Will a Partial Posterior Annuloplasty Ring Prevent Acute Ischemic Mitral Regurgitation?

Tomasz A. Timek, MD; Paul Dagum, MD, PhD; David T. Lai, FRACS; Frederick Tibayan, MD; David Liang, MD, PhD; George T. Daughters, MS; Motoya Hayase, MD; Neil B. Ingels, Jr, PhD; D. Craig Miller, MD

Background—Acute posterolateral ischemia in sheep results in ischemic mitral regurgitation (IMR). While complete ring annuloplasty prevents acute IMR, partial annuloplasty rings may offer a more physiologic repair, but are untested in animal models of IMR.

Methods—Radiopaque markers were placed on the LV, mitral annulus (MA), and leaflets in 13 sheep. Seven sheep served as controls, and 6 had a St. Jude Tailor partial flexible ring implanted (29 mm in 5, 31 mm in 1). After 8±1 day, the animals were studied with biplane videofluoroscopy and echocardiography before and during acute posterolateral LV ischemia (balloon occlusion of circumflex artery). Mitral annular area (MAA), septal-lateral annular diameter (SL), annular perimeters, and leaflet edge separation were calculated from 3-D marker coordinates.

Results—The average degree of mitral regurgitation increased from 0.0±0.0 to 2.1±0.7 (P=0.0006) in the control group during acute ischemia but remained unchanged in the Tailor group (0.1±0.2 for both conditions). The change in MAA throughout the cardiac cycle before ischemia was 17±4% in control animals, but only 5±2% (P=0.0002) in the Tailor ring group. Unlike the control animals, there was no increase in MAA (5.4±0.8 and 5.5±0.7 cm², respectively; p=NS) nor dilatation of the muscular annulus (6.2±0.3 and 6.2±0.4, respectively; p=NS) during ischemia with the Tailor ring. Mitral SL dimension increased slightly with ischemia (2.3±0.2 versus 2.2±0.2 cm, P=0.03). Although posterior leaflet motion was limited, as observed with complete rings, normal annular flexion was maintained with the Tailor ring before and during acute ischemia.

Conclusions—The Tailor partial annuloplasty ring prevented acute IMR probably by limiting SL diameter dilatation during acute ischemia. In this animal model of acute IMR, a partial, flexible posterior annuloplasty ring is as effective as a complete ring. (Circulation. 2002;106[suppl I]:I-33-I-39.)

Key Words: ischemia ■ mitral valve ■ regurgitation ■ valvuloplasty

Mitral valve repair has become the operation of choice for mitral insufficiency of various etiologies. Although more studies are still needed, there is a growing body of evidence suggesting the superiority of mitral repair over replacement in patients with ischemic mitral regurgitation (IMR). In most cases, valve repair for IMR consists solely of mitral annular reduction using a complete annuloplasty ring. Complete semi-rigid and flexible annuloplasty rings prevent mitral insufficiency in ovine models of acute posterolateral left ventricular (LV) ischemia. Complete annuloplasty rings, however, abolish normal mitral annular dynamics and perturb posterior leaflet motion.

Cosgrove and colleagues introduced a partial flexible annuloplasty band that offers annular size reduction yet appears to preserve normal annular dynamics. A similar partial annuloplasty ring, the Tailor partial flexible annuloplasty band, preserved normal annular flexion in experimental ovine experiments, although mitral annular area was fixed during the cardiac cycle. As annular dilatation and displacement primarily of the posterior annulus are associated with ischemic mitral regurgitation, partial posterior ring annuloplasty may offer a simpler and more physiologic repair. Although partial ring annuloplasty has been used in patients with IMR, its efficacy in models of acute LV ischemia has not been assessed. Therefore, we investigated a partial posterior flexible annuloplasty ring in an acute model of LV ischemia in sheep. This current study extends the observations of our previous report to include the response to acute posterolateral LV ischemia.


