Cardiac Trauma from Angiographic Injections

A Quantitative Study

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SUMMARY To relate angiographic injections to potential cardiac trauma, we verified a mathematic theory that allows quantitative definition of the kinetic energy content of contrast jets emanating from the exit holes of angiographic catheters. Cineangiographic recordings of a range of jets of known energy content were obtained in 18 cardiac canine experiments and energy content and dissipation were quantified precisely from center line to jet edge. All contrast jets produced in clinical angiographic practice were turbulent, even those from hand injections into the coronary arteries. Energy content was related to an estimated cardiac wall damage threshold. At energy levels and damage thresholds predicted by the theory and computations, a traumatic spectrum was found by cine radiology and microscopic examination.

A unique curve independent of jet Reynolds number was discovered relating the penetration of the contrast jet into the intravascular blood to the potential for cardiac trauma. This curve allowed ready calculation of hydraulic energy dissipation for any clinically used angiographic catheter and the definition of safe operational injection flow rates. Thus potential cardiac trauma can be anticipated and prevented.

MODERN ANGIOGRAPHIC PUMP INJECTORS may cause catheter recoil and generate high velocity contrast jets that may result in iatrogenically induced cardiovascular trauma. Indeed, adequate angiography is dependent on rapid delivery of large boluses of contrast medium into the vascular and ventricular chambers. Moreover, experienced angiographers empirically vary both the volume and flow rates of contrast injections and it is not surprising that a wide variety of traumatic angiographic complications have been reported in detail. Fortunately, clinically recognized episodes of such trauma are infrequent although the true incidence of subclinical trauma is unknown. One difficulty in analyzing and documenting such trauma has been an inability to separate the effects of catheter whip from those of the contrast jet. In addition, no information is available concerning the precise energy content of such jets and whether they actually are traumatic. To solve these problems, we developed and reported a mathematic theory that allows isolation and definition of angiographic jet energy separately from other variables related to angiographic cardiovascular damage. A series of in vivo canine experiments was designed and performed to test this theory. The results of these experiments form the basis of this paper.

Background Concepts

A detailed mathematic description of our theory has been reported previously. Briefly, the theory is a first order approximated for an angiographic jet that is round or axially symmetric and is discharging into an unbounded medium.

The Angiographic Jet

The Reynolds number in any type of fluid flow is the most important parameter governing the final fluid characteristics. The Reynolds number of a jet is a dimensionless number derived from the ratio of inertial to viscous forces. Depending on the jet Reynolds number, jet flow is either laminar or turbulent. A laminar jet has well defined stream lines and is without fluctuations, whereas a turbulent jet has a flow composed of chaotic random movements superimposed on the main jet. When the jet Reynolds number reaches 77, the motion of a round laminar jet becomes disturbed and eventually develops into full turbulence at higher Reynolds number (Re ≈ 2500).

The Mathematic Solution

There are two approaches by which a free round jet may be analyzed. All angiographic jets emanating from the exit orifice(s) of catheters may be analyzed individually. However, it is a tedious and time consuming venture to analyze all possible permutations of contrast injection conditions and catheter configurations. A more economic technique is an approach that allows for solution of any type of angiographic jet by expressing essential parameters (see appendix) by a few key nondimensional variables. A well established fluid mechanical technique of “dynamic similitude” allows the dynamic characteristics of any types of different jets to be quantitatively calculated in a straightforward fashion. Similarity methods have been applied to the solution of the hydrodynamic equation to obtain solutions for free jets. Fortunately, with only minor modifications, the theoretic solution for laminar jets is also applicable to disturbed and turbulent jets. This mathematic approach and modifications were incorporated into our theoretic approach.

An important result from our formulation is the fact that the strength of any angiographic jet can be quantified by a parameter that we label Cw. This parameter is a ratio of the initial kinetic energy of the jet to the intravascular pressure and has the following formula:
\[ C_p = \frac{1}{2} (\rho) \bar{U}^2 / P_A \]

where \( C_p \) = nondimensional initial jet kinetic energy, \( \rho \) = density of injectate (g/cm\(^2\)), \( \bar{U} \) = mean velocity of the jet at catheter exit (cm/sec), and \( P_A \) = intravascular pressure (dynes/cm\(^2\)).

