In addition, during the last two decades, an increasing trend in the number of patients suffering from coronary artery disease (CAD) has been observed. The anesthesiologist may be required to deal with such patients when they are subjected to either cardiac surgery, non-cardiac surgery or interventional procedures. The preoperative assessment of these patients carries significance, as it can be helpful in making recommendations concerning the cardiac risk in the perioperative period and providing a clinical risk profile that can be utilised for making the choice of an appropriate anaesthetic plan. In addition, the risk stratification may be useful for efficient assessment of new therapy and technology. Sometimes it may be necessary to alter the medical therapy to optimize the patient's condition before subjecting him/her to surgery.

AGE

With the improvement in medical care and consequent increase in the life expectancy of the population, an increasing number of patients from older age group will be presenting for surgical procedures. In this context, the traditional cut-off value of 65 years dividing the elderly from non-elderly patients may no longer be valid, especially in view of the improved general health of the population. The prevalence of cardiovascular disease increases with age, and age is considered a significant predictor of cardiac death following surgery.[1] It has also been shown that the response of the elderly heart to different forms of stress (exercise, catecholamine stimulation) is depressed.[2] It has been shown that for patients undergoing coronary artery bypass grafting (CABG), the operative mortality is 11.8 percent for patients more than 90 years of age, 7.1 percent for those 80 to 89 years and 2.8 percent for those 50 to 79 years.[3] However, as the overall physiological status of an elderly patient might be affected by other associated diseases, it is difficult to evaluate age as an independent predictor factor. Nevertheless, age is considered an important factor for predicting the risk that has been found to increase with an increasing age.

BODY SIZE

Obese patients generally carry a higher statistical risk of death.[4] In a retrospective analysis of 13637 consecutive patients undergoing cardiac surgery, it was shown that morbidly obese patients (body mass index, BMI >40) incurred nearly 60% greater observed mortality than normal weight patients. In addition, morbidly obese patients had greater than 2-fold increase in renal failure and 6.5-fold increase in deep sternal wound infection.[5] A significant association was found after risk adjustment between BMI and mortality and morbidity. In contrast, another study performed on a large database spanning a period of 11 years, demonstrated a lower mortality in overweight and obese patients relative to normal-weight patients and increased mortality in underweight individuals.[6] Thus, there appears to be a paradox in relation to obesity and mortality following cardiac surgery. It seems that existing knowledge gaps in the matter should be addressed by further research.

SEX

Female sex has been quoted as a risk factor for either morbidity or mortality.[7,8] Some studies, however, have examined both sex and body size in the same population and have shown that it is the small body size, and not sex, that increases the risk.[9,10] Without considering the body size, women do appear to be at a higher risk, as they commonly have smaller body surface area compared with men. In addition, a study has shown that the higher mortality rate that was observed in women after cardiac surgery was because of a higher baseline risk resulting from the presence of more concurrent risk factors.[11] A recent paper has reported that female sex was associated with an increase in long-term mortality after cardiac surgery.[12] Another retrospective analysis has concluded that female patients undergoing CABG are at a greater risk of in-hospital death and 30-day and 90-day readmission compared with men.[13] However, female sex is not a risk factor following coronary artery bypass grafting in patients more than 75 years of age.[14] Since, female sex is a component of most risk scores, risk evaluation, especially in elderly females should be performed with caution.

PREVIOUS MYOCARDIAL INFARCTION

As compared to the general population, the risk of developing perioperative myocardial infarction (MI) is increased considerably in patients with previous MI (older than 1 month). Such re-infarctions occur postoperatively and silently, making them difficult to detect. In addition, the mortality associated with re-infarctions is also much higher. Recent preoperative MI is an important predictor and the risk of infarction exceeds 30 percent within 3 months, and it is 15 percent at 3 to 6 months and approximately 6 percent after 6 months of infarction.[15] However, it has been reported that the mortality rates were improved considerably when the patients were subjected to aggressive monitoring and management during the intraoperative and postoperative periods.[16, 17] Although it is reasonable to believe that such monitoring and treatment modalities are likely to improve the outcome, the beneficial effects have not been confirmed.

In any case, the management of MI has been revolutionised in the recent times so that these recommendations have a limited role to play. For instance, the importance of intervening time interval between MI and surgery may not be relevant in the current era of thrombolytics and angioplasty. Risk stratification should be performed during convalescence.[18] The American College of Cardiology (ACC)/American Heart Association (AHA) guidelines[19] recommend coronary revascularisation in patients with stable angina having significant left main coronary artery stenosis, three-vessel disease, or two-vessel disease with significant proximal left anterior descending stenosis, high-risk unstable angina or non-ST segment elevation MI, or with acute ST-elevation MI. In patients in whom coronary revascularisation with percutaneous coronary intervention is appropriate, and non-cardiac surgery is required in subsequent 12 months, a strategy of balloon angioplasty or bare metal stent placement followed by 4 to 6 weeks of dual anti-platelet therapy is probably indicated. In patients who have received drug eluting coronary stents and who must undergo urgent surgical procedures, it is suggested to continue dual anti-platelet therapy unless the risk of bleeding is greater than the risk of stent thrombosis.[19] For details, the reader should refer to the guidelines and the focussed update.[19,20]

In 2009, the first European Society of Cardiology guidelines on perioperative care were developed.[21] Like the ACC/ AHA guidelines, the decision-making process integrates clinical markers, early coronary evaluation, functional capacity, and the type of surgery involved.

In the developing countries, however, the treatment of acute MI in the form of thrombolysis, angioplasty and surgery may not be available to all the patients so that the earlier recommendations regarding the intervening time interval between MI and surgery should not be considered invalid in them. Even the recent literature suggests that 60 days should elapse after MI before noncardiac surgery in the absence of a coronary intervention.[22]

ANGINA

Patients suffering from CAD can be identified by history of a classical angina. The pain is of strangulating nature, often occurring with exercise, emotional stress or during meals and is relieved with rest. Anterior chest pain is most common, but may also present as left arm, neck and right arm pain. Other rare sites are chin, forehead and epigastrium. Typical duration of anginal pain is from 5 to 15 minutes. It is important to understand different types of anginal pain as they have different prediction of risk.

Stable Angina

It is a substernal pain or discomfort that is precipitated by exercise, relieved by rest or nitroglycerin (NTG) or both in less than 15 minutes and is typically radiated to the shoulder, jaw or the inner aspect of the arm. It is a controversial predictor in non-cardiac surgical patients,[23] but the patient who develops dyspnoea on mild exertion is at a high-risk for developing perioperative ventricular dysfunction, myocardial ischaemia and possible MI. Such patients have a high probability of having extensive CAD, and additional monitoring or cardiovascular testing should be performed. A meta-analysis has shown that male sex, reduced ejection fraction, diabetes, prior MI and high C-reactive protein were the most powerful predictors of cardiovascular events in a patient with stable coronary disease.[24] These findings may help clinicians in identifying the most appropriate diagnostic and therapeutic approach for a patient with stable coronary artery disease presenting for non-cardiac surgery.

Angina can be frequently present in patients suffering from aortic stenosis (AS) in the absence of CAD. The reason for this is the increased left ventricular (LV) mass (concentric hypertrophy) as well as decreased coronary perfusion pressure. As AS commonly occurs in young patients in the developing countries (due to rheumatic fever), associated CAD is usually not present. Nevertheless, preoperative coronary angiogram may be performed in all patients with AS above 40 years of age to rule out associated CAD. The combination of AS and ischaemic heart disease (IHD) increases the perioperative risk.[25]

Unstable Angina

It may reflect cyclic coronary obstruction produced by an unstable thrombus associated with varying degrees of vasospasm and is defined as: (1) Newly developed angina occurring within the past 2 months; (2) Progressively worsening angina occurring with increased frequency, intensity or duration, being less responsive to medicine, and/or rest; or (3) Angina lasting longer than 30 minutes exhibiting transient unresponsiveness to standard therapeutic manoeuvres including NTG and rest, and which is associated with transient ST-T wave changes without development of Q waves or diagnostic elevation of enzymes. The presence of unstable angina has been associated with a high perioperative risk of MI.[26] Such patients should, therefore, be referred for further medical or coronary interventions before elective noncardiac surgery. Only vital or emergency surgical procedures should be considered for these patients as they carry a five times greater perioperative risk.[27]

Variant Angina

It was described in 1959 by Prinzmetal and coworkers.[28] It usually occurs at rest and is not associated with exercise or emotional stress. These patients have a high incidence of arrhythmia and conduction abnormalities. Although, the perioperative risk in these patients has not been studied, a review article has suggested that patients with pre-existing vasospastic angina are at much greater risk for perioperative attacks, and special precautions such as prophylactic vasodilator treatment are justified to reduce this risk.[29]

It should be remembered, however, that the characteristics of an anginal pain have no relation to the anatomical lesions in the coronary arteries in terms of number of vessels involved or the degree of narrowing.

SILENT MYOCARDIAL ISCHAEMIA

Silent myocardial ischaemia is now recognised as an important predictor of cardiac risk. It has been shown that as many as 75 percent of episodes of significant ST depression are not accompanied by angina and occur at significantly lower heart rates (HR) than symptomatic episodes.[30] The mechanism of silent ischaemia and infarction is unknown, but sensory neuropathy has been labelled as the causative factor, particularly in the diabetics. These events can be detected by ambulatory (Holter) electrocardiography performed during normal daily activities in normal population or patients with CAD. However, poor sensitivity and specificity make these tests poor screening tools.[31]

HYPERTENSION

IHD is commonly associated with hypertension. As a risk factor, hypertension is less today since the perioperative management of high blood pressure (BP) has improved. The degree of hypertension is more important and the risk of MI is increased in hypertensive patients, especially in the presence of hypercholesterolaemia, cigarette smoking and ECG abnormality. The significance of isolated systolic hypertension in surgical patients is not well established. Patients with untreated, poorly treated or labile preoperative hypertension are more likely to suffer perioperative BP lability, dysrhythmia, myocardial ischaemia and transient neurological complications, thereby increasing the perioperative cardiac morbidity. However, some authors have demonstrated that such is not the case and the importance of preoperative hypertension as a risk factor for postoperative morbidity is controversial.[23] Furthermore, there is a lack of consensus regarding treatment thresholds and the blood pressure targets. However, hypertensive patients with significant target organ involvement are at high risk of complications and should be treated adequately before surgery.