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were performed after a 7 to 10 day recovery period.

Results

graphic marker implantation only and served as a control group. The annular and leaflet dynamics in this historical control group have been reported.20 All animals were recovered in the experimental animal care facility with no untoward events. Seven animals underwent mitral annuloplasty due to mitral insufficiency as determined by echocardiographic assessment.

Data Analysis

End-systole was defined as the videofluoroscopic frame preceding maximum negative dP/dt (-dP/dt max); end-diastole was defined as the videofluoroscopic frame containing the peak of the ECG R-wave. An instantaneous estimate of LV volume was calculated every 16.7 msec from the epicardial LV markers using a multiple tetrahedral model reconstructed from the marker coordinates.24 External LV pressure-volume stroke work (SW) was calculated as the integral of LV pressure (P) on volume (V) over a cardiac cycle for several beats at baseline and during caval occlusion as:

\[ SW = \int P \, dV \]

Preload recruitable stroke work (PRSW) was computed by linear regression of SW on EDV as:

\[ SW = MW(EDV - VW) \]

Where MW and VW are the slope and volume axis intercept, respectively. Adequate volume occlusion for PRSW calculations was obtained in 6 animals for the control and 4 animals in the Tailor groups.

Mitrail Annular Geometry

Mitrail annular area, perimeters, and dimensions where computed from the 3-D marker coordinates without assuming circular or planar approximation.
TABLE 1. Hemodynamics

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Ischemia</th>
<th>Control</th>
<th>Ischemia</th>
<th>Tailor</th>
<th>Ischemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (b/min)</td>
<td>106±11</td>
<td>98±24</td>
<td>115±10</td>
<td>99±31</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EDP (mm Hg)</td>
<td>16±4</td>
<td>18±10</td>
<td>20±9</td>
<td>23±4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ESP (mm Hg)</td>
<td>89±12</td>
<td>53±11*</td>
<td>101±10</td>
<td>71±20*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVP max (mm Hg)</td>
<td>139±18</td>
<td>95±17*</td>
<td>119±15</td>
<td>89±20*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EDV (mL)</td>
<td>145±33</td>
<td>149±36</td>
<td>175±22</td>
<td>182±23</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ESV (mL)</td>
<td>115±23</td>
<td>126±25*</td>
<td>143±15†</td>
<td>153±14*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>+dP/dt max (mm Hg/s)</td>
<td>2034±374</td>
<td>1501±399*</td>
<td>1517±402†</td>
<td>1135±365</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PRSW (mm Hg/mL)</td>
<td>75±13</td>
<td>52±14*</td>
<td>66±12</td>
<td>44±12*</td>
<td></td>
<td></td>
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<tr>
<td>MR (0–4)</td>
<td>0.0±0.0</td>
<td>2.1±0.7*</td>
<td>0.1±0.2</td>
<td>0.1±0.2</td>
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</tbody>
</table>

Heart rate (HR), end-diastolic pressure (EDP), end-systolic pressure (ESP), peak LV pressure (LVP max), end-diastolic volume (EDV), end-systolic volume (ESV), maximum LV +dP/dt (dP/dt max), pre-load recruitable stroke work (PRSW), and mitral regurgitation (MR).

*P<0.05 versus pre-ischemia by t-test for paired observations; †P<0.05 pre-ischemia Tailor versus pre-ischemia control.