The parameter \( C_p \) represents a measure of the energy of motion initially possessed by the angiographic jet. It is proportional to the square of the velocity of the contrast medium being injected. Because the potential vascular damage during angiography is caused primarily by the impact of the contrast jet, the kinetic energy content of the jet becomes the main determinant of trauma.

Our computations revealed that all angiographic jets are turbulent for conditions normally encountered in clinical angiography. A graphic example of the energy content, flow field, and energy dissipation calculated with the aid of a high speed digital computer (CDC 6400) is displayed in figure 1. Note that the angiographic jets have a narrow jet spread angle and are long and dart-like. The jet retains its initial kinetic energy level for a distance of seven exit hole diameters downstream of the catheter orifice. Thereafter, the energy, along the centerline axis of the jet, decays by viscous dissipation.

A criterion for potential tissue trauma was established by relating a theoretic cardiovascular damage to the jet kinetic energy content. Energy threshold for tissue damage was extrapolated from the quantitative stress and strain measurements determined by Fry\(^m\) as causing vascular endothelial changes. This threshold was determined to correspond to a kinetic energy level of the jet of 0.6. We hypothesize that an endothelial injury would develop from the impact of contrast jet if the kinetic energy of the jet just before impinging on the vascular wall was above the trauma threshold.

The Universal Curve

Because the nondimensional jet energy ratio, \( C_p \), determines if the theoretic tissue trauma level is exceeded and the distance that jet energy remains above the trauma threshold, we constructed a graph relating \( C_p \) to the jet penetration distance and demarcated the trauma and no trauma zone for an infinite series of contrast jets (fig. 2). The curve dividing the zones was at the kinetic energy level of 0.6. The remainder of this paper delineates in detail how we verified this graph by canine experiments and the practical implications of our experiments.

Methods

Eight mongrel dogs were anesthetized by intravenous administration of sodium pentobarbitol (1 ml/5 lbs) and subjected to thoracotomy. A special hand-held device was constructed that allowed a No. 7 French end-hole catheter, tapered to an exit-hole orifice of 0.038 inches, to be rigidly held at right angles against the walls of the cardiac chambers at preselected distances from 0 to 20 mm (fig. 3). A single end-hole catheter was used in order to ensure that the angiographic jet impact was perpendicular to the cardiac wall. The holding device could be placed in either the atrium or the ventricle at any predetermined site via careful manipulation under fluoroscopy. The device has a radio-opaque footplate that ensures that the orifice of the catheter is in a plane perpendicular to the cardiac wall and that cineangiographic film strip is recorded in the plane of the injection. Once position and alignment were verified by fluoroscopy, 76% Renografin at body temperature was injected at a series of flow rates predicted from our theory to be either traumatic or harmless. Only one injection was made near a given site. Before injection of the contrast material, the heart rate was markedly decreased or temporarily stopped via the use of right vagal nerve stimulation at 30 Hz and 6 volts (SD-5 Stimulator Grass Medical Instrument Co. Model SD-5C) to allow injection of contrast material during diastole. In addition, the catheter-holding device allowed intravascular pressure recording before and after injection of Renografin. The electrocardiogram was continuously monitored. In one animal (1), two separate injections into the right atrium at different atrial sites were carried out. In another (8), two injections were made into the