As per the ACC/AHA guidelines, uncontrolled hypertension is considered as a minor clinical predictor, i.e. it does not independently increase the perioperative risk.[19]

DIABETES MELLITUS

Diabetes mellitus is a very common systemic disorder associated with CAD and has many important implications. It is considered to be an independent risk factor for preoperative cardiac morbidity. Painless MIs occur more frequently and the infarct size as well as the mortality is reported to be higher in diabetics.[32] In addition, it can also lead to diabetic cardiomyopathy, which by itself can increase the cardiovascular morbidity.[33] According to the ACC/AHA guidelines, diabetes is considered a clinical risk factor.[19] Therefore, diabetes should be considered a potential predictor of perioperative cardiac morbidity.

CIGARETTE SMOKING

The adverse cardiovascular effects of cigarette smoking are well known and smokers are at an increased risk of MI.[34] Acute effects of smoking include increased coronary vascular resistance caused by direct vasoconstrictor effect of nicotine.[35] The myocardial oxygen consumption (MVO_2) may be increased by increasing the rate-pressure product. In addition, the decreased systemic oxygen transport caused by an increase in carboxyhaemoglobin levels can disturb the supply–demand balance. The major mechanisms of chronic cigarette use leading to cardiovascular disease include among others, oxidative injury, endothelial damage and dysfunction, enhanced thrombosis and chronic inflammation.[35] All these adverse cardiovascular effects, along with the well-known detrimental effects of smoking on the respiratory system may increase the risk for cardiac morbidity in these patients.

PREVIOUS CARDIAC SURGERY

The increasing number of cardiac operations performed everyday has led to more patients returning for reoperation. The technical difficulties arising out of adhesions between the heart and surrounding tissues are well known. It has been observed that the tendency to scar formation is more in black as compared to the white population, and therefore, they are likely to have dense adhesions. Bleeding from the adhesions can be profuse and lead to increased blood transfusion requirements. In addition, difficult dissection may require excessive handling of the heart leading to arrhythmias and reduced cardiac output.

In valve replacement surgeries, inadequate mobilisation of the LV apex may impair the deairing of cardiac chambers before release of the aortic cross clamp leading to a higher risk of air embolisation. In addition, these patients are on anticoagulants such as warfarin, and unless the medicine has been discontinued for an adequate period (3 to 7 days), the postoperative bleeding can be increased further.

In general, the patients undergoing reoperation are sicker and carry a higher risk of morbidity and mortality. Previous CABG in patients undergoing non-cardiac surgery has been shown to improve the outcome, and the post-operative incidence of MI in these patients is reported to be 0 to 1.2 percent and mortality 0.5 to 0.9 percent. (MI, 1.1 to 6 percent and mortality, 1 to 2.4 percent in patients without prior CABG).[23]

COMBINED PROCEDURES

Combined procedures such as CABG and valve replacement increase the operative mortality. Yadav *et al*[36] have examined the risk factors associated with early mortality in patients undergoing CABG and mitral valve replacement. Significant factors related to early death were, New York Heart Association (NYHA) functional class, urgency of surgery, valvular lesions secondary to an ischaemic event, increased pulmonary artery pressure (mean pulmonary artery pressure of >30 mm Hg), low ejection fraction (<40 percent) and low cardiac index. In another study, it was shown that mortality among patients undergoing mitral or aortic valve replacement along with CABG was higher as compared with those who underwent CABG alone. However, mitral valve surgery in combination with CABG was found to be an independent significant predictor for death, but aortic valve surgery in combination with CABG was not.[37]

RISK INDICES

A number of risk indices have been reported for quantifying the overall risk. These include the American Society of Anesthesiologists (ASA) classification,[38] the NYHA classification[39] (Table 1.1), and the Canadian Cardiovascular Society (CCS) classification of angina.[40] In addition, Goldman *et al*[41,42] were the first to develop a multivariate risk index using prospective analysis of a large group of patients. Factors such as preoperative cardiac failure and the recent MI that were found to be associated with a high-risk were given a high score. Other factors such as hypertension and smoking were not found to be associated with increased risk and were, therefore, not included as risks. These were later modified by Detsky and Colleagues.[43]

TABLE 1.1: New York Heart Association functional classification

Class I
Patients with cardiac disease but no limitation of physical activity. There is no undue fatigue, palpitation, dyspnoea or anginal pain on ordinary physical activity.
Class II
Patients with cardiac disease leading to slight limitation of physical activity. There is fatigue, palpitation, dyspnoea or anginal pain on ordinary physical activity, but they are comfortable at rest.
Class III
Patients with cardiac disease leading to marked limitation of physical activity. There is fatigue, palpitation, dyspnoea or angina! pain on less than ordinary physical activity, but they are comfortable at rest.
Class IV
Patients with cardiac disease leading to inability to carry out any physical activity without discomfort. Symptoms may be present even at rest. The discomfort is increased if any physical activity is undertaken.

Subsequent indices showed similar performance in predicting cardiovascular risk.[44] However, Lee's revised cardiac risk index (RCRI) performed better than previous indices and was also validated.[45,46] The clinical risk factors include high-risk surgery, IHD, history of congestive cardiac failure, history of cerebrovascular disease, insulin therapy for diabetes, and a preoperative serum creatinine of more than 177 μmol/L. It is now incorporated into the ACC/AHA perioperative algorithm.[19] Presence of 2 or more of these risk factors predicts elevated risk of major adverse cardiac events.

All these risk indices are useful in quantifying not only the preoperative cardiac status of the patient but also in estimation of the risk to which the patient is exposed. However, it should be remembered that the accuracy of these indices is controversial and not consistent. It has been shown that major risk stratification models do not predict perioperative outcome after CABG in patients with previous percutaneous intervention.[47] Patients with previous elective percutaneous coronary interventions had increased perioperative mortality and higher rates of major adverse cardiac events as compared with patients without prior percutaneous coronary interventions.[46] It is suggested that the risk assignment should occur throughout the perioperative period (preoperative, intraoperative and postoperative) and the risk factors chosen for model inclusion should vary depending on when the assignment occurs.[48]

Functional status is considered a reliable predictor of perioperative and long-term cardiac events. Patients with reduced functional capacity are at an increased risk of complications as compared to those who have good functional capacity. Functional capacity is expressed as metabolic equivalent of task (MET), where 1 MET is the resting oxygen consumption of a 40-year-old, 70 kg man. A functional capacity of <4 MET is considered as poor and >10 is considered excellent, 7–10 good and 4–6 moderate. Evaluation of the patient's functional capacity can be estimated by the Duke Activity Status Index questionnaire,[49] which can then be converted to MET. A score is given to each of the activities that the patient can perform and constitutes a self-reported functional capacity. A multicentre trial has shown that a Duke activity status index score of 34 represents a threshold for identifying patients at risk of myocardial injury, myocardial infarction, moderate-to-severe complications, and new disability.[50]

A cardiac risk calculator has been developed by the American College of Surgeons National surgical quality improvement program myocardial infarction cardiac arrest (NSQIP-MICA) that allows more precise calculation of surgical risk.[51] An App-based or online performa (www.surgicalriskcalculator.com) related to patient details can be filled up to obtain a precise perioperative cardiac risk.

Some risk stratification methods more specific to cardiac surgery have also been described. One of the earliest reported methods identified poor ejection fraction (<30 percent), unstable angina or recent MI, clinical evidence of heart failure, age greater than 65 years, severe obesity, emergency surgery, reoperation and other uncontrolled systemic disturbances as risk factors.[9] Patients are assigned to three risk levels: Normal (no risk factor, mortality 0.4 percent), increased (one of the above factors present, mortality 3.1 percent) or high (two or more factors present, mortality 12.2 percent).

The Society of Thoracic Surgeons has developed a model for risk stratification based on more than 80,000 patients undergoing CABG.[52] Thirteen risk factors were identified among which, cardiogenic shock, renal failure and reoperation were labelled as those carrying significant risk.

CARDIAC STATUS

The most important factor for predicting the outcome after surgery is the severity of cardiac disease. The clinician is constantly striving to accurately estimate the severity of disease by history, physical examination and performing several routine as well as specialised tests.

Congestive Heart Failure

Impairment of LV function can lead to an elevation of the pulmonary capillary pressure leading to the classical signs of dyspnoea. Occurrence of dyspnoea and its grading (NYHA classification) can be utilised for estimating the severity of LV dysfunction. However, it has been shown that physical signs may be absent in the presence of marked to severe elevation of LV filling pressure in chronic heart failure.[53]

The most frequent causes include hypertension, IHD, VHD and various cardiomyopathies. In VHD, the breathlessness is not related to LV dysfunction and in fact, may occur in the presence of normal or even supranormal LV function. For instance, in mitral stenosis (MS), the breathlessness is related to the degree of obstruction and the resulting increases in flow gradient across the mitral valve, and in AS it is related to the increased LV end-diastolic pressure (LVEDP) due to concentrically hypertrophied LV that has a poor compliance. Presence of dyspnoea, therefore, does not usually signify LV dysfunction in patients with VHD, unless the disease is in an advanced stage. The possibility of associated VHD in patients with IHD should also be considered, if dyspnoea is present. In fact VHD is an additional risk for a patient undergoing simultaneous CABG and valve replacement, especially for mitral regurgitation (MR) or AS.[37] The severity of symptoms is also related to the rapidity of onset of cardiac failure. Acute insults such as MI, may lead to severe symptoms even though only a small portion of the myocardium is damaged. In conditions that develop slowly such as aortic regurgitation (AR), AS, etc. there is time for compensatory mechanisms to develop and significant portions of the heart muscle may be impaired before symptoms appear.

