synthesis directly and limit any subsequent increase in total body levels of potassium,[5] but potassium-mediated mineralocorticoid production by the adrenal requires the presence of angiotensin II and does not occur during converting-enzyme inhibition.[6] Consequently, the excretion of potassium becomes notably impaired during captopril therapy, and therefore, serum potassium concentration varies directly with changes in dietary potassium. Hyperkalemia may result if potassium intake is increased (either by the administration of prescribed potassium supplements or by the use of potassium-containing salt substitutes) or the renal excretion of potassium is further impaired (by treatment with potassium-sparing diuretics or by decreases in glomerular filtration rate).[2] On the other hand, hypokalemia can occur in patients being treated with diuretic drugs if the renal disposal of potassium is restored by the withdrawal of converting-enzyme inhibition and dietary potassium is not supplemented. Although in most cases changes in serum potassium concentration are small, our findings indicate that in some patients, hypokalemia or hyperkalemia may be sufficiently severe to cause cardiac arrest. Hence, we recommend close attention to and reevaluation of dietary potassium intake and prescribed potassium supplementation whenever treatment with converting-enzyme inhibitors is instituted or discontinued.

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Balloon Angioplasty for Congenital and Rheumatic Mitral Stenosis

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Balloon dilation has been used successfully to treat many forms of congenital heart disease.[1] Inoue et al[2] reported in 6 adults the successful use of a specially designed balloon catheter to treat rheumatic mitral stenosis (MS). This report describes balloon angioplasty in 2 children with congenital MS and in 1 adolescent with rheumatic MS.

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Case 1: *P.P., an 18-year-old woman with rheumatic MS, underwent cardiac catheterization on January 28, 1984 because of severe dyspnea and chest pain. Catheterization revealed severe MS, mild aortic stenosis and regurgitation and mild pulmonary hypertension. After the patient was informed that balloon dilation of the mitral valve was an experimental but potentially therapeutic alternative to surgical mitral commissurotomy, she consented to the procedure. On February 6, 1984, using the percutaneous femoral approach, predilation hemodynamics and angiography were performed (Table I, Fig. 1). The balloon dilation was performed as follows: The left atrium was entered with a No. 8Fr Mullins transeptal sheath. After heparinization, a No. 7Fr wedge catheter was positioned into the left ventricle. A 0.035-inch J exchange (250 cm) guidewire was advanced through the wedge catheter into the left ventricular apex. The wedge catheter and sheath were removed and a No. 9Fr angioplasty cath-*

TABLE I Hemodynamic Data Before and After Angioplasty

	Age (yr)	Mitral Anulus Size (mm)	Balloon Size (mm)	PA s/d (m) (mm Hg)	LA (mm Hg) a-wave	LA (mm Hg) Mean	MVA (cm^2/m^2)	CI (liters/min/m^2)
Patient 1								
Before	17	28	20	48/28 (34)	36	24	0.95	3.2
After				30/19 (26)	20	18	1.25	2.9
Patient 2								
Before	13	18	15	70/36 (54)	39	32	0.70	3.3
After				52/37 (40)	28	21	0.85	3.3

After = after angioplasty; Before = before angioplasty; CI = cardiac index (thermodilution); LA = left atrial pressure; m = mean; MVA = mitral valve area (Gorlin and Gorlin formula); PA = pulmonary artery pressure; s/d = peak systolic/end diastolic.

TABLE II Two-Dimensional Echocardiographic and Doppler Data Before and After Balloon Angioplasty of the Mitral Valve

Time	Pressure ½ Time (ms)	Mitral[3] Orifice Size (cm^2)	Peak A-Wave Velocity (m/s)	Mean Pressure Drop Across Mitral Valve (mm Hg)
Patient 1				
Before	220	.96	2.3	12
Immed. after	210	1.0	1.7	7.5
15 months after	200	1.1	1.8	8.8
Patient 2				
Before	—	—	3.0	16
Immed. after	—	—	2.2	11.8
2 months after	—	—	2.2	9.0

Mean pressure drop = 4 (mean velocity)^2.

