Balloon-Induced Right Ventricular Outflow Obstruction

A New Approach to Control of Acute Interventricular Shunting after Myocardial Infarction in Canines and Swine

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SUMMARY Current management of ventricular septal defect (VSD) after myocardial infarction (MI) is aimed at improving left ventricular (LV) performance by afterload reduction as a means of hemodynamic stabilization or shunt control. The current investigation was undertaken to determine whether primary manipulation of right ventricular (RV) performance by afterload enhancement was an effective means of reducing MI-VSD shunting.

POST-MYOCARDIAL infarction ventricular septal defect (VSD) is a rare but serious complication of myocardial infarction. Its incidence is 0.5-1.0% of all infarcts and this condition accounts for about 2% of all deaths due to myocardial infarction. Although there have been a few reports of long-term survival, this lesion usually is fatal, with up to 24% of the patients dying within 24 hours and 80-90% within 2 months. Earlier reports of surgical therapy have recommended waiting up to 2 months, if possible, before undertaking closure of the VSD. Because of the very high early mortality rate from post-myocardial infarction VSD, it has been suggested that if greater salvage is to occur, it would be necessary to operate on these patients earlier, during the acute phase. Recent experience with such an approach reveals a distressingly high mortality rate of 70-100%. Subsequently, however, improved operative techniques plus aggressive supportive therapy, including use of the intra-aortic balloon pump, have yielded more encouraging results from early operative intervention. Most efforts at temporary hemodynamic support have been aimed at improving left ventricular (LV) performance and reducing systemic afterload to combat hypotension, shock, and pulmonary vascular congestion.

The purpose of the present paper is to report a new mode of therapy for temporary stabilization of cardiovascular function in the presence of an acute VSD, i.e., primary manipulation of right ventricular (RV) afterload. In these studies, it was demonstrated that increasing RV outflow impedance by inflating a balloon in the pulmonary artery (PA) can significantly reduce VSD shunt flow. It is suggested that the limiting factor in the use of this form of intervention may be a deterioration of RV function. By using an inotropic agent (dopamine) that also reduces LV afterload, it was found that shunt flow could be further reduced while RV and LV function were improved.

Methods

To test the hypothesis that primary RV afterload manipulation, with or without inotropic support, could control ventricular shunting, it was necessary to construct an experimental model in which acute ventricular shunting could be measured and controlled and critical hemodynamic variables continuously monitored. This was accomplished by using an external left ventricle to right ventricle...
shunt with an interposed flowmeter (Fig. 1). Two series of studies using the external shunt are described: the first in dogs and the second in swine. The data from each model are presented and analyzed separately.

CANINE MODEL OF VSD

In the studies on canines, five mongrel dogs with an average weight of 20.2 kg were anesthetized with pentobarbital (30 mg/kg, iv) and ventilated with a variable speed, volume-cycled respirator (Harvard Apparatus) via an endotracheal tube. A central venous (right atrial) catheter was placed via the jugular vein and a central aortic catheter inserted from the brachial artery. Through a sternotomy, the heart and great vessels were exposed (Fig. 1). A snuggly fitting electromagnetic blood flow transducer (Statham Flo-Probe) was placed around the ascending aorta, and the right and left ventricles were selectively catheterized with 8F pigtail catheters via the respective atrial appendages. A 7F balloon-tip catheter with separate end-hole then was inserted into the RV outflow tract and the balloon was positioned in the main PA immediately above the pulmonic valve. This catheter was firmly secured with opposing mattress sutures. Finally, we used specially designed metal cannulas with snugly fitting obturators and cannulated the right and left ventricles through their free walls near their apices. The cannulas were firmly secured with Teflon-felt buttressed sutures. With the obturators removed, the cannulas were attached to Silastic tubing with an interposed 10-mm cannulating blood flow transducer (Statham Flo-Probe model SP7517). In this manner, an external left ventricle to right ventricle shunt could be created by opening a clamp to allow free communication between the right and left ventricles, and shunt flows could be measured directly by the flow transducer and controlled by a screw clamp applied to the Silastic tubing. All pressures were measured with Statham P23Db pressure transducers. Recordings of flow and pressure were made at film speeds of 25 and 100 mm/sec with an Electronics for Medicine DR-12 recorder with rapid writer attachment.

