Peripheral vasodilators in open heart surgery

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The concept of pharmacologically induced vasodilatation has gained a new place in the therapy of inadequate hemodynamic function. Vasodilators are now being used in the treatment of myocardial ischemia and low-output cardiac failure. There is now substantial evidence that myocardial ischemia and refractory heart failure can be improved by appropriate vasodilator therapy. These drugs are being applied in the operating room and in the intensive care units of many institutions. Many of these drugs are familiar to us, such as trimethaphan, but are now being used for a different purpose, *not* for deliberate hypotension, but instead to improve tissue perfusion and myocardial function. The hemodynamic effects are listed in the *Table*.

Treatment of myocardial ischemia with vasodilators

In the past 5 years, many studies in man and animals have demonstrated that vasodilators may decrease the extent of infarction as measured by creatine phosphokinase levels, ST-segment changes, and improved systemic hemodynamics. Nitroglycerin has been used in most of these studies, but some authors have used nitroprusside, phentolamine, trimethaphan, and other vasodilators. Even better myocardial preservation has been shown with

Table. Hemodynamic effects of vasodilators

Arterial vasodilatation
Reduction of afterload (systemic vascular resistance or mean arterial pressure)
Decreased ventricular wall tension
Decreased myocardial oxygen consumption
Decreased mitral or aortic regurgitant flow
Venous vasodilatation
Decreased preload of the right and left sides of the heart (pulmonary capillary wedge pressure, central venous pressure)
Decreased ventricular wall tension
Decreased myocardial oxygen consumption
Coronary vasodilatation
Increased myocardial oxygen supply
Redistribution of coronary blood flow to subendocardial areas

a combination of a vasodilator, such as nitroglycerin, and an alpha-adrenergic stimulant, such as methoxamine. The alpha-adrenergic agonist is added to maintain the coronary perfusion pressure. Intravenous nitroglycerin is being used by many in preference to other vasodilators because (1) the dose can be easily regulated when given in a dilute intravenous solution; (2) little overshoot and hypotension occur; (3) minimal increases in heart rate occur; (4) nitroglycerin has little known toxicity; and (5) the effects on coronary blood flow are known and, presently, there is no evidence of an intracoronary steal.[1–4]

In the perioperative period, intravenous nitroglycerin is used for the treatment of (1) intraoperative and postoperative hypertension; (2) coronary artery spasm; (3) ischemic changes on the electrocardiogram; (4) pulmonary hypertension; and (5) ischemic mitral regurgitation.

Afterload reduction in heart failure

A decrease in impedance to left ventricular ejection by afterload-reducing agents is used in the management of acute and chronic congestive heart failure and low output syndromes.[5–6] The mechanism of action of vasodilators in heart failure is fairly clear. Low output states are characterized by increased total peripheral resistance secondary to sympathetic tone to support blood pressure in the face of a decreasing cardiac output. With the increased resistance and increased outflow impedance, wall tension remains high during ejection, and, therefore, stroke volume is decreased. The vasodilators decrease the left and right ventricular outflow impedance and thus allow for increased stroke volume, decreased ventricular chamber size, and decreased ventricular work. Indeed, the increase in stroke volume maintains the blood pressure or even increases it in the face of a decreased total peripheral resistance. Guiha et al[5] have shown dramatic improvement in hemodynamics in patients with congestive heart failure, using nitroprusside infusions at very low doses, 10 to 50 μg/min. In addition, Chatterjee et al[7] have demonstrated the benefits of arterial vasodilators in patients with severe mitral or aortic regurgitation in whom drugs such as sodium nitroprusside greatly increase the forward cardiac output and reduce regurgitant flow.

Nitroprusside or phentolamine are the preferred drugs for patients with a low output syndrome. They reduce afterload more than they reduce the preload, leading to an increase in cardiac output. Nitroglycerin frequently has the opposite effect, a larger reduction in preload. All three drugs reduce pulmonary artery pressures.

The newest therapeutic interventions for patients with severe low output syn-

dromes are the combinations of inotropes and vasodilators.[8] Both an inotropic drug and a vasodilator tend to effect a change up and to the left on the Starling curve. Choosing the correct doses of both an inotrope and a vasodilator will move the patient further toward normal left ventricular function than either drug alone. Inotropic drugs such as dopamine, dobutamine, or epinephrine are frequently combined with nitroprusside.

References

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