eter with a 20-mm balloon (Medi-Tech) was positioned across the mitral valve. The balloon was inflated to 4 atm for less than 10 seconds. During the procedure left ventricular and pulmonary artery pressures and heart rate were continuously monitored. The angioplasty catheter was then exchanged for the wedge catheter. Complete right- and left-sided heart hemodynamic values and a repeat left ventricular angiogram indicated a reduction in left atrial and pulmonary artery pressure without an increase in mitral regurgitation (Table I, Fig. 1). The patient was discharged from the hospital the next day. On her most recent clinic visit, 15 months after angioplasty, she reported a definite improvement in her symptoms. Her exercise tolerance on the treadmill (Bruce protocol) improved from 9 minutes to 11.5 minutes, with an increase in maximal heart rate response (80 to 171 beats/min vs 86 to 184 beats/min). In addition, the chest pain, which had been present during stages II and III of the prior treadmill, was no longer observed. She has also continued to show hemodynamic improvement of MS as assessed by Doppler examination[3] under resting conditions and similar heart rate (Table II).

Case 2: B.S., a 13-year-old girl with a repaired coarctation of the aorta, had mild subaortic stenosis and severe congenital MS. Because of severe dyspnea, March 20, 1985 she underwent cardiac catheterization, which revealed severe MS and pulmonary artery hypertension (Table I). Balloon angioplasty was performed. The technique was identical to that described for case 1, except that a 15-mm angioplasty balloon was used. Two months after angioplasty there has been mild improvement in symptoms and continued hemodynamic improvement documented on Doppler examination[3] under resting conditions and a similar heart rate (Table II).

Case 3: K.K., a 2-year-old boy, had severe congenital MS, coarctation of the aorta, patent ductus, left ventricular hypoplasia, subaortic stenosis and severe pulmonary artery hypertension. Because of severe exercise intolerance and differential cyanosis, mitral valve angioplasty was attempted on October 17, 1984. Using the aforementioned technique, a 12-mm angioplasty catheter was advanced across the atrial septum and into the left atrium. The catheter could not be advanced across the mitral valve, and angioplasty could not be performed. Two months after the attempted angioplasty a large left atrial thrombus was

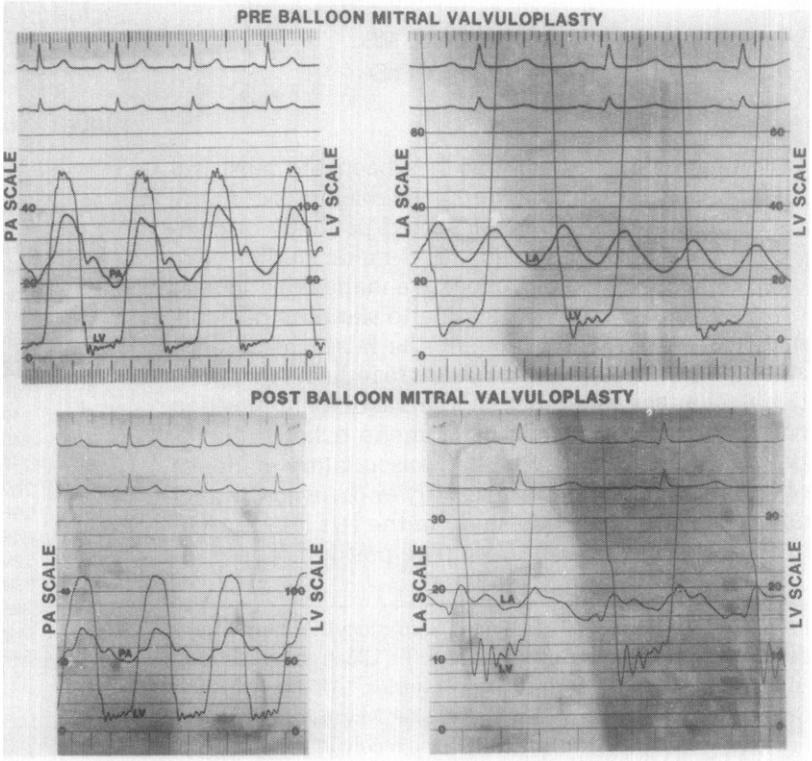

FIGURE 1. Left atrial (LA), left ventricular (LV) and pulmonary artery (PA) pressures from patient 1 before and after angioplasty.

identified and because of increasing symptoms surgical removal of the thrombus was attempted. The child, however, died from sepsis 2 months later. We do not know if the left atrial thrombus was related to the transseptal puncture, angioplasty balloon manipulation, low blood flow or polycythemia.