![Figure 1: Jet kinetic energy decay. A computer-generated mapping of jet energy and its decay over distance (abscissa) is plotted. Jet energy length is expressed in nondimensional units of exit-hole diameters (X/D) (abscissa). Above is a two-dimensional dart-shaped contour map of a jet with an energy value of 3.5 exiting from an end-hole catheter. Each isobar has the calculated jet energy listed on it, i.e., 2.5, 1.5, and 0.5. Note that no energy is lost for the first seven exit holes; thereafter, energy decay is rapid. The center line jet energy content of 3 jets (below) is plotted in a plane perpendicular to the upper two-dimensional display. Center line jet energies (ordinate) for three different jets demonstrate that the distance required to fall below the derived trauma threshold of 0.6 is a direct function of the initial energy content. The lower jet has so little energy that it is never potentially traumatic. Y = radial spread; X = axial distance from exit hole; D = diameter of exit hole.](http://circ.ahajournals.org/content/57/1/92.full)
right atrium and one into the right ventricle. In three animals (2–4), one injection into the right atrium and two into separate right ventricular sites were performed. In two animals (5 and 6), a total of three left ventricular injections at various sites within the ventricle were carried out. All of these seven animals were sacrificed immediately after the injections. In one animal (7), a single injection into the right ventricle was performed at a level predetermined to be traumatic, and thereafter the cardiac and chest wounds were repaired. One week after thoracotomy, this animal was killed and the heart was subjected to detailed examination. All injections were delivered normal to the vascular wall and recorded at right angles to jet impact by cineangiography. The films were analyzed for evidence of angiographic staining. Prior to death, a large dose of heparin (5 ml of a 1000 units/ml solution) was administered intravenously. After the animals were killed, the areas subjected to jet impact were identified, photographed, and examined for gross pathologic changes.

The specimens were rinsed briefly in normal saline and fixed in 10% buffered formalin. Representative portions were dehydrated in alcohol and a Polaron Critical Point drying apparatus. They were then coated with carbon and gold palladium and examined with an ISI Mini-Sens II scanning electron microscope (International Scientific Instrument, Inc.). Similar portions were imbedded in paraffin and sections stained with hematoxylin and eosin, phosphotungstic acid hematoxylin (PTAH), and Gomori’s trichrome stains. The results of the angiography and pathologic examination were coded by independent observers (ML and JK, respectively) and without knowledge as to whether the injections were predicted to be traumatic or harmless. The angiographic and pathologic findings were analyzed independently and without knowledge of the injection forces.

Results

Contrast material was injected into 18 separate sites in the eight dogs. Eight injections were made into the right ventricular cavity, seven into the right atrium, and three were delivered into the left ventricle. One injection into the right ventricular cavity (table 1) was designed as a chronic experiment. Eight experiments were predicted to be traumatic and all proved to be so. These injections were at flow rates employed in clinical practice. Ventricular injections predicted to be traumatic always revealed transient staining of the ventricular wall and frank myocardial contusions on gross and pathologic examination. The ventricular injection in the chronic experiment (7) also initially revealed myocardial wall staining, and pathologically, fibrosis was found in the area of jet impact. Staining was not seen, however, in the four atrial sites of injections predicted to be traumatic, although myocardial contusions were found in three and endothelial rupture with subendothelial damage was found in the fourth. Two of the 11 injections predicted to be harmless revealed injury (3a and 6). In one (3a), injury consisted of endothelial and subendothelial hemorrhage; in the other (6), a myocardial rupture with extravasation of blood and radiopaque contrast medium occurred. In this experiment, the catheter tip directly abutted the ventricular wall throughout the entire injection, although the injection flow rate was only 4 ml/sec. In the remaining seven cases in which the injec-

![Figure 2](https://example.com/f2.png)

**Figure 2.** The universal curve. The distance (L) nondimensionalized by D (abscissa) that the center line jet energy travels until the energy reaches the derived trauma threshold of 0.6, the curve, is charted for any number of initial jet energies (Cr) (ordinate). This curve shifts slightly to the right if the intravascular pressure is elevated. D as in figure 1. Cross-lined curved bar = 55–120 mm Hg; stippled curved bar = 0–55 mm Hg.

![Figure 3](https://example.com/f3.png)

**Figure 3.** A diagram of the hand-held catheter-fixing device. The triangular-shaped tip can be seen clearly on fluoroscopy and insures that the contrast jet exiting from the end-hole catheter strikes the vascular wall at a right angle. The catheter tip can be placed and held rigidly at any desired distance from 0–20 mm by the adjustable screw on the proximal shaft of the device.
4). The electrocardiogram always revealed ventricular irritability upon injection of the contrast medium into either right or left ventricle and once with a traumatic atrial injection (3c). Neither type nor frequency of ventricular ectopy correlated with the presence or absence of tissue damage. Postinjection electrocardiograms, even in the chronic case, never revealed a pattern of transmural myocardial infarction, although transient repolarization changes were always seen.

