Reaction of the Myocardium to Cryosurgery: Electrophysiology and Arrhythmogenic Potential

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SUMMARY The acute and chronic electrophysiological effects of a cryolesion produced in the left ventricle were studied in six dogs. All dogs had frequent ventricular premature beats (VPB) and five of six dogs had ventricular tachycardia during the first 4 days after the cryolesion; only one of the six dogs continued to have VPBs after 1 week, and this dog had identical VPBs before the creation of the cryolesion. Neither control dog had VPBs. Two additional dogs underwent epicardial and tranmural mapping studies immediately after production of a cryolesion. VPBs in these animals were shown to originate at the border of the cryolesion. Epicardial activation sequence during normal sinus rhythm was not altered by the chronic cryolesion. The border zone of the chronic cryolesion was sharply demarcated with normal potentials recorded outside of the lesion and “extrinsic” potentials recorded within.

SURGICAL TREATMENT has been demonstrated to be an alternative approach to management of intractable arrhythmias in selected patients.1,3 A successful surgical outcome depends on accurately locating the site of abnormal impulse formation or conduction by mapping techniques and subsequently ablating it. Several approaches are available for the ablation of regions of myocardial tissue responsible for alterations of rhythm.4,7 The most commonly used technique in ventricular tachycardia has been a combination of surgical excision or transection of the area involved.2,8,9 In addition to requiring the use of cardiopulmonary bypass, surgical excision and ventriculotomy disrupt anatomic continuity of structure and may have as yet unexplored consequences on ventricular function.

We have previously described a cryosurgical technique for the controlled ablation of selected myocardial tissues that does not require the use of cardiopulmonary bypass.10-13 Here, we describe the electrophysiological properties of a controlled cryosurgical lesion in the canine left ventricle.

Methods

Initial Studies

Six mongrel dogs weighing 18–20 kg were used in this part of the study. The dogs underwent ambulatory Holter monitoring for 24 hours before surgery and each was given a standard 12-lead ECG. For the initial surgical procedure, the dogs were anesthetized with intravenous sodium pentobarbital (30 mg/kg) and ventilated with room air through an endotracheal tube attached to a Harvard pump respirator. The heart was exposed through a left thoracotomy in the fifth interspace and suspended in a pericardial cradle. A reference electrode was sewn onto the anterior right ventricle. The heart was then paced from the surface of the right ventricle using programmed premature stimulation (PPS) as a provocative test for ventricular arrhythmias.14 A drive of eight beats (cycle length 350–400 msec) was followed by a premature stimulus (S1) of increasing prematurity until the effective refractory period (ERP) of the ventricle was reached. This sequence was repeated using a premature stimulus at 50 msec above the ERP of the ventricle and progressive prematurity of a second consecutive premature stimulus (S2) until the ERP of the ventricle was reached. The epicardial surface of the ventricles was mapped in sinus rhythm using techniques described previously.15 A hand-held probe with two electrodes 1 mm apart recorded activation times with respect to the reference at 53 epicardial points. Unipolar and bipolar inputs from the probe, together with the reference electrogram and the ECG, were connected to amplifiers with an input impedance of 1011 Ω and a frequency response of 0.1 Hz–1.5 kHz (unipolar data) or 5 Hz–1.5 kHz (bipolar data). All data were recorded on a 32-channel FM analog tape recorder and reproduced subsequently at paper speeds of 250 mm/sec. The data were measured relative to a fixed reference point and subsequently corrected to relate to the onset of ventricular depolarization.

A site on the anterior wall of the left ventricle beside the left anterior descending artery was selected for cryosurgery. Large epicardial vessels were avoided. The cryosurgical unit used (Frigitronics, Inc, Shelton, CT) has been described previously.10 Expanding nitrous oxide was used to cool a probe tip to −60°C. Two cryoprobes were used in this study. One probe (five dogs) had a flat, circular cooling surface 14 mm in diameter which produced an “iceball” on the epicardial surface approximately 25 mm in diameter (fig. 1). The probe was applied to the myocardium and...
cooled to $-60^\circ$ C for 4 minutes. The area was allowed to thaw and again subjected to a 4-minute freeze at $-60^\circ$ C. The second probe used (one dog) is better suited for obtaining transmural cryoablation. This trocar probe has a cutting tip 1 cm long which allows intramural insertion of a $12 \times 5$ mm copper cooling sleeve. The copper sleeve in turn is attached to a graduated shaft which allows the sleeve to be moved to the desired level. A superficial pursestring suture was placed on the epicardial surface around the area to be frozen to provide hemostasis when the trocar probe was removed upon completion of the freeze. The freezing process was identical for both types of probe.