TABLE 2. Mitral Annular Geometry and Dynamics

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Ischemia</th>
<th>Control</th>
<th>Ischemia</th>
<th>Tailor</th>
<th>Ischemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAmax (cm²)</td>
<td>4.7±1.9</td>
<td>3.3±1.2*</td>
<td>5.42±0.77</td>
<td>5.45±0.69</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAV (cm²)</td>
<td>7.76±0.61</td>
<td>8.39±0.77*</td>
<td>5.42±0.77</td>
<td>5.45±0.69</td>
<td></td>
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<tr>
<td>MAVd (cm²)</td>
<td>6.80±0.58</td>
<td>7.95±0.78*</td>
<td>5.32±0.72</td>
<td>5.40±0.73</td>
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<tr>
<td>SL (cm)</td>
<td>2.81±0.12</td>
<td>3.06±1.8*</td>
<td>2.30±0.22</td>
<td>2.32±0.22</td>
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<tr>
<td>SLM (cm)</td>
<td>2.53±0.11</td>
<td>2.93±0.18*</td>
<td>2.22±0.21</td>
<td>2.28±0.22*</td>
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<tr>
<td>CC (cm²)</td>
<td>3.70±0.20</td>
<td>3.67±0.20</td>
<td>3.15±0.38</td>
<td>3.13±0.39</td>
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<tr>
<td>CCd (cm²)</td>
<td>3.51±0.21</td>
<td>3.59±0.23</td>
<td>3.13±0.36</td>
<td>3.13±0.38</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MA-Mmax (cm²)</td>
<td>7.79±0.40</td>
<td>8.10±0.47*</td>
<td>6.24±0.36</td>
<td>6.24±0.36</td>
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<tr>
<td>MA-Md (cm)</td>
<td>7.21±0.40</td>
<td>7.85±0.46*</td>
<td>6.18±0.34</td>
<td>6.20±0.38</td>
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<tr>
<td>MA-Fmax (cm)</td>
<td>2.88±0.15</td>
<td>2.87±0.22</td>
<td>2.77±0.37</td>
<td>2.74±0.31</td>
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<tr>
<td>MA-Fd (cm)</td>
<td>2.74±0.08</td>
<td>2.77±0.14</td>
<td>2.71±0.37</td>
<td>2.68±0.31</td>
<td></td>
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<tr>
<td>MA-Tmax (cm)</td>
<td>10.62±0.40</td>
<td>10.94±0.48†</td>
<td>8.96±0.61</td>
<td>8.93±0.56</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MA-Td (cm)</td>
<td>9.95±0.39</td>
<td>10.62±0.42*</td>
<td>8.89±0.60</td>
<td>8.88±0.60</td>
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</tbody>
</table>

MA=maximal mitral annular area; SL=mitral annular septal-lateral dimension; CC=mitral annular commissure-commissure dimension; MA-M=muscular mitral annulus perimeter; MA-F=fibrous mitral annulus perimeter; MA-T=total mitral annulus perimeter; MAX=maximum; MIN=minimum; ED=end-diastole.

*P<0.05 versus pre-ischemia by t-test for paired observations.

Figure 3. Group mean data for muscular annular perimeter (top panel), fibrous annular perimeter (middle panel), and total annular perimeter (bottom panel) throughout the cardiac cycle for control (left) and Tailor (right) groups before (closed symbols) and during (open symbols) acute posterolateral ischemia. A 650 ms time interval centered at end-diastole (t=0) is illustrated for both groups. Error bars represent ±1 SEM.
Mitral Leaflet Dynamics
To assess leaflet mobility throughout the cardiac cycle, angular position of the anterior leaflet edge was calculated as the angle ($\theta_{AML}$) between the line from the anterior leaflet edge marker to the “saddle horn” and the septal-lateral annular diameter. Posterior leaflet edge angular position ($\theta_{PML}$) was calculated in similar fashion. Leaflet excursion was calculated from diastolic maximum to systolic minimum angle. Anterior and posterior leaflet lengths were measured as the sum of distances between adjacent markers placed on the central meridian of each leaflet. Leaflet edge separation was determined as the distance in 3-D space between the 2 edge markers on the anterior and posterior mitral leaflets with valve closure defined as the time at which this distance reached its minimum plateau. Because of loss of the marker on the anterior leaflet edge in some animals, leaflet separation distance was calculated in 5 control and 4 Tailor animals.