Pathologic findings demonstrated a spectrum from mild and subtle endothelial abnormalities (fig. 5, panel D), identified only by scanning electron microscopy, to frank and severe gross damage. Experiment 5b (fig. 5, panels A-C) illustrates these findings. The angiographic cardiac injections were predicted to be traumatic, and myocardial staining and gross damage were found and readily identified by light microscopy (panels A and B). Endothelial disruption and fibrin deposition with entrapment of red and white blood cells and platelets were observed by electron microscopy (panel C). Similar endothelial changes were noted in experiment 4c (fig. 5, panel D). In the single dog not killed soon after trauma, right ventricular staining was noted on injection of contrast material and an area of trauma was readily identified one week after surgery. Microscopic and scanning electron examination of this area revealed fibrosis within the myocardium overlaid by a thrombus and a re-endothelialized right ventricular wall (fig. 6), indicating an appreciable and more or less permanent sequelae of the injury.

Discussion

These results appear to validate the theoretic concept in a physiologic setting and should allow a quantitative approach to angiographic jets and cardiovascular trauma. Previously, Lim and Cadwallader11 performed experiments seeking the energy content of angiographic jets but their approach of measuring multiple pressures in vitro for each and every individual catheter at various flow rates had obvious limitations.11 Furthermore, they did not relate jet impact to cardiovascular damage.

End hole catheters direct the entire force of a contrast injection out of one orifice and should never be used for left ventricular angiography. Our method was designed to validate a theory that predicts that trauma from angiographic injections can occur at critical energy levels generated at clinically used injection flow rates. Since the critical stress level was verified, the theory can be applied to any catheter regardless of the number of side holes. Thus the technique described in the present study has practical application for the clinical angiographer. Provided the catheter exit-hole diameters, the injection flow rate, and the viscosity of the contrast medium are known, the range of potential vascular damage for an angiographic jet issuing from any catheter exit-hole can be predicted prior to angiography. With these parameters, rapid and accurate calculation of the initial kinetic energy of angiographic jets is possible. Using the universal curve, the distance the energy forces must travel prior to falling below the traumatic kinetic energy level can be determined.

The spectrum of cardiac trauma we predicted and identified included endothelial disruption and subendothelial hemorrhages. Endothelial rupture was always present and was associated with a myocardial contusion, except in one case (3c), when a traumatic angiographic injection was predicted. In addition, endothelial disruption was always accompanied by thrombus formation, which conceivably could give rise to postangiographic emboli. The presence of ventricular wall staining even when transient can be equated with a myocardial contusion. "Washing out" of contrast material occurred with all but two of the myocardial stains, one a ventricular rupture and the other a false aneurysm. We believe the "washing out" phenomenon may lead the clinician to a false sense of security regarding the extent of cardiac damage that has occurred. Ventricular contusions might also explain, in part at least, electrocardiographic conduction delays or repolarization abnormalities encountered in clinical practice. Myocardial staining did not occur with any of the atrial injections even though endothelial disruption and atrial wall contusions were associated with traumatic injections in all but one instance in which there was only endothelial disruption and subendothelial hemorrhage. Probably, the small amount of contrast material that was entrapped in the atrial wall was
Figure 5. a) Cardiac left ventricular septum of dog No. 5a immediately after catheter injection of 26 ml of 76% Renografin (37°C) at a flow rate of 13 ml/sec. Note massive myocardial-dissecting hemorrhage with elevation of endocardium into the lumen (1). (Hematoxylin and eosin stain; magnification × 20.) b) Same heart as in 5a. Note wide separation of myocytes by blood (rbc) and granular changes and apparent loss of nuclear staining in many myocytes. (Hematoxylin and eosin stain; magnification × 100.) c) Same area in heart as in 5a and b. Note smooth endocardial surface above and below a tear (arrows) to the floor of which platelets, fibrin, and white blood cells have already adhered. This animal was killed 2 min after the contrast injection. (Scanning electron micrograph; magnification × 2000.) d) Similarly traumatized endocardium in dog No. 3b killed 20 min after catheter injection. Note extensive roughening and adherence of fibrin, platelets, and red blood cells. (Scanning electron micrograph; magnification × 1000.)