After completing the cryolesion, selected epicardial electrograms over and around the lesion were recorded, the chest was closed, and the dog was again attached to Holter monitoring equipment. Twenty-four-hour Holter records were obtained daily for 4 days after the freeze procedure and at 1-week, 2-week and 4-week intervals. PPS of the ventricles was carried out as described above one-half hour, 2 days, 1 week, 2 weeks, and 4 weeks after the freeze procedure. PPS of the ventricles, with the exception of the one-half hour study, was done while the dogs were awake and intact. Another 12-lead electrocardiogram was obtained 4 weeks after the freeze procedure.

A second surgical procedure was performed 4 weeks after the initial freeze procedure. Under anesthesia with $\alpha$-chloralose (100 mg/kg), the heart was exposed through a median sternotomy and suspended in a pericardial cradle. Epicardial mapping was again performed as described above. Selected epicardial electrograms over and around the cryolesion were obtained. Five plunge electrodes were inserted to include myocardium within the lesion, at the border of the lesion, and outside the border of the lesion. These electrodes contained 15 points positioned 1 mm apart along the shaft of a beveled 23-gauge needle. Unipolar and bipolar data were recorded from five electrode pairs 3 mm apart using the system described for epicardial mapping. By convention, a downward polarity of the bipolar electrogram indicated spread from...
endocardium to epicardium. The dog was sacrificed with the plunge electrodes in place. The heart was fixed in 10% formalin for 48 hours and subjected to gross and microscopic examination.

Additional Studies

The consistent occurrence of ventricular arrhythmias within the first week after production of the cryolesion led to the study of four additional dogs for further clarification of this observation. Two control dogs underwent thoracotomy and mapping studies as described for the initial surgical procedure above, but did not receive a cryolesion. Twenty-four-hour Holter records were obtained daily for 2 days after the procedure and again after 1 week. PPS of the ventricles was carried out, as previously described, one-half hour, 2 days, and 1 week after surgery to determine whether the surgical procedure and manipulation of the heart would in themselves produce arrhythmias appearing immediately after surgery.

Two other animals received cryolesions solely for the purpose of mapping any ventricular arrhythmias that developed within 2 hours after the freeze. These animals received cryolesions with the trocar probe as previously described. The sinus node was crushed. Ventricular escape beats appearing during the pause after cessation of rapid atrial or ventricular pacing were mapped in addition to spontaneously occurring ventricular arrhythmias. This was done using a method we have previously described for the rapid determination of epicardial activation sequence requiring only one beat of a tachycardia or an isolated beat. A mesh containing 26 bipolar electrode pairs (separation 1 mm) was uniformly spread over the epicardium. The data were recorded in the same fashion as described for the hand-held probe and stored on the 32-channel analog recorder described above. In addition to the data leads, three limb leads, a reference electrogram, a time code, and a voice log completed the 32 channels of input. A 32-channel analog-to-digital converter digitizes the data, which are then fed to a DEC PDP 11/34 computer. The data are displayed on a Tektronic 4014 graphics terminal. Activation times can be selected automatically by computer or manually. Another program generates isochronous maps for each beat of selected data. A plaque electrode with 25 bipolar pairs (1 mm apart) arranged in a square array with a 7-mm separation between electrode pairs is then used to obtain a detailed activation sequence in an area of interest. Five plunge electrodes can be implanted in and around the site of early breakthrough of activation for three-dimensional localization of the origin of the desired beat.

Results

Arrhythmias Following the Freeze Procedure

Acute and chronic studies spanning 4 weeks were completed in six dogs as described above. During the final operation, before electrophysiological studies of the chronic lesion could be completed dog C died, and was found to have heart worms. All six dogs had frequent ventricular ectopic beats during the period after the freeze procedure. These data are summarized in Table 1. Ventricular tachycardia was seen in five of six dogs during the first 4 days after cryosurgery. Ventricular tachycardia was of two different QRS configurations in each of four dogs.

