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Sodium Nitroprusside-induced Improvement in Cardiac Function in Association with Left Ventricular Dilatation

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Patients with severely dilated ventricles occasionally have difficulty generating adequate cardiac output after extracorporeal circulation has been discontinued following open-heart surgery. The low cardiac output is partly due to the physiologic and biochemical alterations imposed by the extracorporeal circulation, the trauma of operation, and the anesthetic agents. However, in dilated ventricles, the abrupt change in geometric dynamics following correction of the causative defect may play a more important role in decreasing the cardiac output. The dimensional changes brought about by correction of the defect are important because of Laplace's relation, in which pressure is directly related to wall stress and inversely related to the radius of the intraventricular volume.¹ The latter mechanism is especially important when the dilatation is the result of pre-existing mitral insufficiency.

In compensated mitral insufficiency, the values for peak systolic wall stress and the ejection fraction compare favorably with those found in normal hearts. There is a marked difference in forward stroke volumes, since in mitral insufficiency, a fraction of stroke volume regurgitates back into the left atrium.² This regurgitant fraction allows the ventricular volume to be decreased. With this smaller volume, the ventricular wall stress can generate sufficient pressure to overcome

the afterload and eject an adequate stroke volume into the aorta.

When the regurgitant valve is replaced with a competent valve, the ventricular volume can no longer be vented into the left atrium. As a result of the large volume (radius), the peak systolic wall stress may not be able to generate enough pressure to eject an adequate stroke volume.

REPORT OF A CASE

The patient, a 56-year-old woman weighing 44 kg, had severe mitral insufficiency as a result of chronic rheumatic heart disease. She was intolerant to exercise and completely incapacitated. Physical findings included marked generalized cardiomegaly and pansystolic murmur. The electrocardiogram revealed atrial fibrillation with frequent premature ventricular depolarizations. The patient's medications were digoxin, furosemide, procainamide, and potassium. Cardiac catheterization revealed: right atrial pressure 7 torr; left atrial pressure 28 torr—V waves to 36 torr; right ventricular pressure 60/8 torr; left ventricular pressure 110/15 torr; pulmonary arterial pressure 60/25 torr; systemic arterial pressure 110/80 torr; pulmonary vascular resistance 329 dynes/cm/sec²; systemic vascular resistance 3,670 dynes/cm/sec². Cardiac output (Fick) was 1.7 l/min, with a cardiac index of 1.2 l/min/m². Angiography demonstrated mitral-valve regurgitation with a large left atrium. The left ventricle was dilated and hypertrophied, with normal filling compliance but poor contractility. Localized areas of dyskinesia could not be demonstrated. The coronary arteries were normal.

The patient was fully informed and consented to mitral-valve replacement.

Anesthesia was induced and maintained with morphine sulfate (1.5 mg/kg) and diazepam (0.5 mg/kg). Muscle relaxation was achieved with pancuronium bromide and ventilation was mechanically controlled. P_{aCO_2} was maintained at 40 torr and the inspired oxygen concentration was 100 per cent. Prior to bypass, 500 ml of blood were

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removed for subsequent re-infusion and replaced with 250 ml Protinate† and 1,000 ml of balanced salt solution.

Prior to bypass, blood pressure was stable at 100–110/70–80 torr, urine flow was 100 ml/hour, and there was no base deficit. A test with epinephrine (10 µg/min) resulted in increases in heart rate and blood pressure and an accelerated rise in the ascending limb of the arterial pressure waveform.

During cardiopulmonary bypass, the patient was cooled to 25°C. The average arterial pressure was 70 torr, the total urinary output was 110 ml, and serum potassium and base deficit were maintained within their normal ranges. The mitral valve was replaced with a Lillehei-Kaster prosthetic valve; the duration of bypass was one hour. On rewarming to 37°C, the heartbeat spontaneously converted to a sinus rhythm.

After bypass, left ventricular performance was poor in spite of the administration of calcium chloride (1 mEq/l extracellular fluid), 0.25 mg digoxin, isoproterenol, and epinephrine. More than 200 µg/min epinephrine or 20 µg/min isoproterenol induced ventricular irritability that could not be controlled by lidocaine.

Droperidol, a mild alpha-adrenergic blocker, in combination with epinephrine, increased cardiac output but did not promote urinary output in spite of concurrent administration of ethacrynic acid and the presence of increased left atrial pressure (38 torr).

Over the next 30 minutes, the patient's skin remained vasoconstricted, cold, and cyanotic, and the base deficit increased in spite of repeated correction. Cardiac output, measured with a magnetic flowprobe,‡ was found to be 1.8 l/min. Left atrial pressure was 38 torr and the systemic arterial pressure was 70–80/60–65. The prosthetic valve functioned properly without a significant gradient.