At the end of each study the flow transducers and flowmeters were calibrated with blood in vivo by connecting the RV shunt cannula directly to a precalibrated peristaltic pump (Sarns) with a 200°. 7-inch perfusion head. The left atrium was clamped to prevent backflow and the great vessels arising from the aorta were ligated up to and including the left subclavian. To construct accurate calibration curves for each experiment the descending aorta flow was directed into a beaker and heparinized blood was pumped through the shunt and aortic transducers at four different flow rates that bracketed the flows recorded during the study.

SWINE MODEL OF VSD

In this series of studies, two phases were used. In the first, 11 swine of either sex, with an average weight of 46.9 kg, were used. This change in experimental animal was undertaken to demonstrate efficacy of the proposed interventions in more than one experimental model and because of the similarity of the swine heart to the human heart. These swine were prepared in a manner identical to that used for the canine model except that a tracheostomy was used for ventilation rather than an endotracheal tube. The heart and great vessels were exposed through a sternotomy. Cannulation procedures and creation and control of shunting were as previously described. Whereas the balloon catheter in the canine model required an inflation of only 1–2 ml for hemodynamic effect, the swine, because of its larger size, required inflation of 2–4 ml. In all other respects, including instrumentation and calibration, the swine model was prepared in a manner identical to that used for the canine model.

In the second phase we used four swine of either sex, with an average weight of 37.5 kg. Initial preparation of the model was identical to that described above. In addition, the left anterior descending coronary artery was dissected free and a ligature was placed around it at its midportion. This vessel was occluded prior to initiation of the protocol described below in which epicardial S-T segment mapping was used to evaluate ischemic injury during the experimental intervention.

EXPERIMENTAL PROTOCOL

In the canine model direct measurement of right atrial (RA), RV, LV, and systemic arterial pressures plus aortic and shunt flows was possible. Pulmonary to systemic flow ratio (Qp/Qs) was calculated as (aortic + shunt flow)/aortic flow, and systemic vascular resistance in mmHg/liter per min was derived by the formula: [mean aortic pressure – mean atrial pressure (mm Hg)]/aortic flow (liters/min).

Frequent determinations of blood gases were made throughout the protocol and used to ensure acid-base stability and adequate oxygenation in order to obviate any changes in vascular resistance or ventricular performance secondary to these variables.

After the initial instrumentation, a period of 20–30 minutes was allowed to elapse. When all hemodynamic parameters had reached a stable plateau the clamp was opened and the effects of acute ventricular shunting were observed. After a period of approximately 20 minutes, to allow either hemodynamic stabilization or all hemody-
nomic parameters to reach a stable level for at least 5 minutes, the pulmonary balloon was inflated. The effects of this inflation on ventricular pressures, systemic pressure, and shunt and systemic blood flow were continuously observed. When shunt flow had been diminished maximally without significantly affecting systemic flow, balloon inflation was halted. At this point, all pressures and flows were recorded at amplifications necessary to maximize accuracy of readings. These determinations then were repeated at 10-minute intervals over the next hour. Subsequently the protocol was repeated or the animal was killed. In no instance was right to left shunting observed.

In the first swine model, a similar protocol was followed in terms of pressure and flow measurements and PA balloon inflation. Also, the period of balloon inflation before a subsequent intervention or balloon deflation was limited to 20 minutes instead of 60 minutes because of the more complex protocol. In addition we studied the effects of infusion of dopamine at varying rates, with or without PA balloon inflation. Studies with and without balloon inflation and/or dopamine were performed so that each animal served as its own control. The sequence of balloon inflation followed by added dopamine infusion or balloon deflation with dopamine infusion alone was randomized. Dopamine was infused with a variable speed infusion pump (Harvard Apparatus) at rates of 24, 60, and 120 μg/min. At each infusion rate, sufficient time was allowed for all pressures and flow readings to be stable for at least 5 minutes before recordings were made. After recording, the infusion pump was turned off and all values were allowed to return to their stable, preinfusion levels before moving to the next infusion rate (usually 10–12 minutes).