In all dogs, ventricular tachycardia most frequently started with premature ventricular contractions (VPC) of varying coupling intervals to the last sinus beat. Fusion beats were seen in many of these runs. The tachycardias would appear and disappear sporadically. In addition, two dogs showed fixed-coupled VPCs that would at times lead to ventricular tachycardia. No dog had ventricular ectopic activity after 1 week with the exception of dog E, which had frequent unifocal PVCs in the control records which were unchanged in subsequent records. During the actual freezing and thawing of the lesion, two dogs had ventricular tachycardia, while all dogs had some ventricular ectopic beats. Only infrequent VPCs were noted in the 6–12 hours after the cryolesion was produced, but thereafter ventricular ectopic activity increased as noted in Table 1. Representative monitor tracings are shown in figure 2. No arrhythmias were noted in two dogs subjected to control operations.

Effects of PPS

PPS of the right ventricle was carried out before freezing, immediately after freezing, and 2, 7, 14 and

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<th>Table 1. Ventricular Arrhythmias After Cryosurgery.</th>
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*Ventricular tachycardia present.
†Ventricular couplets present.

Abbreviation: PVC = premature ventricular contraction.
28 days after the freeze procedure as described above. This was done as an additional test of arrhythmogenicity. PPS did not elicit ventricular tachycardia in any animal. One or two reciprocating beats could be consistently elicited after two consecutive premature stimuli in dog E 2 weeks after the initial freeze. The beats were identical in morphology to the stimulated beats. This is the same dog that showed frequent unifocal PVCs before any intervention.

**Mapping Studies of Premature Ventricular Ectopics Appearing After the Freeze Procedure**

Two additional dogs had cryolesions with acute mapping studies of the ensuing arrhythmias. Dog G had infrequent spontaneous PVCs identical to those produced during the pause after cessation of rapid atrial or ventricular pacing. Data from this study are shown in figure 3. Figure 3A is a computer-generated, isochronous map of the ventricular complex shown in the inset. Earliest epicardial activation is noted near the apex. This early area was adjacent to the cryolesion. The plaque electrode already described was then placed over the early site. This provided a more detailed map of epicardial activation with more data points in the area of interest (i.e., the earliest breakthrough of epicardial activation). Five plunge electrodes were then inserted around the site of epicardial breakthrough. Figure 3B shows unipolar data from the plunge electrode nearest to the origin of the ventricular ectopic beats. Electrode 1 is closest to the cavity and electrode 5 is 1–2 mm from the epicardium. The first beat at the left is a sinus beat. Activation of electrode 2 precedes activation at electrode 1 during sinus rhythm, presumably due to penetration of the myocardium by the Purkinje system. Electrodes 4 and 5 show cavity complexes without a rapid intrinsic deflection and are within nonfunctioning myocardium inside the cryolesion. The second beat is a PVC. The impulse spreads from the endocardium to the epicardium. Activation at electrode 1 clearly precedes the beginning of activation in limb lead 2 and the reference electrode and precedes earliest epicardial activation by 24 msec. Electrode 1 activates 8 msec after the onset of the cavity potential and consequently is very close to the point of origin of this ectopic beat.

A second dog underwent mapping of arrhythmias immediately after the freeze procedure. Ventricular tachycardia was noted in this animal during the freeze procedure; however, only a few PVCs were noted during the mapping period in the next 2 hours. These were of two types, one breaking through at the site of the
Fig. 3. Mapping of post-cryosurgical premature ventricular contractions (PVC). Panel A) A computer-generated, isochronous map of epicardial activation sequence for a PVC recorded within 2 hours of freezing (shown in inset). The heart is displayed as if cut from the crux to the apex and flattened. Earliest epicardial breakthrough has been assigned a value of 0 msec and occurs at the margin of the cryolesion (circle). Panel B) Unipolar electrograms for a sinus beat (left) and a PVC identical to that recorded in panels A. The data is obtained from a plunge electrode inserted into the myocardium at the site of epicardial breakthrough of the PVC in panel A. Timing is given assigning a value of 0 msec to the onset of the left ventricular cavity potential and also (in parentheses) relative to earliest epicardial breakthrough, which has been assigned a value of 0 msec. Electrode 1 is very close to the origin of the PVC, activating 8 msec after onset of the cavity potential and 24 msec before epicardial breakthrough.