There is an increasing trend in the number of elderly population undergoing surgery, and the prevalence of heart failure in the general population. Simultaneously, there have been improvements in the perioperative care and care of chronic heart failure. However, despite advances, patients with congestive heart failure (CHF) undergoing common surgical procedures have a substantially higher risk of operative mortality and hospital readmission than other patients.[54]

In patients with CAD, a history of CHF has been shown to be an important risk factor for poor short-term and long-term outcomes after CABG regardless of preoperative ejection fraction.[55] Preoperative CHF is identified as a risk factor and the presence of third heart sound and jugular venous distention are labelled as significant signs having prognostic value. Echocardiography should be performed in patients with CHF, results of which can suggest strategies for preoperative optimisation of cardiovascular status.

Arrhythmias

These are usually benign in healthy patients, but their occurrence in patients with CAD signifies serious nature of CAD and ventricular dysfunction. Many patients with acute MI still die before reaching the hospital, presumably due to dysrhythmias, and complex dysrhythmias are one of the important factors that influence the prognosis in them.[56]

Frequent premature ventricular contractions increase the risk in patients with chronic IHD. Although, few data are available regarding preoperative arrhythmias as a risk factor, frequent premature atrial contractions, rhythms other than normal sinus or atrial fibrillation (AF) appear to be risk factors.[42] In patients undergoing cardiac surgery, preoperative atrial fibrillation has been shown to significantly increase the mortality, patient's mechanical ventilation needs, infection, acute kidney injury and ICU and hospital length of stay.[57] Likewise, new onset perioperative arrhythmias have been shown to be associated with long-term cardiovascular events in patients undergoing vascular surgery.[58]

ECG ABNORMALITIES

ECG abnormalities excluding arrhythmias are considered as preoperative risk factors. ST-T wave changes and signs of LV hypertrophy are the commonest ECG abnormalities detected in patients with CAD. The predictive value of preoperative ECG is controversial. Some studies have shown prognostic value of resting ECG in terms of cardiovascular mortality,[59] while others failed to show any effect on postoperative outcomes.[60,61] The American Association of Family Physicians recommend that the decision to order preoperative tests should be guided by the patient's clinical history, comorbidities and physical examination findings. ECG is recommended for patients undergoing high-risk surgery and those undergoing intermediate-risk surgery who have additional risk factors. Patients undergoing low-risk surgery do not require ECG.[62] The ACC/AHA guidelines recommend that routine preoperative resting 12 lead ECG is not useful for asymptomatic patients undergoing low-risk surgical procedures. It is reasonable for patients with known CAD, significant arrhythmia, peripheral arterial disease, cerebrovascular disease, or other significant structural heart disease, except for those undergoing low risk surgery.[19]

PRIORITY OF CARE

The risk of operation may be influenced by emergency versus elective surgery.[9–10] The patient undergoing emergency surgery may have hypovolaemia, systemic vasoconstriction, acidosis or other physiological derangements. There may not be time for the usual careful preparation of the patient. The actual condition of the patient before operation is more important, as an emergency might mean a patient receiving cardiopulmonary resuscitation or may be undergoing a semi-elective or urgent procedure for left main coronary artery disease or mitral stenosis plus left atrial (LA) thrombus. With more experience and improvements in overall technique, the risk in patients undergoing emergency procedures is decreasing. In a recent study comprising of 100 patients undergoing CABG, it was shown that urgent CABG increased the risk of MI and prolonged hospital stay in comparison to elective CABG, but the mortality rate was similar.[63]

PREOPERATIVE CARDIAC TESTS

As a part of assessment of the cardiovascular disease, several tests are performed prior to anaesthesia and surgery. The anaesthesiologist may have to interpret and sometimes request these tests prior to anaesthesia. It is, therefore, necessary to have knowledge of the tests and be aware of their role in the preoperative assessment of the patient with cardiac disease.

Laboratory Tests

The epidemiological studies suggest that lower blood cholesterol is associated with a reduced risk of CAD and that there is a continuous positive relationship between risk of CAD and cholesterol levels.[64] Increased levels of low density lipoproteins are strong predictors of CAD and an inverse association has been demonstrated between high density lipoprotein (HDL) cholesterol level and CAD incidence. The incidence of CAD has been shown to be eightfold higher in men and women with HDL cholesterol of 35 mg/dL or less as compared with men and women with HDL cholesterol of 65 mg/dL or greater.[65] Thus HDL cholesterol seems to provide protection from CAD. Triglycerides, however, are relatively unimportant for prediction of CAD. The perioperative risk associated with hypercholesterolaemia is not known.

The most important laboratory changes associated with myocardial ischaemia and infarction are cardiac enzyme elevations. The levels of creatine kinase (CK), glutamic oxaloacetic transferase (GOT) and lactate dehydrogenase (LDH) are elevated. The isoenzyme of CK (MB) that is identified by electrophoresis reflects extraction from cardiac muscle. The CK MB level is a fairly accurate method of diagnosing acute MI and its serial measurements can be useful for predicting the infarct size.[66] GOT and LDH on the contrary are not specific enzymes and false positive readings can occur in patients with other non-cardiac diseases.

In general, the specificity and sensitivity of CK MB for MI are high, however, some drawbacks remain. For example, skeletal muscle content of CK MB (which is normally low) may be increased in patients with acute skeletal muscle injury (rigorous exercise) or chronic myopathies. Consequently, skeletal muscle injury occurring during surgery may increase postoperative concentration of CK MB, thus decreasing CK MB specificity for MI. In addition, the sensitivity of CK MB is limited to a short period as the increase persists for at most, 72 hours. Therefore, early and frequent measurements are necessary.

In this regard, cardiac troponins are relatively new markers that may prove to be more beneficial. With acute MI, plasma troponin concentrations (both troponin T and I) increase rapidly within 3 to 5 hours and continue to remain so for 5 days or more.[67] This allows for earlier detection of MI, as well as late diagnosis. Cardiac troponin I also offers greater specificity in patients with skeletal muscle disease or injury.[68] It has also been suggested that cardiac troponins may provide an insight into the preoperative risk stratification in patients with unstable angina undergoing CABG.[69] A recent paper has demonstrated that positive troponin was associated with more significant comorbid conditions and more extensive CAD. Furthermore, operative mortality, major morbidity and long-term mortality were increased in the positive troponin group. However, after risk adjustment, positive troponin was not independently associated with increased mortality.[70]

B-type natriuretic peptides (BNP) have been shown to be independent predictors of cardiovascular events in noncardiac and vascular surgery. It has been shown that preoperative BNP levels can be used to independently predict cardiovascular events in the first 30 days after vascular surgery and to significantly improve the predictive performance of the revised cardiac risk index.[71] A systematic review and meta-analysis has revealed that preoperative BNP concentrations were associated with mortality after cardiac and noncardiac surgery. However, the positive predictive value for mortality was weaker as compared with the negative predictive value. This implies that although BNP risk threshold would exclude patient who are likely to die after surgery, having a BNP value above the risk threshold may or may not mean that the patient has a higher risk of mortality.[72] Nevertheless, since BNP is a biomarker of subclinical and clinical heart failure, preoperative BNP is likely to be associated with postoperative morbidity and mortality related to cardiac failure.

In the developing countries, rheumatic fever is the most common cause of VHD. Repeated attacks of rheumatic fever are not unknown in these patients. Thus, patients subjected to valvular surgery should be investigated for evidence of endocarditis. The erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels are almost always elevated during acute stages of the disease. The surgical outcome in the presence of endocarditis may be compromised due to cardiac failure or increased risk of bleeding due to adhesions and friability of the tissues. Therefore, surgery should be delayed and endocarditis treated with aspirin and steroids, unless the valvular lesion warrants urgent surgery.

The Chest Radiograph

The routine postero-anterior (PA) and lateral radiographs of chest continue to be used extensively as a preoperative screening test. It provides useful information in both patients with CAD and VHD. Figure 1.1 shows the typical PA projection of the heart. The two boundaries of the heart (right and left) are in proximity with the respective lung fields. The right border of the heart is formed by the superior vena cava (SVC) and the right atrium (RA). The left border is formed by the aorta, the main pulmonary artery (PA), the LA appendage and the anterolateral border of the LV. RA enlargement can be detected by broadening of the right heart contour. The LA enlargement leads to displacement of the LA appendage laterally and the left bronchus upwards. In massive LA enlargements that can occur in some patients with mitral valve disease, the right border of the LA may overlap the right heart border giving an appearance of a double density (Fig. 1.2). Right ventricular (RV) enlargement is difficult to detect in the PA view except in conditions such as tetralogy of Fallot (TOF) where the left border may be formed by the RV with LV rotated posteriorly. On the contrary, LV enlargement can be easily detected using the PA view. In regurgitant lesions of the aortic and

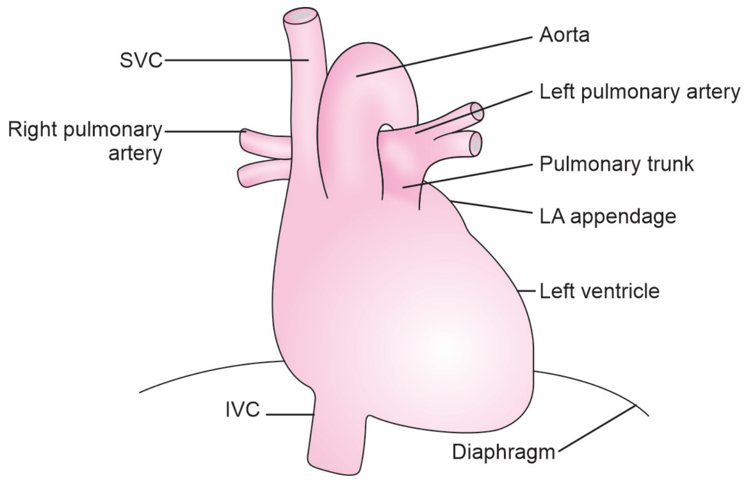

Fig. 1.1A: Diagrammatic representation of the frontal projection of heart. (SVC: superior vena-cava, IVC: Inferior vena-cava, LA: Left atrium).

