3 Cardiac Protection for Noncardiac Surgery

P. FOEX AND G. HOWARD-ALPE

Introduction

Cardiovascular complications of anesthesia and surgery remain, unfortunately, very frequent. In the USA, Mangano and Goldman concluded that approximately 27 million anesthetics were given every year, including 8 million to patients with coronary artery disease. They estimated the number of cardiovascular complications to be approximately 1 million per annum, including 500,000 postoperative myocardial infarctions [1]. This represents one cardiovascular complication for every 27 anesthetics. The complications considered in this context include myocardial infarction, unstable angina, life-threatening arrhythmias, and acute left ventricular failure.

In the UK the number of anesthetics is estimated at 3 million per annum. The number of perioperative cardiac deaths has been found to be approximately 20,000 per annum for many years [2]. Sixty percent of the patients who die within 30 days of surgery have evidence of coronary heart disease [3], and the number of cardiac deaths is approximately 9000 per annum [2]. A systematic review and meta-analysis of randomised controlled trials (2005) shows that for each cardiac death there are ten major cardiovascular complications [4], therefore, the total number of cardiac deaths and cardiovascular complications is likely to be in the region of 100,000 per annum, or one cardiovascular complication for every 30 anesthetics. It is known that perioperative cardiac complications are associated with a significant reduction of the patient’s life expectancy [5], and these complications represent a major health problem.

Postoperative myocardial infarction is one of the major complications of
anesthesia and surgery. It occurs in 2.5% of unselected patients aged over 40 years and 8.6% of patients in whom suspicion of coronary artery disease is sufficiently strong to justify myocardial perfusion scintigraphy [6]. In patients with confirmed significant coronary artery disease on dobutamine-sensitized echocardiography or myocardial perfusion scintigraphy, vascular surgery may be associated with a 30% risk of myocardial infarction or cardiac death [7, 8]. In the face of such a major health risk, active steps must be taken to protect patients as there are very large health costs associated with the treatment of perioperative adverse cardiac events.

**Mechanisms of Myocardial Ischemia and Its Complications**

Ischemic complications result from the presence of underlying cardiac disease and the stress of surgery with its associated increase in sympathetic activity and other stress hormones such as corticosteroids.

In the presence of fixed coronary artery stenoses with limited coronary flow reserve, myocardial ischemia can occur because increases in myocardial oxygen requirements cannot be met by commensurate increases in coronary blood flow. In the presence of dynamic coronary stenoses, myocardial ischemia is caused by sudden increases in coronary vascular tone. α-adrenergic stimulation, release of endothelins, and thromboxane, as well as inhibition of vasodilators such as nitrous oxide, cause vasoconstriction and curtail oxygen supply. In addition, the probability of vasoconstriction is increased because of endothelial damage. This tends to alter the local balance of vasodilators and vasoconstrictors in favor of vasoconstrictors. Thus, many factors contribute to myocardial ischemia (Fig. 1).

Myocardial ischemia causes an immediate reduction in regional cardiac function. Depending upon its duration, myocardial ischemia may be followed by complete recovery, albeit after a period of stunning, or by myocardial infarction. Repeated episodes of ischemia followed by stunning may result in myocardial hibernation, a prolonged, but potentially reversible, depression of function. Paradoxically, myocardial ischemia may also be protective; short episodes of ischemia can reduce the extent of damage after coronary occlusion, as shown in ischemic preconditioning.

Over the past decade, however, it has become increasingly obvious that acute coronary syndromes may be caused by the release of inflammatory mediators. Indeed, in patients with elevated C-reactive protein (CRP), the prognosis of coronary artery disease is worse than in those with normal CRP, especially in acute coronary syndromes [9, 10]. Other inflammatory markers are also elevated. Major surgery causes the release of inflammatory mediators. This can be followed by adverse cardiac events resulting from unstable