To our knowledge, this is only the second report of the use of balloon angioplasty to treat rheumatic MS and the first report of its use to treat congenital MS. In the 2 patients in whom we successfully performed mitral valvuloplasty, left atrial and pulmonary artery pressures decreased significantly and mitral valve area increased significantly, without any increase in mitral regurgitation. In addition, follow-up of 2 and 15 months has shown both clinical and hemodynamic improvement. Our results are similar to those previously reported by Inoue et al[3] and suggest that balloon angioplasty is feasible in both rheumatic and congenital MS. Despite these results 2 additional technical obstacles must still be overcome. First, although for an individual patient the ideal size of the angioplasty balloon is unknown, based on our experience with pulmonary stenosis[4] we believe a balloon approximately the size of the mitral anulus should be used. Only a 20-mm balloon was used in case 1, since this was the largest size available. Had we used a larger balloon or possibly 2 balloons side-by-side, a better result might have been achieved. Second, it was impossible to manipulate the large stiff angioplasty balloon across the stenotic mitral valve in case 3. It is conceivable that either with the aid of a large (12 to 15Fr) sheath or using a precurved catheter that it might have been possible to perform angioplasty in this patient. Although mitral valve angioplasty is, at present, an experimental procedure, if these technical problems can be solved, it is likely that balloon angioplasty may be an alternative for selected patients with MS to surgical mitral commissurotomy.

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Comparison of Supine and Left Lateral Decubitus Positions on M-Mode Echocardiographic Findings in Mitral Valve Prolapse by Auscultation

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M-mode echocardiography has been the most frequently used tool in confirming the diagnosis of mitral valve prolapse (MVP) in suspected patients. Insensitive or false-negative results in M-mode studies have been reported[1,2] and a provocative maneuver, such as postural change from the supine to standing position, has been reported to overcome the false-negative results.[2] There have been no reports to indicate that positional changes even within the recumbent position could change the results of M-mode echocardiography. In some patients who had auscultatory evidence of MVP, we found that apparently or equivocally negative results on M-mode study in the left lateral position became positive in the supine position. These selected patients were the subjects for further evaluation in this study.

Sixteen patients who met the M-mode criteria for MVP only in the supine position were selected for further evaluation. In addition, 10 patients without auscultatory evidence of MVP served as a control group. Clinical profiles of these 16 selected patients, including the presence or absence of midsystolic click or systolic murmur are listed in Table I. Left ventricu-

TABLE I M-mode Echocardiographic Findings and Patient Data

Pt.	Age (yr) & Sex	M-Mode Echo: Supine	M-Mode Echo: Left Lateral	LVEDD (mm): Supine	LVEDD (mm): Left Lateral	Click	Murmur
1	15F	+	±	43	46	+	+
2	16F	+	0	47	49	+	+
3	17F	+	±	47	50	+	0
4	20F	+	±	40	44	0	+
5	21F	+	±	45	45	0	+
6	21F	+	0	49	49	+	+
7	24F	+	0	40	40	+	+
8	25F	+	±	43	43	0	+
9	27M	+	0	46	48	+	0
10	28F	+	0	49	52	0	+
11	31F	+	±	47	49	+	+
12	32M	+	±	46	49	+	0
13	33M	+	0	48	49	+	+
14	38F	+	±	43	47	+	0
15	46M	+	0	47	46	+	0
16	54F	+	±	50	54	0	+
Mean ± SEM				45.6 ± 0.9	47.5 ± 0.9 ($p < 0.001$)		

Echo = echocardiography; LVEDD = left ventricular end-diastolic dimension; SEM = standard error of the mean; + = positive; 0 = negative.

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Manuscript received February 28, 1985; revised manuscript received June 5, 1985, accepted June 7, 1985.