Effect of the Chronic Cryolesion on Epicardial Activation

The global epicardial activation sequence 4 weeks after the freeze procedure was essentially unaffected by the cryolesion. Epicardial electrograms (unipolar) recorded directly over the acute cryolesion showed a significant loss of RS amplitude, loss of the rapid intrinsic deflection, and elevation of the ST segment. Epicardial electrograms adjacent to the acute cryolesion were unaffected. Four weeks later, the loss of RS amplitude and the loss of the rapid intrinsic deflection persisted over the cryolesion. Representative data from one experiment are shown in figure 4.

Three animals manifested QS complexes over the acute cryolesion which reverted to rS complexes at the time of the 4-week study.

Effect of the Chronic Cryolesion on Intramural Activation

Conduction in and around the chronic cryolesion was studied by recording unipolar and bipolar electrograms from plunge electrodes. The anatomical location of the electrodes was then ascertained by multiple histological sections through the needle tracts. The tracts left by the plunge electrodes were clearly visible on histological sections and a good estimate of electrode location was made in most cases.

Correlation of the intramural electrograms in sinus rhythm with the pathological material showed normal complexes (rapid intrinsic deflection > 2.5 V/sec) and activation sequence (spread from endocardium to epicardium) in histologically normal myocardium immediately adjacent to the scar. There was a distinct transition at the edge of the scar to broad, lower voltage complexes with a slow intrinsic deflection (< 2.5 V/sec) within the scar. The mean RS voltage for the data points adjacent to the scar was 31.9 mV (sd = 10.3, n = 71). The mean RS voltage for the data points within the scar was 13.9 mV (4.2 sd, n = 22). Late activation or fragmentation of complexes was not observed. A representative example is shown in figure 5.

Discussion

The production of controlled, predictable lesions in the myocardium by freezing techniques was first described by Hass in 1948.11, 22 Although cryoablation of many other tissues has been studied since that time,20 the freezing of myocardium has received little attention. We have recently described a cryosurgical technique for the ablation of the His bundle,10 accessory pathways associated with the preexcitation syndrome,11 and a right ventricular focus causing ventricular tachycardia in a patient with a variant of scleroderma.12

The cryolesion has many advantages as an ablative technique in the surgical treatment of intractable arrhythmias. The lesion has smooth borders, sharply demarcated from normal myocardium.13, 21, 22 The scar is firm and minimally disrupts the anatomic continuity of adjacent myocardium. The size and shape of the lesion can be controlled by adjusting certain vari-
figures in the text

Figure 4. Effect of cryolesion on overlying epicardial electrograms. Unipolar epicardial electrograms from 5 points are illustrated before and after freezing. The inset diagram shows the location of points 1–5 relative to the lesion. A reasonable constancy of these surface points for studies at separate times was attained by the use of photographs and careful attention to surface landmarks. Panel A) Normal electrograms at points 1–5 before freezing. For the three electrograms recorded over the myocardium to be frozen (sites 2–4), the mean RS amplitude is 18 mV and the mean slope of the intrinsic deflection is 5 V/sec. Panel B) These same five points 10 minutes after thawing of the cryolesion. Electrograms from points 2–4 (over the lesion) show an impressive decrease in RS amplitude, loss of the fast intrinsic deflection, and slight ST elevation. The mean RS amplitude for the electrograms overlying these points has decreased to 8.3 mV and the mean slope of the intrinsic deflection has decreased to 0.68 V/sec. Points 1 and 5 are adjacent to the cryolesion (approximately 1 cm from the hemorrhagic border) and show essentially normal electrograms. The reason for ST elevation at point 1 is not clear but may be due to excessive pressure exerted on the recording probe. Panel C) These same five points 4 weeks after freezing. Electrograms over the lesion (2, 3 and 4) show persistence of the decrease in RS amplitude and absence of a rapid intrinsic deflection (mean RS amplitude 7.3 mV and mean slope of intrinsic deflection 0.43 V/sec).

The trocar probe is the only reliable method for producing a transmural cryolesion of a deep (> 6–8 mm) myocardial cryolesion from the epicardial surface. The stylet penetrates the myocardium, allowing the freezing sleeve to follow and be placed adjacent to the area to be frozen. The epicardial probe may initially cause transmural damage, as evidenced by QS complexes found over the lesion in three dogs immediately after freezing. Four weeks later, however, these QS complexes had reverted to rS complexes, with only subepicardial scars 5–8 mm deep observed pathologically. Normal potentials, including Purkinje activity, can also be recorded from this endocardial area. The large heat source of circulating intracavitary blood probably prevents “lethal” temperatures from developing at the periphery of the iceball.