In the second phase of experiments on the swine model, the mid-left anterior descending coronary artery was occluded before initiating the protocol outlined above for phase 1. In addition to the interventions and determinations described above epicardial S-T segment maps were obtained as described by Maroko and Braunwald from five to six sites in the infarcting, peri-infarcting, and normally perfused areas of the LV myocardium for each stage of the protocol. Since S-T segment elevation was present from 5 to 6 minutes, the pulmonary balloon was inflated. The effects of inflation on ventricular pressures, systemic pressure, and shunt and systemic blood flow were continuously observed. When shunt flow had been diminished maximally without significantly affecting systemic flow, balloon inflation was halted. At this point, all pressures and flows were recorded at amplifications necessary to maximize accuracy of readings. These determinations then were repeated at 10-minute intervals over the next hour. Subsequently the protocol was repeated or the animal was killed. In no instance was right to left shunting observed.

In the canine model, releasing the Silastic tubing clamp created shunt flows that averaged 783 ± 148 ml/min as read by the external electromagnetic flow transducer (range 280–1,290 ml/min) and heart rate remained unchanged at 141/min. RV systolic pressure rose from 32 ± 2.3 to 47 ± 3.3 mm Hg (P < 0.01) and RV end-diastolic pressure increased from 7.7 ± 1.5 to 12.1 ± 2.4 mm Hg.

**Results**

**CREATION OF VSD**

In the canine model, releasing the Silastic tubing clamp created shunt flows that averaged 783 ± 148 ml/min as read by the external electromagnetic flow transducer (range 280–1,290 ml/min) and heart rate remained unchanged at 141/min. RV systolic pressure rose from 32 ± 2.3 to 47 ± 3.3 mm Hg (P < 0.01) and RV end-diastolic pressure increased from 7.7 ± 1.5 to 12.1 ± 2.4 mm Hg.

**FIGURE 2** Data from five dogs demonstrating changes in right ventricular (RV) systolic pressure (panel A), RV end-diastolic pressure (panel B), aortic flow (panel C), left ventricular (LV) systolic pressure (panel D), LV end-diastolic pressure (panel E), and aortic mean pressure (panel F) from control state (C) to opening of LV-RV shunt (S), and inflation of pulmonary artery balloon (B). Comparisons are between C and S and S and B. Asterisks indicate level of statistical significance.

(P < 0.05) (Fig. 2). Also, LV end-diastolic pressure rose from 5.1 ± 2.1 to 7.7 ± 1.9 mm Hg (P < 0.05), whereas aortic mean pressure fell from 95 ± 5 to 80 ± 8 mm Hg (P < 0.05) and LV systolic pressure and aortic mean flow fell slightly but insignificantly. These hemodynamic changes are consistent with a left to right ventricular shunt with a value of Qs/Qo averaging 2.26 ± 0.43 to 1 (range 1.40 to 4.23 to 1).

**PA BALLOON INFLATION**

Inflation of the PA balloon caused a significant fall in shunt flow from 783 ± 148 to 243 ± 74 ml/min (P < 0.01), whereas Qs/Qo fell from 2.26 ± 0.43 to 1.38 ± 0.17 to 1 (P < 0.05). Aortic flow did not change significantly with balloon inflation (Figs. 2 and 3). Other hemodynamic findings of note that occurred during PA balloon inflation included an insignificant fall in heart rate to 135/min and a further significant increase in RV systolic pressure (54.9 ± 3.8 mm Hg, P < 0.05) and RV end-diastolic pressure (14.9 ± 2.1 mm Hg, P < 0.01) (Fig. 2).
PA BALLOON INFLATION WITH INOTROPIC SUPPORT

In the swine model, we observed virtually identical changes secondary to release of the external ventricular shunt clamp and inflation of the PA balloon catheter (Fig. 4). RV systolic and end-diastolic pressures rose significantly with shunting and showed a further significant increase with balloon inflation. LV end-diastolic pressure also rose significantly with shunting but fell slightly although insignificantly with balloon inflation. Differences from the data for dogs included significant increases in heart rate with shunting and balloon inflation (101/min during control to 111/min with shunt open. P < 0.01 and 117/min with balloon inflated. P < 0.05). a significant fall in aortic flow with shunting (1.524 ± 117 to 1.315 ± 98 ml/min. P < 0.01), and significant increase in LV systolic pressure with balloon inflation (110 ± 6 to 120 ± 7 mm Hg. P < 0.05). Also, although aortic mean pressure fell in the swine model as it did in the canine model, this fall was not significant.

