

## Effects of Anesthetics on the Coronary Circulation and Myocardial Metabolism

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Most anesthetic agents affect the coronary circulation indirectly by their effects upon systemic hemodynamics and by their effect on inotropy. Since almost every anesthetic decreases inotropy in a dose-dependent fashion, oxygen requirements and hence myocardial blood flow can be expected to decrease in parallel to the administered dose.

The coronary circulation is autoregulated within a wide range of perfusion pressures. This range may be altered by significant coronary stenoses which restrict vasodilator reserve and by increases in coronary back pressure. In the presence of maintained autoregulation, alterations in myocardial oxygen requirements are met by changes in coronary blood flow. Hence, myocardial oxygen extraction remains unaltered. Myocardial oxygen extraction may increase to compensate for an insufficient coronary flow response to a rise in energy demands. Conversely, a decline in myocardial oxygen extraction indicates flow in excess of demand, e. g. coronary vasodilatation.

Recent data indicate that some anesthetics interfere with normal coronary autoregulation. It is also evident that surgical stimulation and other noxious stimuli may override normal autoregulation. This abstract presents a summary of the effects of common anesthetic agents upon the coronary circulation and

myocardial metabolism.

### Barbiturates

All barbiturates which are able to induce sleep also decrease inotropy and blood pressure. Heart rate increases in healthy patients. The net result is increased metabolism, hence coronary flow. Patients with cardiac disease may not demonstrate any rise in heart rate. Under such circumstances, myocardial oxygen requirements and blood flow decrease in parallel. Barbiturates do not affect coronary vascular tone directly.

### Propofol

Propofol induces hemodynamic changes indistinguishable from those of the short-acting barbiturates. The agent thus produces comparable myocardial metabolic and circulatory changes without interference with coronary autoregulation.

### Ketamine

Ketamine administered without pretreatment with benzodiazepines or droperidol increases heart rate and blood pressure as a consequence of centrally mediated catecholamine release. Coronary blood flow increases in parallel to myocardial oxygen demand. There is no interaction with coronary autoregulation. Benzodiazepines and droperidol largely eliminate the hemodynamic effects of ketamine and

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therefore also the cardiac metabolic and flow responses.

### **Benzodiazepines**

All benzodiazepines reduce myocardial oxygen requirements somewhat by their effects on cardiac contractility. In addition, a slight decrease in myocardial oxygen extraction has been observed. This minor interference with coronary autoregulation has not been shown to be of clinical importance.

### **Etomidate**

This compound has minimal effects on systemic hemodynamics and inotropy. Under these circumstances neither myocardial metabolism nor coronary blood flow is affected.

### **Opioids**

With the exception of pethidine, opioids used in anesthesia do not significantly affect cardiac contractility. Their main effect is to decrease vascular tone. Morphine may cause increase in heart rate. The myocardial metabolic effect of opioids is directly related to their effects on the peripheral determinants of myocardial oxygen demand. These may vary depending on the ability of cardiac reflexes to respond to changes in vascular tone, endogenous sympathetic tone etc. None of the opioids interferes with coronary autoregulation.

### **Inhalation agents**

All three commonly used inhalation agents decrease myocardial metabolism by their effects on inotropy and systemic hemodynamics. They are also coronary vasodilators. This effect has been demonstrated in animals and in humans. The order of coronary vasodilator potency is isoflurane  $\gg$  enflurane  $>$  halothane. The site of action seems to be mostly on epicardial arterioles. None of the agents affect large coronary arteries. The coronary vasodilatation by isoflurane is dose-dependent and results in

limitation of coronary vasodilator reserve. With steal-prone coronary anatomy, this may cause regional maldistribution of blood flow. In the face of a single coronary stenosis, isoflurane has been shown to produce transmural maldistribution of blood flow as well. Since coronary vasodilator reserve may be markedly reduced with high doses of isoflurane, patients with coronary artery disease may be less tolerant to tachycardia, hypotension and increased coronary back pressure. Neither halothane, nor enflurane have been shown to adversely affect coronary blood flow distribution.

### **Nitrous oxide**

Nitrous oxide has a dual effect on the circulation. It stimulates the sympathetic nervous system and causes mild depression of contractility. Therefore, myocardial metabolism and coronary blood flow are usually not affected. Cardiodepression may however dominate over the sympathetic stimulatory effect in patients with high endogenous sympathetic tone, for instance related to left ventricular dysfunction. Under these circumstances, nitrous oxide results in reductions of cardiac function and blood pressure. This is accompanied by a decline in myocardial metabolism. Experimentally, nitrous oxide has been shown to constrict large coronary arteries slightly without effect on resistance vessels.

### **Effects of noxious stimuli**

Endotracheal intubation has been shown to reduce coronary blood flow without affecting myocardial metabolism in patients with coronary artery disease. This indicates a coronary vasospastic reaction which seems to be mediated neurally rather than humorally. A decline in coronary blood flow without simultaneous change in the peripheral determinants of myocardial oxygen requirements has also been observed occasionally during abdominal surgical

stimulation. During isoflurane-induced coronary vasodilatation, surgical stimulation with increased myocardial oxygen demand has been demonstrated without a concomitant rise in myocardial oxygen extraction. Thus, it seems as if coronary vasodilatation and vasoconstriction might coexist.

### Suggested reading

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