Table 1. Summary of the Pathologic Findings Induced by Angiographic Injections in Dogs

<table>
<thead>
<tr>
<th>Animal no</th>
<th>Injection site</th>
<th>Flow rate (ml/sec)</th>
<th>Total time of injection (sec)</th>
<th>Distance from wall (mm)</th>
<th>Predicted traumas</th>
<th>Observed traumas</th>
<th>Ventricular ectopy</th>
<th>Angiographic staining</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 a) RA-high</td>
<td>10</td>
<td>5 4 5 0</td>
<td>None</td>
<td>-</td>
<td>No</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 b) RA-low</td>
<td>13 2 10 0</td>
<td>None</td>
<td>-</td>
<td>No</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 a) RV-apex</td>
<td>15 2 10</td>
<td>ed, mc</td>
<td>+</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 b) RV-septum</td>
<td>11 2 5</td>
<td>ed, mc</td>
<td>+</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 c) RA-septum</td>
<td>11 2 15</td>
<td>ed, mc</td>
<td>+</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 LV</td>
<td>4 2 10 0</td>
<td>ed, mc, mr</td>
<td>+</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7* RV (chronic)</td>
<td>13 2 10 +</td>
<td>fibrosis</td>
<td>+</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 a) RV-apex</td>
<td>20 2 20 0</td>
<td>None</td>
<td>+</td>
<td>No</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 b) RA-low</td>
<td>20 2 20 0</td>
<td>None</td>
<td>-</td>
<td>No</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Killed 7 days after angiography.

Abbreviations: RA = right atrium; RV = right ventricle; LV = left ventricle; ed = endothelial damage with subendothelial hemorrhage; mc = myocardial contusion; mr = myocardial rupture; false A = false aneurysm.
below the resolution of our cinefluoroscopy system. The pathologic changes we induced were at times minor because we attempted to define a critical traumatic energy level. Obviously, the greater the energy content of the angiographic jet the more traumatic is the injection. In experiment 2b, a traumatic ventricular aneurysm resulted from the injection. Our results also demonstrate that ventricular irritability is an invalid index of cardiovascular trauma. Ventricular irritability was seen with every ventricular injection whether traumatic or harmless and was present even with one traumatic atrial injection. Others have also noted the inability to relate the flow rate of the contrast injection to ventricular ectopy.18

The results of our lone chronic experiment lead us to believe that the pathologic changes we demonstrated have lasting consequence in that myocardial fibrosis was found in an area that revealed myocardial staining during the injection of the angiographic contrast material. We did not measure creatine phosphokinase isoenzymes and thus cannot say with certainty that myocardial necrosis resulted from any of our traumatic injections. However, others report that a rise in creatine phosphokinase does not occur with angiographic injection.18 Possibly, this is because any area of myocardial necrosis is small and falls below the threshold of the creatine phosphokinase isoenzyme sensitivity. We also suspect any area of myocardial contusion to be greater than the area of potential necrosis. Whatever the explanation, the dart-like angiographic jet, if of enough strength, acutely causes a contusion and can result in residual myocardial scarring. The practical implication in a patient with a decreased ventricular function is that a scar may further compromise the myocardium.

