# Risk of anesthesia in patients with heart disease

Sait Tarhan, M.D.

*Rochester, Minnesota*

Patients with moderate to severe valvular dysfunction represent a challenge to anesthesiologists because of their specific cardiac pathology. Certain cardiac lesions increase the risk of operation even though cardiac reserve seems good. Patients with severe aortic stenosis or incompetence are particularly susceptible to the development of ventricular fibrillation. Patients with mitral stenosis who have few symptoms can tolerate general surgical procedures reasonably well, but care should be taken not to increase the left atrial pressure and consequent pulmonary edema.[1]

Management of these patients requires understanding of the altered cardiovascular state, hemodynamic impairment caused by the anatomic lesions, and their altered response to the support of cardiac function. Hemodynamic requirements of different lesions dictate the appropriate choice of anesthetic drugs and techniques. Careful titration of agents to preserve circulatory stability is essential while the patient is undergoing surgery. Selection of anesthetic agents for the patient with certain types of cardiac lesions is also important, such as enflurane and halothane, which are associated with junctional rhythm. Gallamine and ketamine may increase the heart rate; therefore, those agents may not be the optimum choice for tight mitral stenosis.

However, narcotics would not depress contractility and change the rhythm, and therefore would be more suitable. The number of patients who have valve disease and undergo operation has declined in the past decade because of the progress made in replacing diseased valves with prosthetic valves. However, replacement of an old, damaged valve with a prosthesis does not guarantee perfect myocardial function. A degree of cardiac compromise still may exist, and therefore these patients require special care.

The most common cardiac disease among patients having anesthesia and surgery is coronary heart disease and previous myocardial infarction. The high incidence and pathogenic mechanisms are discussed.

One million three hundred thousand persons experience myocardial infarctions each year in the United States.[2-4] Therefore, the number of patients with coronary heart disease or myocardial infarction who require some form of surgical operation is increasing steadily and these patients present problems related to the specific cardiac pathologic findings.

Some authors have assumed that patients with coronary artery disease and good cardiac reserve tolerate general surgical procedures well,[1] but others have reported that presence of three-vessel coronary artery disease is associated with approximately 20% risk of myocardial infarction after anesthesia and noncardiac operations. Some studies indicate that risk of postoperative myocardial infarction after noncardiac operations in patients with one- or two-vessel coronary artery disease appears to be low.[3] Patients with coronary heart disease who had undergone coronary artery bypass graft surgery have a greater chance to undergo anesthesia and noncardiac surgery without having postoperative myocardial infarction than patients with coronary heart disease and those who did not have coronary artery bypass graft surgery.[5]

In a study of myocardial infarction after general anesthesia in which the diagnosis was supported by electrocardiography and appropriate enzyme studies, patients with previous myocardial infarctions, 28 of 422 (6.6%) experienced another infarction during the first week after the operation.[6] They had a 50-fold greater risk of reinfarction than patients who did not have a history of myocardial infarction; of 32,455 patients, only 43 (0.13%) had infarctions after surgery.

Fifty-four percent of them died as a result of reinfarction. The relation between time of infarct and surgery is shown in *Table 1* and the relation of reinfarction to the site of surgery is shown in *Table 2.*

Since this study, new anesthetic agents and techniques have been developed; major changes in medical management have come about and high-risk patients are more often monitored in intensive care units. Therefore, all these measures should reduce the reinfarction ratio. To verify this assumption, we recently conducted another study; how-

**Table 1.** Relation between time of infarction and surgery

| Time of surgery, mo | No. of patients | | No. of postoperative reinfarctions (%) | |
|---|---|---|---|---|
| | 1972 | 1978 | 1972 | 1978 |
| 0–3 | 8 | 15 | 3 (38) | 4 (27) |
| 4–6 | 19 | 18 | 3 (16) | 2 (11) |
| 7–12 | 42 | 31 | 2 (5) | 2 (6) |
| 13–18 | 27 | 30 | 1 (4) | 1 (3) |
| 19–24 | 21 | 17 | 1 (5) | 1 (6) |
| >24 | 232 | 383 | 11 (5) | 15 (4) |
| Unknown | 73 | 93 | 7 (10) | 11 (12) |
| Total | 422 | 587 | 28 (7) | 36 (6) |