The addition of the dopamine infusion to balloon inflation caused an increase in RV and LV systolic pressures with a decrease in RV and LV end-diastolic pressure; these effects were most pronounced at the highest infusion rates (Fig. 4A, B, D, and E). In addition, aortic flow increased significantly with each increment in dopamine infusion rate (Fig. 4C), as did heart rate (117/min to 123/min. P < 0.05, to 124/min. P < 0.05, to 129/min. P < 0.005), while aortic mean pressure remained stable (Fig. 4F). Dopamine also caused a decrease in systemic vascular resistance in parallel with the increase in aortic flow (see Fig. 6A) and caused further reduction in shunt flow and Qo/Qs (see Fig. 6C).

THE EFFECTS OF DOPAMINE ALONE ON SHUNT FLOW

The effects of dopamine alone on acute left to right ventricular shunting also were investigated in the swine model. The results with acute ventricular shunting were identical to those reported for the preceding experiments. as evident in Figure 5. with the exception that the heart rate increase from 109 to 112 beats/min was not significant. RV systolic and end-diastolic pressures rose (Fig. 5A and B). as did LV end-diastolic pressure (Fig. 5E), while aortic flow fell significantly and LV systolic and aortic mean pressure remained stable (Fig. 5C, D, and F). With the addition of the dopamine infusion alone, at the same rates described above and without PA balloon inflation. heart rate increased to 115, 120, and 123 beats/min at infusion rates of 24, 60, and 120 µg/min, respectively. The latter two values are significant at P < 0.05. Significant falls in RV end-diastolic pressure (P < 0.01) and LV end-diastolic pressure (P < 0.05) occurred at the higher infusion rates. coincident with moderate but insignificant increases in respective ventricular systolic pressure (Fig. 5A, B, D, and E). Again, significant increases in aortic flow occurred at higher infusion rates (Fig. 5C) and systemic vascular resistance fell significantly and in parallel with these increases in aortic flow (Fig. 6B). The influence of dopamine alone on shunt flow and Qo/Qs is shown in Figure 6D. where it is evident that with each increment in dopamine infusion rate shunt flow and Qo/Qs fell significantly and in parallel. However, on comparing Figure 6C and D. it is evident that shunt flow and Qo/Qs are not influenced as greatly by dopamine alone as by PA balloon inflation alone or PA balloon inflation in conjunction with dopamine infusion.

CORONARY OCCLUSION AND EPICARDIAL S-T SEGMENT ELEVATION DURING PA BALLOON INFLATION AND DOPAMINE INFUSION

Epicardial S-T segment mapping during coronary occlusion was used as a measure of ischemic injury following acute ventricular shunting with PA balloon inflation and dopamine infusion. Following coronary occlusion, hemo-
dynamic changes with acute ventricular shunting. PA balloon inflation and dopamine infusion were similar to those described above except that LV end-diastolic pressure was higher than in the animals without coronary occlusion (LVED preshunt = 8.8 ± 1.8 mm Hg; LVED postshunt = 11.8 ± 1.9 mm Hg). The effects on epicardial S-T segments of acute ventricular shunting, PA balloon inflation, and dopamine infusion at 60 and 120 μg/min in the setting of coronary occlusion are presented in Table 1. Compared to control values (after coronary occlusion but before any other interventions), the summed S-T segment elevation was unchanged by acute ventricular shunting. With the addition of PA balloon inflation either alone or with infusion of dopamine at 60 and 120 μg/min, summed epicardial S-T segment elevations increased but the increase was significant only at the lower rate of dopamine infusion.

**Discussion**

**CURRENT APPROACHES TO TREATMENT OF ACUTE VSD CAUSED BY MYOCARDIAL INFARCTION**

With the proven ability to salvage patients early in the course of post-myocardial infarction VSD, it has become of paramount importance to intervene aggressively to combat shock and maintain hemodynamic stability. The ready availability of bedside catheterization to confirm the suspected diagnosis has simplified prompt and accurate diagnosis of this highly lethal condition.19, 20 For the small subgroup of patients that do not develop serious congestive failure, hypotension, and shock, intensive medical management may suffice and allow full tissue healing to occur postinfarction before operative correction of the defect. However, the larger group of postinfarction VSD patients do experience abrupt hemodynamic deterioration and it is this group that needs urgent assistance.