In an attempt to decrease the afterload, infusion of sodium nitroprusside, 100 µg/ml, was begun. The epinephrine infusion, 10 µg/ml, was continued. The sodium nitroprusside was administered at a rate sufficient to maintain a mean systemic arterial pressure of no less than 70–80 torr and the rate of epinephrine administration was adjusted to prevent an increase in left atrial pressure. Pulse pressure gradually increased. Left atrial pressure decreased to 24 torr. The ascending limb of the arterial pressure wave increased its slope and the waveform became normal. Urinary output increased to 200 ml/hour/m². The skin became pink and warm, with good capillary refill. Base deficit decreased and remained normal.

The rates of drug administration used to maintain these values were 20–30 µg/min epinephrine and 100–150 µg/min sodium nitroprusside.

† Plasma protein fraction (human), Hyland.

‡ Square-wave magnetic flowprobe, Carolina Medical Products, Inc., Model 321, accuracy ± 5 per cent.

After eight hours, epinephrine was discontinued, but the sodium nitroprusside infusion was continued for 30 hours to reduce the afterload. This allowed the ventricle to maintain an adequate output and prevented pulmonary wedge pressure from exceeding 24 torr.

The patient's condition continued to improve, as evidenced by easily controlled oxygenation, stable acid-base and electrolyte balance, and adequate urinary output. Cardiac output is adequate. The patient is now home and has returned to full activity.

DISCUSSION

In valvular heart disease, the ability of the ventricular wall to normalize peak systolic wall stress appears to be the limiting factor in determining whether the heart is compensated. If the heart is compensated, the hypertrophy of the left ventricular wall is appropriate and the peak systolic wall stress approaches normal values. This appears true for all valvular diseases whether volume overload, *e.g.*, dilatation, is or is not present.²

In mitral insufficiency, the left ventricle dilates. The dilatation is necessary to ensure adequate stroke volume, since a large part of the ejection fraction regurgitates back into the low-pressure left atrium. In order to compensate for the increased left ventricular volume (radius) which gradually decreases as contraction progresses, the left ventricle must hypertrophy appropriately to remain compensated. However, since a large part of the left ventricular volume goes into the left atrium, the hypertrophy is appropriate only so long as the relief area exists. When one removes this relief area, *i.e.*, makes the mitral valve competent, the large left ventricular diastolic volume remains. The wall stress necessary to increase the intraventricular pressure to overcome the afterload and eject an adequate stroke volume must increase immediately on correction in obedience to Laplace's law. The Frank-Starling relationship and the inotropic mechanism may be adequate to accomplish the new wall stress requirement and if so, stroke volume will be adequate. If not, the left ventricle will fail and output will decrease.

If the Frank-Starling relationship and the inotropic mechanism have been exceeded, then the ejection pressure requirement can be decreased by reducing the afterload.³

A mechanical method in clinical use is the counterpulsating intra-aortic balloon technique. In addition to the sudden reduction of the afterload by deflation of the balloon, additional hydraulic energy is supplied by inflating the balloon during diastole.⁴ This method, at present, is limited to a few medical centers. It also requires that the patient be kept on anticoagulants, which may be disadvantageous to some patients following open-heart surgery.

A pharmacologic method to decrease the afterload is to use an agent which decreases systemic vascular resistance. When one negates sympathetic tone either chemically or surgically, there is still inherent tone in the vascular muscle. One can further decrease this tone and hence, systemic vascular resistance, by using an agent which acts directly on the muscle. Sodium nitroprusside is such an agent. It is particularly advantageous because it is very potent, has a short duration of action, does not appear to cause tachyphylaxis, and has no effect on myocardial contractility.^{5,6}

By reducing the systemic vascular resistance with this easily controlled drug, one can reduce the afterload to the appropriate extent which produces the maximum stroke volume for the left ventricular contractile force available.

In summary, a case of advanced mitral insufficiency in which the left ventricle was contracting at its maximum capability is pre-

sented. When the geometric dynamics were suddenly changed by replacing the incompetent valve, the left ventricular wall was unable to generate enough wall stress to eject an adequate stroke volume. As a result of decreasing systemic vascular resistance and thereby the afterload with sodium nitroprusside, the ventricular wall stress capability was sufficient to eject an adequate output. During the subsequent 30 hours, the ventricle adjusted to the newly imposed geometric dynamics. The patient survived and is back to full activity six weeks after operation.

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A Simple Method for Monitoring Twitch Height

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We have developed an inexpensive way to monitor twitch height.

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MATERIALS AND METHODS

The materials required are a peripheral-nerve stimulator (Wellcome Peripheral Nerve Stimulator), a 20-inch length of 2-0 silk, a large curved endotracheal tube adapter, a metal stylet, a 3-inch plastic needle, a paper measuring tape, and an intravenous pole.