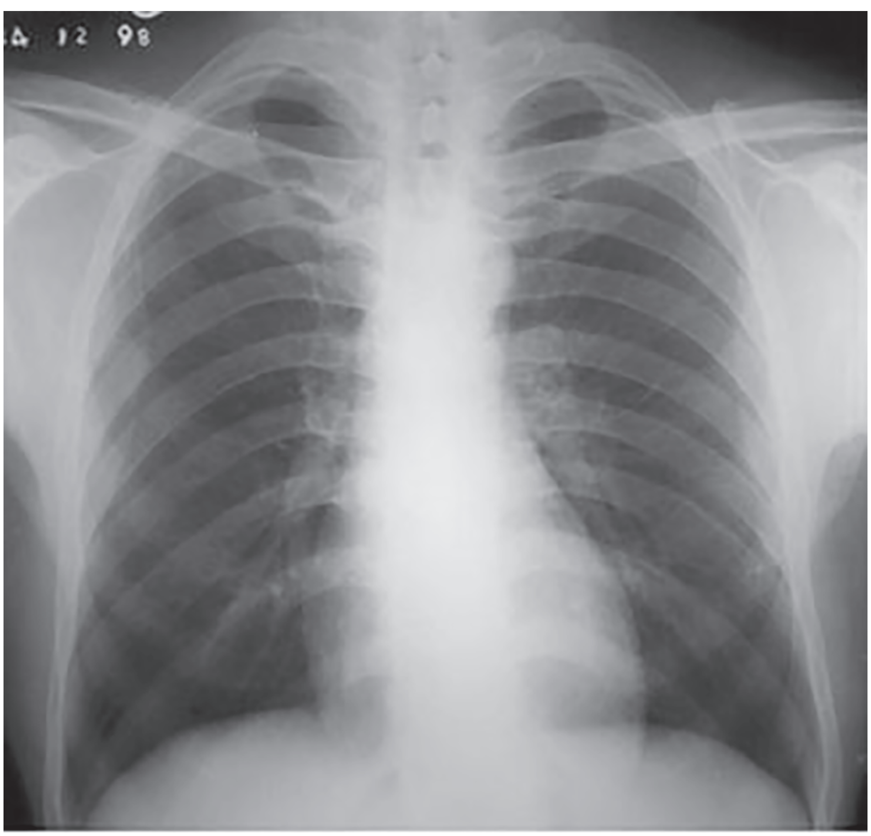

Fig. 1.1B: Normal X-ray of chest, postero-anterior view. The right border of heart is formed by the superior vena-cava and right atrium. The left border is formed by the aorta, main pulmonary artery, left atrial appendage, and anterolateral border of the left ventricle.

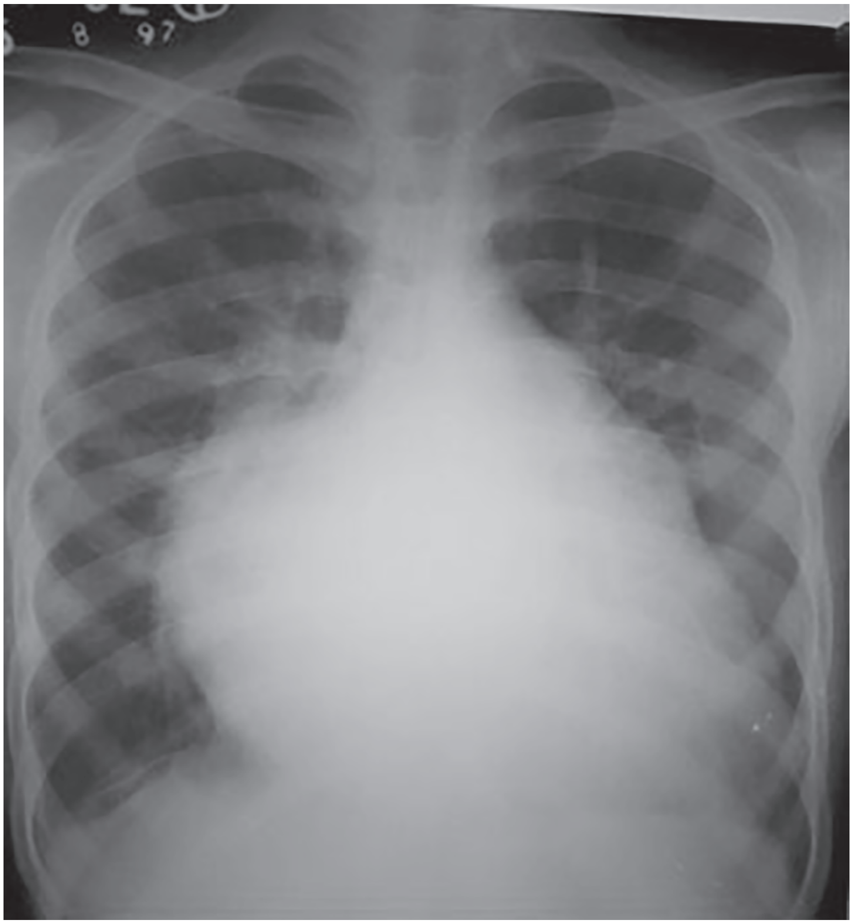

Fig. 1.2: Massively enlarged left atrium producing double density of the right heart border.

mitral valve, the long-axis is elongated with downward and leftward displacement of the apex. In patients with CAD, both long- and short-axis enlargement leads to globular shaped heart. The cardiac valves are not normally visualised unless they are heavily calcified.

The lateral view of chest is most useful in detecting the RV enlargement, which is indicated by obliteration of the retrosternal space along the upper two-thirds of sternum (Fig. 1.3). It can also be useful in patients undergoing reoperation to know if the RV is adherent to the sternum.

Radiographic Changes in Valvular Diseases

Individual valvular diseases can be differentiated with ease based on the radiographic presentation. In MS, LV size is normal, but LA, PA and RV enlargements are present. In MR, both LA and LV enlargement occur that can further lead to pulmonary hypertension and RV enlargement. In AR, there is dilatation of the LV along with dilatation of the ascending aorta. The concentric hypertrophy of the LV in AS is not perceived radiographically. However, with advanced AS, LV dilatation and cardiomegaly occur.

In patients with CAD, the chest radiograph provides useful information in the sense that cardiomegaly has been shown to be a specific predictor of low ejection fraction (<0.40).[73]

In patients with cardiomyopathy, the degree of LV enlargement depends on whether the cardiomyopathy is dilational (moderate to marked LV enlargement), restrictive (mild LV

Fig. 1.3: The lateral view of chest X-ray showing right ventricular enlargement causing obliteration of the retrosternal space.

enlargement), or hypertrophic (mild to moderate LV enlargement).

In addition to the size of cardiac chambers, the chest radiograph provides useful information by way of lung fields. The signs of CHF and pulmonary venous hypertension are useful in determining the cardiac status of the patient.

Chest radiography, thus provides a useful noninvasive method of estimating the cardiac function. However, it should be remembered that it provides an indirect assessment and it is useful to correlate the radiological findings with other clinical findings in order to arrive at definite conclusions.

Electrocardiogram

The ECG is one of the most important preoperative tests for patients with cardiac disease, especially for patients with CAD. However, the resting ECG can be normal in large number of patients with CAD and in some, the ECG may be difficult to interpret because of conditions such as left bundle branch block or Wolff-Parkinson-White syndrome. Also, ECG provides no information about the ventricular function and may be normal in the presence of severe CAD or VHD. ECG is typically utilised for detection of arrhythmias, conduction changes, myocardial ischaemia, injury and infarction. In patients with CAD, the occurrence of ST-T wave abnormalities may correlate with the severity of underlying heart disease, including the number of vessels involved and the presence of LV dysfunction.[74] In contrast, a normal resting ECG in patients with suspected or definite CAD is suggestive of a more favourable long-term prognostic sign.[75]

The earliest ECG change following ischaemia is usually the ST elevation. Later on, there is diminution in the size of R wave and in transmural (full thickness) infarction, a Q wave begins to develop. Subsequently, the T wave becomes inverted, which persists after the ST segment has returned to normal. The Q wave appears within hours and the T wave inversion takes place within days. The T wave inversion becomes less marked in several weeks or months.

In contrast to transmural infarct, subendocardial infarction causes ST/T wave changes without Q wave or prominent ST elevation. This is accompanied by the loss of R wave in the leads facing the infarct.

The ECG changes are best seen in the leads which face the infarcted area. For instance, in antero-septal infarction, abnormalities are found in one or more leads from V_1 to V_4, while antero-lateral infarction produces changes in leads V_1 to V_6 , AVL and I. Inferior infarction is best seen in leads II, III and AVF. Infarction of the posterior wall of LV is not recorded in the standard leads by ST elevation or Q waves. The leads V_1 to V_4 may record reciprocal changes by way of ST depression and a tall R wave.

Exercise Stress Testing (Treadmill)

With exercise, the major determinants of MVO_2 such as HR and myocardial contractility are increased. The increased oxygen demand is met primarily by an increase in the coronary blood flow that is achieved by marked vasodilatation of the coronary vascular bed. Any impairment in this vascular reserve due to coronary obstruction or vasospasm may lead to myocardial ischaemia and its sequelae. In exercise stress testing, the myocardial work is progressively increased by graded physical exercise and the signs and symptoms of ischaemia, arrhythmias and pump dysfunction are simultaneously measured.

Currently, many treadmill protocols are available. They include those introduced by Bruce, Balke, Ellestad, Astand, Naughton and Sheffield. The most familiar of these, the Bruce protocol consists of 3-minute stages that have different grade and speed. Stage 1 has a speed of 1.7 miles/hour with a 10 percent grade. Patients with moderate CAD usually exercise to stages 3 and 4 before termination of the test because of symptoms or HR limitations. The principal indicator of ischaemia during exercise as well as immediate recovery period is ST segment deviation. Three types of ST segment responses have been described.[76] The first type is characterised by ST depression occurring during the exercise period that reverts to normal during the early post-exercise period. In the second type, the ST depression worsens during the recovery period and indicates poor prognosis. The third type of response is ST segment elevation. The conventionally accepted criterion is a threshold elevation of 1.5 mm or more, regardless of the slope. In addition to ST segment response, changes in T wave or R wave, the occurrence of chest pain, alteration in HR, hypotension or arrhythmias are also considered.