To date, attempts to support these patients have been those used to treat cardiogenic shock of any etiology. Pressor and inotropic agents alone have had little success in acute stabilization; this has been the case in cardiogenic shock in general.20 Pressor agents help to increase central aortic pressure and, therefore, coronary perfusion pressure. However, the elevated LV pressures increase left to right shunting through the VSD and may result in a critically reduced systemic flow plus increased pulmonary congestion and hypoxia. Conversely, vasodilator therapy, by reducing systemic vascular resistance, should assist forward flow and LV ejection and reduce left to right shunting. Although this mode of therapy has met with some success in acute myocardial infarction for control of congestive symptoms, recurrent pain, and potential reduction in infarct size,27, 28 its use in cardiogenic shock reduces coronary perfusion pressure and may promote extension of infarction.

Use of mechanical assist devices, such as the intra-aortic balloon pump (IABP) has met with slightly greater success in stabilizing patients with post-myocardial infarction VSD.20, 21 IABP has been shown to reduce left to right shunting by up to 20% while improving systemic flow and reducing LV filling pressures.21 This has been achieved by a reduction in resistance to LV ejection by balloon deflation during systole with the added benefit of raising coronary perfusion pressure during diastole. Although useful, this approach requires the existence of a skilled cardiovascular surgical team for IABP insertion and operation such as usually is present only at major medical centers.

The primary difficulty in post-myocardial infarction VSD is the presence of a low resistance venting system for the high pressure left ventricle. Thus, the already compromised left ventricle must pump not only an adequate aortic flow, but also the large flow now traversing the VSD. In most patients it fails to do this and shock results. There is a critical reduction in systemic blood flow as well as acute flooding of the lungs, with resultant pulmonary edema and hypoxia. In patients with a congenital VSD with conge- nitive failure and evidence of decreased systemic flow, these problems have been attacked in the past by banding of the PA for temporary reduction of shunting.29 This, however, involves thoracotomy, and it has been reasoned that if such were to be undertaken, primary repair of the VSD could be performed. This is being done in many centers for infants with large ventricular septal defects. However, the fragile hemodynamic status of adult patients with post-myocardial infarction VSD makes preoperative stabilization highly desirable.

**BALLOON-INDUCED RV OUTFLOW OBSTRUCTION**

The purpose of the present studies was to determine whether balloon inflation in the PA could accomplish such stabilization in an experimental model of acute VSD by increasing the impedance to RV ejection. In our preparation, an external VSD was created by cannulating the right and left ventricular free walls and connecting the cannulas through an interposed flow transducer. Thus, when the tubing clamp was opened, flow through the ventricular shunt occurred and could be measured accurately. That we...
had created an acute ventricular shunt that was similar to the clinical situation reflected by the rise in RV systemic and end-diastolic pressures as well as LV end-diastolic pressure with a concomitant fall in systemic flow. These changes are consistent with an acute left to right shunt, and the absence of decline in systemic oxygen saturation or pH indicated that there was no right to left shunting. Pulmonary to systemic flow ratios of 1.35–4.23 to 1 were achieved and these values are in the range commonly encountered in acute ventricular shunting post-myocardial infarction. RV outflow impedance was increased by inflating a balloon-tipped catheter positioned in the main PA. The balloon was inflated with saline to prevent compression, as would occur with a gas inflation, and this inflation was effective in increasing RV outflow impedance and in reducing left to right shunt flow. As is evidenced in Figures 2 and 3.

Thus, mechanical impedance to RV ejection is, indeed, effective as a means of reducing shunting at the ventricular level. However, as is suggested by the increased RV systemic and end-diastolic pressures (Figs. 2 and 4), a deterioration in RV function might limit such mechanical intervention. Therefore, an inotropic agent was needed which would provide support for the right ventricle without increasing LV afterload and increasing left to right shunting.