The experimental design has some limitations. Our model does not take into account catheter recoil during the angiographic injection. Indeed, catheter whip may be an inherent safety feature of angiography, although the final position of the catheter exit holes cannot be predicted with certainty. However, we have observed that catheter recoil from most clinically used ventriculography catheters such as pigtail catheters is absent or slight at most clinically used injection flow rates. Moreover, in selective vascular angiography such as coronary, cerebral, or abdominal visualization, the catheter tip remains in a relatively fixed position. The results of our model are directly applicable in these situations and predict with greater certainty iatrogenically induced cardiovascular damage. A further question relates to the applicability of the model, the results of which only strictly apply when the catheter tip lies free of the vascular wall. Our theory is valid only when the angiographic jet is unbounded. The limitation of our theory was shown in that

**Figure 6.** a) Right ventricular subendocardium and wall of dog no. 7 seven days after catheter injection of 26 ml of 76% Renografin (37°C). Arrows demarcate mural thrombi, and areas of myocardial damage (md) are clearly visible beneath m = undamaged myocytes. (Phosphotungstic acid-hematoxylin stain; magnification × 20.) b) Same area as in 6a. Mural thrombus (thr) seven days after the traumatic injection is already covered by endothelium (end), which separates lumen above from damaged myocardium (md) and intact myocardium (m) below. (Phosphotungstic acid-hematoxylin stain; magnification × 100.) c) Right ventricular subendocardium from the same heart as in 6a and b. A typical area of myocyte damage (arrows) showing fibrocyte replacement of myocytes. (Hematoxylin and eosin stain; magnification × 400.)
in one instance when no trauma was predicted a myocardial perforation occurred when the catheter tip was at the ventricular wall. When the exit orifice of the catheter is in proximity to a vascular wall, our theory is inaccurate and likely underestimates cardiovascular damage. However, in clinical practice every effort is made to avoid this situation. Thus, mathematical solutions for the impact damage due to entrapped angiographic jets are impractical. In addition, our theory did not predict with absolute certainty lack of trauma in one instance. Because our estimates of trauma were based on stress and strain values derived from arterial wall measurements, this presence of trauma should not have been surprising. The trauma found in this lone instance was minor and could only be identified by scanning electron microscopy. In the seven other experiments, injections predicted to be nontraumatic always proved to be so.

Finally, our studies were performed in animals with a normal cardiovascular system. A decreased cardiovascular trauma threshold may be present in diseased arteriosclerotic vessels and hearts; we did not determine the effects of angiographic jets in this disease.

Our findings have practical implications. The clinician can now determine the upper safety limit for flow rate for any given catheter and therefore avoid the risks of cardiovascular trauma. For a practical example of our calculations, see the dimensional presentation in figure 7. Here a No. 7 French tapered end-hole catheter, a type commonly employed for coronary angiography, might be expected to cause cardiovascular trauma at a flow rate of 8 ml/sec if the angiographic jet were delivered in a plane perpendicular to the wall of the coronary vessel. In procedures in which the vessel is in proximity to the jet stream, flow rates above this level should not be exceeded. Although injection of contrast material by hand is commonly employed in coronary procedures, it cannot be assumed that this flow rate is not exceeded. We measured the flow rate of hand injections of contrast material through a No. 7 end-hole catheter and showed that with a vigorous injection a mean flow of 8 ml/sec can be easily achieved. Moreover, the flow rate transiently exceeds 10 ml/sec. Pump injections in which flow rates are precisely controlled would seem to be indicated. In addition, figure 7 demonstrates qualitatively the importance of adding side holes to a catheter used for ventriculography or opacification of the great vessels. The calculations for side hole additions assume equal velocities exiting from all orifices. The larger the effective end-hole diameter, the greater the margin of safety of the flow rate injection. In this figure, a trauma zone is quantitatively shown to be shifted upward, allowing the safe use of greater flow rates. Furthermore, we have recently found that flows emanating from the exit holes of multiholed catheters are not equally distributed, but are dependent upon the injection flow rate, the size and geometry of the catheter and the area and the location of each exit-hole. Thus, we also developed and applied general hydraulic formulae to calculate angiographic flow partitioning, as well as the limiting predicted or potential traumatic flow rates, for some commercially available catheters (unpublished observations). Results from the calculations are shown in table 2.

The NIH closed end and the pigtail types of ventriculography catheters have fairly high safe flow rates, whereas the tapered, end-hole coronary catheter permits only a very low injection rate and is unsafe to use in ventriculography. Also, the geometry of the pigtail catheters have higher safe flow rates than the Sones coronary catheters, which are used for ventriculography in some centers, even though both catheter types have four side holes and one end hole.