Statistical Analysis
All data are reported as mean plus or minus 1 standard deviation (±1 SD). Hemodynamic and marker-derived data from 2 to 3 consecutive steady state beats that were time-aligned at end-diastole, and data from these beats were averaged for each animal. The data were analyzed for 20 frames before and 20 frames after end-diastole. Data were compared using Student’s t test for paired comparisons.

Results
Hemodynamics
The animals in the Tailor group were larger than those in the control group (77±9 kg versus 63±9 kg; P=0.02); additionally, they had longer cardiopulmonary bypass (171±21 versus 94±18 minute; P=0.0001) and aortic cross clamp (117±17 versus 65±24 minute) times. Group mean hemodynamic parameters before and after induction of acute posterolateral LV ischemia in each group are shown in Table 1. Before ischemia, the 2 groups were comparable in all hemodynamic parameters except end-systolic volume was greater in the Tailor group and the control animals had a higher baseline LV dP/dt. Peak and end-systolic LV pressures decreased while LV end-systolic volume increased in both groups with proximal circumflex occlusion. Furthermore, preload recruitable stroke work (PRSW), a load independent index of LV function, fell a similar degree in both groups during circumflex occlusion suggesting an equivalent ischemic insult. During acute IMR, mitral regurgitation increased from none to moderate in the control group, but remained only trace in the Tailor group.

Mitral Annular Dynamics
Group mean data for mitral annular area and SL and CC diameters as well as lengths of the fibrous and muscular portions of the mitral annulus are tabulated in Table 2 and shown in Figures 3 and 4. During acute posterolateral ischemia in the control group, the mitral annulus (particularly in the SL diameter) dilated substantially, and the length of the muscular annular perimeter increased significantly. Conversely, during circumflex occlusion in the Tailor group, annular area did not change, and there was no appreciable increase in either the SL diameter or the muscular annular perimeter. Mitral annular area changed only slightly during the cardiac cycle before and during ischemia in the animals fitted with the partial flexible annuloplasty ring. Although annular area remained fixed in the Tailor group, flexion of the septal annulus, as shown in Figure 5, was similar in both groups before ischemia (1.9±0.9 and 1.5±0.3 mm for control
were effective in preventing acute IMR, 11 but ring implanta-
tions were not universally effective. 25 In prior experimental
terapy in correcting ischemic mitral regurgitation 8
Currently, ring annuloplasty represents the mainstay of sur-
urgical therapy in correcting ischemic mitral regurgitation 8

and Tailor, respectively; P=0.4) and did not change during
circumflex occlusion.

Mitral Leaflet Dynamics
Group mean anterior and posterior leaflet edge angular
positions during the cardiac cycle for both groups before
ischemia are illustrated in Figure 6. Anterior leaflet excursion
from minimum to maximum during the cardiac cycle was
similar between the 2 groups (45±12 and 50±10 degrees for
control and Tailor, respectively; P=0.5), posterior leaflet
motion was severely limited in the Tailor group (38±3 versus
14±7 degrees; P=0.0001). Changes in leaflet excursion in
both groups during ischemia along with valve closure times
and lengths of the anterior and posterior leaflets at end-
systole are summarized in Table 3. During circumflex occlu-
sion, posterior leaflet excursion decreased in control animals
but did not change in the Tailor group. The observed mitral
regurgitation in the control group was associated with de-
layed valve closure and early systolic leaflet “loitering”
(Figure 7). In the Tailor group, although there was a trend
toward later valve closure at baseline compared with the
control group (P=0.054), delayed valve closure or leaflet
“loitering” during acute posterolateral LV ischemia did not

Discussion
Currently, ring annuloplasty represents the mainstay of sur-
gical therapy in correcting ischemic mitral regurgitation 8–10
although it is not universally effective. 25 In prior experimental
studies, complete flexible and semi-rigid annuloplasty rings
were effective in preventing acute IMR, 11 but ring implanta-

![Figure 5](image-url) Elevation of the “saddle horn” marker (#1, Figure 2)
avove a plane fitted to the posterior annular markers (#3–#7,
Figure 2) throughout the cardiac cycle for control animals (top)
and with posterior flexible Tailor ring annuloplasty (bottom)
before (solid symbols) and during (open symbols) acute posterolat-
eral ischemia. A 650 ms time interval centered at end-diastole
(t=0) is illustrated for both groups.