PHARMACOLOGICAL MAINTENANCE OF RV FUNCTION

Dopamine, an endogenous catecholamine and the immediate precursor of norepinephrine, would appear to be a reasonable agent to stabilize RV performance in this situation. Since it also has the capacity to reduce LV afterload. Dopamine increases myocardial contractility and heart rate by direct action on the myocardial β-adrenergic receptors and also releases norepinephrine from myocardial sites. In addition, it causes vasodilatation of the mesenteric and renal vascular beds, presumably by affecting specific dopaminergic receptors. Larger doses, however. recruit α-adrenergic receptors and cause vasoconstriction in all vascular beds. Thus, the net effect on peripheral resistance and systemic blood pressure will depend on the dose. When dopamine has been used primarily to combat hypotension in shock, larger doses, generally in excess of 2 μg/kg per min. have been used. On the other hand, doses in refractory congestive heart failure, where the aim was to reduce LV filling pressures and promote systemic flow, have generally been smaller, ranging from 0.5 to 1.0 μg/kg per min for recommended starting doses and from 1.0 to 3.0 μg/kg per min as average effective therapeutic doses.

That dopamine was effective in these studies as an inotropic and systemic vasodilating agent is evident in Figures 4, 5, and 6. The doses of dopamine used in these studies closely correspond to those used in refractory congestive failure, since 24. 60. and 120 μg/min correspond to average infusion rates of 0.51. 1.28. and 2.56 μg/kg per min. respectively, in the swine model. Although the lowest infusion rate produced little change, the higher two rates generally produced dramatic improvement in hemodynamic state. Positive inotropic effect was demonstrated by a rise in ventricular systolic pressures with a concomitant fall in end-diastolic pressures. That dopamine alone, by reducing afterload, can improve systemic flow and reduce shunt flow is clearly shown in Figure 6. This demonstrates that as systemic vascular resistance falls, aortic flow rises and shunt flow and Qc/Qs decline. However, the degree of shunt reduction and improvement in aortic flow is not as great as when RV afterload is first increased by balloon inflation in the PA (Fig. 6).

EFFECT OF INTERVENTIONS TO REDUCE VSD SHUNT FLOW ON EXTENT OF ISCHEMIC INJURY

We recognize that the positive inotropic effect of dopamine might have a deleterious effect on the extent of ischemic injury during myocardial infarction. Therefore, a second swine model was evaluated in which an acute VSD was superimposed on occlusion of the mid-left anterior descending coronary artery. (PA balloon inflation did not significantly increase the extent of summed S-T segment elevation in the ischemic area.) As anticipated, dopamine infusion increased S-T segment elevation, although this change was of statistical significance only at a rate of 60 μg/min. Although the hemodynamic effects of dopamine on ventricular performance and shunt flow were clearly beneficial (Figs. 4, 5, and 6), this enhancement of ischemic injury and an accompanying increase in heart rate must be carefully considered in evaluating the net result. It may be that dopamine administration in the presence of acute myocardial infarction and VSD acts in a manner similar to that of digitalis when myocardial infarction is accompanied by LV failure.
hemodynamic effects may partially counterbalance the unfavorable increase in myocardial oxygen consumption. It would be of interest to evaluate other pharmacological agents. Since LV systolic blood pressure increased with PA balloon inflation (Fig. 4), perhaps systemic afterload-reducing agents, such as nitroprusside, would be of help.

Our data support the thesis that primary manipulation of RV afterload provides an effective means of controlling acute ventricular shunting. Further, the addition of low infusion rates of dopamine provides modest isotropic support for the ventricles while promoting a further decrease in shunting and increase in systemic flow. This increased flow secondary to presumed vasodilation is of particular importance in the renal and mesenteric bed thought to contain dopaminergic receptors because these are the beds most likely to be compromised in the setting of cardiogenic shock. Despite this pharmacological benefit, however, mechanical manipulation of RV impedance offers the most dramatic means for acute shunt control.

