LABORATORY INVESTIGATIONS

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Transmural Redistribution of Myocardial Blood Flow during Isoflurane Anesthesia and Its Effects on Regional Myocardial Function in a Canine Model of Fixed Coronary Stenosis

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Background: **The effects of isoflurane on the transmural distribution of myocardial blood flow distal to an acute critical coronary stenosis and the relationship between the changes in regional blood flow and function were studied to determine whether isoflurane can produce a transmural "steal" phenomenon and to assess the role of this phenomenon in producing changes in regional myocardial function.**

Methods: **After production of acute critical coronary stenosis under baseline chloralose and fentanyl anesthesia, the animals were exposed to increasing end-tidal concentrations of isoflurane (0.7%, 1.4%, and 2.1%) without control of the hemodynamic parameters. At 2.1% isoflurane, the blood pressure then was restored to the baseline level by administration of phenylephrine. Changes in the following parameters were assessed: global contractility (measured by changes in pressure with time), regional myocardial function (assessed by systolic wall thickening and measured by sonomicrometers), transmural distribution of myocardial perfusion (measured by the radioactive microsphere method), and regional oxygen consumption and extraction.**

Results: **Distal to the critical stenosis, a transmural redistribution of myocardial blood flow (endocardial–epicardial ratio < 1) occurred with all concentrations of isoflurane. With higher concentrations (1.4% and 2.1%), a significant decrease in subendocardial blood flow occurred only in the presence of hemodynamic changes and was restored by phenylephrine. In this area, changes in regional myocardial function correlated most strongly with changes in subendocardial perfusion ($y = -0.17 + 1.70x - 0.58x^2$, $r^2 = 0.90$). In the stenotic region, oxygen extraction remained stable, but oxygen consumption decreased in parallel with reductions in regional myocardial function. In the normal region, oxygen consumption did not change, but oxygen extraction decreased with increasing isoflurane concentrations.**

Conclusions: **These results show that isoflurane is a coronary vasodilator able to induce a transmural redistribution of myocardial blood flow distal to an acute critical coronary stenosis. A true transmural steal, however, was not produced reliably in the absence of hemodynamic changes, suggesting that isoflurane either is only a moderate vasodilator, or that the decrease in subendocardial blood flow is offset by the negative inotropic action of the drug. When regional myocardial dysfunction distal to a severe coronary stenosis occurs, this correlates with decreasing subendocardial blood flow during isoflurane anesthesia, suggesting ischemia as the cause. (Key words: Anesthetics, volatile: isoflurane. Arteries, coronary: steal. Heart: coronary hemodynamics; coronary steal; myocardial oxygen consumption; coronary artery stenosis; regional myocardial performance.)**

ISOFLURANE causes coronary and systemic vasodilatation and impairment of coronary blood flow autoregulation.[1–3] In the presence of coronary stenosis severe enough to vasodilate distal arterioles maximally, isoflurane may produce ischemia by reducing arterial perfusion pressure.[4,5] Because subendocardial flow reserve is lost at higher perfusion pressures than is subepicardial flow reserve, subendocardial blood flow may be reduced even if subepicardial blood flow is increased, the condition described as transmural coronary steal.[6,7] This effect is particularly noticeable with drugs that have a predominant effect on small coronary vessels, the proposed site of action of isoflurane.[8,9] A reduction of myocardial blood flow distal to a stenosed coronary artery can lead to myocardial dysfunction when the oxygen supply to that part of the myocardium becomes inadequate to meet its metabolic needs.[10,11] Within the myocardium, changes in regional myocardial dysfunction correlate closely with reductions in subendocardial blood flow in both anesthetized and conscious dogs.[12,13]

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