![Figure 6](image-url) Group mean data for angular displacement of
the anterior mitral leaflet (top panel) and posterior mitral leaflet
(bottom panel) throughout the cardiac cycle for control (squares)
and Tailor (circles) animals before induction of acute ischemic
mitral regurgitation (IMR). Leaflet edge angular displacement
was calculated with respect to the line between mid-septal and
mid-lateral annulus. A 650 ms time window centered at end-
diastole (t=0) is illustrated. Error bars represent ±1 SEM.

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![Table 3](image-url) Leaflet Angular Motion, Closure Timing, and Lengths

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Tailor</th>
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<tbody>
<tr>
<td></td>
<td>Pre-ischemia</td>
<td>Ischemia</td>
</tr>
<tr>
<td>AML_{pre} (°)</td>
<td>45±12</td>
<td>38±16</td>
</tr>
<tr>
<td>PML_{pre} (°)</td>
<td>38±3</td>
<td>32±4*</td>
</tr>
<tr>
<td>Valve closure (ms)</td>
<td>17±41</td>
<td>84±12*</td>
</tr>
<tr>
<td>AML-Length_{pre} (cm)</td>
<td>2.08±0.13</td>
<td>2.19±0.09*</td>
</tr>
<tr>
<td>PML-Length_{pre} (cm)</td>
<td>1.33±0.17</td>
<td>1.33±0.17</td>
</tr>
</tbody>
</table>

AML_{pre} = angular excursion of anterior mitral leaflet from maximum
to minimum during the cardiac cycle; PML_{pre} = angular excursion of posterior
mitral leaflet from maximum to minimum during the cardiac cycle; valve closure =
time at which leaflet separation distance reached its minimum
plateau; AML-Length = length of the anterior mitral leaflet; PML-Length = length
of the posterior mitral leaflet; ES = end-systole.

*P<0.05 versus pre-ischemia by t test for paired observations; †P<0.05
pre-ischemia Tailor versus pre-ischemia control (data for anterior leaflet length
and excursion and valve closure time derived from 5 animals in the control
group and four animals in the Tailor group).
ischemic normal mitral annular dynamics and partially restricted posterior leaflet motion, similar to what has previously been observed in sheep acute IMR models following implantation of complete rings.12,13

Previous studies demonstrated that complete ring annuloplasty abolished acute IMR by reducing the annular septal-lateral (or, in clinical jargon, “antero-posterior”) dimension and prevented mitral leaflet “loitering”27 and perturbations of leaflet geometry.27 The Tailor ring effectively fixed annular size throughout the cardiac cycle and did not permit dilatation of the muscular annulus during acute ischemia. Consequently, no IMR was observed, and the mitral septal-lateral annular dimension increased only slightly; which is the main direction of annular enlargement during acute posterolateral LV ischemia.18,20 Although the Tailor ring is a partial ring, annular dilatation was not observed with circumflex occlusion because the fibrous annulus did not change: The dynamics of the fibrous annulus did not change throughout the cardiac cycle in either group, nor did they change during ischemia. Therefore, perhaps all that is needed in annular reduction procedures aimed at correcting IMR is reduction of the muscular annulus; however, caution must be exercised because dilatation of the fibrous annulus has actually been observed in certain circumstances, for example, patients with28 and experimental models of dilated cardiomyopathy.29

Avoiding circumferential reduction of the mitral annulus may be related to preserved flexion of the anterior annulus that we recently reported in animals with a Tailor ring, as such was not seen with a complete flexible ring:17 maintained annular flexion may play a role in reducing the likelihood of systolic anterior motion of the anterior mitral leaflet which might cause postoperative LV outflow tract obstruction after ring annuloplasty.30 In the current experiment, annular flexion was also conserved with the Tailor ring during acute posterolateral LV ischemia.