CLINICAL IMPLICATIONS

A number of other factors must be considered if one wishes to extrapolate the data from the experimental laboratory to the clinical setting of a patient with post-myocardial infarction VSD. Since the PA in the human is larger than that in either of the experimental models used, a larger balloon would be necessary to manipulate RV impedance in the clinical setting. On the other hand, it might be more feasible to use two smaller balloons positioned in the right and left PA rather than a single large balloon in the main PA. Since pulmonary infarction secondary to wedging of currently available flow directed balloon tip catheters has been reported, it clearly would be necessary to monitor pressures both at the tip of the PA balloon catheter(s) and in the right ventricle simultaneously in order to continuously and accurately assess the influence of balloon inflation on RV performance and to prevent pulmonary infarction by immediately detecting wedging of the catheter in a major pulmonary vessel. This technique of balloon inflation in the main PA has been used in the past in children with VSD to assess pulmonary arteriolar reactivity. The "test banding" was accomplished by inflating a balloon-tipped catheter in the main PA and, among other changes, a decrease in PA pressure with a concomitant increase in systemic pressure was noted and thought to suggest reduced transfer of volume across the interventricular connection. This test in children is used only acutely, however, and the present data suggest that a longer duration of inflation may well be feasible without risking acute RV decompensation; this is particularly likely if appropriate inotropic/systemic resistance pharmacology is employed. Although data regarding chronic use of such a technique are not presently available and cannot be extrapolated from the present study, this information is clearly needed. Ideally, however, the intervention described in this report might be used in smaller centers with catheterization facilities to bring about acute stabilization of a patient with post-myocardial infarction VSD. Transfer of the patient to a major medical center then could be undertaken for further diagnostic or therapeutic interventions, including intra-aortic balloon counterpulsation or acute surgical correction of this defect.

We believe, therefore, that the studies presented support the thesis that primary RV afterload manipulation offers a feasible means of temporarily controlling acute left to right ventricular shunting. With the addition of appropriate pharmacological interventions, the limitations imposed by potential deterioration of RV function may be largely overcome. Further testing and careful clinical trials, including the investigation of other pharmacological agents besides dopamine, are clearly indicated for this technique; however, before it can be safely used in this high mortality complication of myocardial infarction.

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References


Renal Cortical Blood Flow Distribution in Obstructive Nephropathy in Rats

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SUMMARY To examine the role of intrarenal hemodynamics in obstructive nephropathy, we determined cortical blood flow distribution (CBFD) in rats with bilateral ureteral occlusion (BUO) and unilateral ureteral occlusion (UUO) during and after release of obstruction. Prior to release of obstruction of 24 hours' duration, we found that outer cortical perfusion decreased by 20 ± 5% in both BUO and UUO rats. Furthermore, one hour after release of BUO, there was rapid normalization of CBFD associated with a modest return of glomerular filtration rate (GFR), an almost complete return of renal blood flow (RBF), and a marked postobstructive diuresis. In contrast, after release of UUO, we observed that outer cortical perfusion remained decreased by 21 ± 3%, both GFR and RBF remained markedly depressed, and no diuresis occurred. These data demonstrate (1) marked ischemia of the outer cortex in both BUO and UUO during obstruction, (2) a rapid return of CBFD to a normal pattern after release of BUO, but (3) persistent outer cortical ischemia following release of UUO.

DESPIE availability of an adequate and reproducible animal model, the mechanism responsible for the acute renal injury resulting from obstruction of the collecting system and the factors involved in the recovery process following release of the obstruction remain obscure. It has been well documented in the rat that a postobstructive diuresis follows release of bilateral ureteral occlusion (BUO) of 24 hours' duration but does not occur following release of unilateral ureteral occlusion (UOO) in the presence of an intact contralateral kidney. This observation offers a unique opportunity to investigate those factors that may be involved in obstructive nephropathy by studying rats that have been subjected to a similar renal injury, i.e., complete ureteral occlusion, but may have a different sequence or pattern of recovery.

Recent studies have focused attention on the role of renal hemodynamics and renal vascular resistance in obstructive nephropathy. In these studies, changes in the pattern of cortical perfusion have not been completely appreciated because (1) animals were studied only after release of obstruction but not during the period of obstruction, and (2) a comparison of changes in animals with unilateral occlusion and animals with bilateral occlusion has not been made using the same techniques for determination of cortical blood flow distribution (CBFD). Consequently, the present study was undertaken to determine (1) whether the pattern of cortical perfusion prior to release of obstruction was similar in rats with UUO and BUO; (2) whether alterations in renal CBFD are associated with the diuresis that follows relief of bilateral ob-
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