The epicardial activation sequence in all dogs was essentially unchanged by the cryolesion. The lesion was not detectable on the standard surface electrocardiograms in any dog. Unipolar complexes recorded directly over the scar showed diminished R voltage and reduced slope of the intrinsic deflection compared with potentials obtained from the same area prior to freezing. The reduced R wave voltage is not surprising as much of the R wave voltage is generated in the outer layers of myocardium which have been replaced by fibrous tissue. The slope of the intrinsic deflection from these complexes falls to less than 2.5 V/sec and can no longer be considered to represent local activation.

Plunge electrodes inserted in and around the chronic cryolesion revealed that the sharp transition between fibrous scar and normal muscle observed pathologically is present electrophysiologically.
Intramural activation around a transmural freeze lesion. This figure shows a histological section through the cryolesion in dog F. The needle tracts left by the plunge electrodes are labeled 1–5. The edge of the scar is well defined. Disruption of the epicardium between needles 2 and 3 was caused by suture material. This transmural lesion was made with the trocar cryoprobe. Unipolar and bipolar data during sinus rhythm recorded from needles 1–5 respectively are illustrated. Normal muscle immediately adjacent to the scar is activated from the endocardium to the epicardium without conduction delay (needles 1, 5 and endocardial electrodes of needles 2, 4). The intrinsic deflections are very rapid (mean slope 17.4 V/sec). Electrograms recorded within the scar (needle 3 and epicardial electrodes of needles 2, 4) have a mean intrinsic deflection slope of 1.75 V/sec. Bipolar pair 5–6 on needle 3 shows baseline artifact due to a fractured electrode wire. A slight current of injury is noted on these records, taken 15 minutes after insertion of the plunge electrodes. $P$ = Purkinje activation.

Mal bipolar and unipolar complexes were recorded in histologically normal myocardium with a clear transition to abnormal complexes in the substance of the scar. Bipolar complexes within the lesion were broad and smooth. Their beginning and end coincided with the beginning and end of the cavity potential. The voltage was low and no fast deflections were present. These potentials are very similar to those recorded from an inert mass implanted in myocardium and are undoubtedly “extrinsic” complexes originating from myocardium outside of the cryolesion. Spread of activation beneath and adjacent to the lesion was from the endocardium to the epicardium. It proceeded without conduction delay to the border of the lesion, where it stopped. The chronic cryolesion is therefore very much like an inert plug that has displaced a portion of myocardium with minimal disruption to the surrounding architecture. The lack of arrhythmogenicity of this chronic lesion is not unexpected, since the usual substrates commonly associated with arrhythmias, namely areas of slowed conduction and areas where normal and gradations of abnormal myocardium are related in a complex irregular fashion, are absent.

VPCs and ventricular tachycardia appearing in the period immediately after the freeze procedure were not seen after 1 week in any dog. This ventricular ectopic activity probably originates at the margins of the acute cryolesion. Mapping ventricular ectopic activity in the period immediately after the freeze procedure in two dogs showed this activity to originate at the border of the cryolesion. These transient arrhythmias probably arise from injured myocardium at the periphery of the cryolesion. The border of injury is very narrow because tissue is a poor thermal conductor and large, abrupt thermal gradients

\[ \text{delay to the border of the tissue} \]

\[ \text{spread of activation} \]

\[ \text{VPCs and ventricular tachycardia} \]

\[ \text{ Margins of the acute cryolesion} \]

\[ \text{Myocardium at the periphery of the cryolesion} \]

\[ \text{Border of injury} \]

\[ \text{Large, abrupt thermal gradients} \]
develop when a focal heat sink (the freeze probe) is applied. The tissue ice front moves slowly from the probe with frozen tissue 1–2 mm from non frozen tissue. At equilibrium, a gradient of temperature exists from the myocardium beside the probe to the heat source, namely intracavitary blood. This creates a narrow zone of cells within a temperature range that is damaging but not necessarily permanently destructive. This zone is not evident in the chronic cryolesion, as it is likely that the more damaged cells closer to the probe die and those damaged distally recover.