Exercise stress testing is a noninvasive test that is useful in patients with chest pain of unknown aetiology and for quantification and prognosis in patients with known CAD. It has a significant prognostic value when the ST changes of significant magnitude occur during early stages of the test (1 to 3), do not revert during recovery period, are associated with subnormal increases in HR or BP and are accompanied by angina or arrhythmias. It should be remembered, however, that negative tests do not imply absence of disease as many patients undergoing CABG have negative exercise stress testing results. Apart from the fact that the results of exercise stress test are valuable in offering the diagnosis of CAD, the anaesthesiologist should take note of the haemodynamic changes that are associated with ST segment deviation. In addition, the therapies used during exercise stress testing to reverse ischaemia should also be noted. These may help to set the guidelines regarding the dangerous HR and BP response during anaesthesia as well as the therapy that should be used to reverse intraoperative ischaemia. A few studies[77,78] have demonstrated that a positive ischaemic response and a low exercise capacity predict adverse outcome following non-cardiac surgery. The ACC/AHA guidelines recommend that routine screening with noninvasive stress testing is not useful for patients at low-risk for noncardiac surgery and it is reasonable to forgo it in patients with elevated risk and excellent (>10 MET) functional capacity.[19]

Pharmacological stress testing with dobutamine stress echo can be used in patients who cannot perform exercise. Although randomised controlled trials on the use of stress testing are lacking, several meta-analysis have shown the clinical utility of pharmacological stress testing in the preoperative evaluation of patients undergoing noncardiac surgery.[19]

The 6 min. walk test (6 MWT) has been shown to be a useful clinical tool to screen and risk stratify patients in departments where cardiopulmonary exercise testing (CPET) is not available. It has been shown that patients walking more than 563 meters in the 6 MWT do not routinely require CPET, those walking less than 427 meters should be referred for further evaluation, and in those walking more than 427 meters but less than 563 meters, the number of clinical risk factors and magnitude of surgery should be incorporated into decision-making process.[79]

Echocardiography

The echocardiography is based on the principle of detection of reflected sound waves from the surfaces of the internal organs. Pulses of ultrasound waves with a frequency of 2.5 to 7.5 million cycles per second (MHz) are utilised in echocardiography. (Precordial echo: 2 to 3 MHz and transoesophageal echo (TOE): 3.5 to 5 MHz.) Frequencies in this range provide the optimum penetration (10–25 cm) and resolution of objects (objects 1 mm or less in size). When the ultrasound waves strike an interface of tissues of differing densities, a portion is reflected. The amount reflected is directly related to the difference in tissue densities. For instance, air in the LV appears as a brighter signal on the screen as it reflects a much greater portion of the transmitted ultrasound than the blood. The duration taken by the sound waves to bounce back to the transducer determines the location of the tissue. The longer a sound wave takes to bounce back, the greater is its distance from the transducer.

Echocardiography has undoubtedly become an important means of reliably and non-invasively assessing the cardiac function. The valvular function as well as the myocardial function is accurately assessed by this technique. The Doppler technique allows the detection of intracardiac shunts and valvular insufficiency and can estimate cardiac output (CO), valve gradients and intracardiac pressures.

M-mode Echocardiography

A typical M-mode echocardiogram is shown in Fig. 1.4. The tracing that is obtained by placing the transducer on chest wall represents the one-dimensional view against time. The motion of valves, and linear dimensions of intracardiac and pericardial structures can be visualised. These views are useful in estimating the end-diastolic and end-systolic LV dimensions and calculation of ejection fraction. The LV dimensions can be useful in quantifying the LV enlargement. It has now been suggested that patients with severe MR with LV end-systolic diameter greater than 40 mm and patients with severe AR with LV end-systolic diameter greater than 50 mm have already developed LV systolic dysfunction. Therefore, such patients are likely to be associated with adverse surgical outcome.[80,81]

Two-dimensional Echocardiography

Multiple views can be obtained by using phased array transducers that can be collated into a two-dimensional image. The two-dimensional picture helps to recognize anatomical and pathological landmarks. The operator can alter the angle and position of the ultrasound beam so that multiple cross-sectional images can be produced, helping to identify the anatomy of the heart and great vessels. Moreover, images are displayed in "real-time" on a monitor screen and can be recorded on a digital format. Two-dimensional echo enables the visualisation of valves and ventricular wall motion along with measurement of LV volumes (ejection fraction), areas and wall thickness. Figure 1.5 shows a typical 2-D echo using parasternal long-axis view. The other common views are parasternal short-axis, apical (two-chamber and four-

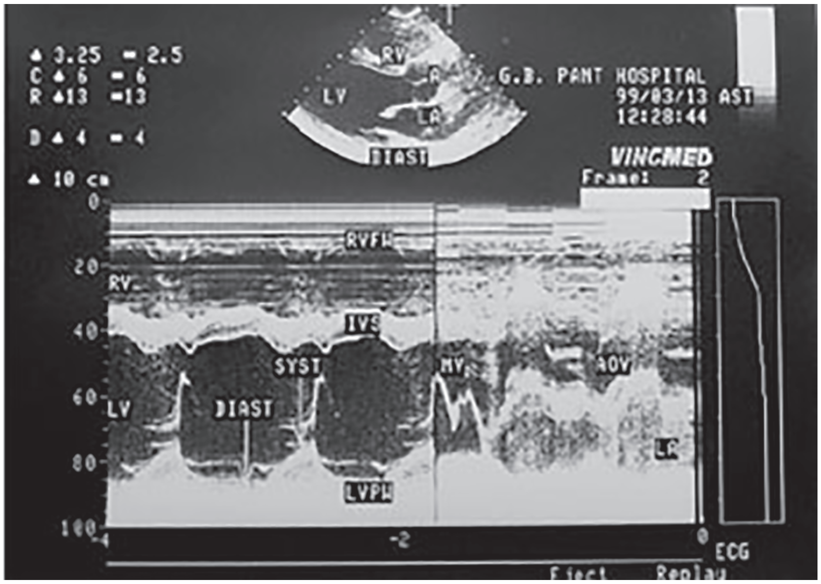

Fig. 1.4: A typical M-mode echocardiogram (LA: left atrium, LV: left ventricle, RV: right ventricle, A: aorta, RVFW: right ventricular free wall, IVS: interventricular septum, LVPW: left ventricular posterior wall, MV: mitral valve, AOV: aortic valve).

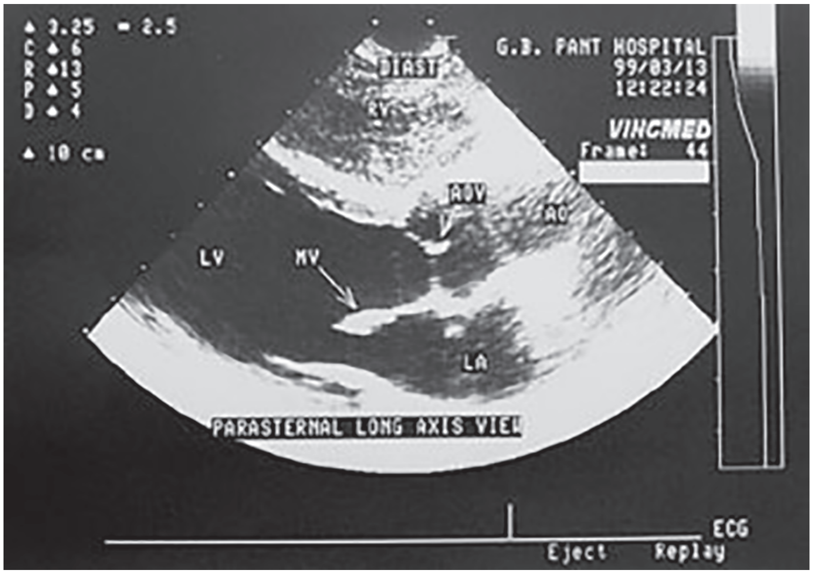

Fig. 1.5: A typical 2-D echocardiogram showing parasternal long-axis view (RV: right ventricle, LV: left ventricle, MV: mitral valve, LA: left atrium, AOV: aortic valve, AO: aorta).

chamber), suprasternal long-axis and short-axis and subcostal views.

Transoesophageal Echocardiography

In this technique the transducer is placed at the distal end of a gastroscope that is passed into the oesophagus. As the transducer is close to the heart and the sound waves do not have to penetrate the chest wall or lung, very high quality images are obtained.

The simplest TOE probe has one phased array transducer. The ultrasound beam is oriented at right angles to the gastroscope to produce transverse imaging planes. In biplane transducers, a second transducer is mounted immediately proximal and at right angles to the first to provide a longitudinal imaging plane. In multiplane transducers, a single transducer is mounted on a rotating device that allows 0 to 180 degrees rotation of the transducer on its own axis. The whole assembly is housed within the tip of the gastroscope. The advances in technology have helped the manufacturers to produce transducers that are small enough for use in infants and neonates.

The advances in Doppler technique have allowed for noninvasive estimation of morphology as well as function of valves, intracardiac shunts, intracardiac pressures and CO. As a preoperative test, precordial echocardiography provides a noninvasive and accurate method for estimating the overall cardiac performance. Of importance, is its value in the diagnosis of acute MI [presence of regional wall motion abnormalities, (RWMA)] when other technique such as ECG is inconclusive or uninterpretable. In VHD, it provides invaluable information in terms of valve size, degree of regurgitation, various chamber sizes, pressure gradients and degree of pulmonary hypertension. The severity of VHD can thus, be objectively assessed by echocardiography. In addition, it provides accurate assessment of the global ventricular function. Thus, it is helpful in deciding optimal preoperative management and perioperative monitoring and care. Its preoperative prognostic value is unknown. However, because preoperative ventricular dysfunction is associated with perioperative ventricular dysfunction, echocardiography can be potentially useful as it provides the information less invasively. More recently, stress echocardiography is being used as a preoperative test. The appearance of a new or worsened RWMA is considered a positive test and represents areas at risk for myocardial ischaemia. In this respect, dobutamine stress echocardiography has been found to be very useful.