Complete ring annuloplasty abolishes leaflet “loitering” and prevents delayed leaflet coaptation in models of acute ovine IMR.11 The current data suggest that the Tailor ring is equally effective in facilitating timely valve closure and preventing IMR during acute LV ischemia; however, annular reduction with the Tailor ring was also associated with restriction of posterior leaflet motion as found previously with complete rings.13,14 We believe that the observed “frozen” posterior mitral leaflet in the setting of mitral annuloplasty is related to reduction of annular area and altered geometric relationships between the posterior mitral annulus and the papillary muscle tips. As the reduction in annular area induced by the Tailor ring is comparable to that achieved with complete rings,12 this limited posterior leaflet motion may not be surprising. Additionally, “reefing” of the posterior leaflet by the annuloplasty band must also be considered and may be reflected in the baseline differences in posterior leaflet lengths between the 2 groups. Extension of the anterior leaflet during acute IMR has been described in control sheep during acute IMR in a prior report27 and attributed to papillary muscle “tethering”. In animals with complete annuloplasty rings, systolic anterior leaflet length remained unchanged during acute ischemia, a finding corroborated by the current study. Thus, the Tailor partial flexible annuloplasty ring has essentially the same effects on mitral leaflet motion and geometry during acute LV ischemia in this model as do complete flexible and semi-rigid annuloplasty rings, except that annular flexion is preserved.

**Summary**

Ring annuloplasty is frequently used in the surgical correction of ischemic mitral regurgitation.9,10 and partial flexible ring annuloplasty has been shown to be effective clinically in treating IMR.16,21 The current data support this practice as the Tailor partial flexible ring effectively prevented acute IMR in healthy sheep hearts. Surprisingly, as already described under non-ischemic conditions,17 the Tailor ring altered annular and leaflet in vivo dynamics during acute LV ischemia almost identically to that associated with use of complete annuloplasty rings. The Tailor ring prevented mitral annular dilatation, especially in the septal-lateral dimension, facilitated timely valve closure, and converted the mitral valve into a single-leaflet valve by “freezing” the motion of the posterior leaflet. Unlike complete flexible rings,17 however, the Tailor ring permitted normal annular flexion that was sustained during acute LV ischemia, but the physiologic importance of this annular flexion, if any, in a clinical setting needs further investigation.

**Study Limitations**

The results of the current study must be interpreted in the context of several limitations. This experimental design does not imitate clinical practice where prior ischemia or infarction is present and then is treated surgically with placement of a ring- in this study, an annuloplasty ring was implanted in a normal heart before the ischemic insult and therefore represented a “prophylactic” surgical correction that is distinctly different than the clinical situation. It is also important to
emphasize that these findings can only be interpreted in the setting of acute LV ischemia in a normal sheep heart and should not be extrapolated directly to patients with chronic IMR where LV dilatation, sub-valvular geometric perturbations induced by previous infarction and ischemia, and LV remodeling may play a predominant role. This animal model also has other limitations in that sheep have a less well defined posterior annulus than do humans and more atrial tissue above and below the line of leaflet insertion;\(^3\) however, annular dynamics are similar in both humans and sheep.\(^2,3\) While the myocardial marker method requires suturing small metal markers to numerous intra-cardiac structures, previous echocardiographic studies show that the markers do not interfere with normal mitral annular or leaflet motion as they are very small (aggregate mass = 20±6 mg). Although there were baseline differences in animal size and some hemodynamic parameters between the 2 groups, we believe that these differences did not contribute in an important way to the observed differences in annular and leaflet dynamics between the control sheep and the ring animals.

**Acknowledgments**

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**References**


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