**Table 2.** Relation of myocardial reinfarction to site of surgery

| | No. of patients | | No. of reinfarctions (%) | |
|---|---|---|---|---|
| | 1972 | 1978 | 1972 | 1978 |
| Great vessels | 54 | 69 | 5 (9) | 11 (16)* |
| Other intrathoracic | 18 | 24 | 8 (44) | 3 (13)† |
| Upper abdominal | 59 | 84 | 3 (5) | 7 (8)† |
| Total of above | 131 | 177 | 16 (12)* | 21 (12) |
| Other sites | 291 | 410 | 12 (4)* | 15 (4)*† |
| Total | 422 | 587 | 28 (7) | 36 (6) |

* $p < 0.001$.
† $p < 0.05$.

ever, the results were disappointing.[7] The incidence and severity of reinfarctions were identical with those in the previous study (6.1% reinfarctions). The time of the infarction and surgery showed the same relationship as the previous study (*Table 1*). Mortality among these patients was 69%. It was apparent in both studies that the incidence of a new myocardial infarction stabilized after a 6-month interval. In the second study the relation of reinfarction to the site of surgery also gave similar results (*Table 2*). In both studies, age, sex, anesthetic techniques, and site of previous infarct had no significant effect on reinfarction ratio.

In the second study, we tried to correlate preoperative hypertension requiring medical treatment, diabetes, angina, and blood pressure changes during anesthesia to the reinfarctions. Patients with preoperative hypertension had significantly higher reinfarction rates than normotensive patients (9.4% versus 4.7%, $p < 0.05$). Diabetes and angina did not increase the infarction rate. Of 145 patients in whom systolic pressures decreased by at least 30% one or more times for 10 minutes or more during anesthesia, the reinfarction rate was significantly higher (15.2% versus 3.2%, $p < 0.001$) than for patients who did not have such intraoperative hypotensive episodes. However, a 30% or more increase in blood pressure did not increase the reinfarction ratio significantly ($p < 0.15$). Duration of anesthesia increased the reinfarction rate for the entire group from 1.9% for procedures lasting less than one hour to 16.7% for procedures lasting more than 6 hours ($p < 0.001$). Twenty-five percent of the patients in the second study were admitted to the intensive care unit after surgery, which did not influence the reinfarction ratio.

## Discussion

Reinfarction after anesthesia and surgery is a serious complication with high mortality. Elective operations should be delayed at least 6 months after the first infarct. We have not been able to reduce (statistically) the number of reinfarctions after anesthesia and surgery, despite apparent major changes in perioperative management.

The pathogenic mechanism of myocardial infarction is still largely speculative.[8] It is reasonably easy to explain the occurrence of myocardial infarction in patients with atherosclerotic coronary vessels with superimposed hypotension or hypertension, or an acute thrombosis, and this could be accounted for in some of our cases. Yet, some infarctions have occurred without any hemodynamic changes during or after surgery.

The critical degree of narrowing of the coronary arteries is about an 80% diameter reduction. Narrowing above 80% is consistently associated with limitations of blood flow and less than 80% narrowing causes flow limitations only under conditions of increased demand.[9]

Myocardial infarction can be precipitated by coronary vasospasm, usually but not always superimposed on atherosclerotic plaque, which may obstruct 60% to 80% of the luminal diameter, an acute increase of that narrowing from 90% to 100% by vasoconstriction, or the addition of a relatively small thrombus[9] would complete the process. This pathogenic mechanism may be operative on patients during and after anesthesia. Several different factors can cause coronary artery spasm such as an increase of calcium ion in the blood,[10] parasympathomimetic agents,[11] hyperventilation,[12] increased parasympathetic tone,[10] and released thromboxane $A_2$ by aggregated platelets in the thrombi.[13] Little information is available about their effects on the coronary arteries under anesthesia. Unquestionably, coronary artery spasms occur much more frequently than has been suspected.[14] Hyperventilating the patients rather than hypoventilating, intravenous injection of calcium salts to the patients, and a high incidence of vasovagal reflexes during abdominal and thoracic surgery are a few of the conditions that commonly occur during anesthesia and should be considered as a cause of coronary artery spasm, even myocardial infarction. Also ST-segment elevations observed during and after anesthesia and surgery that suggest transmural ischemia and characteristic of Prinzmetal's angina[15] are no rarity. Prinzmetal's variant angina in the immediate postanesthetic state also has been reported.[16] One must be aware of the possibility of coronary vasospasm during and after anesthesia. This subject merits further investigation.

## References


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