Tissue Doppler

The tissue Doppler velocity of the mitral annulus is used for the assessment of LV function. The ratio of early trans-mitral blood flow velocity (as measured by colour Doppler) to early diastolic velocity of the mitral annulus by tissue Doppler (E/e′) is an indicator of LV diastolic function and is relatively independent of the systolic function and rhythm abnormalities. The normal E/e′ is less than 8. It has been shown that E/e′ ratio of more than 15 is an independent predictor of composite endpoints of postoperative morbidity.[82]

Nuclear Imaging

Nuclear imaging is now a safe and accurate method for assessment of myocardial perfusion and infarction, and ventricular function. The regional distribution of myocardial blood flow can be visualised using the radio-isotopes that accumulate proportional to the regional myocardial blood flow. Thallium-201 has been employed successfully for this purpose. Recently, new technetium 99^m labelled compounds with better imaging characteristics and novel biological properties have been introduced. The relative distribution of myocardial blood flow can be visualised using these imaging agents. For the diagnosis of MI, two types of imaging exist: the myocardial scintigraphy (hot spot imaging) and perfusion scintigraphy (cold spot imaging).

The hot spot technique uses technetium 99^m pyrophosphate as the radionuclide. The infarcted segment of the myocardium that has a selective affinity for technetium is detected as a hot spot by the gamma camera. Normal tissue or areas of old infarction do not have affinity for technetium, and these areas are not visualised. The image can be detected after 12 to 16 hours following the event with maximum abnormality occurring from 48 to 72 hours. The intensity of the image returns towards normal within 5 to 7 days.

In perfusion scintigraphy or cold spot imaging, thallium-201 is used. The isotope is taken up by the areas of heart with normal perfusion and thus allows imaging of normal myocardium. Defects in the normal pattern (cold spots) represent areas of decreased perfusion as well as acute or old MI. The technique is useful in detecting the stress response of coronary circulation by obtaining scans during exercise or infusion of coronary vasodilator, dipyridamole. Because of rapid myocardial clearance rate of thallium-201, redistribution of thallium occurs quickly and allows visualisation of the reperfusion process. Perfusion defects of the cold spots last for approximately 30 to 60 minutes with redistribution occurring during the next 2 to 3 hours. Repeat imaging is performed approximately 3 to 4 hours later. The initial perfusion defects that persist, indicate infarction or prolonged ischaemia, and those that disappear are indicative of reversible perfusion defect or transient myocardial ischaemia without infarction.

For the assessment of ventricular performance and wall motion indices, first-pass radionuclide angiography and gated blood-pool imaging is used. Thus, radionuclear imaging is useful in enhancing the preoperative assessment of MI and quantification of the ventricular function. The predictive value of preoperative radionuclear imaging has been studied in vascular surgery patients. Initial results suggested preoperative gated-pooled-determined ejection fraction of less than 0.35 as an independent predictor of perioperative cardiac morbidity.[83] More recently, however, it has been shown that there is no association between the redistribution defects and adverse cardiac outcomes in patients undergoing elective vascular surgery.[84, 85]

Perfusion tracers and imaging technology have evolved and more recently dual tracer protocols with thallium and Technetium 99^{m} (99^{m} Tc) agents and Rubidium 82 (82 Rb) Positron Emission Technology (PET) imaging are used.[86]

A normal perfusion imaging study indicates a low risk of MI or cardiac deaths, whereas moderate to large reversible perfusion defects, which reflect myocardial ischaemia, carry the greatest risk of perioperative cardiac death or MI.[19]

Pharmacological Stress Testing

Many patients with CAD are unable to perform exercise tests. Such patients who cannot exercise adequately can now be stressed by pharmacological agents. These techniques have been found to give results that are equivalent to exercise testing. Myocardial perfusion can be increased artificially by infusing the small vessel vasodilators, dipyridamole or adenosine. This is followed by thallium imaging to detect myocardial ischaemia. Areas of myocardium surrounding a coronary vessel (that has fixed stenosis) have small vessel vasodilation at rest to maintain normal resting flow. Such areas have a diminished hyperaemic response to vasodilator compared with normal myocardium. This leads to a relative defect in myocardial perfusion imaging at peak flow. In fixed defects, the decreased uptake persists, whereas in reversible defects, later resting images reveal improved flow. Reversible defects are more likely to indicate myocardium at risk. The perioperative cardiac events occurring in patients undergoing non-cardiac surgery have been shown to be related to the size of jeopardised viable myocardium (myocardium at risk) determined by thallium imaging.[87] Abnormal scans are also useful to clinically select patients for therapeutic intervention such as revascularisation.

Dobutamine stress echocardiography is a newer technique that has rapidly established itself as a useful clinical tool. The coronary flow reserve is tested by increasing the heart rate by dobutamine infusion. A normal test result is defined as the absence of a new or worsening RWMA. Most studies have evaluated the role of dobutamine stress echo in patients undergoing abdominal aortic or peripheral vascular surgery (high perioperative cardiovascular risk). The data suggest that the test is safe and feasible as a part of preoperative assessment and has a high negative predictive value (if the test is negative, there is very less chance of nonfatal MI or death) as compared with positive predictive value.[19]

Cardiac Catheterisation

Cardiac catheterisation is the most important investigation that is undertaken in patients with CAD. Coronary angiography provides information about the coronary artery and the presence and pattern of atherosclerotic disease. For a cardiac surgeon, the coronary angiography helps to decide how many bypass grafts should be performed and where the distal anastomosis should be placed. Physiological information in terms of intracardiac pressures and CO can also be obtained. By knowing CO and ventricular filling pressure, i.e. LVEDP, a reliable index of cardiac function can be derived. An increase in LVEDP (>15 mm Hg), is indicative of pulmonary congestion. Likewise, a sudden large increase in LVEDP (an increase of more than 5 mm Hg) during ventriculography (when the low oxygen containing contrast material displaces blood from coronary circulation) is indicative of transient episode of cardiac failure. Such information provides a rough estimate of the risk of similar episodes of altered cardiac performance during anaesthesia and surgery. In addition, LV angiogram can provide valuable information such as ejection fraction, LV volumes and the presence of associated MR.

The type of coronary artery lesion can be useful. According to one study, proximal left circumflex artery stenosis was an independent predictor of mid-term mortality, especially in patients with a history of heart failure.[88]

Cardiac catheterisation provides detailed anatomical information about the patient with complex congenital heart disease and contributes to the planning of surgical correction. The role of cardiac catheterisation in other simple congenital anomalies such as atrial septal defect, ventricular septal defect, TOF, etc. is now limited due to advances in the two-dimensional echocardiography and colour Doppler techniques. It is rarely performed in situations where the echocardiography is not able to provide definitive diagnosis. Similarly, it is rarely performed in patients with VHD, as most of the information is obtained by echocardiography.[89] It is, however, indicated in patients with VHD before surgery to rule out associated CAD, especially in elderly patients. At many centres, coronary angiogram is routinely performed in patients more than 45 years of age. In the developing countries, however, rheumatic fever is still the commonest cause of VHD so that the patients are usually young and do not require coronary angiogram.

For patients undergoing non-cardiac surgery, cardiac catheterisation has a limited role, as alternative less costly and less invasive techniques are available to assess the ventricular as well as valvular function. However, it is perhaps, indicated in patients with suspected left main or triple vessel disease or patients with unstable angina in whom revascularisation either with angioplasty or bypass surgery may be useful. Although, previously successful myocardial revascularisation appears to reduce the risk for subsequent non-cardiac surgery,[77,78] it is doubtful, if elective revascularisation should be performed prior to surgery. Since the risk of CABG itself often exceeds the risks of noncardiac surgery, CABG is rarely justified simply to lower the risk of non-cardiac surgery. The current ACC/AHA guidelines endorse this view.[19] Therefore, routine angiography should not be performed in all high-risk patients undergoing non-cardiac surgery, but only in those patients who warrant revascularisation for medical reasons, independent of surgery. In addition, relative urgency of the non-cardiac problem should also be considered.

ICU ADMISSION RISK ASSESSMENT

Patient outcome is also influenced by the events in the operation theatre (OT). These may relate to the anaesthetic technique, adequacy of valve repair and myocardial protection. In other words, the OT events can alter the risk (increase or decrease) that was based on preoperative status. Any other adverse events in the OT such as excessive surgical bleeding, difficulty in separating the patient from CPB, and use of intra-aortic balloon pump can change the outcome in a patient but cannot be predicted accurately at the time of preoperative evaluation. Therefore, assessment of the patient for the second time on arrival in the intensive care unit (ICU) may be necessary.

Acute Physiology and Chronic Health Evaluation (APACHE II) that was designed for general ICU patients has also been evaluated for cardiothoracic surgical patients.[90] APACHE III (refinement of APACHE II) has also been tested.[91] The independent predictors of outcome are the acute physiology score, age, emergency and reoperation status, number of grafts and sex of the patient. The most recent scoring system has identified seven factors at ICU admission that were independent predictors of morbidity and mortality outcome.[92] Amongst these, the use of intra-aortic balloon pump to separate the patient from CPB was the single most important predictor of poor outcome, followed by prolonged CPB time (>160 min.).