**Table 2. Predicted Safe Flow Rates for Catheters Used in Ventriculography**

<table>
<thead>
<tr>
<th>Type of catheter</th>
<th>Manufacturer</th>
<th>Size (French)</th>
<th>Number of exit-holes*</th>
<th>Highest injection flow rate (ml/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pigtail</td>
<td>Cook</td>
<td>6</td>
<td>4 SH</td>
<td>35</td>
</tr>
<tr>
<td></td>
<td></td>
<td>7</td>
<td></td>
<td>37</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8</td>
<td>1 EH</td>
<td>40</td>
</tr>
<tr>
<td>NIH</td>
<td>Cordis</td>
<td>6</td>
<td>4 SH</td>
<td>37</td>
</tr>
<tr>
<td>(closed end)</td>
<td></td>
<td>7</td>
<td></td>
<td>39</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8</td>
<td>52</td>
<td></td>
</tr>
<tr>
<td>Gensini</td>
<td>USCI</td>
<td>7</td>
<td>6 SH</td>
<td>37</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>+ 1 EH</td>
</tr>
<tr>
<td>Shirey</td>
<td>USCI</td>
<td>8</td>
<td>6 SH</td>
<td>25</td>
</tr>
<tr>
<td>Sones</td>
<td>USCI</td>
<td>7 (tapered to</td>
<td>4 SH</td>
<td>19</td>
</tr>
<tr>
<td>(coronary catheter)</td>
<td></td>
<td>8 #7 tip</td>
<td></td>
<td>20</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>+ 1 EH</td>
</tr>
<tr>
<td>Judkins</td>
<td>Cordis</td>
<td>4</td>
<td>1 EH</td>
<td>8</td>
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<tr>
<td>(coronary catheter)</td>
<td></td>
<td>5 (tapered to</td>
<td></td>
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</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.038&quot; tip</td>
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</tbody>
</table>

*SH = side hole; EH = end hole.
Our technique defines the maximum safe injection flow rate for any type of ventriculography catheter rather than leaving this to the clinician to decide on a case by case basis. Arbitrarily selecting a contrast flow rate does not guarantee lack of clinical or subclinical ventricular trauma. In addition, with the use of higher quality image intensification systems, flow rates for angiography can be quantitatively reduced into safe flow ranges without sacrificing film quality.

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Coronary Sinus Pacing

Clinical Follow-up

PAUL GREENBERG, M.D., MARK CASTELLANET, M.D., JOHN MESSENGER, M.D., AND MYRVIN H. ELLESTAD, M.D.

SUMMARY Coronary sinus pacing is a safe and effective means of pacing from the atrium. In 66 patients with an average follow-up of 14 months, there was a 14% failure rate. There were transient problems in 14% which were subsequently corrected. There was a 6% death rate which was not pacemaker related. Successful atrial pacing with thresholds up to 6.0 mA is feasible. Atrial pacing was successful in 18 of 19 patients with varying degrees of atioventricular block. Our experience with a new electrode has been very satisfactory.

INDICATIONS FOR AND THERAPEUTIC USES of atrial pacing have rapidly expanded in recent years. Successful long-term atrial pacing from the coronary sinus has been reported in a large series by Moss et al. Atrial pacing has been shown in several studies to be hemodynamically superior to ventricular pacing. Improvement in ventricular performance has been demonstrated consistently in a damaged ventricle when atrial pacing is compared to ventricular pacing. However, difficulties with pacing leads and lead positioning have hampered the widespread use of atrial pacing. This article presents our experience and long-term follow-up with coronary sinus pacing.

Methods

A retrospective study was done reviewing the clinical records on all patients who had a permanent coronary sinus pacemaker inserted from 1970-1976. A total of 71 hospital charts and pacemaker records were reviewed for indications of pacemaker insertion, type of pacemaker, type of pacemaker lead, thresholds, ECG, atrial pacing studies, early complications, pacemaker failures and causes of death. Complete records and follow-up could be obtained on 66 patients. Of the five excluded cases, three patients were lost.
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