Ventricular arrhythmias after cryosurgery are in many ways similar to the arrhythmias noted after Harris two-stage ligation of the left anterior descending coronary artery in the dog. In dogs subjected to cryosurgery and in dogs in Harris' study, ectopic activity appeared after several hours. Harris noted long runs of tachycardia between 6 hours and 4 days which generally stopped within 5 days. After coronary ligation, Harris also noted that the discharges were often multifocal. He further noted the frequent competition between ventricular foci and the sinus node, with each “alternately gaining and losing dominance of the cardiac rhythm.”

Enhanced automaticity has been suggested as the mechanism for arrhythmias appearing several hours to days after acute coronary ligation in the dog and in acute myocardial infarction in man. These arrhythmias appear to originate in the surviving endocardial Purkinje network in the peri-infarction zone. Enhanced automaticity as the mechanism for arrhythmias that appear after cryosurgery is suggested by their electrocardiographical features (usually beginning at varying coupling intervals to the previous sinus beat and competing against the dominant sinus rhythm with frequent fusion beats) and their lack of inducibility by PPS. Ventricular arrhythmias that appear immediately after cryosurgery are important considerations in any attempt at therapeutic cryosurgical ablation of ventricular tissue responsible for ventricular tachycardia. Ventricular arrhythmias lasting as long as 1 week may be seen starting immediately after freezing as a direct result of the cryolesion. Furthermore, the cryolesion will be made in the region where the ventricular tachycardia to be treated originates. Theoretically, ventricular tachycardia originating from the margin of the cryolesion may then have a similar QRS morphology to the tachycardia to be ablated. Lack of awareness of this phenomenon may create some confusion in the postoperative period.

We have demonstrated the feasibility of cryosurgical ablation of selected left ventricular sites. The desired size and shape of the lesion can be achieved by selecting the appropriate probe and cooling parameters. The lesion heals by fibrosis into a firm scar that is sharply demarcated from normal myocardium and does not disrupt the surrounding anatomy. The chronic lesion behaves electrophysiologically like an inert plug with no disruption of surrounding transmural and epicardial activation. Ventricular ectopic activity is present in the acute phase after freezing (up to 1 week), but is not present thereafter.

This cryosurgical technique may be useful for ablating regions of left ventricular myocardium responsible for arrhythmias in man.

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References

Contrast M-Mode Echocardiography in Diagnosis of Atrial Septal Defect in Acyanotic Patients

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SUMMARY Contrast echocardiograms during normal quiet respiration and during the Valsalva maneuver were performed in 15 patients with atrial septal defect (ASD) by injecting saline solution into an antecubital vein. Contrast shunting (the appearance of contrast echoes in the left heart) was observed not only in four patients with severe pulmonary hypertension (group 2), but also in 11 patients with uncomplicated ASD (group 1). Contrast shunting was more pronounced during the Valsalva maneuver than during normal respiration, although there were exceptions. The amount of contrast appearing in the left heart did not correlate with the size of the defect. Small right-to-left shunts which are clinically insignificant but detectable by contrast echocardiography are present, or can be provoked by the Valsalva maneuver, in most patients with ASD. Contrast echocardiography is a useful, noninvasive method to detect interatrial communication, even in acyanotic patients.

CONTRAST ECHOCARDIOGRAPHY is a very sensitive method to detect right-to-left (R-L) shunts, but it is usually considered of little value in patients with left-to-right (L-R) shunts. Patients with atrial septal defect (ASD) may be an exception to this rule and contrast echocardiography may be successfully used to detect interatrial communications even in the absence of pulmonary vascular disease and severe pulmonary hypertension.

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 Patients and Methods

We examined 15 patients with ASD by contrast echocardiography. The presence of ASD was documented by standard cardiac catheterization. The 11 patients in group 1 had normal pulmonary vascular resistance, normal to moderately elevated pulmonary artery pressure, and no oxymetric evidence of a R-L shunt. The four patients with clinically obvious R-L shunts (group 2) had high pulmonary arteriolar resistance and severe pulmonary hypertension documented at cardiac catheterization. The most important clinical and hemodynamic data of both groups are presented in table 1.

After a complete M-mode echocardiographic study had been obtained, an antecubital vein was punctured and a plastic cannula with a luminal diameter of 1 mm