In summary, the growing demands of improved outcome following cardiac surgery make it essential for each patient to undergo thorough perioperative evaluation. Preoperative cardiac complications are also an area of clinical interest and concern in patients undergoing non-cardiac surgery.[93] Over the years, perioperative risk assessment has evolved significantly. There is now evidence to suggest that reduced functional capacity is an independent predictor of mortality in patients undergoing non-cardiac surgery and should be a part of routine pre-operative evaluation.[94,95] It has also been shown that the use of a routine preoperative cardiac assessment allows to obtain satisfactory peri-operative results in patients undergoing abdominal aortic surgery.[96] Risk stratification of a patient is therefore important so that probabilities of the difficulties during the procedure as well as the outcome of surgery can be anticipated. By identifying the high-risk patient, it may be possible to alter the therapy or technique to reduce the risk. For instance, an appropriate method of myocardial preservation may be chosen in high-risk patients. This will not only be more cost- effective, but can also reduce the morbidity that is related to poor myocardial performance in the postoperative period. In addition, time may be spent preoperatively to improve the status of the patient by additional investigations and necessary therapy. Pre-operative statins have been shown to provide protective effect in the perioperative period in terms of cardiac morbidity and mortality[97] as well as renal insufficiency.[98] Most of the risk stratification has been described for patients suffering from CAD. There is a need to develop risk stratification in patients undergoing valve surgery or repair of congenital heart defects.

REFERENCES

1. Sprung J, Abdelmalak B, Gottlieb A, et al. Analysis of risk factors for myocardial infarction and cardiac mortality after major vascular surgery. Anesthesiology, 2000; 93:129–40.
2. Port S, Cobb FR, Coleman RE, et al: Effect of age on the response of the left ventricular ejection fraction to exercise. N Engl J Med 1980;303:1133–7.
3. Bridges CR, Edwards FH, Peterson ED, et al: Cardiac surgery in nonagenarians and centenarians. J Am Coll Surg 2003; 197:347–56.
4. Manson JE, Willet WC, Stampfer MJ: Body weight and mortality among women. N Engl J Med 1995; 333:677–85.
5. Ghanta RK, LaPar DJ, Zhang Q, et al. Obesity increases risk adjusted morbidity, mortality, and cost following cardiac surgery. J Am Heart Assoc 2017, Mar 8. doi 10.1161/JAHA.116-003831.
6. Mariscalco G, Wozniak MJ, Dawson AG, et al. Body mass index and mortality among adults undergoing cardiac surgery: A nationwide study with a systematic review and meta-analysis. Circulation 2017; 135; 850–63.
7. Tuman KJ, McCarthy RJ, March RJ: Morbidity and duration of ICU stay after cardiac surgery. A model for preoperative risk assessment. Chest 1992; 102:36–44.
8. O'Connor GT, Morton JR, Diehl MJ: Differences between men and women in hospital mortality associated with coronary artery bypass graft surgery. The Northern New England Cardio-vascular Disease Group. Circulation 1993; 88:2104–10.
9. Parsonnet V, Dean D, Bernstein AD: A method of uniform stratification of risk for calculating the results of surgery in acquired adult heart disease. Circulation 1989; 79(6 pt 2):13–12.
10. Higgins TL, Estafanous FG, Loop FD: Stratification of morbidity and mortality outcome by preoperative risk factors in coronary artery bypass patients. JAMA 1992 267:2344–8.
11. Koch CG, Weng Y, Zhou SX, et al: Prevalence of risk factors, and not gender per se, determines short and long-term survival after coronary artery bypass surgery. J Cardiothorac Vasc Anesth 2003; 17:585–93.
12. Johnston A, Mesana TG, Lee DS, et al. Sex differences in long-term survival after major cardiac surgery: A population-based cohort study. J Am Heart Assoc 2019; 8:e013260.
13. Gupta S, Lui B, Ma Y, et al. Sex differences in outcomes after coronary artery bypass grafting. J Cardiothorac Vasc Anesth 2020; 34:3259–66.

14. Faerper G, Zacher M, Reents W, et al. Female sex is not a risk factor for post-procedure mortality in coronary bypass surgery in the elderly: A secondary analysis of the GOPLABE trial. PLoS one 2017; 12:e0184038.
15. Goldman L: Cardiac risks and complications of non-cardiac surgery. Ann Intern Med 1983; 98:504–13.
16. Rao TK, Jacobs KH, EL-Etr AA: Reinfarction following anesthesia in patients with myocardial infarction. Anesthesiology 1983; 59:499–505.
17. Wells P, Kaplan JA: Optimal management of patients with ischemic heart disease for non-cardiac surgery by complementary anesthesiologist and cardiologist interaction. Am Heart J 1981; 102:1029–37.
18. Ryan TJ, Anderson JL, Antman EM, et al: ACC/AHA guidelines for the management of patients with acute myocardial infarction: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 1996; 28:1328–428.
19. Fleisher LA, Fleischmann KE, Auerbach. AD, et al. 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: A report of the American College of Cardiology/American Heart Association Task Force on practice guidelines. Circulation 2014; 1; 30:000–000.
20. Levine GN, Bates ER, Mauri L, et al. 2016 ACC/AHA guideline focused update on duration of dual antipletelet therapy in patients with coronary artery disease. Circulation 2016; 133:000–000.
21. Hoek SE, Poldermans D. European Society of Cardiology 2009 guidelines for preoperative cardiac risk assessment and perioperative cardiac management in non-cardiac surgery: key messages for clinical practice. Pol Arch Med Wewn 2010; 120:294–9.
22. Mashour GA, Shanks AM, Kheterpal S. Perioperative stroke and associated mortality after noncardiac nonneurologic surgery. Anesthesiology 2011; 114:1289–96.
23. Mangano DT: Perioperative cardiac morbidity. Anesthesiology 1990; 72:163–84.
24. Barbero U, D'Ascenzo F, Nijhoff F, et al. Assessing risk in patients with stable coronary disease: When should we intensify care and follow-up? Results from a metaanalysis of observational studies of the COURAGE and FAME era. Scientifica 2016, doi.org/10.1155/2016/3769152.
25. Higgins TL. Quantifying risk and assessing outcome in cardiac surgery (review article). J Cardiothorac Vase Anesth 1998; 12:330–40.
26. Shah KB, Kleinman BS, Rao T, et al: Angina and other risk factors in patients with cardiac diseases undergoing non-cardiac operations. Anesth Analg 1990; 70:240–7.
27. Chassot PG, Delabays A, Spatin DR. Preoperative evaluation of patients with, or at risk of, coronary artery disease undergoing non-cardiac surgery. Br J Anaesth 2002; 89:747–59.
28. Prinzmetal M, Kennamer R, Merliss R, et al: Angina pectoris. I. A variant form of angina pectoris; preliminary report. Am J Med 1959; 27:375–88.
29. Mac Alpin R. Observation on coronary spasm complicating non-cardiac surgery. J Cardiol Cardiovasc Med. 2018; 1:112.
30. Deanfield JE, Selwyn AP, Chierchia S, et al: Myocardial ischemia during daily life in patients with stable angina: Its relation to symptoms and heart rate changes. Lancet 1983; 2(8353):753–8.
31. Cohn PF, Fox KM, Daly C. Silent myocardial ischemia. Circulation 2003; 108:1263–77.
32. Rennart G, Saltz-Rennart H, Wanderman K, et al: Size of acute myocardial infarcts in patients with diabetes mellitus. Am J Cardiol 1985; 55:1629–30.
33. Fein FS, Sonnenblick EH: Diabetic cardiomyopathy. Prog Cardiovasc Dis 1985; 27:255–70.
34. AHA. ACC. National Heart Lung and Blood Institute. et al. AHA/ACC guidelines for secondary prevention for patients with coronary and other atherosclerotic vascular disease. 2006 update endorsed by the National Heart Lung and Blood Institute. J Am Coll Cardiol 2006; 47:2130–9.
35. Benowitz NL, Burbank AD. Cardiovascular toxicity of nicotine: Implications for electronic cigarette use. Trends Cardiovasc Med 2016; 26:515–23.
36. Yadav KS, Ross JK, Monro JL1 et al: Study of the risk factors related to early mortality following combined mitral valve replacement and coronary artery bypass grafting. Thorac Cardiovasc Surg 1985; 33:16–9.
37. Herlitz J, Brandrup-Wognsen K, Caidahl M, et al. Motality and morbidity among patients who undergo combined valve and coronary artery bypass surgery. Early and late results. Eur J Cardiothorac Surg 1997; 12:836:46.

