Autoregulation and Heart Rate

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Autoregulation is an important physiological mechanism whereby blood flow to an organ is kept relatively constant as perfusing pressure changes. For all organs except the heart, autoregulation can be examined by changing aortic pressure and measuring the resulting organ flow. In the left ventricle, however, changing aortic pressure alters myocardial oxygen consumption, which itself alters myocardial blood flow. Therefore, autoregulation in the left ventricle can be tested only by cannulating the left coronary artery so that coronary perfusing pressure can be changed while aortic pressure (and myocardial oxygen consumption) remain constant. If this is done and coronary perfusing pressure is suddenly changed, coronary blood flow initially changes transiently in the direction of the pressure change (decreasing when pressure is lowered and increasing when pressure is raised) and then returns towards its control value.\(^1\)\(^-\)\(^5\) Autoregulation takes place in all layers of the left ventricle, thus preserving the approximate equality of flows per gram across the ventricular wall as coronary perfusing pressures change.\(^6\)\(^,\)\(^7\)

There is a limit to how low coronary perfusing pressure can decrease before autoregulation fails to keep coronary flow relatively constant. This limit is lower in the superficial subepicardial muscle than in the deep subendocardial muscle;\(^6\) in other words, as perfusing pressure decreases, autoregulation fails first in the subendocardium. Therefore, as coronary perfusing pressure decreases, flow first fails to match demand in the subendocardium, and subendocardial dysfunction occurs.\(^8\)\(^-\)\(^10\) Studies done in anesthetized dogs\(^6\) showed that autoregulation failed at a mean perfusing pressure of 70 mm Hg in the subendocardium and at 40 mm Hg in the subepicardium.\(^6\) An important study in conscious dogs by Canty\(^8\) confirmed both the earlier loss of autoregulation in the subendocardium and the reduced subendocardial muscle function that ensued but also showed that autoregulation in the subendocardium did not fail until coronary perfusing pressure decreased below 38–40 mm Hg. Evidently, the effects of anesthesia and the acute surgical preparation alter coronary regulatory mechanisms, and this difference must be considered whenever applying the results of acute animal experiments to humans.

In a subsequent study published in this issue of Circulation, Canty and his colleagues\(^11\) explored the effects of tachycardia on regional autoregulation in conscious dogs. They observed that when heart rate was increased from about 100 to 200 beats/min, the lower pressure limit of subendocardial autoregulation was increased from 38 to 61 mm Hg mean coronary perfusing pressure. The effect of tachycardia on the lower limit of subendocardial autoregulation is thought to be due to two mechanisms. Tachycardia increases myocardial oxygen consumption and so leads to an increase in autoregulated myocardial flow per minute.\(^12\) In addition, the reduction in diastolic time per minute due to the tachycardia decreases maximal coronary flow.\(^13\) Both these changes contribute to a reduction in coronary flow reserve.\(^14\)\(^,\)\(^15\) Several studies,\(^16\)\(^-\)\(^20\) some done also in conscious animals, have shown that tachycardia to 200 or 250 beats/min decreases maximal flow and flow reserve mainly in the inner half of the ventricular muscle. As shown by Hoffman,\(^14\) any decrease in coronary flow reserve is automatically associated with an increase in the pressure at which autoregulation fails, especially in the subendocardium. Canty and colleagues, however, have not only shown this principle to be true, but have also shown that subendocardial autoregulation fails at a perfusing pressure that is not far below normal.

What can we learn from this experimental animal study that might be of use in the management of human heart disease? First, the fact that these studies have been done in conscious dogs probably allows us to apply the results to conscious humans with little fear of serious error. Second, in normal hearts with normal perfusing pressures, a heart rate of 200 beats/min will reduce coronary flow reserve but will not by itself lead to subendocardial ischemia. If, however, coronary flow reserve is already reduced by anemia, hypoxemia, polycythemia, or acute cardiac dilatation, then a heart rate of 200 beats/min will reduce coronary flow reserve even more, and so is likely to produce subendocardial ischemia even at normal coronary perfusing pressures. Third, as Canty
et al.\textsuperscript{11} concluded, such a degree of tachycardia has ominous consequences for patients with atheroma of the extramural coronary arteries. Tachycardia increases myocardial oxygen demand, dilates the distal coronary bed, and decreases the pressure beyond the obstruction to the point that autoregulation fails and subendocardial ischemia occurs.\textsuperscript{21,22} In fact, another recent experimental study\textsuperscript{23} has demonstrated that in dogs with a chronic coronary stenosis, exercise-induced regional myocardial dysfunction can be eliminated by administering a bradycardic agent. Fourth, when there is left ventricular hypertrophy, tachycardia further reduces the already decreased coronary flow reserve, and subendocardial ischemia and dysfunction may occur at normal perfusing pressures.\textsuperscript{24,25} Finally, when an increase in contractility is added to any of the changes listed above, there is likely to be a further reduction of coronary flow reserve.\textsuperscript{25} Many of the deleterious changes described above may be seen after surgery, particularly cardiac surgery, and the risk of subendocardial ischemia and its consequences is high unless these variables are controlled and excessive tachycardia is prevented. Studies like the one reviewed here not only improve our understanding of coronary pathophysiology but also indicate that in the future a safe, controllable, selective bradycardic agent may need to be added to the armamentarium of the cardiologist.

\textbf{References}

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