38. American Society of Anesthesiologists: New classification of physical status. Anesthesiology 1963; 24:111.
39. Braunwald E: The history. In: Braunwald E (Ed); Heart Disease: A Textbook of Cardiovascular Medicine, Philadelphia, WB Saunders Company, 1984; 1–13.
40. www.ccs.ca/images/Guidelines/Guidelines_POS_Library/Ang-1976.pdf accessed on November 10, 2020.
41. Goldman L1 Caldea DL, Nussbaum SR, et al: Multifactorial index of cardiac risk in noncardiac surgical procedures. N Engl J Med 1977; 297: 845–50.
42. Goldman L: Multifactorial index of cardiac risk in non-cardiac surgical procedures: Ten year status report. J Cardiothorac Anesth 1987; 1:237–44.
43. Detsky AS, Abrams HB, Forbath N1 et al: Cardiac assessment for patients undergoing noncardiac surgery. A multifactorial clinical risk index. Arch Intern Med 1986; 146:2131–4.
44. Gilbert K, Larocque BJ, Patrick LT: Prospective evaluation of cardiac risk indices for patients undergoing noncardiac surgery. Ann Intern Med 2000; 133:356–9.
45. Lee TH, Marcantanio ER, Mangione CM, et al: Derivation and prospective validation of a simple index of prediction of cardiac risk of major non-cardiac surgery. Circulation 1999; 100:1043–9.
46. Boersma E, Kertai MD, Schouten O, et al: Perioperative Cardiovascular mortality in non-cardiac surgery: Validation of the Lee Cardiac risk index. Am J Med 2005; 118:1134–41.
47. Bonaros N, Vill D, Wiedemann D, et al: Major risk stratification models do not predict peri-opertive outcome after coronary artery bypass grafting in patients with previous percutaneous intervention. Eur J Cardiothorac Surg 2011; 39: e164–9.
48. Biccard BM, Rodseth RN: Utility of clinical risk predictors for preoperative cardiovascular risk prediction. Br J Anaesth 2011; 107:133–43.
49. Hlatky MA, Boineau RE, Higginbotham MB, et al. A brief self-administered questionnaire to determine functional capacity (the Duke Activity Status Index) Am J Cardiol 1989; 64:651–4.
50. Wijeysundera DN, Beattie WS, Hillis GS, et al. Integration of Duke Activity Status Index into preoperative risk evaluation: a multicentre prospective cohort study. Br J Anaesth 2020; 124:261–70.
51. Bilimoria KY, Liu Y, Paruch JL, et al. Development and evaluation of the universal ACS NSQIP surgical risk calculator: a decision aid and informed consent tool for patients and surgeons. J Am Coll Surg 2013; 217:833–42.
52. Edwards FH, Clark RE, Schwartz M: Coronary artery bypass grafting: The Society of Thoracic Surgeons National Database Experience. Ann Thorac Surg 1994; 57:12.–9.
53. Stevenson LW, Perloff JK. The limited reliability of physical signs for estimating hemodynamics in chronic heart failure. JAMA 1989; 261:884–8.
54. Hammill BG, Curtis LH, Bennett-Guerrero E, et al. Impact of heart failure on patients undergoing major noncardiac surgery. Anesthesiology 2008; 108:559–67.
55. Dalen M, Lund LH, Ivert T, et al. Survival after coronary artery bypass grafting in patients with preoperative heart failure and preserved vs reduced ejection fraction: JAMA Cardiol 2016; 1:530–8.
56. Solomon SD, Zelenkofske S, McMurray JJ, et al: Sudden death in patients with myocardial infarction and left ventricular dysfunction, heart failure or both. N Engl J Med 2005; 352:2581–8.
57. Anghel D, Anghel R, Corciova F, et al. Pre-operative arrythmias such as atrial fibrillation: Cardiovascular surgery risk factor. Biomed 2014; doi.org/10-1155/2014/584918.
58. Winkel TA, Schouten O, Hoeks SE, et al. Risk factors and outcome of new onset cardiac arrhythmias in vascular surgery patients. Am Heart J 2010; 159:1108–15.
59. De Bacqer D, De Backer G, Kornitzer M, et al. Prognostic value of ECG findings for total, cardiovascular disease, and coronary heart disease death in men and women. Heart 1998; 80:570–7.
60. Lin LL, Dzankic S, Leung JM. Preoperative electrocardiogram abnormalities do not predict postoperative cardiac complications in geriatric surgical patients. J Am Geriatr Soc 2002; 50:1186:91.
61. US Preventive Services Task Force: Screening for coronary heart disease: Recommendations statement. Ann Intern Med 2004; 140:569–72.

62. Feely MA, Collins CS, Daniels PR, et al. Preoperative testing before noncardiac surgery: Guidelines and recommendations. Am Fam Physician 2013; 87:414–8.
63. Mohammad WA, Ashraf Z, Sayed HF, Urgent versus elective coronary artery bypass grafting in acute coronary artery syndrome. J Egyptian Soc Cardiothorac Surg 2018; 26:17–23.
64. Huxley R, Lewington S, Clarke R. Cholesterol, coronary heart disease and stroke: a review of published evidence from observational studies and randomized controlled trials. Semin Vasc Med 2002; 2:315–23.
65. Gordon T, Castelli WP, Hjortland ML, et al: High density lipoprotein as a protective factor against coronary heart disease: The Framingham study. Am J Med 1977; 62:707–14.
66. Grande P, Hansen BF, Christiansen C: Estimation of acute myocardial infarct size in man by serum CK-MB measurements. Circulation 1982; 65:756–64.
67. Katus HA, Remppis A, Scheffold T, et al: Intracellular compartmentation of cardiac troponin T and its release kinetics in patients with reperfused and nonreperfused myocardial infarction. Am J Cardiol 1991; 67:1360–7.
68. Adams TE 3rd, Bodor G S, Davila-Roman VG, et al: Cardiac troponin I: A marker with high specificity for cardiac injury. Circulation 1993; 88:101–6.
69. Machler H, Metzler H, Sabin K, et al: Preoperative myocardial cell damage in patients with unstable angina undergoing coronary artery bypass graft surgery. Anesthesiology 1994; 81:1324–31.
70. Beller JP, Hawkins RB, Mehaffey JH, et al. Does preoperative troponin level impact outcomes following coronary artery bypass grafting? Ann Thorac Surg 2018, Mar 8. doi:10.1016/j.athoracsur.2018.01–085.
71. Rodseth RN, Lurati Buse GA, Bolliger D, et al: The predictive ability of pre-operative B-type natriuretic peptide in vascular patients for major adverse cardiac events: an individual patient data meta-analysis. J Am Coll Cardiol 2011; 58:522–9.
72. Lurati Buse GA, Koller MT, Burkhart C, et al. The predictive value of preoperative natriuretic peptide concentrations in adults undergoing surgery: a systematic review and metaanalysis. Anesth Analg 2011; 112:1019–33.
73. Ostojic MC,. Young JB, Hess KR, et al: Prediction of left ventricular ejection fraction using a unique method of chest X-ray and ECG analysis. Am Heart J 1989; 117:590–8.
74. Miranda CP, Lehmann KG, Froehlicher VF: Correlation between resting ST segment depression, exercise testing, coronary angiography, and long term prognosis. Am Heart J 1991; 122:1617–28.
75. Crenshaw JH, Mirvis DM, EL-Zeky P, et al: Interactive effects of ST-T wave abnormalities on survival of patients with coronary artery disease. J Am Coll Cardiol 1991; 18:413–20.
76. Scheffield LT: Exercise stress testing. In: Braunwald E (Ed). Heart Disease: A Text Book of Cardiovascular Medicine, WB Saunders Company, Philadelphia, 1984; 267–268.
77. Mangano DT. McSPI Research Group: Preoperative assessment of the patient with cardiac disease. Curr Opin Cardiol 1995; 10:530–42.
78. Mangano DT, Goldman L. Preoperative assessment of patients with known or suspected coronary disease. N Engl J Med 1995; 333:1750–6.
79. Sinclair RCF, Batterham AM, Davies S, et al: Validity of the 6 min walk test in prediction of the anaerobic threshold before major noncardiac surgery. Br J Anaesth 2012; 108:30–5.
80. Nishimura RA, Otto CM, Bonow RO, et al. 2017 AHA/ACC focused update of the 2014 AHA/ACC guideline for the management of patients with valvular heart disease: A report of the American College of Cardiology/American Heart Association task force on clinical practice guidelines. Circulation 2017; 135:e1159–e1195.
81. Nishimura RA, Otto CM, Bonow RA, et al. 2014 AHA/ACC guidelines for the management of patients with valvular heart disease. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2014; 63:2438–88.
82. Jun NH, Shim JK, Kirn JC, et al: Prognostic value of a tissue Doppler-derived index of left ventricular filling pressure on composite morbidity after off-pump coronary artery bypass surgery. Br J Anaesth 2011; 107:519–24.
83. Pasternack PF, Imparato AM, Rites TS, et al: The value of radionuclide angiogram in the prediction of perioperative myocardial infarction in patients undergoing lower extremity revascularisation procedures. Circulation 1985; 72(3 pt 2):II13–7.
84. Mangano DT, London MJ, Tubau JF, et al: Dipyridamole thallium 201 scintigraphy as a preoperative screening test: A re-examination of its predictive potential. Circulation 1991; 84:493–502.

85. Baron JF, Mundler O, Bertrand M, et al: Dipyridamole thallium scintigraphy and gated radionuclide angiography to assess cardiac risk before abdominal aortic surgery. N Engl J Med 1994; 330:663–9.
86. Weinstein H, Steingart R. Myocardial perfusion imaging for preoperative risk stratification. J Nud Med 2011; 52:750–60.
87. Brown KA, Rowen M: Extent of jeopardised viable· myocardium determined by myocardial perfusion imaging best predicts perioperative cardiac events in patients undergoing noncardiac surgery. J Am Coll Cardiol 1993; 21:325–30.
88. Goto M, Kohsaka S, Lee VY, et al: Risk stratification of coronary revascularization patients by using clinical and angiographic data. J Cardiovasc Surg. 2011; 52:863–71.
89. Popovic AD, Thomas JD, Neskovic AN, et al: Time related trends in the preoperative evaluation of patients with valvular stenosis. Am J Cardiol 1997; 80:1164–8.
90. Turner JS, Mudaliar YM, Chang RW: Acute Physiology and Chronic Health Evaluation (AP ACHE II) scoring in cardiothoracic intensive care unit. Crit Care Med 1991; 19:1266–9.
91. Becker RB, Zimmerman JE, Knaus WA: The use of APACHE III to evaluate ICU length of stay, resource use and mortality after coronary artery surgery. J Cardiovasc Surg 1995; 36:1–11.
92. Higgins TL, Estafanous FG, Starr NJ: ICU admission score for predicting morbidity and mortality after coronary artery bypass grafting. Ann Thorac Surg 1997; 64:1050–8.
93. Smilowitz NR, Berger JS. Perioperative cardiovascular risk assessment and management for noncardiac surgery: A review. JAMA 2020; 324:279–90.
94. Wilson RJT, Davies S, Yates D, et al: Impaired functional capacity is associated with all cause mortality after major elective intra-abdominal surgery. Br J Anaesth 2010; 105:297–303.
95. Visnjevac O, Davari-Farid S, Lee J, et al. The effect of adding functional classification to ASA status for predicting 30-day mortality. Anesth Analg 2015; 121:110–6.
96. Troisi N, Dorigo W, Lo Sapio P, et al: Preoperative cardiac assessment in patients undergoing aortic surgery: analysis of factors affecting the cardiac outcomes. Ann Vase Surg 2010; 24:733–40.
97. Gan HL, Zhang JQ, Bo P, et al: Statins decrease adverse outcomes in coronary artery bypass for extensive coronary artery disease as well as left main coronary stenosis. Cardiovasc Ther 2010; 28:70–9.
98. Virani SS, Nambi V, Polsani VR, et al: Preoperative statin therapy decreases risk of postoperative renal insufficiency. Cardiovasc Ther 2010; 28:80–6.